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Abstract English

Dysfunctions of the autonomic nervous system such as reduced heart rate variability (HRV) and maladaptive emotion regulation (ER) are assumed to contribute to the development and progression of somatic symptom disorder (SSD). There is growing evidence for reduced HRV activity and more maladaptive ER use in comparison to healthy controls (HC). However, stressor-related HRV-reactivity is not well understood. Further, ER research often refers to formerly called somatoform disorders, does not necessarily cover both adaptive and maladaptive ER strategies, and is neglecting contextual factors influencing ER use. Aim of this doctoral thesis was to examine those physiological and psychological aspects within SSD and its subgroups, namely subjects with medically unexplained (MUS) and medically explained (MES) symptoms, with help of a quasi-experimental study (study 1 and 2) and baseline-data of a multicentric intervention study (study 3). Results from study 1 (*N* = 94) showed that HRV in SSD responded less flexible to changes in experimental conditions than in HC, and to social stressors, which caused more symptom disability than health-related stressors. Study 2 (*N* = 108) showed more maladaptive and less adaptive ER use in SSD in comparison with HC. Even though ER predicted health anxiety (HA) within SSD, HA differences between SSD and HC could only partially be explained by ER. Subgroup differences within SSD were found neither in Study 1 nor 2. Study 3 contrasted acceptance as an ER strategy and specific coping strategies with regard to predicting somatic burden in SSD-MUS (*N* = 255). Both, acceptance and coping, were considerable predictors. However, contextual variables like the intensity of symptom-related emotions moderated relationships between strategy use and somatic burden in specific ways. In sum, both autonomic rigidity and ER deficits in SSD might be the result of reduced cardiac vagal control and appear to have similar relevance in SSD-MUS and SSD-MES. A HRV biofeedback and ER training might enhance self-regulatory skills, enabling SSD patients to cope better with symptom-related internal and external demands.

Abstract German

Es wird angenommen, dass Störungen des autonomen Nervensystems in Form einer reduzierten Herzratenvariabilität (HRV) und eine maladaptive Emotionsregulation (ER) mit zur Entstehung und Aufrechterhaltung der Somatischen Belastungsstörung (SBS) beitragen. Es gibt immer mehr Belege für eine reduzierte HRV und mehr Anwendung maladaptiver ER-Strategien im Vergleich zu einer gesunden Kontrollgruppe (GK). Über die stressorbezogene Reaktivität ist hingegen wenig bekannt. Außerdem ist die ER-Forschung häufig auf die vormals somatoformen Störungen ausgerichtet, berücksichtigt häufig nicht sowohl adaptive als auch maladaptive Strategien, und vernachlässigt den Einfluss kontextueller Faktoren auf die Anwendung jeweiliger ER-Strategien. Ziel der Dissertation war die Untersuchung physiologischer und psychologischer Aspekte bei SBS und ihrer Subgruppen, nämlich Personen mit medizinisch unerklärten (MUS) und medizinisch erklärten Symptomen (MES), was mittels einer quasi-experimentellen Studie (Studie 1 und 2) und den Ausgangsdaten einer multizentrischen Interventionsstudie (Studie 3) angestrebt wurde. Die Ergebnisse von Studie 1 (N = 94) wiesen darauf hin, dass die HRV bei SBS weniger flexibel auf sich ändernde experimentelle Bedingungen reagierte als die der GK, und dass soziale Stressoren eine stärkere Symptombeeinträchtigung hervorriefen als Gesundheitsbezogene. Studie 2 (N = 108) zeigte, dass bei Personen mit SBS im Vergleich zur GK in stärkerem Ausmaß maladaptive und in geringerem Ausmaß adaptive ER-Strategien verwendeten. Obwohl Krankheitsangst (KA) bei SBS durch ER vorhergesagt wurde, wurden unterschiedliche KA-Ausprägungen zwischen SBS und GK nur partiell durch ER erklärt. Weder in Studie 1 noch 2 konnten innerhalb der SBS-Stichprobe Subgruppenunterschiede gefunden werden. In Studie 3 (N = 255) wurde die ER-Strategie der Akzeptanz bestimmten Copingstrategien zur Vorhersage somatischer Belastung bei SBS-MUS gegenüber gestellt. Sowohl Akzeptanz als auch

Copingstrategien waren nennenswerte Prädiktoren. Kontextabhängige Variablen, wie die Intensität symptombezogener Emotionen, moderierten auf spezifische Art Beziehungen zwischen Strategieanwendung und somatischer Belastung. Zusammengefasst könnten sowohl die autonome Rigidität als auch Emotionsregulationsdefizite das Ergebnis einer reduzierten kardialen, vagalen Kontrolle sein und scheinen eine ähnliche Relevanz bei SBS-MUS und SBS-MES zu haben. Ein HRV-Biofeedback und ER-Training könnten die selbstregulatorischen Fähigkeiten verbessern, was SBS-Patienten dazu befähigen könnte besser mit internen und externen Anforderungen umzugehen.

1 Introduction

The newly labeled category of the fifth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) ´somatic symptom and related disorders´ includes the diagnosis SSD, which is defined by affected people suffering from at least one somatic symptom lasting at least six months accompanied by either excessive cognitive, affective, or behavioral symptom-related reactions (APA, 2013). The presence of somatic symptoms is manifesting mono- or polysymptomatic, for example in the form of pain syndromes of all kind, fatigue, or other functional somatic syndromes (FSS). The former requirement of somatic symptoms having to be medically unexplained or not fully explained by medical disease factors is no longer relevant (Henningsen, 2018). Even though the DSM-5 section ´somatic symptom and related disorders´ received positive reception, difficulties in differential diagnostics were criticized as potentially causing excessive overdiagnosis of SSD (Mayou, 2014). The DSM-IV (APA, 2000) section somatoform disorders (SFD) was viewed as problematic for several reasons, which is why a conceptional revision was considered necessary (e.g. Dimsdale et al., 2013). There are good reasons for the omission of medical explanation in SSD classification (Klaus et al., 2013). However, there is no evidence yet accounting for the same relevance of illness-related mechanisms in somatoform as in medically explained physical symptoms. Etiological assumptions for SSD are based on SFD (Rief & Martin, 2014).

Biological abnormalities in SFD have been examined in different ways (Rief & Barsky, 2005). Study results related to autonomic characteristics indicate elevated heart rates (HR; Rief, Shaw, & Fichter, 1998) and reduced HRV (Tak et al., 2009), but have to be regarded with caution. The role of HRV has been investigated in several psychopathologies because it is regarded to be an indicator for stress processing

(Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012) or rather for the ability to adapt to changing environmental conditions (Appelhans & Luecken, 2006). So far, several studies investigated HRV under resting conditions (e.g. Huang et al., 2017). On the contrary, HRV-reactivity after application of specific stressors has only been investigated in few, recent studies (Huang et al., 2019; Lee et al., 2018). Script driven imagery is one possibility to induce stress. This method has been successfully adapted to patients with multiple MUS (Schwarz, Gottschalk, Ruckmann, Rief, & Kleinstauber, 2016). According to standardized guidelines, participants created individualized scripts dealing with their somatic complaints. Those scripts were recorded. Later on, participants were exposed to their own audio material, which caused increased symptom disability. Script driven imagery can also be used to create other stressors dealing with interpersonal conflicts, which are acknowledged as risk factor for the development of FSS (Van Houdenhove, Egle, & Luyten, 2005). The aim of this doctoral thesis was the examination of autonomic characteristics in SSD and their subgroups. Specifically, the dissertation intended to examine physiological and subjective reactivity after induction of health-related and interpersonal stressors.

ER is another underlying mechanism that was examined in more details. While there are numerous, empirical findings regarding relationships between maladaptive ER and depressive disorders (Liverant, Brown, Barlow, & Roemer, 2008), anxiety disorders (Campbell-Sills, Barlow, Brown, & Hofmann, 2006), or other psychopathologies (Aldao, Nolen-Hoeksema, & Schweizer, 2010), its relevance in SSD is not well understood. In the 1970s the concept of alexithymia (Sifneos, 1973) was introduced to describe deficits in emotion processing in somatoform patients. Contrarily to alexithymia, ER focusses on the actual dynamics of emotions (Aldao, 2013) and not primarily on its perception and expression. Most studies, especially in chronic pain, investigated effects of specific ER strategies on symptom perception and tolerance (e.g. Kohl, Rief, & Glombiewski, 2014).

Only a few studies examined differences in ER use between SSD and HC (Schwarz, Rief, Radkovsky, Berking, & Kleinstäuber, 2017), not providing a broad picture of adaptive and maladaptive ER use differences. According to DSM-5 classification, patients with SSD may experience HA as an affective symptom (APA, 2013). Following the concept of ER, the adaptive regulation of HA is an important issue. Therefore, it is important to know which ER strategies particularly predict HA. As contextual variables like emotion intensity (Dixon-Gordon, Aldao, & De Los Reyes, 2015) seem to influence an individual´s choice of ER use, a further objective of this doctoral thesis was to understand to what extend the intensity of symptom-related negative emotions moderates certain relationships between strategy use and somatic burden.

Hereafter, SSD is characterized in accordance with epidemiological findings and etiological models. In this context, assumed disorder-relevant factors and mechanisms like adverse life events, negative affect, ER, and physiological characteristics are described. The three studies of this doctoral thesis present results regarding subjective and physiological reactivity, the relevance of ER, and distinct relationships between acceptance/coping use and somatic burden. Finally, the study results will be discussed in the light of existing empirical findings.

2 Theoretical Background

This section deals with phenomenological aspects and classification of SSD at first, followed by current research regarding epidemiology and etiology. Negative affect, ER, and autonomic characteristics in the form of HR and HRV gain particular attention with respect to etiology.

2.1 Phenomenology and classification of SSD

SSD was introduced in the fifth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), namely in the section ´somatic symptom and related disorders´. Patients are diagnosed with SSD if they experience at least one somatic symptom lasting at least six months, accompanied by one of three psychological criteria. These include disproportionate thoughts about the seriousness of the symptoms, HA, and excessive time devoted to these symptoms. Whereas the diagnoses of a somatoform disorder required medically unexplained or at least not fully explained physical symptoms, SSD classification neglects the medical explanation of existing symptoms. SSD can be specified as presenting with ´predominant pain´ or as ´persistent´. Its severity can be classified as mild, moderate, and severe, depending on the number of psychological criteria being fulfilled. In case of severe SSD, multiple somatic problems or one severe physical symptom are required with two psychological criteria that have to be fulfilled (APA, 2013). Depending on whether burdensome physical symptoms exist or not, health anxious people get diagnosed with SSD or illness anxiety disorder (Witthöft, Gropalis, & Weck, 2018). In the DSM-IV-TR (APA, 2000) section SFD patients were diagnosed with hypochondriasis in case of being health anxious contrary to medical reports. This diagnosis was dropped in the new DSM version. Hypochondriasis subdivides into SSD and illness anxiety disorder, although newer

findings suggest that it predominantly passes to SSD (Bailer et al., 2016). Further diagnoses included in the DSM-5 section somatic symptom and related disorders are called conversion disorder, psychological factors affecting other medical conditions, factitious disorder, other specified somatic symptom and related disorder, and unspecified somatic symptom and related disorder (APA, 2013). The doctoral thesis primarily focuses on SSD.

The fundamental revision of the first DSM criterion is based on findings in studies of primary care. Depending on the doctor´s special field, patients with the same physical complaint (PC) received different diagnoses (Fink, Rosendal, & Olesen, 2005). Persisting somatic symptoms that were initially rated as MUS or MES were partially evaluated differently one year later (Klaus et al., 2013). Besides different kinds of medical diseases like cancer or diabetes, patients with SSD might also suffer from MUS, manifesting in different forms of pain relating to the back, head, hips, extremities, or in the form of food intolerances, lack of sexual desire or sexual dysfunctions (Hiller, Rief, & Brähler, 2006). MUS might also manifest in the form of functional complaints, relating primarily to one single organ system. Examples of those functional problems are the irritable bowel syndrome, fibromyalgia, and the chronic fatigue syndrome (Henningsen, Zipfel, Sattel, & Creed, 2018). The number of MUS within the general population may be subsumed in two clusters, ranging from few to many symptoms with four symptoms as cut-off value (Rosmalen, Tak, & De Jonge, 2011). Psychological criteria according to DSM-5 seem to be the reason why patients with MUS fulfilling SSD-criteria have more severe and disabling, physical symptoms than those fulfilling only DSM-IV criteria. SSD severity seems to depend on the number of fulfilled psychological criteria (van Dessel, van der Wouden, Dekker, & van der Horst, 2016).

Rief and Martin (2014) questioned whether SSD patients with MUS and those with medical diseases like diabetes or cancer are comparable concerning psychological

illness mechanisms. They proposed a subdivision of SSD into five subgroups, namely mono- or polysymptomatic unexplained somatic symptoms, medically explained symptoms, mono- or polysymptomatic pain syndromes with episodic or chronic course, and hypochondriasis. According to Güney and colleagues (2019) this suggestion represents a ´splitting´ view regarding SSD classification, as Rief and Martin (2014) doubt whether the commonalities between symptom clusters cover all patient groups within SSD and its specific symptom presentation. The so called ´lumpers´ assume that commonalities between symptom clusters are more significant than their differences. So far, there is little systematic research on whether illness mechanisms are equally relevant for development, maintenance, prognosis and treatment of the various manifestations of SSD.

2.2 Epidemiology

A recently published cross-sectional study investigated the distribution of SSD in the German general population (Häuser et al., 2020). According to their findings, the prevalence of SSD accounted for 4.5 %. This corresponds with the APA´s assumptions, estimating the prevalence in the adult general population to be 5 to 7 %, which is higher than somatization disorder and lower than undifferentiated somatic symptom disorder (APA, 2013). The prevalence is assumed to be distinctly higher in women and believed to be underdiagnosed in older people with existing medical conditions. Patients with underlying medical conditions reveal a higher risk of fulfilling psychological criteria and might therefore more likely be diagnosed with SSD (Kop, Toussaint, Mols, & Löwe, 2019). In the meantime, there are time- and cost-efficient self-report measures covering both somatic symptom burden and psychological criteria in order to evaluate individual risk to come down with SSD (Toussaint, Hüsing, Kohlmann & Löwe, 2020). Course and

prognosis of SSD cannot be completely evaluated yet (APA, 2013; Witthöft & Jasper, 2015; Witthöft, Gropalis & Weck, 2018).

Physical complaints are quite common in the general population. Results of a representative study sample show that 81.6 % of surveyed participants report to have had at least one mild MUS within the last seven days. In addition to that, 22.1 % report to be severely impaired by at least one somatic symptom. The average number of symptoms was 6.6, showing a wide spread of MUS (Hiller et al., 2006). MUS are no rarity in primary care as general practitioners classified 76 % of the reported symptoms as MUS (Körber, Frieser, Steinbrecher, & Hiller, 2011). The prevalence of SFD in primary care is estimated to be between 16.1 % (De Waal, Arnold, Eekhof, & Van Hemert, 2004) to 22.9 % (Steinbrecher et al., 2011). Affected individuals usually consulted their general practitioners with symptoms like stomach pain, dizziness, pain in the chest, pelvis, and hips, food intolerances, or palpitation. Even though half of the patients did not consult their general practitioner five years later again, one third of the symptoms were still classified as medically unexplained (Jackson & Passamonti, 2005). Some of the affected individuals repeatedly consult their general practitioner due to their MUS (Verhaak, Meijer, Visser & Wolters, 2006). Consequently, there is an increased risk of chronic course of the somatic symptom burden (Arnold, de Waal, Eekhof, & van Hemert, 2006). These facts potentially lead to increased health care use and 2.2-times higher health care costs in relation to the national average (Hiller, Fichter & Brähler, 2003).

Existing research reveals that somatic distress across different symptom clusters rather appears dimensional than categorial (Jasper, Hiller, Rist, Bailer, & Witthöft, 2012) and that there is a general factor (Witthöft, Hiller, Loch, & Jasper, 2013). Furthermore, there is evidence that this general factor unlike single symptom clusters is associated with cognitive-affective characteristics such as HA (Witthöft, Fischer, Jasper, & Rist,

2016). HA is assumed to be an important predictor of symptom persistence (McKenzie, Clarke, McKenzie, & Smith, 2010), accompanied by increased health care use (Fink, Ørnbøl, & Christensen, 2010), and is regarded to be a negative prognostic factor in pain patients (Hadjistavropoulos & Hadjistavropoulos, 2003).

The presence of a comorbid disorder has the effect that somatoform symptoms are experienced more severely and more disabling in daily life (De Waal et al., 2004). Alongside anxiety and affective disorders, SFD reveal the highest 12-months-prevalence (Jacobi et al., 2004) with 26 % of this patient group suffering from a comorbid depressive or anxiety disorder.

2.3 Etiology

In terms of etiology, multi-causal processes involving cognitive, affective, behavioral, and physiological aspects are thought to be relevant in the development and maintenance of SSD (Witthöft et al., 2018). Next, prominent explanatory models get introduced, covering illness mechanisms based on empirical findings on SFD (Rief & Martin, 2014). One model dealing with somatization stems from Barsky and Wyshak (1990), focusing on perceptive and cognitive factors in hypochondriasis. The authors suggest that bodily changes are perceived first. Due to catastrophizing and drawing attention to these changes, they are perceived as more intense and are evaluated as signs of severe illness. This vicious circle describes the mechanism of somatosensory amplification (Barsky & Wyshak, 1990; Rief & Broadbent, 2007), which is proposed to be relevant in SSD (Rief & Martin, 2014; Witthöft et al., 2018). Another explanatory approach also covering perception processes is the signal-filtering-model (Rief & Barsky, 2005), assuming higher cortical structures to receive constant, neural information from organs, skin, and body parts (Rief & Broadbent, 2007) that underlie neural filtering

processes. According to the model, somatic sensations either result from strong sensory input, e.g. due to chronic hypothalamic pituitary adrenal (HPA) axis stimulation or decreased filter activity, for example due to increased HA influencing selective attention processes (Rief & Barsky, 2005). With respect to functional somatic syndromes, genetic and epigenetic factors are considered to be relevant only to a limited extend (Henningsen et al., 2018). On the contrary, endocrinological characteristics, like abnormalities of the HPA-axis, are of more interest (Kirmayer & Looper, 2006). With respect to chronic fatigue syndrome there are findings supporting HPA-axis dysfunction and related hypocortisolism. However, there are no corresponding findings for other functional syndromes (Tak et al., 2011). The role of the autonomic nervous system (ANS) will be discussed in chapter 2.3.3.

Van den Bergh and colleagues (2017) suggest that the perception of somatic distress is the result of a constructivistic process from the central nervous system (CNS). It is argued that somatic distress rests on top-down processes based on expectations. These processes can only be modified by peripheral information (bottom-up) in case of discrepancy between this information and CNS predictions. Brown (2004) considers the role of memory and its interaction with perceptive factors. In his opinion experiences of stress and illness are represented in the memory. Subordinately, attention is selectively directed to somatic symptoms in case of primary attentional processes activating those representations. Consequently, perceived physical sensations get interpreted as a sign of serious illness causing a distinctive negative affect (Rief & Broadbent, 2007). Kirmayer & Taileffer´s (1997) cognitive-behavioral model, which is depicted in figure 1 considers aforementioned perceptive-cognitive factors as well as maladaptive behavior. According to this model, physical sensations result from medical or physiological conditions like medical illnesses or emotional arousal caused by psychological conditions such as traumatic experiences.

Affected persons experience worries, catastrophization, and demoralization in case of interpretations of these sensations as a sign of illness. This enhances emotional arousal on the one hand and inadequate reassurance and help-seeking behavior on the other hand. Further, avoidance behaviors aiming to reduce physical or social activity may promote physical disability. Depending on the social response by doctors, family, or social environment, maladaptive behavior patterns may be reinforced (Looper & Kirmayer, 2002; Rief & Broadbent, 2007; Rief & Martin, 2014).

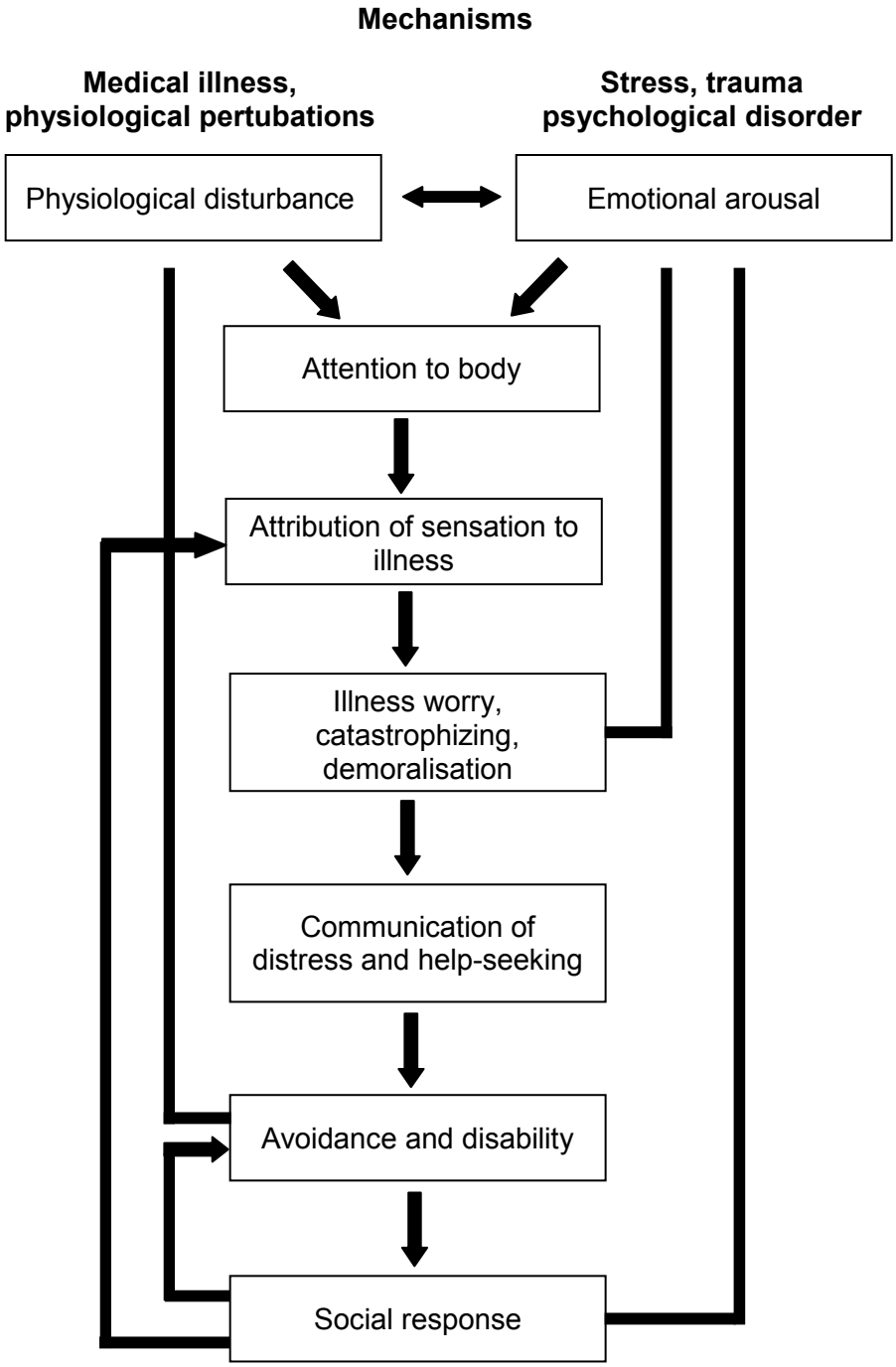

Figure 1 A model of somatoform disorders (Kirmayer & Taileffer, 1997)

2.3.1 Adverse life-events and negative affect

As the thesis aimed to investigate ER deficits in SSD, two related factors deserve particular consideration: one factor is adverse-life events like early, maladaptive interactions with primary caregivers that are believed to disrupt neural circuits. This consequently promotes difficulties in affect regulation, leading then again to somatoform symptoms through various stages (Landa, Peterson, & Fallon, 2012). The other factor refers to negative affect, which patients with somatoform complaints have difficulties to reduce or change due to impaired ER skills (Witthöft, Loch, & Jasper, 2013).

Explanatory models like Kirmayer and Taillefer´s (1997) suggest that emotional arousal caused by trauma or psychosocial stressors provokes somatoform symptoms through several stages. Even though the term ´negative affect´ is not used, the model implies that the development of somatoform symptoms depends on physiological and emotional factors.

Existing findings indicate that traumatic childhood experiences are predisposing, psychosocial factors for the development of functional syndromes (Henningsen et al., 2018). There are significant relationships between adverse life events over the life span and functional complaints within the previous year (Tak, Kingma, van Ockenburg, Ormel, & Rosmalen, 2015). These relationships were independent of socioeconomic status or sensitivity to stress. However, gender dependent differences appeared in consideration of the temporal occurrence of these events, meaning that associations between adverse childhood events and functional complaints were only found in women. With respect to men, associations between functional complaints and adverse life events could only be revealed if these life events occurred during adulthood. Expellees that experienced traumatic life events during displacement revealed increased somatic distress compared to people without any experience of displacement (Kuwert, Braehler, Freyberger, &

Glaesmer, 2012). Besides future risks of suffering from anxieties and depressions, individuals who have been sexually abused during childhood also carry the risk of experiencing MUS in the future (Nelson, Baldwin, & Taylor, 2012).

On the one hand, this could be due to an abnormal stress response (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). On the other hand, it could be caused by negative affect and shame in particular. It is assumed that emotional abuse predicts somatic symptoms during adulthood through existing feelings of shame (Kealy, Rice, Ogrodniczuk, & Spidel, 2018). This suggests that negative affect particularly influences associations between adverse life events and somatic burden. Relationships between negative affect and somatic complaints have been assumed for centuries (Watson & Pennebaker, 1989). Experimental studies using pictures with different affective qualities in order to influence negative affect levels, showed similar changes in state negative affect as well as symptom reporting in high habitual symptom reporters (Bogaerts, Janssens, de Peuter, van Diest, & van den Bergh, 2010). This provides an argument for a causal role of negative affect. In addition, there are findings suggesting associations between reported mood and functional complaints (Burton, Weller, & Sharpe, 2009) as well as effects of positive and negative affect regulation on pain perception (Connelly et al., 2007).

2.3.2 Emotion regulation

According to Aldao (2013), ER aims to influence emotional states in order to adaptively react to situational demands. ER refers ´to the processes by which individuals influence, which emotions they have, when they have them, and how they experience and express these emotions.´ (Gross, 1998, p. 275). Systematic research on this issue began in the 1970s and was heavily influenced by stress and coping research (Gross, 1998). A prominent definition of coping refers to coping as ´cognitive and behavioral

efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person.´ (Lazarus & Folkman, 1984, p. 141). Coping can be problem- or emotion-focused. The latter aims to reduce the negative, emotional impact of a stressor, whereas ER aims at regulating negative and positive emotions at the same time (Gross, 1998). Unlike ER, coping is intentional and non-automatic (Lazarus & Folkman, 1984; Jensen, Turner, Romano, & Karoly, 1991). On the contrary, ER-processes can be automatic or controlled, conscious or unconscious. According to the hierarchical conception of affect regulation, both coping and ER are subordinate to affect regulation (Gross, 1998). ER can be viewed from various perspectives. The intrapersonal view focuses on the efforts by which individuals aim to feel better or to avoid aversive emotional states, whereas the relational view deals with ER in the presence of others and particularly with conflicting goals of a single person and those of others (Campos, Walle, Dahl, & Main, 2011). ER strategies can be need-, goal-, or person-oriented and target cognitive, attentional, or bodily expressions (Koole, 2009).

There are various models of ER; the most prominent being the process model of ER (Gross, 1998). It describes the so called antecedent-focused, regulatory processes like situation selection, situation modification, attentional deployment, and cognitive change. Situation selection aims to regulate emotions by avoiding people or certain situations, situation modification refers to efforts intending to influence the emotions while being in an emotion-eliciting situation. Attentional deployment focuses on efforts to move attention towards less aversive aspects of a situation, cognitive change aims to modify the evaluation of a perceived situation. Altogether, antecedent-focused ER becomes effective before emotional response tendencies are fully generated, while response modulation as the fifth regulatory process aims to modulate emotional response tendencies (Gross, 1998). Sheppes and colleagues (2015) proposed

conceptional extensions of Gross´s process model. They suggested three regulatory stages (identification, selection, implementation) being involved in the process. The identification stage refers to the initial decision whether emotions should be regulated or not. The selection valuation system decides which of the antecedent- or response-focused, regulatory categories should be used. Finally, the implementation valuation system decides which specific strategy within the selected category should be used. Reappraisal is an example of antecedent-focused ER and becomes effective before emotional response tendencies are fully generated, while suppression is an example of response-focused ER (Gross & John, 2003). Research using functional magnetic resonance imaging (fMRI) to investigate neural effects of ER strategies found that reappraisal use provokes early prefrontal-cortical reactions, reducing negative affects that have been previously induced by specific films. Suppression use also provokes prefrontal-cortical reactions reducing negative affect. However, these reactions occur later and are accompanied by increased amygdala and insula activity (Goldin, McRae, Ramel, & Gross, 2008). Investigations of the relationship between ER strategy use and psychopathology point to rumination, avoidance, and suppression being associated with increased psychopathology, and problem solving and reappraisal being associated with mental health (Aldao et al., 2010). Berking´s (2007) model of adaptive coping with emotions suggests nine relevant skills to improve ER, namely awareness of emotions, identification and labeling of emotions, adaptive interpretation of emotion-related body sensations, understanding causes for emotions, self-support in challenging situations, confront situations, abilities to actively modify emotions, accept emotions, be resilient/tolerate aversive emotions. The abilities to actively modify and/or to accept and tolerate aversive emotions seem to be crucial and are highly associated with mental health (Berking et al., 2008).

However, ER strategies are not per se adaptive or maladaptive. Their adaptiveness depends on contextual factors. Recent studies imply that emotion type and intensity influence choice and adaptability of the respective ER strategy (Dixon-Gordon et al., 2015; Zimmermann & Iwanski, 2014). Recently, increasing numbers of experimental studies dealt with the relevance of ER in specific mental disorders (e.g. Aldao, 2013; Aldao et al., 2010; Gross & Munoz, 1995). The majority of the publications refer to anxiety (Campbell-Sills et al., 2006; Levitt et al., 2004) as well as depressive, (Liverant et al., 2008), compulsive (Najmi, Riemann, & Wegner, 2009), or borderline personality disorder (Jacob et al., 2011).

Relevance of ER in SSD

Initially, Thompson´s (1994) and Gross´ (1998) ER concepts did not provide stimulation to explore the relevance of ER in somatoform complaints. Instead, Sifneos (1973) already described patients with somatoform complaints as having ´difficulty identifying and describing feelings, difficulty in distinguishing between feelings and the bodily sensations of emotional arousal, constrictive imaginative fantasy life, and the tendency to focus on the concrete detail of external events´ (Deary, Scott, & Wilson, 1997, p. 552). Sifneos (1973) subsumed these characteristics under the term alexithymia. Studies using the Toronto-Alexithymia-Scale (TAS, TAS-R; Taylor et al., 1992) point to elevated alexithymia levels in somatoform patients compared to HC (De Gucht & Heiser, 2003). Furthermore, an alexithymia trait is discussed in patients with functional syndromes who seem to have difficulties recognizing day-to-day anxiety and depression levels (Burton et al., 2009). However, Rief, Heuser and Fichter (1996) emphasize that associations between alexithymia measured by TAS and somatoform symptoms disappear when controlled for depression. Therefore, they concluded that a direct relationship between alexithymia and somatization appears to be questionable.

Overall, the concept of alexithymia focuses on initial experience and expression of emotions (Witthöft et al., 2013). In contrast, ER focuses on how to affect the course of an emotional reaction (Aldao, 2013).

As negative affect influences the reporting of somatic symptoms (Bogaerts et al., 2010; Bogaerts et al., 2015), it seems obvious that there are ER deficits in somatoform patients (Witthöft et al., 2013).

Even though there are good arguments to assume the existence of ER deficits in SSD (Rief & Martin, 2014), the empirical basis for such an assumption is much smaller in comparison to anxiety and affective disorders (Schwarz et al., 2017; Witthöft et al., 2013). Previous studies refer to the use and effects of specific ER strategies mostly based on DSM-IV classification. Existing research points to deficits in emotion recognition (Subic-Wrana, Beutel, Knebel, & Lane, 2010). Experimental studies show that strategies like reappraisal and acceptance are associated with short-term symptom reduction in patients with multiple MUS (Kleinstäuber, Gottschalk, Ruckmann, Probst, & Rief, 2018). In addition the use of reappraisal is further assumed to increase pain tolerance in patients with fibromyalgia (Kohl et al., 2014). Effects of ER strategy use are largely investigated in chronic pain patients. Maladaptive ER strategies are supposed to be a risk factor for the development and maintenance of pain syndromes (Koechlin et al., 2018). Several study results emphasize adaptive effects of acceptance use regarding pain management (Vowles, McNeil, Gross, McDaniel, & Mouse, 2007; McCracken & Eccleston, 2006; Kohl, Rief, & Glombiewski, 2012). Studies investigating ER use in SSD according to DSM-5 are rare. However, the few existing studies point to relatively lower ER skills in comparison to HC (Schwarz et al., 2017). As SSD is accompanied by aspects of HA (APA, 2013), important research questions are raised dealing with the manner, in which SSD patients regulate HA, and with the relationship between particular ER strategies and HA. Existing research concerning mostly healthy or subclinical

samples indicate relationships between emotion dysregulation and increased HA (Bardeen & Fergus, 2014; Fergus & Valentiner, 2010; Görgen, Hiller, & Witthöft, 2014).

2.3.3 Autonomic imbalance in SSD: Heart rate and heart rate variability as indicators

This section contains characterizations of ANS functioning and its control of the cardioavascular system in particular. The chapter begins with descriptions of the cardiovascular system, followed by measuring instruments like the electrocardiogram (ECG), specific parameters like HR and HRV. The HRV is characterized in detail, including its neural/cortical pathways, and its proposed relationships with familiar constructs like ER.

In general, the cardiovascular system is responsible for blood transport and consequently ensures transport of oxygen, hormones and metabolites. Regulation of body temperature is another function of this system. Cardiovascular activity responds flexibly to environmental requirements. To cover supply needs of each organ system, cardiovascular activity increases in physical activity and decreases in resting periods. The heart as the center of the cardiovascular system works autorhythmic. That means that it can contract rhythmically on its own (Gramann & Schandry, 2009), which is due to specific characteristics of the heart fibers. Coronary fibers are reticularly connected with each other as a so-called functional syncytium, and partially capable of spontaneous excitation. The excitation can be triggered at a random spot and spreads rapidly across the entire heart. It typically begins in the sinus node, a part of the right atrium, which is why it is regarded as the pacemaker of the human heart (Birbaumer & Schmidt, 1996). From there excitation gets transmitted to the atrioventricular (AV) node. Right after the end of atrial contraction, excitation gets transmitted to the heart ventricle through HIS bundle and purkinje fibers. According to environmental influences, the heart´s

autorhythm gets modulated by sympathetic and parasympathetic heart fibers. These heart fibers belong to the ANS. During stress periods the heart underlies sympathetic, noradrenergic influences, resulting in increased beat frequency and pronounced muscle systole (contraction phase). In consequence, oxygenated blood gets carried through the arteries with more power. On the contrary, the heart underlies parasympathetic influence during resting periods via cholinergic vagus nerve fibers effecting decreased beat frequency (Gramann & Schandry, 2009).

The ECG depicts potential differences between depolarized and non-excited heart muscle cells. In a healthy heart, this translated into five peaks and waves (P, Q, R, S, T). The P-wave represents atrial excitation, PQ time the transmission of excitation from the sinus towards the AV node. The QRS-complex represents excitation expansion within the ventricle and the T-wave the ventricle repolarization. The R-wave is the most salient wave, representing a heartbeat. HR can be calculated by determining the number of heartbeats within one minute (BPM). Furthermore, phasic parameters like the variability of R wave intervals, so called HRV can be examined (Gramann & Schandry, 2009). HRV can be assessed by means of time- or frequency domain methods. The square root of the mean squared differences of successive normal to normal intervals (RMSSD) and the standard deviation of the normal to normal interval (SDNN) are two prominent time-domain measures. Normal to normal intervals describe temporal intervals between consecutive R waves. Frequency-based parametrization of ECG signals is carried out by the help of spectral analysis. Relevant measures are high-frequency (HF; indicator of parasympathetic influences), low frequency (LF; indicator of sympathetic and parasympathetic influences) and the low frequency / high frequency ratio (LF/HF), which is regarded as an indicator of sympatho-vagal balance (Schmidt & Martin, 2017; Task Force of the European Society of Cardiology, 1996). Sympathetic influence results in increased HRs and therefore in smaller intervals of consecutive heart beats. However,

this change in HR manifests rather slowly. On the contrary, parasympathetic nervous system (PNS) activation results in decreased HRs much more quickly, allowing for rapid and flexible modulation of cardiac activity depending on external demands. Consequently, PNS activation enhances autonomic flexibility, which manifests in increased HRV (Appelhans & Luecken, 2006). HRV is heavily influenced by respiratory frequency, resulting in rhythmic fluctuations of HR, caused by in- and expiration. This phenomenon, called respiratory sinus arrhythmia (RSA), is regarded as an index for vagal control of HRV (Berntson, Cacioppo & Quigley, 1993). Decreased HRV is viewed as an indicator of stress response and mental disorders (Thayer et al., 2012; Beauchaine & Thayer, 2015). Relationships between low HRV and psychopathologies have been investigated in various studies: in comparison to HC, lower HRV levels were found in patients with anxiety disorders (Chalmers, Quintana, Abbott, & Kemp, 2014), e.g. social anxiety disorder, patients with posttraumatic stress disorder, borderline personality disorder (Meyer et al., 2016), unipolar (Carney & Freedland, 2009) and bipolar depressive disorder (Hage et al., 2017).

Balzarotti and colleagues (2017) argued that cardiac vagal control might be a marker of ER, citing two prominent models describing neural pathways between the brain and the heart. The polyvagal theory suggests that ANS activity is closely related to emotional experience and types of behaviors like vocal communication, gestures or social behavior. Therefore, reduced vagal break is associated with fight or flight reactions and mental or behavioral disorders, whereas vagal influence appears to mobilize social behavior (Porges, 2009). The model of neurovisceral integration (Thayer & Lane, 2000) assumes that HRV is regulated by a so-called central autonomic network (CAN). Neural structures like the anterior cingulate, insular and ventromediale prefrontal cortex, the central nucleus of the amygdala, hypothalamic nuclei, parts of the medulla, and the periaqueductal gray belong to the network. The CAN receives and integrates visceral,

humoral, and environmental information (e.g. from the baroreceptor). Therefore, HRV gets modulated by central-peripheral feedback loops (Thayer & Lane, 2000). Empirical findings relating cardiac vagal control with ER suggest that participants with high baseline RSA show more reappraisal than suppression use when being exposed to emotional films (Volokhov & Demaree, 2010). Aldao and colleagues (2016) found associations between reduced spontaneous avoidance use and greater resting vagal tone.

Cardiovascular characteristics in SSD

Models of SFD (Kirmayer & Taileffer, 1997) assume both physiological disturbance and emotional arousal to promote somatic distress through various psychological mechanisms. Life stress is discussed to be an etiological factor for the development of FSS (Van Houdenhove et al., 2005). Associations between low vagal influence and increased stress perception (Dishman et al., 2000) seem to support the assumption. Studies on cardiovascular specifics in somatoform syndromes point to increased HRs and low finger pulse volume in comparison to HC, especially during cognitive burden (Rief et al., 1998). After mental stress, the cardiovascular system of somatoform patients appears to adapt less flexibly to resting periods compared to HC, which means that their HRs decelerate slower (Rief & Auer, 2001). Upcoming research adressing heightened, physiological activity did not focus on HR characteristics, but HRV instead. Therefore, the empirical basis to reveal specific HR characteristics compared to HC is not broad.

Associations between resting HRV and physical complaints have been investigated in larger numbers. With respect to FSS, results of a meta-analysis (Tak et al., 2009) revealed evidence for reduced vagal influence on the one hand, but pointed to limitations in the form of comparability of methodology, reported effect sizes, and

publication bias on the other hand. A later published meta-analysis revealed lower HF-HRV levels in patients with irritable bowel syndrome compared to HC (Liu, Wang, Yan, & Chen, 2013). Tak and colleagues (2010) advised to consider age-specific HRV-characteristics. They outlined negative associations between HF-HRV and reported symptom numbers in younger adults, whereas this association could not be found in elderly patients (Tak, Janssens, Dietrich, Slaets, & Rosmalen, 2010). Besides FSS, study results point to associations between higher HRV levels and increased pain tolerance in headache patients (Appelhans & Luecken, 2008). These patients reveal lower parasympathetic influence on HRV levels in comparison to HC (Koenig, Williams, Kemp, & Thayer, 2016). So far, only few studies investigated HRV activity and reactivity in SSD samples according to DSM-5. Huang and colleagues (2017) examined resting HRV in SSD compared to HC. Their results revealed significantly lower LF-HRV and total power-HRV levels in SSD. Regarding HF-HRV as a marker of vagal influence, there were no group differences. However, there were sex-dependent results. Only elder women revealed significant lower LF/HF-HRV than HC indicating less parasympathetic influence. Furthermore, associations between HF-HRV and depression could only be found in men.

HRV reactivity refers to the course of HRV levels during e.g. mental load like facial recognition tasks (Pollatos et al., 2011). So far, only few studies investigated HRV-reactivity in SSD, which is why there is insufficient knowledge about assumed inflexible, autonomic stress-responses. Lee and colleagues (2018) revealed higher SDNN-reactivity scores in SSD in comparison with HC, when being exposed to emotional stimuli. Results point to an inhibition of vagal control leading to inflexible parasympathetic response to emotional stressors. Huang and colleagues (2019) investigated HRV-reactivity in SSD when being exposed to health-related material. They found sex-dependent reactivity differences, outlining female participants having lower HF- and LF-

HRV levels when being exposed to those materials. However, recent findings need further elaboration.

As life stress and early adversive events are associated with the development and maintenance of FSS and SFD (e.g. Van Houdenhove et al., 2005; Kuwert et al., 2012; Nelson et al., 2012), daily-life stressors like a social conflict (SC) might be of interest to future experimental paradigms. As health-related cues are expected to induce illness-specific processing in SSD patients, conventional experimental paradigms would rely on those kinds of cues to gain the most substantial influence on HRV-reactivity in SSD patients. Implementing social stressors in experimental paradigms, would allow to compare the impact of different illness-related stressors on HRV-reactivity in SSD.

3 Aims and research questions of the conducted studies

In contrast to former SFD, SSD has different characteristics as the medical explanation of physical symptoms is not relevant for classification and psychological criteria are defined. These changes raise the question whether illness mechanisms in SFD are equally relevant for the entire SSD population (Rief & Martin, 2014). With respect to development and maintenance of SSD, Witthöft and colleagues (2018) discuss physiological factors besides cognitive, affective, and behavioral factors. Autonomic characteristics like increased physiological activity (Rief et al., 1998) and reactivity (Rief & Auer, 2001) have been assumed in SFD (Rief & Barsky, 2005). A cardiovascular measure that has gained particular attention during the past years is HRV. It is regarded as an indicator for the organism´s adaptability towards changing environmental conditions (Appelhans & Luecken, 2006). Existing research in the field of FSS (e.g. Tak et al., 2009) and first studies regarding SSD (e.g. Huang et al., 2017) point to lower resting HRV in comparison with HC. There are only few experimental studies investigating physiological reactivity (Lee et al., 2018; Huang et al., 2019) following induction of illness-related stress. ER is a further mechanism supposed to be relevant in the development and maintenance of SSD (e.g. Rief & Broadbent, 2007; Rief & Martin, 2014). So far, most results were obtained in pain research, outlining adverse effects of maladaptive ER on pain experience (e.g. Koechlin et al., 2018; Vowles et al., 2007). Even though there is evidence for ER deficits in MUS (Schwarz et al., 2017), empirical findings are not sufficient. Especially, given the modified classification in DSM-5 (APA, 2013), HA as affective SSD-symptom must be particularly considered when examining ER processes in SSD (APA, 2013). Existing findings outline associations between emotion dysregulation and increased HA in healthy or subclinical study samples

(Bardeen & Fergus, 2014; Fergus & Valentiner, 2010; Görgen et al., 2014). Relationships between ER and HA have not been investigated in clinical samples so far.

Assumed autonomic rigidity and ER deficits were examined with the help of a quasi-experimental research project called ´information processing during physical complaints´. It was carried out from 2015 to 2017 at the department facilities of clinical psychology and psychotherapy of the University of Wuppertal. Two studies were conducted referring to research data from the aforementioned project.

The first study ´Subjective and physiological reactivity to emotional stressors in somatic symptom disorder´ examined stressor-related subjective and physiological reactivity differences between SSD patients and HC. The study aimed to examine whether participants with SSD reveal higher HRs and lower HRV in comparison with HC, and whether HRV in SSD responds less flexible to changing experimental conditions (stressor and resting periods). With the help of subjective measures, it was examined whether people with SSD exhibit worse mood and increased tension after health-related and interpersonal stress induction in comparison with HC. Further, reactivity differences depending on the stressor type were investigated within SSD. Against the background of interpersonal stressors being a risk factor for FSS (Henningsen et al., 2018), it remains uncertain whether health-related stressors provoke more reactivity than interpersonal stressors. Another aim of the thesis was to examine potential reactivity differences between SSD subgroups as there is no evidence so far to refer assumed autonomic characteristics in SFD to the entire SSD population (Rief & Martin, 2014).

The second study ´Health anxiety in somatic symptom disorder: The impact of emotion regulation deficits´ aimed to clarify whether patients with SSD exhibit more use of maladaptive and less use of adaptive ER in comparison to HC. Additionally, potential subgroup differences were intended to be examined in SSD. A further aim was to

investigate whether particular ER strategies predict HA like catastrophizing and rumination (Marcus, Hughes, & Arnau, 2008), and whether different HA levels between SSD and HC result from ER differences.

The third study „Context dependent relevance of acceptance and coping in somatic symptom disorder" examined predictive power of coping and acceptance regarding symptom severity and disability. Pre-treatment data was used, obtained from a multicentric, randomized-controlled intervention study called ENCERT (Enriching Cognitive Behavior Therapy with Emotion Regulation Training) that is directed at patients with multiple MUS (Kleinstäuber et al., 2019). As pain research was heavily influenced by coping research up to the 1990s (Geisser, Robinson, & Riley, 1999), this control-oriented approach was critically questioned over the recent years (McCracken & Eccleston, 2003). Following ER research (Aldao et al., 2010), acceptance as an ER strategy received more and more interest in pain research, comparing acceptance and coping regarding their adaptability to manage chronic pain indicates acceptance to be a better predictor of the patient´s functioning level (McCracken & Eccleston, 2006). However, it is not clear whether these findings are transferable to SSD. A further research question dealt with contextual factors affecting relationships between acceptance or rather coping use and somatic burden. Existing findings suggest that the adaptability of specific strategy use depends, besides others, on emotion intensity (Dixon-Gordon et al., 2015). This study aimed to examine in what way the intensity of different symptom-related affects interacts with these aforementioned relationships.

4 Information processing during physical complaints

This chapter contains descriptions of two studies and additional analyses referring to data of the research project ´information processing during physical complaints´. It begins with the study ´Subjective and physiological reactivity to emotional stressors in somatic symptom disorder´, followed by the second study ´Health anxiety in somatic symptom disorder: The impact of emotion regulation deficits´. The chapter closes with a presentation of additional analyses that were not included in either one of the aforementioned studies. Procedures/participant instructions of the whole research project are presented in A-1.4.

4.1 Study 1: Subjective and physiological reactivity to emotional stressors in somatic symptom disorder

Objective: To date, little is known about autonomic reactivity in SSD. This study investigated the influence of health-related and social stressors on subjective and physiological reactivity in two types of SSD, with and without medically unexplained symptoms, in comparison to HC. The aim was to examine whether patients with SSD reveal less autonomic flexibility to experimentally presented stressors using HRV as an indicator.

Methods: In this experimental study, the total sample of N = 94 consisted of 29 participants with SSD-MUS, 33 participants with SSD-MES and 32 HC. Participants were exposed to both a health-related and a social stressor. Subjective and physiological variables were measured before, during and after stressor exposure, using state impairment measures, HR and HRV.

Results: Participants experienced higher tension and worse mood after exposure to stressors compared to pre-exposure (p = .002). The two SSD groups showed higher

levels of symptom intensity, impairment, and state tension as well as worse mood (p < .001), the SSD-MUS group showed a higher HR than HC (p = .012) during the experimental session. Compared to pre-exposure, symptom impairment increased after social stressor exposure in SSD-MUS (p < .001), but not in SSD-MES. HRV only decreased in HC during exposure, not in the SSD groups. The two SSD-groups did not differ in their reactivity to stressors.

Conclusion: HRV in SSD patients seems to respond less flexibly to stressors than HC, potentially reflecting overall physiological disturbance through reduced parasympathetic influence of the ANS.

4.1.1 Introduction

SSD is characterized by at least one persistent, burdensome physical symptom lasting for at least six months, accompanied by cognitive, affective, and behavioral health-related symptoms (APA, 2013). Patients with SSD make frequent use of medical services and with an estimated prevalence of 15 – 20 %, they are believed to represent the most widespread mental disorder in primary care (APA, 2013; Witthöft & Jasper, 2015). Contrary to former criteria for SFD (APA, 2000), affected people might not only suffer from MUS, but also from MES. Currently, existing knowledge about mechanisms involved in SSD, mostly relies on SFD (Rief & Martin, 2014).

Various models of SFD postulate an interaction of biological and psychological factors resulting in distressing somatic symptoms. In Kirmayer and Taileffer´s model (1997) for instance, somatic disability results from emotional arousal or physiological disturbance being amplified through perception and misattribution to serious illness and through maladaptive behaviors like communication of distress and avoidance. Despite the lack of a specific medical disease as a cause of somatoform symptoms, the contribution of biological factors is generally considered relevant in eliciting somatic

symptoms. These biological factors might refer to the endocrine and immune system and as well to autonomic physiological arousal (Rief & Barsky, 2005), potentially manifesting in elevated HRs (Rief et al., 1998).

Physiological arousal is generated and modulated by the ANS, which is comprised of two parts, the SNS and PNS. Depending on environmental demands, both SNS and PNS influence HR increases and decreases, and consequently the variation of temporally ordered interbeat intervals, labeled as HRV. Higher HRV seems to reflect physiological arousal being flexibly adjusted depending on changing situational demands, whereas lower HRV reflects autonomic rigidity (Appelhans & Luecken, 2006). Lower HRV resting activity is associated with major risk factors for cardiovascular disease (Thayer et al., 2010) and mental illness such as anxiety disorders (Chalmers et al., 2014; Gaebler et al., 2013), posttraumatic stress disorder, borderline personality disorder (Meyer et al., 2013) and depression (Carney & Freedland, 2009; Hage et al., 2017).

With regard to resting HRV in patients with somatic symptoms, a LF-HRV appears to be related to higher pain tolerance (Appelhans & Luecken, 2008) on the one hand. On the other hand, when compared to HC, patients with chronic pain (Tracy et al., 2016), and headaches (Koenig et al., 2016), showed lower HF-HRV associated with predominant parasympathetic activity. In FSS, results also point to lower HRV, but increased sympathetic activity in fibromyalgia and chronic fatigue (only at night) compared to HC (Meeus et al., 2013). The HRV values for irritable bowel syndrome, however, were not consistently different from the HC, as deviations were only found in specific subgroups (Mazurak et al., 2012). A meta-analysis on 14 studies revealed lower PNS activity (HF-HRV and RMSSD) in FSS in comparison to control participants. These results, however, should be evaluated critically due to heterogeneous effect sizes and risk of publication bias (Tak et al., 2009).

Based on DSM-5 criteria of SSD, one recent study found lower levels of both total-power HRV and low-frequency HRV for SSD patients in comparison to HC (Huang et al., 2017). As HF-HRV-levels and standard deviations of normal to normal R wave (SDNN) and RMSSD were not relatively decreased in SSD, a reduced parasympathetic activity as previously suggested, could not be found. There are therefore a series of empirical indications for HRV abnormalities in persistent somatic symptoms, but the findings are inconsistent and rarely investigated directly in SSD. For example, autonomic function was mostly investigated under resting conditions. Furthermore, to date, only a few studies examined HRV reactivity in patients with PCs. Some experimental tasks only examined HRV reactivity in relation to emotion recognition or pain sensitivity (Pollatos et al., 2011a, b). Few studies investigating SSD pointed to reduced vagal control when being exposed to emotional stressors (Lee et al., 2018). Furthermore, female SSD patients revealed lower HF and LF levels in comparison with HC during exposure to health-related material (Huang et al., 2019). Other stressor types associated with onset and maintenance of PCs like interpersonal stressors (Van Houdenhove et al., 2005) have not been implemented in experimental designs examining HRV reactivity in SSD.

Based on the new DSM-classification (APA, 2013), SSD not only comprises SSD-MUS, as seen in formerly named SFD, but also includes SSD-MES. Rief and Martin (2014) questioned whether illness-related mechanisms in SSD-MUS have the same relevance in SSD-MES. Therefore, this study investigated whether proposed autonomic reactivity in SSD-MUS also applies to SSD-MES.

For a method of stress induction, this study refers to an approach used by Schwarz and colleagues (2016). Using script driven imagery, Schwarz and colleagues showed increased state symptom intensity and disability for patients with MUS, and increased state impairment ratings for HC. This method enables the investigation of psychological and physiological processes associated with different kinds of stressors.

The aim of this study was to examine subjective and physiological reactivity in SSD and HC, resulting from individual health-related and social stressors. Health-related stressors are expected to provoke higher autonomic reactivity for SSD patients than for HC. As previous research in FSS outlined life events to be etiologically relevant for symptom development (Van Houdenhove et al., 2005), this study hypothesized that SCs also provoke reactivity. It was expected to see higher levels of subjective symptom intensity, disability, tension, health beliefs, health worries and negative affect for SSD than for HC with the clinical groups showing higher stressor-related subjective reactivity. Further, higher HRs and lower HRV were predicted for SSD in comparison with HC. Specifically, the study expected higher HR increases and autonomic rigidity in SSD, manifesting in reduced HRV adaption in time domains (RMSSD, SDNN) before and during stress inductions. Additionally, the study explored subgroup differences in SSD and reactivity differences between clinical and control group depending on the stressor type.

4.1.2 Methods

The methods section begins with a description of the study design, followed by participant characteristics, the study procedure, used self-report and physiological measures, and a description of statistical analyses.

Study design

The design of the study was quasi-experimental with a 2 (exposure) × 2 (stimulus type) × 3 (group) mixed design. The study was conducted at the University of Wuppertal, Germany. Following diagnostic interviews, participants were classified as either SSD-MUS, SSD-MES or HC. The study consisted of measuring participants´ physiological activity before and during exposure to stressors. To induce stress, participants listened

to two forms audio scripts that they had put together themselves with support of a study assistant. One audio script was health-related and the other dealt with a SC. Each participant was exposed to both audio-scripts in a randomized order. Participants also provided self-report data on their subjective reactions before and after stress exposure. They were also debriefed after the experiment. All study procedures were performed in accordance with the declaration of Helsinki and the study protocol was approved by the ethics review board at the University of Wuppertal (see A-1.1). All participants received written and oral information about the study procedure and provided written and oral informed consent (see A-1.2) before diagnostic interviews took place. Participating undergraduate psychology students received course credit as an incentive.

Participants

A total of 94 participants were recruited, consisting of 29 patients with SSD-MUS, 33 patients with SSD-MES and 32 HC. Participants were aged between 18 and 70 years. They were included on the basis that they were without the following: drug and/or substance abuse; an acquired brain damage; a current pregnancy; heart pace maker; psychosis or history of bipolar disorder; a current suicidality; a continuous medication with antipsychotics or benzodiazepines. For the control group, only participants who did not suffer from disabling body symptoms or any kind of mental disorders were included in the study.

Participants with SSD-MUS were required to report at least one burdensome bodily symptom that could not be sufficiently explained by any known medical explanation. Patients with SSD-MES were required to report at least one burdensome bodily symptom associated with a medical disease factor. Both groups needed to fulfil DSM-5 criteria of SSD as a primary diagnosis. The presence of comorbid diagnoses and a sporadic intake of pain medication were allowed.

Study procedure

This section depicts the recruitment and assessment process, including a detailed description how cardiovascular activity was measured before and during stressor exposure.

Recruitment and assessment

Participants were recruited in cooperation with general practitioners, specialists at the Helios Medical Clinic Wuppertal, and via notices on the university´s bill-boards, information flyers, announcements on the website of the department. An initial brief telephone interview served for screening of basic inclusion and exclusion resulting in the exclusion of *N* = 38 participants (detailed trial profile in A-1.12). Participants were invited to the facilities of the Department of Clinical Psychology and Psychotherapy at the University of Wuppertal. Following completion of a sociodemographic questionnaire (see A-1.5), participants were examined via the Mini-DIPS (Margraf, 1994) and the SSD-interview (Rief, Mewes, Martin, Glaesmer, & Braehler, 2010; Kleinstäuber, Gottschalk, Berking, Rau, & Rief, 2016). The latter was conducted in order to diagnose SSD according to DSM-5. Following the interview, participants completed self-report measures in a computerized format. They were then randomly allocated to either one of the two experimental conditions (i.e., order of stress exposure). Participants then created the two auditory stressors with support of the study assistant. Once these were complete, electrodes for electrocardiography (ECG) were placed in chest-lead configuration.

Physiological activity under experimental stress inductions

The study examined cardiovascular activity prior and during experimental stress inductions. At first, participants underwent a resting period of two minutes, in which HR

and HRV were assessed. They then underwent a distractor task, which was meant to neutralize any stress or affective reaction prior to the following induction. Participants therefore listened to a two-minute long radio clip dealing with a neutral topic (history of small umbrellas), followed by six multiple choice questions relating to the radio clip (see A-1.9). These questions had to be answered within one minute without receiving any feedback. Before stress induction, they rated their current symptom intensity, symptom impairment, tension and mood. Based on the results of randomization, participants were exposed either to the health-related or to the social stressor first. The auditory script was presented via loudspeakers, and during this one-minute induction HR and HRV were assessed. Following this, participants rated their current state of symptom intensity, impairment, tension, health beliefs, health worries and mood again. Participants underwent a two-minute long period to return their stress level to baseline, followed by a second distractor task (see A-1.10). The procedure was repeated with exposure of the other stressor type. Shortly before and after the second exposure, during which HR and HRV were recorded, participants rated their current status again.

At the end of the experiment, study assistants verified that no participants experienced unpredictable stress throughout both exposures. Participants were then debriefed and received information about the study. The experimental design is depicted in figure 2.

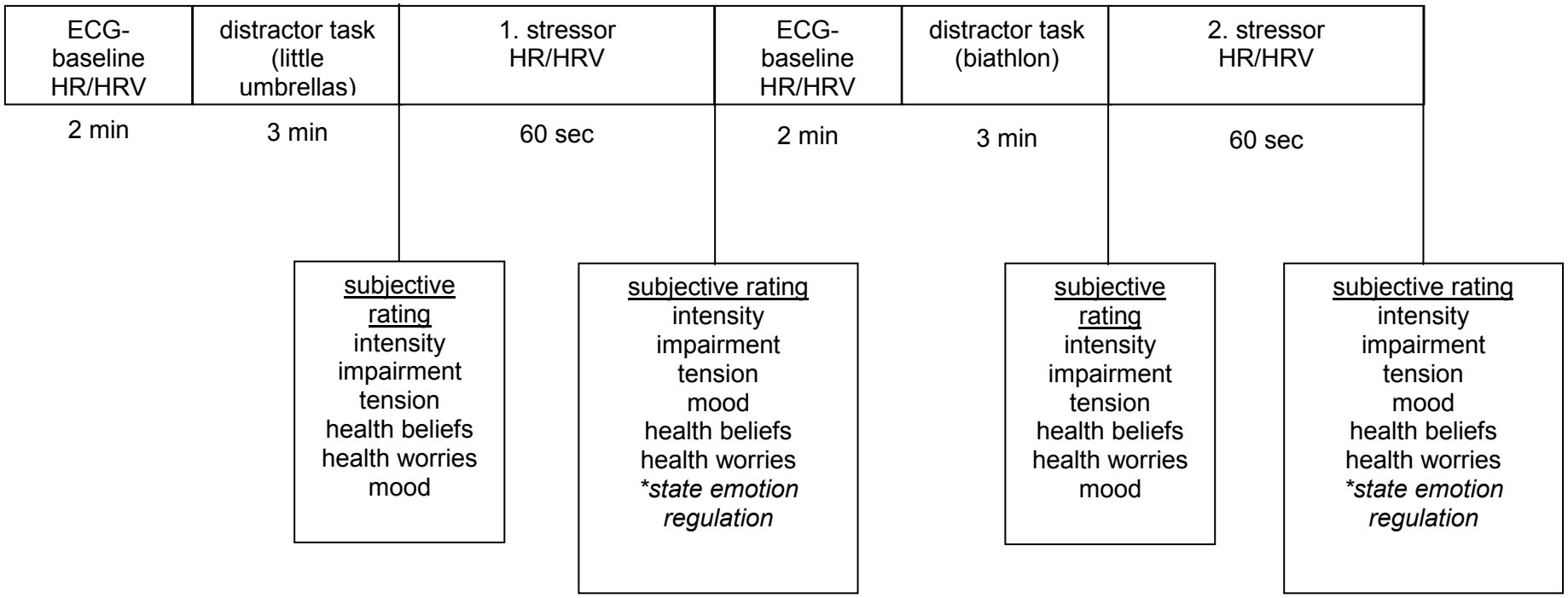

Figure 2 Experimental study sequences

* Further descriptions and analyses of this measure are depicted in section 4.3.

Measures

This section contains a detailed description of used diagnostic and cardiovascular measures, starting with diagnostic interviews (SSD-interview, Mini-DIPS), followed by self-report measures with regard to symptom severity and disability. Afterwards used state rating scales are revealed, followed by a detailed description of ECG application and stressor audiofile creation.

SSD-interview

The *SSD-interview* is a structured diagnostic interview to assess SSD (Kleinstäuber et al., 2016; Rief et al., 2010). It includes three sections designed to ascertain the presence of MUS and MES, related psychological features, and the medical history of a participant. In the first section, the presence of 64 physical symptoms gets assessed with respect to lifetime, during the past 12 months, and within the past 7 days. This section also evaluates impairment, consultation with a doctor and possible medical explanations for each symptom. Here, the presence of physical symptoms of at least moderate severity (≥ 2; scale of 0-4) during the the past 12 months was required. Somatic complaints were rated as MUS when participants reported them as being not, or not fully, medically explained. Symptoms were rated as MES when participants reported them as fully explained by a diagnosed medical condition. Sum scores referring to the numbers of MUS or MES and total symptom scores were computed.

The second section consists of 28 dichotomous items that cover 22 psychological features, which may accompany somatic complaints. According to Rief et al. (2010), nine items are particularly crucial for assessing the presence of DSM-5 B-criteria used in the present study. Catastrophizing about physical symptoms, and somatic illness beliefs represent the first SSD-B-criterion and health worries represent the second criterion. The

remaining items (ruminations about bodily complaints, bodily self-observation, negative self-concept of bodily weakness, avoidance of physical activities that can provoke symptoms, disuse of body parts, need for immediate medical care), comprise the third SSD-B-criterion. For SSD diagnosis, at least one MUS/MES lasting at least six months, and one cognitive, affective or behavioral symptom in accordance with SSD criterion B were required.

Mini-DIPS

The *Mini-DIPS* (Margraf, 1994) is a short version of the Diagnostic Inventory of Mental Disorders (DIPS), and allows for the assessment of common mental disorders such as anxiety, affective and eating disorders, substance abuse and dependency and psychotic symptoms according to DSM-IV criteria. Examination of interrater- and retest-reliability revealed good to very good results (Margraf, 1994). The Mini-DIPS was used to assess comorbid disorders in SSD participants and to exclude mental disorders in HC.

Patient Health Questionnaire-15 (PHQ-15)

The PHQ-15 (Kroenke, Spitzer & Williams, 2002) is a widely-used, self-report measure to assess somatic symptom severity for the previous four weeks on a 3- point scale (0 = *not bothered* to 2 = bothered a lot). The sum score of the 15 symptoms represents total symptom burden. Various studies revealed good validity and acceptable internal consistency of the PHQ-15 (Kroenke et al., 2010). In the present study, the Cronbach´s alpha of .80 shows good internal consistency.

Pain Disability Index (PDI)

The modified version of the PDI (Mewes et al., 2009) is a self-report measure to assess functional impairment due to PCs in seven facets of daily life on 11-point scales (0 = *no disability* to 10 = *total disability*). These facets deal with family/home responsibilities, recreation, social activities, occupation, sexual behavior, self-care, life support activity. This measure provides good internal consistency (α = .90). The PDI total score showed high correlations with measures of somatic complaints and mental health (Mewes et al., 2009).

Somatic symptoms - state ratings

Six items on numeric ratings scales (range 1 - 9) indicated state symptom intensity (´*How intense do you experience your physical complaints at present?´* ; 1 = *not at all*, 9 = *very much*), state symptom impairment (´*How much do your physical complaints impair you at present?´* ; 1 = *not at all*, 9 = *very much*), state tension (´*How tense are you at present?´* ; 1 = *not tensed at all*, 9 = *very tensed*), state health beliefs ("*How strong do you believe at present to suffer from a serious disease?"* ; 1 = *not at all*, 9 = *very much*), state health worries ("*How much do you worry about your health at present?"* ; 1 = *not at all*, 9 = *very much*) and state mood (´*What is your current mood?´* ; 1 = *very bad*, 9 = *very good*) of the participants. Higher scores indicated higher degrees of symptom intensity, symptom impairment, tension and positive mood. The numeric rating scales are presented in A-1.11.

Electrocardiogram

ECG was recorded in chest lead configuration with a Biopac MP 150 system, with a sampling rate of 500 Hz using associated *Acqknowledge* 4.4.2 software. Hardware

configurations of the Biopac MP 150 were adjusted to optimal R-wave detection (R-Wave mode implemented in the hardware), baseline stabilizer, and 0.5 Hz high-pass filter. Electrocardiographical data were analyzed using BPM, and HRV time domains RMSSD and SDNN. The data was cleaned with *EKG-Bio*, Version 2.73 (*Periphysys GmbH*, Potsdam, Germany) using the pre-implemented artefact correction component of the software. Further, visual artefact control was conducted in order to check falsely detected NN intervals, which the software allows to manually adjust. Mean HR and HRV (RMSSD, SDNN) were analyzed during the last 30 seconds of both baseline tracks and the first 30 seconds of both induction tracks, using 15 Hz low-pass filter. The 30 seconds segments were divided into 10-seconds sections. Within one 10-second section, at least two NN-intervals had to be valid in order to include the whole 30 seconds section in the data set.

Stress induction audiofiles

Participants created two emotional, auditory stressors based on scripts, one related to somatic symptom experiences and the other to a SC experience describing accompanying body sensations, thoughts and emotions (following Schwarz et al., 2016). In relation to the impairing physical symptom (see A-1.7) and the current SC (see A-1.8), study participants evaluated the quality of the symptom/conflict (e.g. *´Which somatic symptom/social conflict do you suffer from the most?´*), impairment (e.g. *´How do you rate current somatic/interpersonal impairment on a scale of 0 to 100?´*), accompanying thoughts (e.g. *´What is on your mind now?´*), feelings (e.g. *´Can you describe what you feel at this moment?´*), and body sensations (e.g. *´Can you describe what body sensations you perceived? How did it start? What was the worst?´*). In contrast to SSD participants, healthy participants were asked to describe the last occasion they experienced impairing PCs, for example, due to an injury or a disease.

With the support of the study assistants, participants wrote their notes down in full sentences in order to make an audio recording of them afterwards. With regard to these notes, there were no restrictions except that it should be possible to fully read one script aloud within one minute.

Statistical analyses

A priori power analyses in G*Power 3.1.9.2 (Faul, Erdfelder, Buchner, & Lang, 2009) estimated that an overall sample size of at least n = 78 would be required, based on the detection of small to medium effects (α = .05 and 1-β = .80). Data analyses were conducted using the 22 nd version of the IBM Statistical Package for Social Sciences (SPSS). With respect to categorical sociodemographic data, χ^2 - tests or exact fisher tests in the case of small samples, were conducted. For state symptom intensity, impairment, tension, health beliefs, health worries, mood, HR, and HRV (both RMSSD and SDNN), 3 $\times$ 2 $\times$ 2 mixed ANOVAs with repeated measures were conducted. Group (SSD-MUS, SSD-MES, HC) served as the between-participants factor. Exposure (pre-exposure vs. exposure) and stimulus type (PC vs. SC) served as within-participants factors. With respect to main effects of group, Games-Howell or Bonferroni-post-hoc analyses were conducted depending on the results from Levene´s-tests of homogenity of variances (see A-1.13). In the case of further univariate main or interaction effects post-hoc pairwise comparisons of cell means were conducted with Bonferroni corrections for multiple tests. With respect to both HRV measures, exploratory analyses were conducted, and four subjects taking blood-pressure medication excluded, before performing 3 x 2 x 2 again. Further, control analyses in the form of 3 x 2 x 2 x 2 mixed ANOVAs with repeated measures were conducted (with chronological order of stressor-

presentation as further between-subjects factor) to rule out that the chronological order of stressor-presentation has undesired effects on the results.

4.1.3 Results

The results section contains a presentation of sociodemographic and clinical characteristics.

Participant characteristics

Overall, participants were predominantly female (68.1 %), middle aged (*M* = 39.78) and highly educated (43.6 % with university entrance qualification). Groups did not differ in age, *p* = .419, ratio of women, *p* = .777 and family status, *p* = .814. Compared to controls, degrees of symptoms severity was elevated in SSD-MUS (*M* = 10.27, *SD* = 4.77) and SSD-MES (*M* = 10.18, *SD* = 4.29) with no difference between the two clinical groups, *mean difference* = .09, *p* = .928. Patients with SSD-MUS (*M* = 3.37, *SD* = 1.87) and SSD-MES (*M* = 3.37, *SD* = 2.05) also revealed elevated degrees of symptom disability with no difference between groups, *mean difference* = .00, *p* = .997. Further, the SSD groups did not differ in symptom duration, *p* = .078, and the presence of comorbid disorders, *p* = .768. No difference was found between all groups for BMI, *p* = .450, and beta-blocker intake, *p* = 1.000. Groups differed, however, in the ratio of subjects with opiate, *p* = .013, and antidepressants intake, *p* = .006. Detailed sociodemographic and clinical characteristics of all three groups are presented in Table 1.

Table 1 Sociodemographic and clinical characteristics

Variable	SSD-MUS	SSD-MES	HC	Statistics
N	29	33	32	
Age *M* (*SD*)	42.62 (15.29)	39.77 (15.74)	37.22 (19.28)	$F(2,92)= .78$
Female % (*N*)	65.5 (19)	72.7 (24)	65.6 (21)	$\chi^2(2)= 0.50$, $p = .777$
Family status % (*N*)				$\chi^2(4)= 3.43$, $p = .488$
Single % (*N*)	31.0.(9)	45.5 (15)	34.4 (11)	
Relationship % (*N*)	24.1 (7)	18.2 (6)	34.4 (11)	
Married % (*N*)	44.8 (13)	36.4 (12)	31.3 (10)	
BMI *M* (*SD*)	23.28 (9.94)	21.75 (8.38)	22.72 (5.14)	$F(2,92)= 0.30$
Symptom duration in years *M* (*SD*)	10.78 (11.28)	14.85 (12.16)		$T(60) = -1.36$
Number of MUS *M* (*SD*)	8.17 (7.27)	2.33 (2.91)		$T(35.84) = 4.05$***
Number of MES *M* (*SD*)	2.41 (2.91)	7.09 (4.92)		$T(52.95) = -4.62$***
Number of physical symptoms *M* (*SD*)	10.59 (8.36)	9.42 (6.49)		$T(60) = .62$, $p = .541$
Symptom severity [PHQ-15] *M* (*SD*)	10.27 (4.77)	10.18 (4.29)	2.97 (3.02)	$F(2,92)= 33.55$***
Mean Disability [PDI] *M* (*SD*)	3.37 (1.87)	3.37 (2.05)	.67 (1.09)	$F(2,92)= 25.92$***
Number of fulfilled psychological SSD-features [SSD-Interview] *M* (*SD*)	3.38 (1.63)	3.52 (1.66)		$T(60) = -.32$
Depressive disorder % (*N*)	13.8 (4)	24.2 (8)	-	
Comorbid disorder % (*N*)				$\chi^2(3)= 1.14$
No comorbid disorder % (*N*)	65.5 (19)	57.6 (19)	-	
1 comorbid disorder % (*N*)	27.6 (8)	27.3 (9)	-	
2 comorbid disorders % (*N*)	3.4 (1)	6.1 (2)	-	
More than 2 comorbid disorders % (*N*)	3.4 (1)	9.1 (3)	-	
Medication				
Beta-blocker % (*N*)	3.4 (1)	6.1 (2)	3.1 (1)	$p = 1.000$
Opiate % (*N*)	3.4 (1)	18.2 (6)	0 (0)	$p = .013$*
Other pain medication % (*N*)	3.4 (1)	15.6 (5)	3.1 (1)	$p = .209$
Anxiolytics % (*N*)	3.4 (1)	6.1 (2)	0 (0)	$p = .523$
Antidepressants % (*N*)	24.1 (7)	15.2 (5)	0 (0)	$p = .006$**

Note. *** $p < .001$; ** $p < .01$; * $p < .05$

Group-related effects and reactivity in self-report measures

This section reveals group differences with respect to presented rating scales, starting with state symptom intensity, followed by symptom impairment, tension, health beliefs, health worries and mood. As the chronological order of stressor-presentation has no substantial effect on the results, the aforementioned control analyses are not presented here, but in section A-1.14 and A-1.15 instead.

State symptom intensity

With respect to state symptom intensity, there was a main effect of group, $F(2,92) = 29.73$, $p < .001$, $\eta^2 = .40$. Patients with SSD-MUS ($p < .001$) and SSD-MES ($p < .001$) had higher levels of symptom intensity than HC, with no difference between SSD groups, $p = .560$. The level of symptom intensity did not change after the stressor exposure.

State symptom impairment

Concerning state symptom impairment, a main effect of group was revealed, $F(2,92) = 26.09$, $p < .001$, $\eta^2 = .36$. Patients with SSD-MUS ($p < .001$) and SSD-MES ($p < .001$) had higher levels of symptom impairment than HC, with no difference in levels of symptom impairment between the SSD groups, $p = .511$. Further, there was a main effect of exposure, $F(1,93) = 8.07$, $p = .006$, $\eta^2 = .08$. Compared to pre-exposure, levels of symptom impairment increased after exposure, $p = .006$. A group x stimulus type x exposure interaction was observed, showing a stimulus type x exposure interaction for SSD-MUS only, $F(1,93) = 3.94$, $p = .023$, $\eta^2 = .08$, suggesting impairment level increases after SC exposure within SSD-MUS, compared to pre-exposure, $p < .001$.

State tension

Concerning state tension, there was a main effect of group, $F(2,92) = 17.04$, $p < .001$, $\eta^2 = .27$. Tension was higher in both SSD-MUS ($p < .001$) and SSD-MES ($p < .001$) in comparison with HC, with no differences between SSD-MUS and SSD-MES, $p = 1.000$. Further, there was a main effect of exposure, $F(1,93) = 10.02$, $p = .002$, $\eta^2 = .10$. Levels of tension increased after exposure, compared to pre-exposure, $p = .002$. State tension prior and after health-related stressor-exposure did not differ, $F(1,93) = .87$, $p = .353$. Instead, tension levels prior and after social stressor-exposure differed significantly, $F(1,93) = 17.49$, $p < .001$. Compared to pre-exposure, tension increased after social stressor-exposure, $p < .001$.

State health beliefs

With respect to state health beliefs, there was a main effect of group, $F(2,92) = 17.40$, $p < .001$, $\eta^2 = .28$. Health beliefs were more pronounced in both SSD-MUS ($p < .001$) and in SSD-MES ($p < .001$) in comparison with HC, without any differences between SSD-MUS and SSD-MES, $p = .118$. Health beliefs did not change after exposure, $p = .181$, and there were no health belief differences in dependence of the stressor type, $p = .088$.

State health worries

With respect to state health worries, there was a main effect of group, $F(2,92) = 23.12$, $p < .001$, $\eta^2 = .34$. Health worries were more pronounced in both SSD-MUS ($p = .033$) and in SSD-MES ($p < .001$) in comparison with HC. Patients with SSD-MES revealed more pronounced health worries than patients with SSD-MUS, $p = .002$. Health worries did not change after exposure, $p = .057$, and there were no health worry differences in dependence of the stressor type, $p = .633$.

State mood

With respect to state mood, there was a main effect of group, $F(2,92) = 8.84$, $p < .001$, $\eta^2 = .16$. Mood in both SSD-MUS and SSD-MES was overall worse than for HC, $p < .001$. Further, there was a main effect of exposure, $F(1,93) = 10.27$, $p = .002$, $\eta^2 = .10$. Within participants, mood worsened after exposure, compared to pre-exposure, $p = .002$. However, there were no reactivity differences between groups. Detailed information can be found in Table 2.

Table 2 Group differences in stressor-related subjective reactivity

Variable	Stimulus type	Exposure	SSD-MUS *M (SD)*	SSD-MES *M (SD)*	HC *M (SD)*	Main effects	Interaction effects
Symptom intensity	PC	P	4.45 (2.25)	3.73 (2.25)	1.09 (.39)	$F_{Gr}(2,92) = 29.73$***	$F_{Gr \times Ex}(2,92) = 0.70$
		E	4.31 (2.05)	3.82 (2.34)	1.13 (.42)	$F_{Ex}(1,93) = 2.21$	$F_{Gr \times ST}(2,92) = 0.46$
	SC	P	4.07 (2.09)	3.61 (2.32)	1.13 (.42)	$F_{ST}(1,93) = 1.21$	$F_{Gr \times ST \times Ex}(2,92) = 1.79$
		E	4.38 (2.19)	3.82 (2.20)	1.09 (.30)		$F_{ST \times Ex}(1,93) = 2.38$
Symptom impairment	PC	P	4.31 (2.35)	3.36 (2.34)	1.03 (.18)	$F_{Gr}(2,92) = 26.09$***	$F_{Gr \times Ex}(2,92) = 0.94$
		E	4.14 (2.12)	3.52 (2.32)	1.06 (.25)	$F_{Ex}(1,93) = 8.07$**	$F_{Gr \times ST}(2,92) = 2.13$
	SC	P	3.62 (1.97)	3.33 (2.29)	1.03 (.18)	$F_{ST}(1,93) = 1.71$	$F_{Gr \times ST \times Ex}(2,92) = 3.94$*
		E	4.10 (1.95)	3.58 (2.32)	1.09 (.30)		$F_{ST \times Ex}(1,93) = 6.96$*
Tension	PC	P	4.76 (1.84)	4.67 (2.11)	2.81 (1.47)	$F_{Gr}(2,92) = 17.04$***	$F_{Gr \times Ex}(2,92) = 0.35$
		E	4.90 (1.61)	4.79 (2.23)	4.19 (2.01)	$F_{Ex}(1,93) = 10.02$**	$F_{Gr \times ST}(2,92) = 1.32$
	SC	P	4.55 (1.92)	4.76 (2.12)	3.97 (2.02)	$F_{ST}(1,93) = 0.64$	$F_{Gr \times ST \times Ex}(2,92) = 0.50$
		E	5.21 (1.72)	5.36 (2.23)	4.48 (2.15)		$F_{ST \times Ex}(1,93) = 5.58$*
Health beliefs	PC	P	2.00 (.96)	3.94 (2.93)	1.31 (2.22)	$F_{Gr}(2,92) = 17.40$***	$F_{Gr \times Ex}(2,92) = 1.10$
		E	2.03 (.98)	4.15 (2.93)	1.41 (1.10)	$F_{Ex}(1,93) = 3.72$	$F_{Gr \times ST}(2,92) = 0.24$
	SC	P	2.10 (1.11)	3.94 (2.87)	1.34 (1.10)	$F_{ST}(1,93) = 0.23$	$F_{Gr \times ST \times Ex}(2,92) = 0.00$
		E	2.10 (1.23)	4.12 (2.93)	1.41 (1.10)		$F_{ST \times Ex}(1,93) = 0.21$
Health worries	PC	P	3.52 (1.77)	4.79 (2.64)	1.53 (.72)	$F_{Gr}(2,92) = 23.12$***	$F_{Gr \times Ex}(2,92) = 1.09$
		E	3.66 (1.80)	4.36 (2.79)	1.50 (.72)	$F_{Ex}(1,93) = 1.82$	$F_{Gr \times ST}(2,92) = 0.79$
	SC	P	3.34 (1.78)	4.48 (2.68)	1.50 (.62)	$F_{ST}(1,93) = 2.97$	$F_{Gr \times ST \times Ex}(2,92) = 2.19$
		E	3.17 (1.85)	4.39 (2.63)	1.47 (.57)		$F_{ST \times Ex}(1,93) = 0.00$
Mood	PC	P	5.97 (1.92)	5.91 (1.79)	7.03 (1.38)	$F_{Gr}(2,92) = 8.84$***	$F_{Gr \times Ex}(2,92) = 1.56$
		E	5.66 (1.78)	5.79 (2.03)	7.03 (1.26)	$F_{Ex}(1,93) = 10.27$**	$F_{Gr \times ST}(2,92) = 1.42$
	SC	P	5.93 (1.93)	5.85 (1.79)	7.25 (1.11)	$F_{ST}(1,93) = 1.46$	$F_{Gr \times ST \times Ex}(2,92) = 0.10$
		E	5.21 (1.80)	5.42 (1.92)	7.03 (1.20)		$F_{ST \times Ex}(1,93) = 3.15$

Note. ST = stimulus type; Ex = exposure; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; P = pre-exposure; E = exposure period;*** $p < .001$; ** $p < .01$; * $p < .05$

Group-related effects and reactivity in physiological measures

This section reveals group differences with respect to physiological measures, starting with HR, followed by group difference results concerning RMSSD and SDNN, which were controlled for heart medication.

Heart rate

With respect to HRs, there was a main effect of group, $F(2,92) = 4.53$, $p = .013$, $\eta^2 = .09$. Although HRs were elevated in SSD-MES in comparison with HC, significant higher HRs could only be obtained in SSD-MUS ($p = .012$). However, HRs in SSD-MUS and SSD-MES did not differ, $p = .846$. Further, there was a main effect of exposure, $F(1,93) = 25.51$, $p < .001$, $\eta^2 = .22$. HRs increased during the exposure period compared to pre-exposure, $p < .001$. There was no group x exposure interaction, $p = .850$.

Heart rate variability

With respect to RMSSD, there were no main effects of group or exposure. However, a group x exposure interaction, $p = .045$ was found. This interaction remained significant when four subjects with heart medication were excluded in control analysis, $F(2,92) = 3.61$, $p = .031$, $\eta^2 = .08$. With respect to RMSSD, there was a significant group effect at baseline, $F(2,92) = 3.46$, $p = .036$, $\eta^2 = .07$. Pairwise comparisons did not reveal any significant differences between the groups. In comparison with both SSD-groups, RMSSD levels within HC decreased from baseline to induction, $p = .003$. In contrast, RMSSD levels in both SSD-MUS and SSD-MES did not change from baseline to induction, $p = .874$ - $.957$.

Regarding SDNN, there were no group and exposure effects, instead, a significant group x exposure interaction was found, $p = .046$. This interaction remained

significant, $F(2,92) = 3.50$, $p = .034$, $\eta^2 = .07$, when participants with heart medication were excluded in control analysis. However, groups did not differ during baseline, $F(2,92) = 2.27$, $p = .109$, or during induction period, $F(2,92) = .08$, $p = .921$. SDNN levels did not change in any one of the three groups, $p = .246 – 309$. Detailed information can be found in Table 3.

Table 3 Group differences in stressor-related physiological reactivity

Variable	ST	Ex	SSD-MUS *M (SD)*	SSD-MES *M (SD)*	HC *M (SD)*	Main effects	Interaction effects
Heart rate	PC	P	77.23 (13.67)	73.43 (12.67)	67.56 (10.14)	$F_{Gr}(2,92) = 4.53$*	$F_{Gr \times Ex}(2,92) = 0.16$
		E	78.82 (13.18)	75.75 (12.92)	69.94 (11.39)	$F_{Ex}(1,93) = 25.27$***	$F_{Gr \times ST}(2,92) = 0.03$
	SC	P	77.56 (13.77)	74.54 (12.97)	68.18 (11.10)	$F_{ST}(1,93) = 5.03$*	$F_{Gr \times ST \times Ex}(2,92) = 0.49$
		E	79.59 (13.73)	76.01 (13.10)	70.45 (10.58)		$F_{ST \times Ex}(1,93) = 0.12$
RMSSD	PC	P	2.17 (1.34)	2.39 (1.10)	3.17 (1.57)	F_{Gr} (2,92) = 2.26	$F_{Gr \times Ex}(2,92) = 3.21$*
		E	2.29 (1.29)	2.38 (1.21)	2.64 (1.52)	$F_{Ex}(1,93) = 2.85$	$F_{Gr \times ST}(2,92) = 0.18$
	SC	P	2.36 (1.53)	2.36 (1.24)	3.01 (1.61)	$F_{ST}(1,93) = 0.09$	$F_{Gr \times ST \times Ex}(2,92) = 2.50$
		E	2.23 (1.20)	2.39 (1.09)	2.80 (1.59)		$F_{ST \times Ex}(1,93) = 0.14$
SDDN	PC	P	2.50 (1.40)	2.58 (1.38)	3.15 (1.58)	$F_{Gr}(2,92) = 2.26$	$F_{Gr \times Ex}(2,92) = 3.20$*
		E	2.71 (1.42)	2.92 (1.89)	2.90 (1.74)	$F_{Ex}(1,93) = 0.86$	$F_{Gr \times ST}(2,92) = 0.03$
	SC	P	2.48 (1.38)	2.70 (1.43)	3.30 (1.99)	$F_{ST}(1,93) = 0.87$	$F_{Gr \times ST \times Ex}(2,92) = 0.30$
		E	2.88 (1.59)	2.94 (1.56)	2.98 (1.59)		$F_{ST \times Ex}(1,93) = 0.00$

Note. ST = stimulus type; Ex = exposure; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; P = pre-exposure; E = exposure period;*** $p < .001$; ** $p < .01$; * $p < .05$

4.1.4 Discussion

The aim of the study was to examine subjective and physiological reactivity for patients with SSD and HC in association with individual health-related and social stressors. The first research question referred to general group differences in subjective state measures. Group main effects suggesting higher symptom intensity and impairment ratings in SSD confirmed classificatory diagnostics. Higher tension and worse mood in SSD might reflect affective-physiological consequences of perceived symptom impairment. However, although mood-related group differences were found, mean state mood ratings in SSD were moderate. While the study expected increased physiological arousal in both SSD groups compared to HC, mean HR was only higher in SSD-MUS, which confirms previous findings in SFD (e.g. Rief et al., 1998; Rief & Barsky, 2005). HRV levels in SSD were not generally lower as proposed in previous research (e.g. Meeus et al., 2013; Tracy et al., 2016; Koenig et al., 2016). However, when considering interactions between the group and exposure factors, groups differed with respect to RMSSD at pre-exposure stages. Although post-hoc analyses did not reveal significant group differences due to the Bonferroni multiple testing corrections, RMSSD levels in both SSD groups were descriptively lower in comparison to healthy controls. This might reflect reduced parasympathetic influence on HR in SSD, even in the absence of stressors. However, the results are inconclusive, which might support those of previous studies having not discovered robust HRV group differences (Tak et al., 2009; Mazurak et al., 2012).

With respect to subjective reactivity, state symptom impairment and tension increased and mood decreased after stressor exposure. Even though there was no reactivity with respect to symptom intensity, the study could successfully adopt Schwarz and colleagues´ (2016) method of script driven imagery. The paradigm was modified in order to not only produce health-related, but also social stressors. However, health-related stressors were

assumed to result in stronger subjective reactivity than social stressors in SSD due to health-related symptom characteristics. Absence of interactions between the group and stressor type factors did not allow any conclusion about whether one particular stressor was subjectively more distressing for SSD patients than the other. Although no interactions were found between the group and exposure factors with respect to subjective ratings, an interaction between group, stimulus type and exposure factors was revealed, suggesting increased symptom impairment in SSD-MUS only after exposure to a SC. This increase in disability could not be observed with respect to health-related stressors. This suggests that irregular stressors (like social stressors) appear to be more salient to SSD patients than being regularly exposed to somatic symptoms in daily life. As a consequence, the auditive exposure to social stressors might have caused an unfamiliar and a more intentional shift of attentional focus towards the body when listening to these conflicts. This might explain why the health-related information they listened to was not evaluated as particularly health-threatening. Therefore, SSD patients did not possibly underlie cognitive or interoceptive biases that are usually a consequence of this evaluation (Leonidou & Panayiotou, 2018). Further, this offers the conclusion that dysfunctional processes in SSD leading to increased symptom impairment are not only promoted by health-related information. Any other emotional stressor with certain salience appears to promote them in SSD, too. With respect to state health beliefs and worries, no reactivity could be obtained. The fact that these beliefs and worries were generally higher in SSD in comparison with healthy controls was not particularly surprising as both represent cognitive and affective aspects of HA that are associated with psychological criteria, according to DSM-5 classification of SSD (APA, 2013). More pronounced health beliefs in SSD-MES in comparison with SSD-MUS could be explained by a certain support by their doctors to regard their symptoms as manifestation of a severe illness. They listened to their own voices talking about cognitions, emotions and behaviors they experience on a regular basis. With respect to physiological measures and

especially HR, a main effect of exposure was found, pointing to successful adoption of Schwarz and colleagues´ (2016) method revealed above. Higher stressor-related HR increases were expected for SSD groups in comparison to healthy controls. As HRs in SSD-MUS were generally higher, the absence of interactions/triple interactions between group and exposure factors or triple interactions might be explained by the elevated HR levels in SSD-MUS prior to stress exposure, which limited further increases in cardiovascular activity.

Although groups did not generally differ with regard to HRV levels, the interaction between the group and exposure factors revealed that HRV levels only changed within healthy controls, but not within SSD groups. The absence of changes in the SSD samples might be due to generally higher autonomic arousal (e.g. Rief & Barsky, 2005). In contrast, HRV level changes within HC reflect adaptive dynamics as parasympathetic influence is significantly greater in the absence of stress than during stress exposure. As groups did not differ with respect to heart medication, it was unsurprising that this particular interaction remained significant when excluding the few subjects taking beta-blockers. Groups differed, however, with respect to antidepressant and opiat intake, potentially affecting HRV levels (Kemp et al., 2010).

Finally, the study investigated potential group differences between the two different manifestations of SSD. The results did not reveal any differences between SSD-MUS and SSD-MES regarding subjective impairment and physiological dynamics. This supports criticism to differentiate between ´unexplained´ or ´explained´ physical symptoms (e.g. Mayou 2014), especially as interrater reliability concerning the evaluation of medical explanation seems low (Fink et al., 2005). These results also encourage a shift in attention away from etiological models being restricted to one particular cluster of physical symptoms. Instead etiological models focusing on any kind of bodily distress resulting from constructive CNS-processes might be more adequate (Van den Bergh et al., 2017).

To date, HRV reactivity in SSD is not well understood. As Huang and colleagues (2019) only used a standardized health-related script to provoke reactivity, this study used individualized emotional stressors that also included interpersonal issues which are particularly associated with symptom development.

Another strength of the study was the use of script driven imagery (Schwarz et al., 2016), enabling the provocation of various somatic symptoms, which is especially convenient when using a heterogeneous SSD sample. In contrast to previous research, a structured diagnostic interview (DSM-5) was used, that did not only assess the SSD subtype, but also kind and number of psychological features. Finally, this study allows reliable assertions about differences found between clinical and healthy groups as both groups had similar sociodemographic characteristics like age, sex ratio, etc.

This study is subject to limitations, however, especially with respect to HRV measurement: as the aim was to investigate decreased vagal control in SSD, the use of HF-HRV, which correlates with RMSSD (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (TFESC), 1996), would have produced additional information. As individual depression levels were not measured, observed HRV reactivity differences between SSD and HC cannot potentially be referred to depression-related group differences. Further, the use of ultra short term periods of 30 seconds might offer methodological difficulties (Shaffer & Ginsberg, 2017), although Baek and colleagues (2015) revealed acceptable correlations between a 30 seconds HRV period and five minutes standard short term period (TFESC, 1996). Limited group differences concerning subjective reactivity might be due to the 9-point Likert rating scales used. In contrast to previous studies (Schwarz et al., 2016; Kleinstäuber et al., 2018), this might have caused greater limitation of variance than if visual analogue scales were used.

These study results point to difficulties for SSD patients to flexibly adapt to emotional stressors. Future research directions should also point to HRV biofeedback due to supposed

improvement of autonomic homeostasis (Lehrer & Gevirtz, 2014) and reduction of perceived pain, stress and negative emotions in chronic pain patients (Berry et al., 2014), in SSD.

4.2 Study 2: Health anxiety in somatic symptom disorder: The impact of emotion regulation deficits

Objective: This study discussed HA in SSD in relation to maladaptive ER. It aimed to examine expected deficits in ER for SSD groups compared with HC and differences between SSD subgroups. A further aim was the clarification of which ER strategies predict HA within SSD.

Methods: A total sample of *N* = 108 consisted of 32 patients with SSD-MUS, 40 patients with SSD-MES and 36 HC. The study used a structured diagnostic interview (DSM-5) and questionnaires regarding ER, HA, severity of somatic symptoms, and related disabilities. Analyses were conducted using MANOVAs, correlation, multiple linear regression and mediation analyses.

Results: Patients with SSD reported less adaptive and more maladaptive ER use than HC. The only difference between SSD-MES and SSD-MUS patients manifested in the degree of avoidance, *d* = .62 [95% CI: .14; 1.09]. ER correlated with HA. Expression suppression was the only ER strategy to predict HA within SSD (β = .31, *p* = .039). ER partially mediated HA group differences between SSD and HC.

Conclusion: As ER is related to HA, it would be beneficial for psychotherapeutic approaches to target functional ER skills additionally to promote HA management.

4.2.1 Introduction

SSD requires at least one persistent, burdensome physical symptom often accompanied by HA as an affective symptom (APA, 2013). HA occurs both in patients with SSD-MUS and SSD-MES. Models of SFD like Kirmayer & Taileffer´s (1997) postulate emotional stressors to promote symptom disability through maladaptive interpretation and behavior. The ability to regulate negative affectivity (Watson &

Pennebaker, 1989) therefore deserves attention with respect to somatic burden (Witthöft et al., 2013). ER comprises ´processes by which individuals influence, which emotions they have, when they have them, and how they experience and express these emotions´ (Gross, 1998, p. 275). ER processes have also been related to various psychopathologies (Aldao et al., 2010). When considering specific ER strategies, positive relationships have not only been found between adaptive ER skills like regulation, resilience and acceptance and mental health (Berking et al., 2008), they have also occured between maladaptive ER strategies like rumination, avoidance, suppression and psychopathology (Aldao et al., 2010).

With respect to chronic pain, results point to the relevance of day-to-day affect regulation for the course of pain (Connelly et al., 2007) and to the association between effective ER and quality of life (Agar-Wilson & Jackson, 2012). Several studies have outlined beneficial effects of cognitive restructuring and acceptance-oriented strategies on pain tolerance (Kohl et al., 2014). Other studies have shown a reduction in physical impairment for pain patients following the use of control-oriented strategies (Vowles et al., 2007). Alongside psychological factors like selective attention to bodily sensations, catastrophizing, and other factors (von Baeyer & Champion, 2011), ER appears, in rare cases, to influence the experience of pain directly. In most cases, this relationship seems to be mediated by factors like negative mood (Koechlin et al., 2018).

Regarding MUS, results point in a similar direction, as the use of ´cognitive restructuring´ seems to be associated with reduced symptom disability (Kleinstäuber et al., 2018). To date, only one study investigated ER deficits in SSD-MUS in comparison with HC (Schwarz et al., 2017). While the results revealed a lower degree of adaptive ER for clinical cases than for HC, the study only investigated adaptive ER, and neglected maladaptive ER. Further, clinical and HC groups were not comparable in mean age or educational levels, therefore, potentially affecting group differences in ER use.

Overall, recent research provides accumulating evidence for the relevance of ER deficits in SSD. However, it is not clear whether less adaptive or more maladaptive ER use is more relevant for SSD psychopathology. The current study, therefore, aimed to investigate ER use for patients with SSD in comparison with HC, regarding both adaptive and also maladaptive ER strategies. According to Gross´ (1998) process-model of ER, ER strategies might refer to situational aspects or the emotional experience itself, meaning that acceptance can refer to an adverse event or the emotional experience caused by it. Therefore, two ER measures were used to encompass the various aspects that ER strategies can refer to.

Schwarz and colleagues (2017) only investigated ER processes in SSD-MUS, neglecting SSD-MES. Their results, therefore, are not fully transferable to the whole SSD population. SSD-classification suggests that SSD-MUS and SSD-MES underlie the same dynamics of illness-related mechanisms, however, so far, there is little empirical foundation (Rief & Martin, 2014; Mayou, 2014, Dimsdale et al., 2013). This study not only examined whether ER differs between SSD (with MUS and MES) and a HC group, it also explored potential differences in ER use between SSD-MUS and SSD-MES.

In a second step, the study investigated the relevance of assumed ER deficits for SSD psychopathology. As HA represents a central, affective symptom in SSD, the study aimed to examine the relationship between ER and HA. HA itself is associated with somatosensory amplification and biased interoceptive and exteroceptive awareness (Leonidou & Panayiotou, 2018), with persistence of somatic symptoms (Hadjistavropoulos & Hadjistavropoulos, 2003; McKenzie et al., 2010). HA can also been shown to associate with increased health care use (Fink et al., 2010), dissatisfaction with doctors' care, and in combination with high somatic burden is found to relate to poor therapy outcomes (Lee et al., 2015). So far, existing research points to symptom severity or number of physical symptoms (Tomenson et al., 2013) in the influence of HA levels.

HA resulting from health-threatening information may also be influenced by emotion processing (Leonidou & Panayiotou, 2018). Earlier findings suggest an association between maladaptive ER and HA in samples with subclinical hypochondriasis (Bardeen & Fergus, 2014; Fergus & Valentiner, 2010; Görgen et al., 2014). More specifically, difficulties in describing feelings and symptom-focused rumination were associated with higher levels of HA whereas ‘distraction’ and HA were negatively related (Bailer, Witthöft, Erkic, & Mier, 2017). However, the role of adaptive ER strategies for experiences of HA has received little attention so far. For this reason, the current study examined whether HA levels vary depending on specific ER use, with the expectation that adaptive ER is associated with reduced HA levels and maladaptive ER is related to increased HA levels. In addition, the study investigated which particular strategies are associated with HA.

The study also examined whether ER is an additional feature explaining different HA levels in SSD and HC. More specifically, the study examined whether these differences disappear when controlling for ER.

4.2.2 Methods

This section reveals participants characteristics first, followed by presentations of the recruitment process and study procedure. Afterwards used self-report measures are revealed, followed by a description of statistical analyses.

Participants

A total of 108 subjects was recruited for the cross-sectional study, consisting of two clinical groups with SSD (*N* = 72; SSD-MUS *N* = 32, SSD-MES *N* = 40) and a HC group (*N* = 36). Participants were aged between 18 and 70 years. They were included on the basis that they were without the following: another dominant psychiatric condition; drug and/or substance abuse; an acquired brain damage; a current pregnancy; psychosis or history of bipolar disorder; a current state of suicidality; and/or medication with antipsychotics or benzodiazepines. For both SSD subgroups, at least one burdensome somatic symptom (MUS or MES) was required lasting for at least 6 months and in fulfillment of DSM-5 SSD criterion B. For the HC group, participants did not suffer from disabling body symptoms or any mental disorder. A detailed trial profile is presented in A-1.12.

Recruitment and procedure

The study protocol and the consent forms (see A-1.1 and A-1.2) were approved by the Ethics Committee of the University of Wuppertal. All participants provided informed consent. Patient recruitment was achieved in cooperation with general practitioners, specialists in the Helios Universitätsklinikum Wuppertal and via notices on the university´s bill-boards, information flyers, and announcements on the website of the department.

Participants were invited to take part in the Department of Clinical Psychology and Psychotherapy at the University of Wuppertal. After completing a sociodemographic questionnaire (see A-1.5), the diagnostic interview Mini-DIPS (Margraf, 1994) was conducted in order to diagnose comorbid mental disorders. Following this, the SSD-interview (Rief et al., 2010; Kleinstäuber et al., 2016) was conducted to diagnose SSD

according to DSM-5. Participants were then asked to fill out self-report measures in a computerized format.

Measures

Besides the SSD-interview (Rief et al., 2010; Kleinstäuber et al., 2016), Mini-DIPS (1994), PHQ-15 (Kroenke et al., 2002) and PDI (Mewes et al., 2009), further self-report measures were used described below.

modified Short Health Anxiety Inventory (mSHAI)

The mSHAI (Bailer et al., 2013) consists of 14 items and is a shortened version of the SHAI (Salkovskis et al., 2002). The mSHAI assesses HA, experienced during the previous 12 weeks on a 5-point scale (0 = *strong rejection* to 4 = *strong approval*). The internal consistency of the scale ($\alpha \geq .95$) is excellent (Bailer et al., 2013).

Emotion Regulation Skills Questionnaire (ERSQ)

The prolonged state version of the ERSQ (Berking & Znoj, 2008) is a self-report measure that assesses ER skills on a 5-point scale (0 = *not at all* to 4 = *almost always*) during the past seven days. It consists of 27 items equally distributed on nine subscales (awareness, sensations, clarity, understanding, acceptance, resilience, self-support, readiness to confront, and modification). These subscales refer to the ER skills represented in the model of adaptive ER (Berking, 2007). The prolonged state version was validated (Berking & Znoj, 2008), and the results indicated good internal consistencies for the nine subscales (α = .72 to .81). The ERSQ scales were significantly associated with various indicators of mental health and well-being (Berking & Znoj, 2008). Acceptance, resilience and regulation were the ER skills of interest, as according

to Berking´s (2007) model of adaptive ER, they are crucial to mental health. Corresponding items to those skills, respectively, were as follows: *´I was able to accept my negative feelings.´; ´I felt strong enough to tolerate even negative emotions.´; ´I was able to influence my negative feelings.´*

Heidelberg Form for Emotion Regulation Strategies (HFERST)

The HFERST (Izadpanah, Barnow, Neubauer, & Holl, 2017) is a self-report measure that assesses ER skills on a 5-point scale (1 = never to *5* = always) during the past four weeks. It consists of 28 items measuring four, functional ER strategies (reappraisal, acceptance, problem solving, social support) and four dysfunctional (rumination, avoidance, expressive suppression, experience suppression). The subscales, especially the acceptance-subscale, includes situational aspects and refers to the emotional experience itself (Izadpanah et al., 2017). Corresponding items to those skills were as follows: *´When I feel bad, I try to see positive aspects of a situation.´* (reappraisal); *´When I cannot change something, I accept the situation as it is.´* (acceptance); *´When I am confronted with problems, I think very carefully about how I can deal best with the situation.´* (problem solving); *´I often talk about my emotions with my partner or my close friends.´* (social support); *´When I have negative feelings, I often brood about why I am feeling this way.´* (rumination); *´I prefer to avoid situations that could cause negative emotions in me.´* (avoidance); *´I hide physical expressions of my feelings.´* (expressive suppression); *´When I have strong emotions, I immediately push them aside.´* (experience suppression). The HFERST showed good internal consistencies (α = .78 to .86).

Analyses

The 22 nd version of the IBM SPSS and PROCESS macro (Hayes, 2013) were used for data analyses. Pearson chi-square tests and independent *t* – tests were used to compare groups with respect to demographic and clinical variables. Two MANOVAs were conducted, one with respect to three ERSQ-related ER strategies and the other with regard to the eight HFERST-related ER strategies, using group (SSD-MUS, SSD-MES, HC) as between-subjects factor. In the event of significant multivariate group main effects, two contrasts were planned for each ER strategy. The first contrast tested potential ER differences between SSD and HC. As ER use differences were expected between SSD and HC, both SSD groups were weighted with 0.5 each, and HC weighted with -1. The second contrast tested expected differences in ER use between SSD-MUS (weighted with 1) and SSD-MES (weighted with -1). Pearson´s bivariate corrrelation was used to assess the relationship between ER (ERSQ, HFERST) and HA (mSHAI) in the SSD sample. One-tailed comparisons of correlation coefficients were conducted with a Bonferroni multiple testing correction set at $p = .01$. Multiple linear regression analyses were used to examine explained variance of adaptive and maladaptive ER strategies in predicting HA. Mediation analyses were performed in the total sample ($N = 108$) to assess whether group predicted HA (mSHAI), and whether this direct path would be mediated by ER (ERSQ, HFERST). Total, direct and indirect effects of *X* and *Y* through the potential mediator variable *M* were calculated using PROCESS with 95% bias-corrected (BC) bootstrap confidence intervals based on 5000 bootstrap samples for total, direct and indirect effects of *X* and *Y* through the potential mediator variable *M*. Group as a predictor was defined as a dummy variable with HC coded 0 and SSD groups coded 1.

4.2.3 Results

The result section reveals sociodemographic and clinical characteristics first, followed by presentations of ER-related group differences and of relationships between ER and HA within SSD. Finally, mediator analyses are depicted.

Participant characteristics

Groups did not differ in age, p = .108, ratio of women, p = .303, and family status, p = .340, but differed with respect to education, p = .015. HC had higher levels of school education in comparison with the clinical group, but not necessarily regarding university degrees. The two SSD groups showed higher levels of symptom severity (PHQ-15), disability (PDI), and HA (mSHAI) than HC. The SSD groups did not differ in symptom severity, p = 1.000, disability, p = 1.000, HA, p = 1.000, nor with respect to the presence of comorbid disorders, p = .986. Sociodemographic and clinical characteristics of the three groups are presented in Table 4.

Table 4 Sociodemographic data and clinical characteristics of both SSD groups and HC

Variable	SSD-MUS	SSD-MES	HC	Statistics
N	32	40	36	
Female % (*N*)	62.5 (20)	77.5 (31)	65.7 (23)	$\chi^2(2)= 2.39$, $p = .303$
Age (*M* (*SD*))	44.34 (15.68)	40.26 (16.06)	35.61 (18.74)	$F(2,106)= 2.28$, $p = .108$
Family status				$\chi^2(4)= 4.53$, $p = .340$
Single	34.4 (11)	40.0 (16)	33.3 (12)	
In a relationship % (*N*)	21.9 (7)	20.0 (8)	38.9 (14)	
Married % (*N*)	43.8 (14)	40.0 (16)	27.8 (10)	
Education				$\chi^2(6)= 15.85$, $p = .015$
Main school % (*N*)	15.6 (5)	5.0 (2)	8.6 (3)	
Secondary school % (*N*)	21.9 (7)	30.0 (12)	5.7 (2)	
A level certificate % (*N*)	25.0 (8)	40.0 (16)	63.9 (23)	
University degree % (*N*)	37.5 (12)	25.0 (10)	22.9 (8)	
Comorbid disorders				
No comorbid disorder % (*N*)	65.6 (21)	62.8 (27)	-	$\chi^2(3)= 0.15$, $p = .986$
1 comorbid disorder % (*N*)	25.0 (8)	25.6 (11)	-	
2 comorbid disorders % (*N*)	3.1 (1)	4.7 (2)	-	
More than 2 comorbid disorders % (*N*)	6.3 (2)	7.0 (3)	-	
Symptom duration (*M* (*SD*))	10.11 (10.94)	13.80 (11.49)		$T(70)= -1.38$, $p = 171$
MUS (*N)* [CIDI-SOM]	8.19 (6.97)	2.05 (2.75)		$T(38.75)= 4.70$, $p = <.001$
MES (*N)* [CIDI-SOM]	2.44 (2.93)	7.18 (5.01)	-	$T(64.59)= -5.01$, $p = <.001$
Symptom severity (*M* (*SD*)) [PHQ-15]	10.22 (4.61)	9.70 (4.40)	2.97 (2.89)	$F(2,105)= 37.95$, $p <.001$
Symptom-related disability (*M* (*SD*)) [PDI]	3.50 (1.91)	3.27 (1.96)	0.63 (1.05)	$F(2,105)= 30.98$, $p < .001$
Health anxiety (*M* (*SD*)) [mSHAI]	21.47 (11.12)	20.43 (12.65)	6.31 (6.31)	$F(2,105)= 23.54$, $p < .001$
Number of fulfilled psychological SSD-features (*M* (*SD*)) [SSD-Interview]	3.44 (1.70)	3.23 (1.67)	-	$T(70)= .53$, $p = 597$

Group differences in ER strategies

Regarding adaptive ER (ERSQ), the MANOVA revealed a significant main effect of group, λ = .72, $F(6,206) = 6.23$, $p < .001$, $\eta^2 = .15$. Planned contrasts revealed lower ER scores in SSD in comparison to HC with respect to acceptance, -.69 (*SE* = .14), $p < .001$, resilience, -.77 (*SE* = .14), $p < .001$ and regulation, -.67 (*SE* = .19), $p < .001$. Conversely, planned contrasts did not reveal any ER-score differences between SSD-MUS and SSD-MES.

For the HFERST, the MANOVA (Λ) revealed a significant main effect of group, λ = .69, $F(16,196) = 2.49$, $p = .002$, $\eta^2 = .17$. Planned contrasts revealed lower ER scores for SSD in comparison to HC regarding reappraisal, -.76 (*SE* = .17), $p < .001$, and acceptance, -.61 (*SE* = .18), $p < .001$ and higher scores were shown for rumination, .52 (*SE* = .19), $p = .007$. To compare SSD-MUS and SSD-MES, planned contrasts revealed no difference with regard to problem solving, social support, expression suppression and experience suppression. The two SSD groups differed only in avoidance with higher scores revealed for SSD-MES than for SSD-MUS, -.54 (*SE* = .21), $p = .010$. Detailed information can be found in Tables 5 and 6.

Table 5 Contrasts of SSD (N = 72) and HC (N = 36) for adaptive and maladaptive ER

	SSD		HC				95 % CI		
Variable	*M*	*SD*	*M*	*SD*	*t*	*p*	*LL*	*UL*	Cohen´s *d*
ERSQ									
Acceptance	2.27	.88	2.98	.57	-5.01	< .001	.48	1.32	.90
Resilience	2.10	.84	2.89	.58	-5.71	< .001	.61	1.46	1.03
Regulation	1.79	.96	2.44	.78	-3.61	< .001	.31	1.13	.72
	SSD		HC				95 % CI		
Variable	*M*	*SD*	*M*	*SD*	*t*(105)	*p*	*LL*	*UL*	Cohen´s *d*
HFERST									
Reappraisal	2.83	.89	3.58	.77	-4.35	< .001	.46	1.30	.88
Acceptance	3.11	.93	3.71	.74	-3.37	.001	.28	1.10	.69
Probl. solving	4.08	.72	4.12	.53	-.26	.792	-.34	.46	.06
Social support	3.42	1.12	3.58	.99	-.67	.508	-.25	.55	.15
Rumination	3.66	.97	3.13	.84	2.74	.007	-.98	-.16	-.57
Avoidance	3.26	.92	2.94	.86	1.59	.115	-.76	.05	-.36
Expr. supp.	3.08	.99	3.03	.80	.18	.859	-.45	.34	-.05
Exp. supp.	2.39	.87	2.29	.68	.54	.589	-.52	.28	-.12

Note. Expr. supp. = Expression suppression; Exp. supp. = Experience suppression

Table 6 Contrasts of SSD-MUS (N = 32) and SSD-MES (N = 40) for adaptive and maladaptive ER

	SSD-MUS		SSD-MES				95 % CI		
Variable	*M*	*SD*	*M*	*SD*	*t*	*p*	*LL*	*UL*	Cohen´s *d*
ERSQ									
Acceptance	2.44	.68	2.14	1.01	1.48	.143	-.81	.12	-.35
Resilience	2.27	.67	1.96	.94	1.65	.105	-.85	.09	-.38
Regulation	1.66	.97	1.89	.96	-1.09	.276	-.23	.70	.24
	SSD-MUS		SSD-MES				95 % CI		
Variable	*M*	*SD*	*M*	*SD*	*t*	*p*	*LL*	*UL*	Cohen´s *d*
HFERST									
Reappraisal	2.72	.68	2.92	1.03	-.99	.324	-.23	.69	.23
Acceptance	3.11	.94	3.10	.94	.07	.944	-.47	.45	-.01
Probl. solving	4.14	.64	4.03	.77	.65	.515	-.63	.29	-.17
Social support	3.63	.90	3.25	1.26	1.47	.143	-.78	.15	-.31
Rumination	3.55	.95	3.74	1.00	-.85	.396	-.27	.66	.20
Avoidance	2.96	.83	3.50	.92	-2.61	.010	.14	1.09	.62
Expr. Supp.	2.83	.80	3.28	1.09	-2.00	.050	.00	.94	.47
Exp. Supp.	2.32	.86	2.44	.89	-.64	.525	-.33	.6	.14

Note. Expr. supp. = Expression suppression; Exp. supp. = Experience suppression

Associations between ER and HA wihin patients with SSD

Within the SSD sample, bivariate correlations between adaptive ER (ERSQ) and HA revealed that higher acceptance (r = -.31, p = .009), resilience (r = -.29, p = .013) and regulation scores (r = -.29, p = .014) were significantly related to lower HA levels. Regarding resilience and regulation, the association was only significant at $p < .05$. When all three ER strategies were entered simultaneously into the linear regression model as potential predictors of HA, the total model was significant, and the explained variance (R^2) accounted for 11 % of the model. Neither acceptance, resilience nor regulation were significant single predictors of HA in this model.

Bivariate correlations between adaptive ER (HFERST) and HA revealed that both higher reappraisal (r = -.28, p = .009), and acceptance scores (r = -.28, p = .008) were associated with lower HA levels. The relationship between problem solving and HA (r = -.22, p = .035) was only significant at $p < .05$. In contrast, no correlation was found between social support and HA (r = -.14, p = .124). When all four ER strategies were entered simultaneously into the regression model as potential predictors of HA, the total model was significant, with the explained variance (R^2) accounting for 15 % of the model. Neither reappraisal, acceptance, problem solving, nor social support were significant predictors of HA in this multivariate model. In sum, adaptive ER predicted the degree of HA, but HA was not predicted by any particular strategy.

Bivariate correlations between maladaptive ER (HFERST) and HA indicated that rumination (r = .33, p = .005) and expression suppression (r = .35, p = .001) were associated with higher HA levels. Avoidance (r = .19, p = .056) and experience suppression (r = .04, p = .380) showed no significant relationships with HA. When these ER strategies were entered simultaneously in the second linear regression model, the total model was significant, and the explained variance accounted for 18 % of the model. Expression suppression was a

significant predictor of HA whereas rumination, avoidance and experience suppression were not. Correlation and multiple regression analyses are presented in Table 7.

Table 7 Linear regression analyses: ER as predictor of HA within SSD (N = 72)

ERSQ Predictor	*M*	*SD*	*r*	β	*t*	*p*	R^2	*F*	*p*
Acceptance	2.27	.88	-.31*	-.14	-.59	.558	.11	2.76	.049
Resilience	2.10	.84	-.29	-.08	-.36	.718			
Regulation	1.79	.96	-.29	-.15	-.96	.342			
HFERST Predictor	***M***	***SD***	***r***	β	***t***	***p***	R^2	***F***	***p***
Reappraisal	2.83	.89	-.28*	-.17	-1.34	.184	.15	2.91	.028
Acceptance	3.11	.93	-.28*	-.22	-1.87	.066			
Probl. solving	4.08	.72	-.22	-.16	-1.33	.187			
Social support	3.42	1.12	-.14	-.04	-.38	.705			
Predictor	***M***	***SD***	***r***	β	***t***	***p***	R^2	***F***	***p***
Rumination	3.66	.97	.33*	.19	1.54	.128	.18	3.72	.009
Avoidance	3.26	.92	.19	.07	.53	.600			
Expr. supp.	3.08	.99	.35*	.31	2.11	.039			
Exp. supp.	2.39	.87	.04	-.13	-.91	.367			

Note. Probl. Solving = Problem solving, Expr. supp. = Expression suppression, Exp. supp. = Experience suppression, * $p < .01$

ER as mediator of the relationship between group and HA

A strong effect of group on HA was observed, b = 14.58, p < .001, suggesting that SSD was associated with higher HA levels than those seen in HC. Furthermore, when single adaptive ER strategies (ERSQ) were entered into the model, acceptance did not mediate the relationship between group and HA, b = .09 (95% CI: .00, .20).

Resilience, however, partially mediated the aforementioned relationship, b = .10 (95% CI: .02, .20), as the direct path remained significant, b = 11.92, p < .001. Regulation did not mediate the relationship between group and HA, b = .06 (95% CI: .00, .14).

Likewise, when single adaptive and maladaptive ER strategies (HFERST) were entered into the model, reappraisal partially mediated the relationship between group and HA, b = .07 (95% CI: .01, .16), as the direct path remained significant, b = 12.74, p < .001.

In contrast, the other adaptive ER strategies, acceptance, problem solving, and social support, did not mediate the aforementioned relationship. For the maladaptive ER strategies, only rumination partially mediated the association between group and HA, b = .06 (95% CI: .02, .14), as the direct path remained significant, b = 12.93, p < .001. The relationship between group and HA was not mediated by the remaining maladaptive strategies, avoidance, expression suppression and experience suppression. Detailed information can be found in Table 8.

Table 8 Effects of group on HA through ER (N = 108)

Variable	a path	b path	Total Effect (c path)	Direct Effect (c´ path)	Sobel Test	Indirect effect
			ERSQ			
Acceptance	*b* = -.71, *p* < .001	*b* = -3.21, *p* = .011	*b* = 14.58, *p* < .001	*b* = 12.31, *p* < .001	*Z* = 2.27, *p* = .029	*b* = .09, BC 95 % CI [.00, .20]
Resilience	*b* = -.79, *p* < .001	*b* = -3.36, *p* = .010	*b* = 14.58, *p* < .001	*b* = 11.92 *p* < .001	*Z* = 2.66, *p* = .022	*b* = .10, BC 95 % CI [.02, .20]
Regulation	*b* = -.66, *p* = .001	*b* = -2.51, *p* = .023	*b* = 14.58, *p* < .001	*b* = 12.93, *p* < .001	*Z* = 1.88, *p* = .060	*b* = .06, BC 95 % CI [.00, .14]
			HFERST			
Reappraisal	*b* = -.75, *p* < .001	*b* = -2.47, *p* = .037	*b* = 14.58, *p* < .001	*b* = 12.74, *p* < .001	*Z* = 1.86, *p* = .063	*b* = .07, BC 95 % CI [.01, .16]
Acceptance	*b* = -.61, *p* = .001	*b* = -2.23, *p* = .053	*b* = 14.58, *p* < .001	*b* = 13.23, *p* < .001	*Z* = 1.65, *p* = .100	*b* = .05, BC 95 % CI [-.00, .12]
Problem solving	*b* = -.04, *p* = .778	*b* = -2.03, *p* = .184	*b* = 14.58, *p* < .001	*b* = 14.51, *p* < .001	*Z* = .22, *p* = .823	*b* = .00, BC 95 % CI [-.01, .05]
Social support	*b* = -.17, *p* = .451	*b* = -1.10, *p* = .240	*b* = 14.58, *p* < .001	*b* = 14.40, *p* < .001	*Z* = .52, *p* = .604	*b* = .01, BC 95 % CI [-.01, .05]
Rumination	*b* = .54, *p* = .006	*b* = 3.09, *p* = .004	*b* = 14.58, *p* < .001	*b* = 12.93, *p* < .001	*Z* = 1.98 *p* = .048	*b* = .06, BC 95 % CI [.02, .14]
Avoidance	*b* = .32, *p* = .089	*b* = 1.68, *p* = .134	*b* = 14.58, *p* < .001	*b* = 14.05, *p* < .001	*Z* = 1.04, *p* = .299	*b* = .02, BC 95 % CI [-.01, .08]
Expression suppression	*b* = .06, *p* = .771	*b* = 3.61, *p* = .001	*b* = 14.58, *p* < .001	*b* = 14.38, *p* < .001	*Z* = .28, *p* = .7879	*b* = .01, BC 95 % CI [-.04, .06]
Experience suppression	*b* = .10, *p* = .559	*b* = .87, *p* = .489	*b* = 14.58, *p* < .001	*b* = 14.50, *p* < .001	*Z* = .30, *p* = .764	*b* = .00, BC 95 % CI [-.01, .05]

4.2.4 Discussion

This study aimed to examine ER in SSD in comparison with HC, and explore whether ER use differs between SSD-MUS and SSD-MES. With respect to adaptive ER strategies (ERSQ), less use of acceptance, resilience and regulation was found in SSD groups than in HC. According to Berking´s model of adaptive ER (2007), these abilities are crucial for mental health. Consequently, less use of these particular strategies might contribute to impaired mental health in SSD. These results are in line with the findings of Schwarz and colleagues (2017) who reported less adaptive ER use (ERSQ) in SSD than in HC, but more adaptive ER use than in patients with clinical depression or SSD with comorbid depressions. However, the results from this study go beyond those of Schwarz and colleagues. In the present study, the sample was not restricted to patients with SSD-MUS, but also considered SSD-MES. Furthermore, the HC groups did not differ from the clinical group with respect to age and sex ratio, an important factor, as these variables have been shown to influence ER use (Zimmermann & Iwanski, 2014). Groups did, however, differ in education, potentially affecting ER use. Unlike Schwarz and colleagues (2017), two different measures for the assessment of ER were used, ensuring that strategies like acceptance do not only refer to the emotional experience itself but also, as proposed by Gross (1998), to situational aspects. In that respect, contrasts revealed less acceptance-based strategy use in SSD groups compared with HC, referring both to the ERSQ and HFERST. This means less acceptance use for SSD groups regarding the situation and experiencing aversive emotions, as well as potentially these emotions. The study found less adaptive ER use in SSD regarding reappraisal, but not with regard to problem solving and social support. The investigations of group differences in ER processing were not limited to adaptive ER strategy use as they also included maladaptive ER strategies. Contrasts for maladaptive ER strategy use revealed rumination as the only difference between SSD groups and HC. More rumination in SSD is in line with existing explanatory models of SFD (e.g. Kirmayer & Taileffer, 1997; Witthöft &

Hiller, 2010), representing a dysfunctional interpretation of bodily symptoms, which consequently promote somatic disability.

The clinical group in this study comprised of patients with SSD-MUS and SSD-MES. This was due to classificatory changes in DSM-5 (APA, 2013) that no longer require the exclusion of somatic causes, and therefore unify both subgroups in one SSD population. So far, there is no empirical evidence that SSD-MUS and SSD-MES are comparable with respect to ER as an illness-related mechanism, therefore this study examined ER use in SSD-MUS and SSD-MES. Apart from avoidance, no subgroup differences were found, supporting the assumption that both groups are not substantially different. In addition, the two SSD groups revealed highly increased levels of HA, without subgroup differences. These findings, showing little difference in ER processing, therefore provide support for the DSM-5-classification, and as a consequence the assumption of equally relevant illness-related mechanisms within SSD.

In a further step, this study investigated relationships between ER and SSD-psychopathology, focusing on HA as an affective SSD symptom. HA results from health threatening information and requires certain strategies to deal with elicited emotions (Leonidou & Panayiotou, 2018). Therefore, information is needed regarding which specific ER strategies relate to adaptive or maladaptive HA management within SSD. Apart from problem solving and social support, the study revealed significant negative correlations between adaptive ER use and HA, and therefore supports previous research that revealed an association between cognitive restructuring and lower HA (Bardeen & Fergus, 2014). Linear regression models showed that adaptive ER (referring to both ERSQ and HFERST) explained considerably variance in HA, even though there was no specific ER strategy predicting it.

Further, these results support previous research regarding relationships between emotion dysregulation and HA (Bardeen & Fergus, 2014; Fergus & Valentiner, 2010; Görgen

et al., 2014, Bailer et al., 2017). Greater rumination and expression suppression use was associated with increased HA. Avoidance, however, was not associated with HA. Avoidance not only refers to the emotional experience, but also the situation causing the distress. For example, patients with SSD avoid the emotional experience, but not necessarily related situations like visiting doctors or hospitals as health care offers them reassurance. In contrast to expectations, rumination was not a significant predictor of HA (Marcus et al., 2008). This is a surprising finding as rumination is associated with various psychopathologies (Aldao et al., 2010). This might be due to suppressor effects within the linear regression model. Positive associations between experience suppression and HA replicate Bardeen & Fergus´ (2014) results which measured expressive suppression using the Emotion Regulation Questionnaire (Gross & John, 2003). Taken together, both adaptive and maladaptive ER explain variance in HA, suggesting the relevance of ER in explaining the degree of HA in SSD.

Finally, the study examined whether emotion regulation is particularly responsible for higher degrees of HA in SSD than HC. The results revealed that group differences remained significant when controlling for ER. However, various indirect effects of group on HA through ER were found, indicating that ER partially explained different HA levels in SSD patients and HC. This implies that while ER has considerable impact on these differences, it is not the only factor explaining it. Other factors like sex, age, number of symptoms and symptom severity could be additional relevant predictors (Tomenson et al., 2013).

As far as one knows, this study is one of the first to systematically investigate ER processing in SSD. The study used a structured diagnostic interview (DSM-5) that not only assessed the SSD subtype, but also the kind and number of psychological features. In contrast to previous research (e.g. Schwarz et al., 2017), these results also considered adaptive and maladaptive ER use to evaluate whether either promoting adaptive ER use or limiting maladaptive ER use is associated with lower HA levels.

The study offers some limitations. Firstly, mood-associated recall bias may have caused observed relationships between ER and HA due to the use of self-report measures.

Secondly, as the design of the study was cross-sectional, causal effects of ER on HA cannot be drawn. In order to evaluate those assumed associations, future studies should rely on paradigms, in which ER use is experimentally manipulated. Another limitation refers to the fact that no questionnaire concerning depression was used. As ER deficits in SSD might be explained by elevated depression levels (Schwarz et al., 2017), it was not possible to control in this case whether revealed ER use differences between SSD patients and HC are explained by depression levels. Furthermore, contextual conditions like emotion type and intensity should be considered. This would enable investigations under specific conditions, in which certain ER use is associated with reduced HA levels.

Cognitive Behavioral Therapy (CBT) approaches for SFD and hypochondriasis/pathological HA are regarded as efficacious (Witthöft & Hiller, 2010, Olatunji et al., 2014). However, the effect sizes are moderate with respect to multiple MUS (Kleinstäuber, Witthöft, & Hiller, 2011). Recently, a promising approach extended conventional CBT by ER training to improve treatment outcomes in patients with multiple MUS (Kleinstäuber et al., 2016, Kleinstäuber et al., 2019). As the present results imply that HA levels vary depending on specific ER use, SSD patients might also benefit from the aforementioned ER training. With the help of this training, patients learn to reduce maladaptive ER use in particular, and learn to flexibly use adaptive ER depending on specific situational demands.

4.3 Additional analyses

The design of the quasi-experimental study (see figure 2) included a further self-report measure that was not revealed in section 4.1. This measure examines state ER use during stress-exposures. So far, no experimental studies investigated state ER use in SSD. As previous findings outline ER deficits with respect to habitual ER (e.g. Schwarz et al., 2017) patients with SSD were expected to exhibit less adaptive and more maladaptive state ER use. Referring to assumed ER deficits, SSD patients were expected to evaluate presumably adaptive state ER strategies like reappraisal, acceptance and problem solving as less effective to reduce negative emotions as HCs might do. Further, they were supposed to evaluate presumably maladaptive ER strategies like rumination, avoidance, experience and expression suppression not as ineffective in reducing negative emotions as HCs might do. Researchers like Aldao (2013) emphasize that ER processes are influenced by emotion-eliciting stimuli. Transferring this to the experimental paradigm, ER use during exposure with health-related and social stressors might potentially be different, although both stressors were supposed to respectively induce negative affect particularly in SSD patients.

Balzarotti and colleagues (2017) assumed cardiac vagal control (and therefore HRV) to be a marker of ER. As previous results indicated relations between ER use and HRV levels (Aldao, Dixon-Gordon, & De Los Reyes, 2016; Volokhov & Demaree, 2010), the thesis intended to examine whether assumed maladaptive ER use in SSD was related with decreased HRV levels and adaptive ER use with increased HRV levels. As SSD patients presumably reveal ER deficits, these hypothesized results might not necessarily manifest in SSD as these patients might not appraise ER strategies like reappraisal to be particularly helpful in reducing negative emotions. Following this, ER strategies that SSD patients evaluated as effective in reducing negative emotions might not automatically be associated with increased HRV levels.

4.3.1 Methods

This section reveals participant characteristics first, followed by a description of used measures, and a presentation of statistical analyses.

Participants

Participant characteristics refer to those of section 4.1.3. Results of one participant with SSD-MUS could not be included in these additional analyses. This was due to technical problems not allowing the participant to fill out the state ER questionnaire after stressor exposure. Data of 28 participants with SSD-MUS, of 33 with SSD-MES and of 32 HC were included in the following analyses.

HFERST

The unpublished short version for the retrieval of regulation strategies based on the *Heidelberg Form for Emotion Regulation Strategies* (Izadpanah et al., 2017) is a self-report measure that assesses state ER on a 5-point scale (1 = does not apply at all; *5* = applies) related to a previous experience. Further, it assesses in how far the use of strategies subjectively reduced negative emotions (1 = not at all; *5* = applies). It originally consists of eight items measuring three functional (reappraisal, acceptance, problem solving) and four dysfunctional ER strategies (rumination, avoidance, expressive suppression, experience suppression) and distraction. As distraction was not considered in the published version of the HFERST, this strategy was excluded from the following analyses. Corresponding items to those strategies were as follows: “When I had negative feelings during the experience, I…”I brooded about it” (rumination); “I tried to change the feelings about the current situation.” (reappraisal); “I tried to figure out how to deal best with the situation.” (problem solving); “I tried to stay with my feelings.” (acceptance); “I tried to push the negative feelings aside.” (expressive suppression); “I tried to avoid thinking about it.” (avoidance); “I tried to hide my feelings.” (experience suppression).

Statistical analyses

With respect to state ER use and subjective reduction of negative emotion after state ER use, two 3 x 2 mixed MANOVAs with repeated measures were conducted. Group (SSD-MUS, SSD-MES, HC) served as between-subjects factor and stressor type (PC vs. SC) as within-subjects factor. In case of significant multivariate main effects of group or stressor type, or multivariate group x stressor type interaction effects, further univariate analyses were conducted. With respect to main effects of group, Games-Howell or Bonferroni-post-hoc analyses were conducted depending on the results from Levene´s-tests of homogenity of variances (see A-1.16). Further, control analyses in the form of 3 x 2 x 2 mixed ANOVAs with repeated measures were conducted (with chronological order of stressor-presentation as further between-subjects factor) to rule out that the chronological order of stressor-presentation has undesired effects on the results.

In order to investigate relationships between RMSSD/SDNN levels during stress exposure and state ER strategy use and subjective reduction of negative emotion after state ER use within SSD (N = 61), mean RMSSD/SDNN levels, mean state ER strategy use, and mean subjective reduction of negative emotion after state ER use out of both stress exposures were calculated. In a further step, two-tailed comparisons of correlation coefficients were conducted with Bonferroni-corrected p = .007.

4.3.2 Results

The results section begins with presentations of two MANOVAs regarding state ER use and subjective reduction of negative emotions after state ER use. Afterwards, two correlation analyses are presented, starting with associations between state ER use and HRV levels, followed by relationships between subjective reduction of negative emotions after state ER use and HRV levels. As the chronological order of stressor-presentation has

no substantial effect on the results, the aforementioned control analyses are not presented here, but in section A-1.17 and A-1.18 instead.

State ER use

Concerning state ER use, the MANOVA (*Λ*) revealed a significant main effect of group, λ = .74, $F(14,168) = 1.94$, p = .025, η^2 = .14. However, there was no main effect of stressor type, λ = .92, $F(7,84) = 1.09$, p = .379, and no significant group x stressor interaction, λ = .85, $F(14,168) = .85$, p = .458. Univariate analyses revealed significant main effects of group only concerning rumination, $F(2,90) = 5.03$, p = .008, η^2 = .10. Pairwise comparisons showed that only SSD-MUS showed more rumination use in comparison with HC, p = .006, without any differences between both clinical groups, p = .188. Detailed information can be found in Table 9.

Subjective reduction of negative emotion after state ER use

Concerning subjective reduction of negative emotion after state ER use, the MANOVA (*Λ*) did not reveal a significant main effect of group, λ = .82, $F(14,168) = 1.26$, p = .237. There was also no main effect of stressor type, λ = .89, $F(7,84) = 1.42$, p = .208, and no significant group x stressor interaction, λ = .84, $F(14,168) = 1.08$, p = .384. Due to non-significant multivariate main/interaction effects, further reporting of univariate main/interaction effects is not necessary. Detailed information can be found in Table 10.

Table 9 Group differences in state ER use

ER strategy	Stimulus type	SSD-MUS *M (SD)*	SSD-MES *M (SD)*	HC *M (SD)*	Main effects	Interaction effects
Reappraisal	PC	2.46 (.96)	2.33 (.99)	2.25 (1.19)	$F_{Gr}(2,92) = 0.23$	$F_{Gr \times ST}(2,92) = 1.57$
	SC	2.46 (1.23)	2.33 (1.05)	2.72 (1.37)	$F_{ST}(1,93) = 1.54$	
Acceptance	PC	3.11 (1.13)	2.94 (1.32)	2.88 (1.45)	$F_{Gr}(2,92) = 0.19$	$F_{Gr \times ST}(2,92) = 0.11$
	SC	3.04 (1.10)	2.91 (1.16)	2.94 (1.32)	$F_{ST}(1,93) = 0.01$	
Problem solving	PC	3.04 (1.04)	2.85 (1.28)	2.72 (1.40)	$F_{Gr}(2,92) = 0.36$	$F_{Gr \times ST}(2,92) = 0.14$
	SC	3.00 (1.19)	2.88 (1.11)	2.84 (1.37)	$F_{ST}(1,93) = 0.10$	
Rumination	PC	3.25 (1.17)	2.64 (1.22)	2.31 (1.20)	$F_{Gr}(2,92) = 5.03^{**}$	$F_{Gr \times ST}(2,92) = 0.28$
	SC	3.43 (1.17)	3.03 (1.13)	2.66 (1.26)	$F_{ST}(1,93) = 6.41^{*}$	
Avoidance	PC	2.50 (1.04)	2.67 (1.31)	2.25 (1.22)	$F_{Gr}(2,92) = 1.71$	$F_{Gr \times ST}(2,92) = 1.74$
	SC	2.79 (1.34)	2.39 (1.14)	2.09 (1.15)	F_{ST} 1,93) = 0.14	
Exp. Supp.	PC	2.36 (1.06)	2.36 (1.29)	2.19 (1.31)	$F_{Gr}(2,92) = 0.53$	$F_{Gr \times ST}(2,92) = 0.76$
	SC	2.14 (1.15)	2.52 (1.09)	2.19 (1.15)	F_{ST} 1,93) = 0.03	
Expr. Supp.	PC	2.68 (1.22)	3.03 (1.26)	2.34 (1.26)	$F_{Gr}(2,92) = 2.04$	$F_{Gr \times ST}(2,92) = 0.95$
	SC	2.50 (1.32)	2.97 (1.31)	2.53 (1.27)	$F_{ST}(1,93) = 0.02$	

Note. Exp. Supp. = Experience suppression; Expr. Supp. = Expression suppression; ST = Stimulus type; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; *** $p < .001$; ** $p < .01$; * $p < .05$

Table 10 Group differences in subjective reduction of negative emotions after state ER use

ER strategy	Stimulus type	SSD-MUS *M (SD)*	SSD-MES *M (SD)*	HC *M (SD)*	Main effects	Interaction effects
Reappraisal	PC	2.04 (1.04)	1.82 (1.04)	2.38 (1.29)	$F_{Gr}(2,92) = 1.59$	$F_{Gr \times ST}(2,92) = 0.43$
	SC	2.14 (1.15)	2.09 (1.16)	2.44 (1.19)	$F_{ST}(1,93) = 2.18$	
Acceptance	PC	2.04 (.96)	1.91 (1.01)	2.38 (1.18)	$F_{Gr}(2,92) = 2.48$	$F_{Gr \times ST}(2,92) = 0.04$
	SC	2.07 (.90)	1.94 (1.03)	2.47 (1.22)	$F_{ST}(1,93) = .25$	
Problem solving	PC	2.36 (1.13)	2.24 (1.06)	2.53 (1.37)	$F_{Gr}(2,92) = .84$	$F_{Gr \times ST}(2,92) = 0.08$
	SC	2.29 (1.15)	2.18 (1.21)	2.56 (1.24)	$F_{ST}(1,93) = .08$	
Rumination	PC	2.04 (1.00)	2.09 (.98)	2.28 (1.28)	$F_{Gr}(2,92) = 0.10$	$F_{Gr \times ST}(2,92) = 0.88$
	SC	2.29 (1.18)	2.12 (1.05)	2.13 (.83)	$F_{ST}(1,93) = 0.11$	
Avoidance	PC	2.11 (1.03)	1.82 (1.04)	2.47 (1.34)	$F_{Gr}(2,92) = 0.95$	$F_{Gr \times ST}(2,92) = 4.14^{*}$
	SC	2.00 (.94)	2.09 (1.13)	2.13 (1.13)	$F_{ST}\ 1,93) = 0.43$	
Exp. Supp.	PC	2.21 (1.17)	1.93 (1.17)	1.97 (1.23)	$F_{Gr}(2,92) = 0.33$	$F_{Gr \times ST}(2,92) = 0.28$
	SC	2.07 (.94)	1.97 (1.23)	2.03 (1.18)	$F_{ST}\ 1,93) = 0.14$	
Expr. Supp.	PC	2.29 (1.05)	1.82 (1.16)	2.19 (1.31)	$F_{Gr}(2,92) = 1.15$	$F_{Gr \times ST}(2,92) = 1.26$
	SC	1.86 (.89)	1.73 (.94)	2.03 (.97)	$F_{ST}(1,93) = 0.43$	

Note. Exp. Supp. = Experience suppression; Expr. Supp. = Expression suppression; ST = Stimulus type; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; *** $p < .001$; ** $p < .01$; * $p < .05$

Relationship between state ER and HRV

Correlation analyses within SSD did not reveal any significant relations between reappraisal use and both RMSSD (p = .864) and SDNN (p = .945), no associations between acceptance use and both RMSSD (p = .534) and SDNN (p = .652), no relations between problem solving use and both RMSSD (p = .097) and SDNN (p = .052). Further there were no significant associations between rumination use and both RMSSD (p = .057) and SDNN (p = .334), no relations between avoidance use and both RMSSD (p = .083) and SDNN (p = .512), no associations between use of experience suppression and both RMSSD (p = .332) and SDNN (p = .189), and finally no significant relations between expression suppression use and both RMSSD (p = .486) and SDNN (p = .922). Detailed information can be found in Table 11.

Relationship between subjetive reduction of negative emotions after state ER use and HRV

Correlation analyses within SSD did not reveal any significant relations between subjective reduction of negative emotion after reappraisal use and both RMSSD (p = .535) and SDNN (p = .645), no associations between subjective reduction of negative emotion after acceptance use and both RMSSD (p = .389) and SDNN (p = .496), no relations between subjective reduction of negative emotion after use of problem solving and both RMSSD (p = .848) and SDNN (p = .469). Further there were no significant association between subjective reduction of negative emotion after rumination use and both RMSSD (p = .489) and SDNN (p = .574), no relations between subjective reduction of negative emotion after avoidance use and both RMSSD (p = .913) and SDNN (p = .775), no associations between subjective reduction of negative emotion after use of experience suppression and both RMSSD (p = .283) and SDNN (p = .185), and finally no significant relations between subjective reduction of negative emotion after use of expression suppression and both RMSSD (p = .342) and SDNN (p = .195). Detailed information can be found in Table 12.

Table 11 Correlations between state ER and HRV time domain measures within SSD

Variable	SSD *M (SD)*	*r* (ER * RMSSD)	*r* (ER * SDNN)
HFERST			
Reappraisal	2.39 (.86)	.02	.01
Acceptance	2.99 (.99)	-.08	-.06
Probl. solving	2.93 (.94)	.21	.25
Rumination	3.06 (1.01)	.25	.13
Avoidance	2.58 (1.04)	.08	.09
Exp. Supp.	2.35 (.97)	-.13	-.17
Expr. Supp.	2.81 (1.13)	.09	.01
RMSSD	2.34 (1.13)	-	-
SDNN	2.88 (1.45)	-	-

Note. *$p < .05$

Table 12 Correlations between subjective reduction of negative emotion after use of state ER strategies and HRV time domain measures within SSD

Variable	SSD *M (SD)*	*r* (ER * RMSSD)	*r* (ER * SDNN)
HFERST			
Reappraisal	2.02 (.97)	-.08	-.06
Acceptance	1.98 (.87)	-.11	-.09
Probl. solving	2.26 (1.03)	.03	.09
Rumination	2.13 (.87)	-.09	-.07
Avoidance	2.00 (.95)	.01	.04
Exp. Supp.	2.04 (1.00)	-.14	-.17
Expr. Supp.	1.91 (.92)	-.12	-.17
RMSSD	2.34 (1.13)	-	-
SDNN	2.88 (1.45)	-	-

Note. *$p < .05$

4.3.3 Discussion

With respect to state ER, group differences could only be shown with respect to rumination, suggesting more subjective use of rumination in SSD-MUS compared with HC during stress inductions. This group difference was independent from the stressor type. The few state ER group differences could be due to several reasons. Altough results show that mood considerably worsened after stress exposures there were no group differences with respect to mood decreases. As ER research points out that ER itself depends besides others on emotion-eliciting stimuli (Aldao, 2013), negligent state ER group differences might be due to similar emotional reactions to the stressors. Another explanation refers to potential deficits of the used instrument, as the short-version of the HFERST (Izadpanah et al., 2017) is unpublished and not yet validated. Therefore, items used might not adequately represent the specific ER strategies. Participants were asked to retrospectively evaluate state ER use requiring certain introspective skills. Against the background of elevated alexithymia levels (De Gucht & Heiser, 2003) and deficits in emotion recognition (Subic-Wrana et al., 2010), SSD patients were expected to have greater difficulties to reflect ER strategy use retrospectively than HC. Perhaps the experimental task might have made introspection for all participants difficult, which might explain why only one group difference emerged. Groups did not differ with respect to subjective reduction of negative emotions after state ER use. This might be explained by two aspects: on the one hand, mood decreases in HC might not have been significant enough by which HCs did not necessarily rate certain ER strategies as particular effective. On the other hand, SSD patients are supposed to exhibit ER deficits. Following this, they might not have been aware of which ER strategy could have helped them to reduce negative emotions.

Associations between cardiac vagal control and ER (Aldao et al., 2016; Volokhov & Demaree, 2010) could not be found within SSD. HRV measures were not related with state

ER reporting and subjective reduction of negative emotions after state ER use as well. Even though patients with SSD-MUS reported more rumination use when being exposed with emotional stressors, a negative, correlational relationship between rumination use and RMSSD/SDNN in SSD was not revealed. SSD patients might not necessarily evaluate rumination as a particular maladaptive ER strategy in order to reduce negative emotions. Perhaps they appraise ER strategies as helpful in reducing negative emotions, although they do not actually increase vagal control. This might explain why no significant, correlational relationships between subjective reduction of negative emotions after state ER use and both measures could be found.

5 Acceptance versus coping: Examining their relevance in somatic symptom disorder

The following study investigated the respective relevance of acceptance and coping strategies in somatic symptom disorder.

5.1 Study 3: Context dependent relevance of acceptance and coping in somatic symptom disorder

Objective: Pain research regards acceptance as a stronger predictor of pain management than traditional coping strategies. This study aimed to examine whether this is also applicable to patients with SSD-MUS. Furthermore, this study investigated whether the intensity of symptom-related emotions moderates associations between acceptance, coping and symptom severity and disability.

Method: A total of 255 SSD patients participated in this study. MUS was assessed by using a diagnostic interview, ER, coping strategies, severity of somatic symptoms, and related disabilities by using questionnaires. For statistical analyses, bivariate correlation analyses, hierarchical regression, and moderation analyses were calculated.

Results: Acceptance was a significant predictor of both, symptom severity (β = -.32, p < .001) and symptom disability (β = -.24, p < .001), but did not account for more variance in symptom disability than behavioral coping. Acceptance was negatively related to symptom disability only at mean (b = -0.27 to -0.39) and high levels of anxiety and anger (b = -0.65 to -.67). Cognitive restructuring was negatively related to symptom severity only at low levels of anxiety and anger (b = -0.63 to -1.05). However, depression did not moderate any relationship between strategy use and symptom severity or disability.

Conclusion: The results suggest that a treatment promoting both acceptance and coping as well as considering contextual demands seems promising for SSD patients.

5.1.1 Introduction

SSD refers to bodily complaints such as pain, gastrointestinal or cardiovascular symptoms, accompanied by psychological criteria in the form of cognitive, affective, and behavioral aspects of HA (APA, 2013). Those physical symptoms might be medically explained or unexplained. MUS are classified as such if there is no evidence for a somatic disorder after medical evaluation (Klaus et al., 2013). The occurrence of MUS was a central requirement for classification of former called SFD (APA, 2000). Etiological assumptions of SSD mostly refer to models of SFD (Rief & Martin, 2014). Kirmayer and Taileffer (1997) proposed that emotional and physiological arousal ultimately results in somatic distress through cognitive, perceptional, and interactional processes. Emotional arousal can result from psychiatric disorders, stress, and trauma. This is in line with current research suggesting associations between somatization and negative life events in older adolescents (Bonvanie, Janssens, Rosmalen, & Oldehinkel, 2017). Cognitive processes particularly refer to catastrophizing (Frølund Pedersen, Frostholm, Søndergaard Jensen, Ørnbøl, & Schröder, 2016), meaning that perceived bodily changes a person steers attention to are evaluated as potentially dangerous. Cognitive and emotional reactions resulting from catastrophizing lead to illness behavior like help- and reassurance-seeking. Looper and Kirmayer (2002) therefore proposed that cognitive restructuring reattributes physical sensations to realistic causes, and behavioral techniques like promoting activity to address illness behaviors. These cognitive and behavioral interventions can be subsumed under the term coping, which is defined as ´cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person.´ (Lazarus & Folkman, 1984). These coping strategies are common therapy tools within CBT, which itself represents the most effective therapy approach to treat multiple MUS (Martin et al., 2013). As related effect sizes are moderate (Kleinstäuber et al., 2011), there is room for treatment improvement.

In recent years, acceptance-based strategies became increasingly relevant in psychological research, especially in chronic pain research. Acceptance refers to the ´willingness to remain in contact with and to actively experience particular private experiences (e.g. bodily sensations, emotions, thoughts, memories, behavioral predispositions)´ (Hayes et al., 1999, p. 34) and is widely regarded as an ER strategy (Aldao et al., 2010). ER describes ´processes by which individuals influence, which emotions they have, when they have them, and how they experience and express these emotions´ (Gross, 1998). The relevance of ER in SSD can be understood by means of Kirmayer and Taileffer's model, indicating that somatic distress results from emotional arousal and further processing (Kirmayer & Taileffer, 1997). This leads to the conclusion that regulation of emotional arousal affects somatic distress (Witthöft et al., 2013). Existing research shows beneficial effects of effective ER on somatic complaints (Vowles, McNeil, Gross, McDaniel, & Mouse, 2007; (Kohl et al., 2014). A previous study investigating ER skills in SSD (Schwarz et al., 2017) outlined lower ER skills, especially less use of acceptance as an ER strategy in SSD than in HC. The effects of acceptance as an ER strategy have been investigated previously. With respect to pain tolerance, there is evidence that acceptance-based strategies are superior to other ER strategies, while not generally being superior to other strategies like distraction or reappraisal (Kohl et al., 2012).

Regarding chronic pain, acceptance is known as an alternative to coping. Whilst a coping response is effortful and not automated (Jensen et al., 1991; McCracken & Eccleston, 2003), acceptance addresses disengagement (McCracken & Eccleston, 2003). Some researchers criticize that coping often aims to control aversive experiences, carrying the risk of discouragement when these attempts fail (McCracken & Eccleston, 2003). McCracken and Eccleston (2006) compared coping and acceptance regarding their impact on functional impairment. In comparison to coping, acceptance was more relevant with respect to pain disability or work status.

The specific role of acceptance strategies in comparison to traditional coping has not been investigated in SSD yet. This study examined whether acceptance-based strategies are as crucial in SSD as suggested in chronic pain research. As a first step, associations between strategy use and symptom severity as well as disability were examined. The second step was to investigate whether acceptance is a stronger predictor of symptom severity and disability compared to traditional coping.

There is growing evidence that the intensity of symptom-related emotions is an important contextual variable that may determine the specific strategy use dealing with private experiences. Recent findings point to acceptance only being used when people experience high emotional intensity, e.g. anger (Dixon-Gordon et al., 2015). Contrarily, people seem to use reappraisal in low intensity, but not in high intensity emotional states (Sheppes, 2014). So far, clinical research widely neglected these contextual factors. This is why this study aimed to evaluate the relation between acceptance/coping and symptom severity/disability under varying intensities of specific symptom-related emotions, in this case depressed/helpless mood, anxiety and anger.

5.1.2 Methods

This section reveals participant characteristics first, followed by a presentation of the recruitment process and study procedure. Afterwards, used self-report measures are presented, followed by a description of statistical analyses.

Participants

A total of 255 SSD participants with multiple MUS took part in a multicentric, prospective, parallel-grouped and randomized controlled trial to compare conventional CBT to CBT, enriched with strategies to improve emotion processing and regulation (ENCERT). Detailed information can be found in articles about the trial (Kleinstäuber et al., 2016, 2019).

The present study included baseline data from diagnostic assessments prior to treatment. Inclusion criteria were age between 18 and 69, at least three burdensome MUS lasting for at least 6 months, and a minimum of one psychological symptom (criterion B) of SSD according to DSM-5 (APA, 2013). Exclusion criteria were drug and substance abuse, acquired brain damage, a current psychosis or history of bipolar disorder, a current state of suicidality or another dominant psychiatric condition, continuous medication with antipsychotics, opioids or benzodiazepines, changes in antidepressant medication during the past four weeks, and a current psychotherapeutic treatment.

Recruitment and procedure

The multi-site study was carried out at seven different study sites. The German Psychological Association (DGPs), the Ethics Committee of the Medical Faculty of the Technical University Munich and the Ethics Committee of the Medical Association Hamburg approved the study protocol and the consent forms. All participants provided written consent. The study centers individually conducted recruitment of participants via print media, cooperation with general practitioners, clinical psychologists, and specialists in private practices.

Interested participants first registered at the study centers via phone or email. They received a telephone screening to review basic inclusion and exclusion criteria. If patients met the requirements, a set of consent form documents and questionnaires was sent to them. In order to finally evaluate inclusion criteria, a diagnostic interview was conducted in the facilities of the respective study center.

Measures

Besides the SSD-interview (Rief et al., 2010; Kleinstäuber et al., 2016), PHQ-15 (Kroenke et al., 2002), PDI (Mewes et al., 2009) and the ERSQ (Berking & Znoj, 2008), a further self-report meausure was used described below.

Coping With Chronic Pain Questionnaire (CPQ)

The adapted version of the CPQ (Geissner, 2001) is a self-report measure consisting of 38 items. It assesses the use of cognitive and behavioral pain coping strategies for PCs and related negative emotions like depressed mood, anxiety, and anger on 6-point Likert-scales (1 = *completely false* to 6 = *completely right*) related to the past days. To adjust the CPQ for SSD patients the target symptom *pain* was replaced with *physical complaints*. The present study used mean scores of the cognitive coping strategy ´cognitive restructuring´ and of the behavioral coping strategy ´countersteering activities´. Corresponding items were the following, e.g., *´When I have physical problems, I balance them with the good sides of life.´* (cognitive restructuring); *´When I have physical problems, I cover them by continuing my work.´* (countersteering activites); corresponding items for helpless/depressed mood, anxiety and anger were as follows, e.g., *´Due to my physical problems, I feel helpless.´* (helplessness/depression); *´Due to my physical problems, I am anxious.´* (anxiety); *´Due to my physical problems, I could scream in rage.´* (anger). In the study sample, the analyses of internal consistency revealed acceptable results for cognitive restructuring (α = .72) and countersteering activities (α = .67) and good results for helpless/depressed mood (α = .84), anxiety (α = .85), as well as anger (α = .88).

Statistical analysis

Sample size was calculated with a priori power analyses in G*Power 3.1.9.2 (Faul et al., 2009) based on the detection of small to medium effects (α = .05 and 1-β = .80). Calculations showed that an overall sample of at least n = 126 would be required to reach adequate statistical power, which was achieved by exceeding the required sample size (n = 129).

Data analyses were conducted using the 24 th version of the IBM SPSS. For correlation analyses, Pearson´s r was used to assess the relationship between acceptance

(ERSQ), coping (CPQ), symptom severity (PHQ-15), and symptom disability (PDI). One-tailed comparisons of correlation coefficients with p = 0.1 were conducted. Two hierarchical regressions were calculated to examine incremental variance explained by acceptance beyond the two coping strategies in predicting scores of somatic impairment measures. Criterion variables were symptom severity and symptom disability. Cognitive restructuring and countersteering activities were tested as predictors for entry first, acceptance was tested for entry second.

This study examined whether the particular intensities of symptom-related emotions moderate the relationship between strategy use and symptom severity or disability. The mean scores of each emotion intensity served as dimensional moderators, strategy use (acceptance, cognitive restructuring, countersteering activities) as predictor variables (all mean centered), and symptom severity and disability served as outcome variables. The analyses were conducted in PROCESS macro (Hayes, 2013) with 5000 bias corrected bootstrap samples. In case of significant interactions (p = .05), simple slope analyses, and calculations based on the Johnson-Neyman-procedure were carried out.

5.1.3 Results

The results section begins with a description of sociodemographic and clinical characteristics, followed by a presentation of correlation analyses and two hierarchical regression analyses, starting with the prediction of symptom severity first, followed by the prediction of symptom disability. The section ends with a presentation of moderation analyses.

Participant characteristics

Participants reported considerably more MUS than required for study inclusion (*M* = 10.82, *SD* = 6.93). Average symptom duration indicated chronicity of the symptoms (*M* = 7.23 years, *SD* = 7.87). Participants were significantly impaired by their physical symptoms as indicated by average PHQ-15 (*M* = 13.63, *SD* = 4.51) and the PDI mean score (*M* = 4.82, *SD* = 1.70). For detailed sample characteristics see Table 13.

Table 13 Sociodemographic data and clinical characteristics in the sample of SSD patients

N	251
Female % (n)	63.7 (160)
Age (M, SD)	43.45 (12.97)
German (first language) % (n)	92 (231)
Married % (n)	50.2 (126)
Single % (n)	14.3 (36)
Other % (n)	35.5 (89)
A-levels % (n)	28.3 (71)
Education in years (M,SD)	14.51 (2.9)
Number of MUS (M,SD)	10.82 (6.93)
Symptom duration in years (M,SD)	7.23 (7.87)
Symptom disability [PDI] (M,SD)	4.82 (1.70)
Symptom severity [PHQ-15] (M,SD)	13.63 (4.51)
Acceptance [ERSQ] (M,SD)	1.80 (0.93)
Cognitive restructuring [CPQ] (M,SD)	2.82 (1.15)
Countersteering activities [CPQ] (M,SD)	2.70 (1.03)
Depression [CPQ] (M,SD)	4.28 (1.04)
Anxiety [CPQ] (M,SD)	4.17 (1.25)
Anger [CPQ] (M,SD)	3.54 (1.34)

Note. PDI = Pain Disability Index, PHQ-15 = Patient Health Questionnaire-15, ERSQ = Emotion Regulation Questionnaire, CPQ = Coping With Chronic Pain Questionnaire

Relationships between acceptance, coping, and symptom impairment measures

Correlations indicate that a stronger degree of acceptance is associated with lower symptom severity (r = -.30, p < .001) and disability (r = -.24, p < .001). Bivariate correlational analyses revealed no significant relationship between cognitive restructuring and symptom severity (r = -.07, p = .130) or disability (r = -.05, p = .225). However, countersteering activities did correlate with symptom severity (r = -.13, p = .022) as well as with symptom disability (r = -.28, p < .001).

Predictions of symptom impairment by coping and acceptance

Cognitive restructuring (step 1: β = -.05, p = .467) and countersteering activities did not significantly predict symptom severity (step 1: β = -.12, p = .068). Both coping strategies did not explain incremental criterion variance (step 1: ΔR^2 = .02, p = .101). However, acceptance was a significant predictor of symptom severity (β = -.32, p < .001) and significantly increased the amount of variance explained (step 2: ΔR^2 = .09, p < .001). In total, the carried out regression model carried out explained 9% of the variance in symptom severity.

Cognitive restructuring was not a significant predictor of symptom disability (step 1: β = .01, p = .876), but countersteering activities were (step 1: β = -.28, p < .001). The criterion variance explained was 8%. Acceptance was also a significant predictor of symptom disability (β = -.24, p < .001) and increased the explained variance significantly (step 2: ΔR^2 = .05, p < .001). Overall, the model explained 13% of the variance in symptom disability. Detailed information of the hierarchical regression analyses is displayed in Table 14.

Table 14 Hierarchical regression models predicting symptom severity (PHQ-15) and disability (PDI)

Predictor	PHQ-15				PDI			
	F(2,248)	*ΔR²*	Step 1 β	Step 2 β	*F*(2,248)	*ΔR²*	Step 1 β	Step 2 β
Step 1	2.32	.02			10.23	.08***		
Cognitive restructuring			-.05	.08			.01	.10
CSA			-.12	-.10			-.28***	-.26***
	F(3,247)				*F*(3,247)			
Step 2	9.56	.09***			11.73	.05***		
Acceptance				-32***				-.24***

Note. ****p* <.001, CSA = Countersteering activities

Moderation effects of symptom-related emotions

Moderation analyses (see Table 15) examined associations between strategy use and symptom severity as well as disability among patients with varying levels of depressed mood. The degree of depressed mood did neither moderate the relationship between acceptance use and somatic symptom severity, $F(1,247) = .10$, $p = .702$, nor the association between acceptance use and symptom disability, $F(1,247) = -.12$, $p = .149$. Depressed mood intensity did not moderate the relationship between cognitive restructuring and symptom severity, $F(1,247) = .32$, $p = .115$, and neither between cognitive restructuring and symptom disability, $F(1,247) = -.04$, $p = .556$. Finally depressed mood levels did not moderate the association between countersteering activities and symptom severity, $F(1,247) = -.01$, $p = .975$, and neither between countersteering activities and symptom disability, $F(1,247) = -.04$, $p = .643$.

Table 15 Moderation effect of the intensity of helpless/depression on the relation between strategy use and symptom severity (PHQ-15) and symptom disability (PDI)

PHQ-15					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	13.67	.29	47.91	< .001	[13.11 ; 4.24]
HD (M)	1.23	.29	4.24	< .001	[0.66 ; 1.81]
Acceptance (X)	-.82	.30	-2.69	.008	[-1.42 ; -0.22]
X × M	.10	.26	.38	.702	[-0.41 ; 0.61]
Overall Model: $F(3,247) = 18.13$, $p < .001$, $R^2 = .16$					
Constant	13.73	.28	49.61	< .001	[13.19 ; 4.28]
HD (M)	1.55	.27	5.79	< .001	[1.02 ; 2.08]
CR (X)	.15	.26	.57	.566	[-0.37 ; 0.67]
X × M	.32	.20	1.58	.115	[-0.08 ; 0.72]
Overall Model: $F(3,247) = 16.42$, $p < .001$, $R^2 = .15$					
Constant	13.63	.27	50.15	< .001	[13.09 ; 4.16]
HD (M)	1.54	.25	6.23	< .001	[1.05 ; 2.03]
CsA (X)	-.25	.27	-.95	.346	[-0.77 ; 0.27]
X × M	-.01	.29	-.03	.975	[-0.57 ; 0.55]
Overall Model: $F(3,247) = 14.13$, $p < .001$, $R^2 = .14$					
PDI					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	4.76	.10	46.34	< .001	[4.56 ; 4.97]
HD (M)	.90	.10	9.52	< .001	[0.72 ; 1.09]
Acceptance (X)	.03	.12	.22	.827	[-0.21 ; 0.26]
X × M	-.12	.09	-1.45	.149	[-0.29 ; 0.05]
Overall Model: $F(3,247) = 41.71$, $p < .001$, $R^2 = .29$					
Constant	4.81	.09	51.28	< .001	[4.62 ; 4.99]
HD (M)	.93	.08	11.01	< .001	[0.76 ; 1.09]
CR (X)	.15	.09	1.79	.074	[-0.02 ; 0.32]
X × M	-.04	.07	-.59	.556	[-0.17 ; 0.09]
Overall Model: $F(3,247) = 40.86$, $p < .001$, $R^2 = .30$					
Constant	4.81	.09	51.61	< .001	[4.63 ; 4.99]
HD (M)	.81	.08	9.62	< .001	[0.65 ; 0.98]
CsA (X)	-.28	.10	-2.83	.005	[-0.47 ; -0.09]
X × M	-.04	.09	-.46	.643	[-0.22 ; 0.14]
Overall Model: $F(3,247) = 43.15$, $p < .001$, $R^2 = .31$					

Note. HD = helplessness/depression, CR = cognitive restructuring, CsA = countersteering activities, M = moderator variable, X = predictor variable, Y = criterion variable

Anxiety did not moderate the relationship between acceptance and symptom severity, $F(1,247) = -.13$, $p = .648$. However, there was an interaction between anxiety and the association between acceptance and symptom disability, $F(1,247) = -.32$, $p = .003$. Follow-up analyses showed that this association was only significant at mean anxiety levels or at levels above the mean (centered cut-off value obtained via Johnson-Neyman technique = -.03). Simple slope analyses showed that acceptance was negatively related to symptom disability at mean anxiety levels ($b = -.27$, $p = .041$) and those above the mean (one *SD* above mean: $b = -.67$, $p < .001$). Anxiety did also moderate the relationship between cognitive restructuring and symptom severity, $F(1,247) = .47$, $p = .011$. Follow-up analyses showed that this association was only significant at anxiety levels below mean (centered cut-off value obtained via Johnson-Neyman technique = -.93). Simple slope analyses showed that cognitive restructuring was negatively related to symptom disability at anxiety levels below the mean ($b = -.63$, $p = .019$). However, anxiety did not moderate the relationship between cognitive restructuring and symptom disability, $F(1,247) = -.01$, $p = .946$. Furthermore, neither a moderation effect of anxiety with respect to the association between countersteering activities and symptom severity, $F(1,247) = -.04$, $p = .842$, nor the relationship between countersteering activites and symptom disability was found, $F(1,247) = -.09$, $p = .370$. Further details can be found in Table 16.

Table 16 Moderation effect of the intensity of anxiety on the relation between strategy use and symptom severity (PHQ-15) and disability (PDI)

PHQ-15					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	13.57	.28	48.07	< .001	[13.02 ; 4.13]
Anxiety (M)	1.11	.23	4.83	< .001	[0.65 ; 1.56]
Acceptance (X)	-.91	.29	-3.13	.002	[-1.48 ; -0.34]
X × M	-.13	.29	-.46	.648	[-0.69 ; 0.43]
Overall Model: $F(3,247) = 16.28$, $p < .001$, $R^2 = .17$					
Constant	13.74	.27	50.49	< .001	[13.20 ; 4.28]
Anxiety (M)	1.33	.21	6.18	< .001	[0.90 ; 1.75]
CR (X)	-.04	.25	-.16	.875	[-0.53 ; 0.45]
X × M	.47	.19	2.56	.011	[0.11 ; 0.84]
Overall Model: $F(3,247) = 21.09$, $p < .001$, $R^2 = .16$					
Constant	13.63	.27	51.44	< .001	[13.11 ; 4.15]
Anxiety (M)	1.34	.22	6.18	< .001	[0.91 ; 1.76]
CsA (X)	-.52	.26	-2.02	.045	[-1.03 ;- 0.01]
X × M	-.04	.23	-.17	.862	[-0.50 ; 0.42]
Overall Model: $F(3,247) = 13.42$, $p < .001$, $R^2 = .15$					
PDI					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	4.68	.12	39.72	< .001	[4.45 ; 4.91]
Anxiety (M)	.33	.10	3.34	.001	[0.13 ; 0.52]
Acceptance (X)	-.27	.13	-2.06	.041	[-0.53 ; -0.01]
X × M	-.32	.11	-3.05	.003	[-0.53 ; -0.11]
Overall Model: $F(3,247) = 14.88$, $p < .001$, $R^2 = .13$					
Constant	4.82	.11	44.96	< .001	[4.61 ; 5.03]
Anxiety (M)	.36	.09	3.99	< .001	[0.18 ; 0.54]
CR (X)	-.01	.10	-.08	.935	[-0.21 ; -0.19]
X × M	-.01	.08	-.07	.946	[-0.15 ; 0.14]
Overall Model: $F(3,247) = 5.67$, $p = .001$, $R^2 = .07$					
Constant	4.82	.11	47.42	< .001	[4.62 ; 5.02]
Anxiety (M)	.36	.09	4.08	< .001	[0.18 ; 0.52]
CsA (X)	-.43	.11	-4.02	< .001	[-0.64 ; -0.22]
X × M	-.09	.10	-.90	.370	[-0.29 ; 0.11]
Overall Model: $F(3,247) = 14.00$, $p < .001$, $R^2 = .15$					

Note. HD = helplessness/depression, CR = cognitive restructuring, CsA = countersteering activities, M = moderator variable, X = predictor variable, Y = criterion variable

Finally, moderation analyses (see Table 17) with respect to the relationship between strategy use and symptom severity as well as disability among patients with varying anger mood were conducted. Anger did not moderate the association between acceptance and symptom severity, $F(1,247) = -.01$, $p = .962$. However, anger moderated the relationship between acceptance use and symptom disability, $F(1,247) = -.20$, $p = .040$. Follow-up

analyses showed that this association was only significant at mean anger levels or those above mean (centered cut-off value obtained via Johnson-Neyman technique = .56). Simple slope analyses showed that acceptance was negatively related to disability at mean anger levels (b = -.39, p = .002) and those above mean (one *SD* above the mean: b = -.65, p < .001). Furthermore, anger moderated the relationship between cognitive restructuring and symptom severity, $F(1,247)$ = .55, p = .004. Follow-up analyses showed that this association was only significant at anger levels below mean (centered cut-off value obtained via Johnson-Neyman technique = -0.28). Simple slope analyses showed that cognitive restructuring was negatively related to symptom severity at anger levels below the mean (b = -1.05, p < .001). However, there was no interaction between anger and the association between cognitive restructuring and symptom disability, $F(1,247)$ = .02, p = .809. Anger neither moderated the association between countersteering activities and symptom severity, $F(1,247)$ = .15, p = .436, nor between countersteering activities and symptom disability, $F(1,247)$ = -.09, p = .277.

Table 17 Moderation effect of the intensity of anger on the relation between strategy use and symptom severity (PHQ-15) and disability (PDI)

PHQ-15					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	13.63	.27	50.53	< .001	[13.10 ; 4.16]
Anger (M)	.64	.21	3.01	.003	[0.22 ; 1.06]
Acceptance (X)	-.1.32	.28	-4.69	< .001	[-1.87 ; -0.76]
X × M	-.01	.22	-.05	.962	[-0.44 ; 0.42]
Overall Model: $F(3,247) = 13.06$, $p < .001$, $R^2 = .13$					
Constant	13.62	.27	49.66	< .001	[13.08 ; 4.16]
Anger (M)	.77	.21	3.61	< .001	[0.35 ; 1.18]
CR (X)	-.32	.25	-1.27	.205	[-0.81 ; 0.18]
X × M	.55	.19	2.95	.004	[0.18 ; 0.91]
Overall Model: $F(3,247) = 11.52$, $p < .001$, $R^2 = .10$					
Constant	13.62	.28	49.03	< .001	[13.08 ; 4.17]
Anger (M)	.83	.22	3.76	< .001	[0.39 ; 1.26]
CsA (X)	-.63	.27	-2.38	.018	[-1.15 ;- 0.11]
X × M	.15	.19	.78	.436	[-0.22 ; 0.51]
Overall Model: $F(3,247) = 6.19$, $p < .001$, $R^2 = .08$					
PDI					
Variable	*b*	*SE*	*t*	*p*	*95% CI*
Constant	4.78	.11	44.26	< .001	[4.57 ; 4.99]
Anger (M)	.20	.08	2.45	.015	[0.04 ; 0.36]
Acceptance (X)	-.39	.12	-3.20	.002	[-0.62 ; -0.15]
X × M	-.20	.10	-2.07	.040	[-0.39 ; -0.01]
Overall Model: $F(3,247) = 10.05$, $p < .001$, $R^2 = .10$					
Constant	4.82	.11	45.15	< .001	[4.61 ; 5.03]
Anger (M)	.24	.08	2.91	.004	[0.08 ; 0.40]
CR (X)	-.08	.10	-.74	.461	[-0.28 ; 0.13]
X × M	.02	.08	.24	.809	[-0.13 ; 0.17]
Overall Model: $F(3,247) = 3.19$, $p = .024$, $R^2 = .04$					
Constant	4.82	.11	47.24	< .001	[4.62 ; 5.02]
Anger (M)	.25	.08	3.17	.002	[0.09 ; 0.40]
CsA (X)	-.44	.10	-4.22	< .001	[-0.65 ; -0.24]
X × M	-.09	.08	-1.09	.277	[-0.24 ; 0.07]
Overall Model: $F(3,247) = 10.02$, $p < .001$, $R^2 = .12$					

Note. HD = helplessness/depression, CR = cognitive restructuring, CsA = countersteering activities, M = moderator variable, X = predictor variable, Y = criterion variable

5.1.4 Discussion

This study examined the relevance of acceptance-based strategies in SSD, particularly whether these are stronger predictors of symptom severity and disability than traditional coping. Furthermore, this study investigated whether certain strategy use is associated with reduced symptom severity and disability under specific intensity levels of symptom-related emotions.

The data shows that acceptance and somatic burden were negatively related. The higher the degree of acceptance-based ER, the lower the symptom severity and disability. This is consistent with literature outlining relationships between acceptance and pain intensity as well as tolerance in experimental settings (Kohl et al., 2012). Concerning coping, the results were diverse: cognitive restructuring was neither associated with symptom severity nor with symptom disability in SSD subjects. This contradicts previous results, which indicated negative relations between cognitive restructuring and somatic preoccupation in health anxious participants (Bardeen & Fergus, 2014). The results suggest that potential relations between cognitive restructuring and symptom impairment might depend on further variables like emotion intensity. Countersteering activities were negatively associated with both symptom severity and disability. This illustrates that in comparison to protective behavior, controlled behavior activation is beneficial in dealing with somatic burden.

Previous research suggests that acceptance strategies are better predictors for pain adjustment than coping (McCracken & Eccleston, 2003, 2006). However, this does not apply completely to the present SSD patient sample. The behavioral coping strategy countersteering activities explained more variance in disability than acceptance, but both variables served as relevant predictors. This discrepancy between the present and

previous results might be caused by the different measures that were used. While McCracken & Eccleston (2006) used an acceptance measure that covers behavioral aspects of activity engagement, the measure that was used in the present study primarily referred to the emotional experience. In contrast to the former study, not only cognitive, but also behavioral coping was included.

While current ER research suggests that emotion intensity predicts a person´s choice of ER strategy (Dixon-Gordon et al., 2015; Sheppes, 2014), it is not clear in MUS patients yet, whether the adaptiveness of acceptance and coping depends on the intensity of a person´s emotional state. The results indicate that - under certain circumstances - emotion intensity moderates a relationship between strategy use and somatic burden. At mean and elevated levels of anxiety and anger, acceptance use was negatively related to symptom disability, but not symptom severity. This is in line with previous findings (Hayes et al., 1999), indicating that acceptance may lead to more flexibility in intense, aversive, and uncontrollable inner states. Under these circumstances acceptance does not necessarily reduce somatic burden, but seems to enhance the ability to adapt in daily life. Furthermore, at low anxiety and anger levels cognitive restructuring was negatively associated with symptom severity, but not symptom disability. When experiencing less intense emotions, it seems to be easier to change dysfunctional beliefs about experienced symptom burden in comparison to more intense emotions.

These conditional relations between strategy use and somatic burden were comparable for emotional experiences of anxiety and anger. However, depressed mood did not moderate any association between strategy use and symptom severity and disability. This might show that in comparison to anxiety and anger, emotions of depression are accompanied by limited access to adaptive strategies, therefore

explaining missing associations with symptom severity and disability under specific levels of depressed mood.

In sum, the findings imply that coping or ER strategies are not, per se, adaptive or maladaptive, but their effect may depend on the contextual factors when being used. This shows that a flexible use of a range of strategies (Bardeen & Fergus, 2014) is essential in psychotherapy.

The established CBT approach does not explicitly include ER skills like acceptance to overcome aversive symptoms and emotions. Bleichhardt, Gottschalk and Rief (2014) conceived a promising CBT extension considering an ER training for MUS patients including acceptance strategies. The data shows that a CBT concept promoting not only adaptive coping, but also ER skills like acceptance might be associated with beneficial treatment outcome.

The present study offered several strengths to be mentioned: reported data referred to consecutively recruited clinical participants. Another strength was the multicenter design, allowing conclusions about a representative patient sample. Furthermore, this data refers to a state prior treatment onset. This implies that patients did not have any psychotherapeutic treatment for at least two years. Therefore, the present patients' use of coping and ER was not confounded by undergoing psychotherapy and its interventions at the same time.

However, the present study offers some limitations as well, especially in respect to the cross-sectional design. Therefore, longitudinal effects of acceptance or coping strategy implementation on symptom impairment cannot be examined at this stage. Another limitation related to different periods of time both the ERSQ and CPQ refer to. While some pain researchers (Geisser et al., 1999) stated that the reduction of maladaptive coping might be more crucial for treatment outcome, the present study exclusively focused on adaptive strategy use. Future studies should consider the broader

range of ER and coping strategies and examine under which contextual influences their effects are particularly inconvenient.

The study shows that besides coping, acceptance is also related to symptom severity and disability in SSD patients. As acceptance predicts symptom severity and disability, it might be useful to individually promote this ER strategy at SSD treatment under certain conditions. As the data implies that the beneficial outcome of strategy use depends on contextual factors, future SSD research should integrate recent ER findings in experimental paradigms in order to learn more about the contextual adaptiveness of ER strategies. Furthermore, this research indicates that a tailored treatment approach might be reasonable, teaching patients a flexible use of strategies based on external and internal demands.

6 General discussion

In this section a summary of key findings in relation with current research is presented first. Afterwards, strenghts and limitations of the thesis are revealed, followed by implications for future research and therapy, closing with an overall conclusion.

6.1 Summary of key findings in relation with current research

The doctoral thesis was conducted in order to investigate HRV abnormalities and ER deficits in SSD patients. Provoking psychological and physiological reactivity was a crucial premise to make profound conclusions about HRV rigidity and ER deficits. Script driven imagery (Lang, Levin, Miller, & Kozak, 1983) proved to be a successful method to induce psychological and physiological reactivity. Physiological abnormalities in SSD could be found: in comparison to HC, HRs were generally higher in SSD patients confirming previous results outlining heightened, autonomic arousal in SSD (Rief et al., 1998; (Rief & Barsky, 2005). In contrast to HR, HRV activity in SSD and HC did not differ overall, although SSD revealed marginally lower HRV levels than HC prior to exposure. As previous findings outlined lower HRV activity in SSD compared to HC (e.g. Tak et al., 2009; Huang et al., 2017), the current results do not present a clear picture. Perhaps the comparability of the current results with previous ones is not ensured due to the use of ultra short term periods.

Although there were no reactivity differences with respect to HR, HRV reactivity differences could be found instead. HRV levels were not significantly affected by stress exposures, independent of the stressor type. In contrast to HC, HRV levels in SSD did not decrease during transitions between resting conditions and stress exposures. This might be due to increased HRs prior to stress inductions not necessarily allowing HRV decreases. As HRV is regarded as a marker of the ability to flexibly adapt to changing environmental demands (Appelhans & Luecken, 2006; Schmidt & Martin, 2017), this

finding reveals difficulties in adaptively regulating emotional and physiological reactions. Study results broaden existing knowledge regarding physiological reactivity in SSD as it is one of the first ones revealing autonomic rigidity against the background of different disorder-related emotional stressors. Whereas previous studies used facial recognition tasks (Pollatos et al., 2011) or pain imagination tasks (Pollatos et al., 2011), the emotional stressors in the present study covered a broad range of individual physical symptoms and interpersonal conflicts, also including related cognitions and emotions. Stressor-related group differences could not be found regarding physiological measures, but SSD patients revealed greater symptom disability increases after SC induction in comparison with HC. As SC induction led to greater tension increases than PC inductions in all groups, this particular increase might have caused SSD patients to experience their physical symptoms as more disabling. Although mood worsened after stress exposure, there were no affective reactivity differences between SSD and HC. This might explain why no state ER group differences were found, except for increased rumination use in SSD-MUS compared to HC. Increased rumination use could have been the reason why SSD patients experienced their symptoms as more disabling after SC, although this has not explicitely been investigated. Although there was no affective reactivity in the form of increased health worry levels after stress exposure, generally higher health worry levels might have contributed to more state rumination use in SSD patients.

In contrast to state ER, results concerning habitual ER were clearer. In accordance with previous results (e.g. Schwarz et al., 2017), SSD patients reported less adaptive habitual ER use in comparison with HC. The present study broadened existing knowledge by also revealing more maladaptive habitual ER use in SSD. While adaptive ER use was associated with lower HA levels, maladaptive ER use was related with increased HA levels. These findings correspond with earlier findings in subclinical patients, which outlined emotion dysregulation to be related with increased HA (Bardeen

& Fergus, 2014; Fergus & Valentiner, 2010; Görgen et al., 2014). Although adaptive and maladaptive ER predicted HA, ER in general does not necessarily play a crucial role in explaining different HA levels between SSD and HC as some ER strategies did either not or only partially mediate relationships between both the clinical and HC group and HA levels. The ER strategy acceptance was a significant predictor of symptom severity and disability and in some cases more adaptive as traditional cognitive and behavioral coping strategies. The present results therefore confirm previous findings, which suggest acceptance of emotions to be a better predictor than coping regarding pain management (McCracken & Eccleston, 2003; McCracken & Eccleston, 2006). However, the present results broaden existing knowledge as the adaptiveness of coping and acceptance was investigated under consideration of contextual factors like emotion intensity (Dixon-Gordon et al., 2015). The thesis provides new findings as acceptance and coping use was adaptive under specific circumstances: acceptance use was associated with reduced symptom severity at mean or elevated anxiety and anger levels, whereas cognitive restructuring use was related to reduced symptom severity at low anxiety and anger levels. Taken these results into account, aforementioned predictions of HA by ER strategies might not present the whole picture. Depending on the present state of emotion intensity, relationships between acceptance and HA and reappraisal and HA might have been different.

As researchers propose relationships between vagal control and ER (Balzarotti, Biassoni, Colombo, & Ciceri, 2017; Aldao et al., 2016; Volokhov & Demaree, 2010), results of this thesis do not allow definite conclusions. HRV measures and state ER reporting were not significantly associated. Even though patients with SSD-MUS reported more rumination use when being exposed to emotional stressors, no relationship between rumination use and RMSSD or SDNN as indicators of cardiac vagal control could be found. Positive correlations between increased RMSSD or SDNN levels and stronger subjective reduction of negative emotions after adaptive state ER use could

not be found. This means that an ER strategy appraised as subjectively more helpful in reducing negative emotions is not necessarily associated with increased vagal control. Cardiac control and subjective reduction of negative emotions might be different constructs. The ER strategy expression suppression for example might have a beneficial short-term effect of reducing negative emotions, but might simultaneously be associated with increased tension and therefore with decreased vagal control.

A further aim was to examine whether SSD-groups, namely SSD-MUS and SSD-MES, were comparable regarding ER and autonomic characteristics. This became relevant as SSD, based on DSM-5 classification, includes cases suffering from physical symptoms with different etiological factors. The ´lumpers´ perspective focuses on commonalities between different SSD symptom clusters, whereas the other, the ´splitting´ view, questiones whether these commonalities are valid for the entire SSD population (Güney, Sattel, Witthöft, & Henningsen, 2019). To the best of knowledge, this investigation has not been explicitely conducted within SSD-subgroups. However, Klaus and colleagues (2013) compared MUS- and MES-characteristics within the general population. Irrespective of the medical explanation, pain symptoms appear to be associated with high impairment and changes in lifestyle. Psychological features like illness attributions or avoidance behavior seem to be the reason why former MES “transform” into MUS, for example after certain medical surgeries. MUS and MES altogether seem to be comparably impairing and stable (Klaus et al., 2013). Schroeder and colleagues (2014) compared patients with non-cardiac chest pain with patients with and without cardiac chest pain. In contrast to their expectations, groups did not differ with respect to implicit negative interpretation bias concerning somatosensory sensations. The present results seem to confirm this ´lumper´-perspective as no substantial differences between SSD-MUS and SSD-MES were revealed with regard to autonomic or ER characteristics, except for more habitual ER-avoidance use in SSD-MES compared to SSD-MUS. Whether a physical symptom is medically explained or not,

might not play a crucial role on how memory representations of illnesses influence the perception of somatic distress (according to the model of Brown, 2004).

6.2 Strengths and limitations

Aim of this section is to report overall strenghts and limitations of the studies. To the best of knowledge, this is the first study investigating HRV reactivity in SSD, referring to several illness-related stressors and state ER use during stress-exposure. This allowed a more distinguished view whether decreased vagal control and maladaptive ER manifest equally independent of the stressor type, and whether there are contextual-related reaction patterns. The use of script-driven imagery enabled to expose SSD patients to individual stressors and therefore allowing to examine reactivity patterns. In the quasi-experimental study, subjective ER was assessed with the HFERST, a questionnaire that appeared suitable for several reasons: one reason refers to advantages over other ER questionnaires as it covers broad cognitive and behavioral strategies being significantly related with psychopathology. Another reason refers to the inclusion of understudied ER strategies like experience suppression and using an acceptance scale that refers both to situational and emotional aspects (Izadpanah et al., 2017). Furthermore, the HFERST has a habitual (trait-like) and a state version. It enables to investigate whether habitual ER deficits in SSD manifest in the same way while being exposed to different emotional stressors. The fact that especially SSD patients were asked to label emotions accompanying PCs or SCs in a semi-structured interview is an issue to be adressed. As SSD patients are assumed to reveal emotion recognition deficits (Subic-Wrana et al., 2010), it is questionable whether patients labeled their emotions according to their actual experience or in accordance to certain social norms, which means that they potentially labeled them the way they thought people would normally feel in these situations. The induction method itself underlies certain

disadvantages that have to be discussed. Kleinstäuber and colleagues (2018) previously used this induction method and referred to small effect sizes with respect to reactivity. The present results either indicated no significant reactivity (state symptom intensity, health beliefs/worries, both HRV measures) or the effect sizes were also relatively small with the exception of HR. This raises the question whether this method exposed participants to their acute or chronic symptoms realistically. It would be convenient to implement a further rating scale in the paradigm. Participants might be asked to evaluate how vividly they experienced their individual symptoms during the exposure.

6.3 Implications for future research

Future research should invest in the improvement of the experimental paradigm. While participants create individual scripts dealing with their PCs or SCs, they also label accompanying emotions. One disadvantage of the present paradigm is that rating scales just referred to general mood and not a particular emotional experience participants described in the script previously (like sadness, fear, etc). Therefore, ratings referring to script-related emotions would be convenient. Taken knowledge about moderating influence of emotion intensity into account, state ER results might have been more conclusive if participants would be asked to rate intensities of previously identified emotions just before and after stressor inductions. However, this kind of rating process might bear the risk that the emotional reaction towards the actual stressor is influenced by particular expectations on what to feel based on previously identified emotions in the script. In recent years, virtual reality became a promising tool to expose subjects to computer-generated sensory information and images that encourages them to confront problematic, illness-related situations (Freeman et al., 2017). With respect to pain, virtual reality was previously used as an alternative non-pharmacologic analgesic by encouraging patients to shift their focus away from acute pain (Ahmadpour et al., 2019).

Taken the possibilites of virtual reality into account, implementing it in SSD-related experimental paradigms might be a promising approach. Considering relatively small exposure-related effect-sizes in the present and previous studies (Kleinstäuber et al., 2018), the use of virtual reality might enable to investigate SSD-related illness-mechanisms more effectively: exposing patients to more detailed and graphic illness-related stimuli (e.g. situations in hospitals, patient-doctor-interactions, etc.), might make different information-processing between SSD-patients and HC more obvious.

Manipulating emotion type and intensity and instructing participants using specific ER strategies would provide reliable knowledge about the contextual adaptability of ER strategy use in SSD patients. At the same time, it would be important to investigate further relationships between ER processes and cardiac vagal control, meaning that the adaptiveness of spontaneous ER use should be evaluated simultaneously by examining relationships between ER use and HRV levels. Referring to assumed emotion recognition deficits in SSD, it would be convenient to examine whether HRV rigidity in SSD corresponds with problems to identify the emotional impact of the stressor first in order to effectively regulate it.

6.4 Practical implications

As presented study results point to autonomic rigidity in SSD, the necessity to restore autonomic homeostasis becomes one target of therapeutic approaches. HRV biofeedback aims to enhance respiratory sinus arrhythmia (RSA) and in consequence vagal control of HRV (Schmidt & Martin, 2017). It is a promising intervention, which proved to be effective with regard to various psychopathologies (e.g. Lehrer & Gevirtz, 2014). Existing study results imply that HRV biofeedback might successfully reduce somatic impairment for example in patients with fibromyalgia or veterans suffering from chronic pain (Hassett et al., 2007; Berry et al., 2014). The present study results

emphasize a less flexible, autonomic response of SSD patients to emotional stressors in comparison to HC. Following this, SSD patients might be trained to enhance RSA when being exposed to health-related cues, simulated with the help of virtual reality.

Further, the present study results also reveal ER deficits in SSD. Consequently, these deficits deserve further attention in psychotherapeutic approaches. In the recently published ENCERT-study (Kleinstäuber et al., 2019), conventional CBT was compared with an extension of CBT including an ER training called ENCERT. Whereas CBT included interventions like attention allocation training, stress coping and cognitive restructuring, ENCERT focused on the role of negative emotions and its recognition and regulation. Results revealed comparable treatment effects concerning MUS with the ENCERT group. The ENCERT-group revealed better behavioral coping strategies due to acquired acceptance strategies as adaptive alternative to avoidance behavior (Kleinstäuber et al., 2019). Taken these results into account and drawing on the results of study 3, future, psychotherapeutic approaches might lean on several pillars. The first one is an emotion recognition training. In a second step it might be advisable to apply e-diary-methods, in which patients monitor which emotions they experience and to what extent. Simultaneously, they should also monitor symptom severity and disability. Depending on the type and intensity of emotions, patients should learn to use ER strategies flexibly in order to adaptively regulate aversive emotions. After ER strategy application, they might rate their somatic and emotional state again.

6.5 Conclusion

Inflexible HRV responses to emotional stressors and ER deficits appear to manifest equally both in SSD-MUS and SSD-MES. ER is a significant predictor of HA and symptom severity and disability, not necessarily a crucial one, but apparently context-dependent. Whether autonomic rigidity and ER deficits are a result of decreased

vagal control has to be further investigated. It might be useful to focus on further factors that influence these relationships like emotion recognition. When patients with SSD get exposed to emotional stressors, autonomic rigidity might result from a missing ability to identify the emotional impact of the stressor. As a consequence, they might experience diffuse stress and might not able to adaptively regulate it because they are not aware what exactly they have to regulate.

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Appendix

A-1 Information processing during physical complaints

A-1.1 Vote of the institutional ethics committee

BERGISCHE
UNIVERSITÄT
WUPPERTAL

Prof. Dr. Michael Scheffel
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DATUM 18. Juni 2015

Bergische Universität Wuppertal, Prof. Dr. Michael Scheffel, Gaußstraße 20, 42119 Wuppertal

Frau
Prof. Dr. Alexandra Martin
FB G
Human- und Sozialwissenschaften
Z.02.09

im Hause

Votum der Ethik-Kommission

Sehr geehrte Frau Martin,

hiermit teile ich Ihnen in meiner Eigenschaft als zuständiger Prorektor mit, dass die Ethik-Kommission der Bergischen Universität Wuppertal ausführlich über den von Ihnen gestellten Antrag zur Durchführung des Forschungsvorhabens *„Informationsverarbeitung bei körperlichen Beschwerden"*, beraten und positiv, d. h. für die Unbedenklichkeit der projektierten Studie votiert hat.

Mit freundlichen Grüßen

Prof. Dr. Michael Scheffel

A-1.2 Participant information

BERGISCHE UNIVERSITÄT WUPPERTAL

Bergische Universität Wuppertal
Fachbereich G
Wissenschaftliche Leitung: Prof. Dr. Alexandra Martin
Projektleitung: Dipl.-Psych. Harald Gitzen
Telefon: 0202 439-5053
E-mail: klipsy-projekt@uni-wuppertal.de

Allgemeine Teilnehmerinformation über die Untersuchung

Bergische Universität Wuppertal, Fachbereich G

„Informationsverarbeitung bei körperlichen Beschwerden“

Herzlich willkommen zu unserer Studie „Informationsverarbeitung bei körperlichen Beschwerden“! Wir danken Ihnen für Ihr Interesse an der Studie.
Ziel dieser Untersuchung ist die Erforschung von emotionalen Verarbeitungsprozessen im Kontext körperlicher Beschwerden und zwischenmenschlicher Konflikte.

Ablauf der Studie

Die Untersuchung gliedert sich in zwei Teile, die an zwei möglichst aufeinanderfolgenden Tagen durchgeführt werden:

Im ersten Teil der Studie findet zunächst ein kurzes Eingangsscreening zur Klärung der Ein- und Ausschlusskriterien statt, welches am Computer bearbeitet wird. Daran schließen sich ein strukturiertes und ein standardisiertes Interview zwischen Ihnen und dem Versuchsleiter an. Im Anschluss werden mehrere Fragebögen zu den Themen Emotionsregulation, Krankheitsangst und Körpersymptombelastung alleine am Computer bearbeitet. Dieser Teil der Studie wird insgesamt ca. 1,5 Stunden in Anspruch nehmen. Sie können selbstverständlich jederzeit eine Pause machen.

Zu Beginn des zweiten Studientermins wird gemeinsam mit dem Versuchsleiter ein Skript bezüglich einer Körperbeschwerde und eines zwischenmenschlichen Konflikts erstellt und anschließend jeweils auf Tonband gesprochen. Das wird ca 15 Minuten dauern. Diese Tonaufzeichnungen werden Ihnen im weiteren Verlauf des Experiments noch einmal dargeboten, wozu Ihnen dann verschiedene Fragen gestellt werden. Parallel werden physiologische Ableitungen vorgenommen (EKG, PVA, EDA, EMG). Dabei werden Ihnen 3 Elektroden zur Messung des Herzschlags, 2 Elektroden an der Hand zur Messung der Hautleitfähigkeit, 3 Elektroden im Gesicht zur Messung der mimischen Muskelaktivität sowie ein Licht-Fingersensor angelegt. An den Stellen im

Gesicht müssen wir die Haut dafür mit einem leichten Peeling und medizinischem Alkohol vorbehandeln. Die Elektroden sind gesundheitlich vollkommen unbedenklich und lassen sich nach dem Experiment einfach und rückstandslos entfernen. Alle verwendeten Verbrauchsmaterialen und Pasten sind dermatologisch auf ihre Verträglichkeit getestet.

Es ist anzumerken, dass im Kontext dieser Erhebung diagnostisch relevante Informationen über körperliche und psychische Erkrankungen gesammelt werden. Nach dem Prinzip des Nicht-Schädigens sind wir dazu verpflichtet, Ihnen auffällige Befunde am Ende der Studienteilnahme mitzuteilen. Nach einer Mitteilung steht es Ihnen frei, die Befunde weiter abzuklären. Weitere medizinische bzw. psychologische Abklärungen können mit versicherungsrechtlichen Konsequenzen (z. B. Berufsunfähigkeitsversicherungen, private Krankenversicherung) verbunden sein. Falls Sie keine Aufklärung über auffällige Befunde wünschen, besteht KEINE Möglichkeit an dieser Studie teilzunehmen. Der zweite Teil des Experiments wird insgesamt ca. 45 Minuten in Anspruch nehmen.

Freiwilligkeit und Anonymität

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an der Studie widerrufen, ohne dass daraus Nachteile entstehen. Falls Sie zu irgendeinem Zeitpunkt – aus welchen Gründen auch immer – einen Termin oder auch die gesamte Untersuchung abbrechen möchten, steht Ihnen dies völlig frei. Teilen Sie es uns bitte in einem solchen Fall mit. Sie werden zur Studienteilnahme eine Einverständniserklärung ausfüllen, in der Sie uns bestätigen, dass Sie über diese Möglichkeit aufgeklärt wurden und freiwillig an der Studie teilnehmen.
Die im Rahmen dieser Studie erhobenen persönlichen Informationen (Fragebogen-/ Interviewdaten, Audioaufnahmen, physiologische Messungen) werden streng vertraulich behandelt. Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, sind verpflichtet, diese nicht an Dritte weiterzugeben.

Datenschutz

Alle erhobenen Daten werden pseudoanonymisiert und streng vertraulich nach Vorgaben der Datenschutzrichtlinien behandelt. Die Daten können nicht Ihrer Person zugeordnet werden, d. h. es wird eine Nummer ohne Angabe Ihres Namens verwendet. Eine Verbindung zwischen Ihrem Namen und der Tonaufnahme kann nur mittels der Kodierliste (auf Papier) hergestellt werden. Die Kodierliste ist nur dem Versuchsleiter zugänglich und wird nach Abschluss der Datenerhebung gelöscht. Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, sind verpflichtet, diese nicht an Dritte weiterzugeben. Des Weiteren wird die Veröffentlichung der Ergebnisse der Studie in anonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

Aufklärung über Befunde

Die Untersuchung dient ausschließlich Forschungszwecken. Es könnten uns sowohl in den psychophysiologischen als auch in den Fragebogendaten ungewöhnliche Untersuchungsergebnisse auffallen. In diesem Fall werden wir Sie vertraulich auf die Auffälligkeiten hinweisen und Ihnen eine Kurzberatung zum möglichen weiteren Vorgehen anbieten.

Vergütung

Psychologiestudierende der Bergischen Universität Wuppertal erhalten für die Teilnahme 3 Versuchspersonenstunden. Für die klinischen Stichproben ist für die Teilnahme an der Untersuchung eine Aufwandsentschädigung in der Höhe von 20 € vorgesehen.

A-1.3 Declaration of consent regarding recording and application of audio material

BERGISCHE UNIVERSITÄT WUPPERTAL

Bergische Universität Wuppertal
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Einwilligungserklärung zur Aufnahme und Anwendung von Tonaufnahmen

Bergische Universität Wuppertal, Fachbereich G

„Informationsverarbeitung bei körperlichen Beschwerden“

Ich (Name des Teilnehmers in Blockschrift),

_____

bin mündlich und schriftlich (siehe Allgemeine Teilnehmerinformation) von

Herr/Frau _____

darüber informiert worden, dass im Rahmen der Studie zwei Tonaufnahmen angefertigt werden.

Die Aufnahmen dienen dazu, dass die Teilnehmer/innen ihre eigenen Erfahrungen zu einer körperlichen Beschwerde und zu einem zwischenmenschlichen Konflikt im Versuch

vorgespielt bekommen. Jede(r) Teilnehmer/in der Studie hört nur seine persönlichen Tonaufzeichnungen an.

Ich bin darüber informiert worden, dass die Aufzeichnung und Auswertung der Tonaufnahme pseudoanonymisiert erfolgt. Pseudoanonymisierung bedeutet, dass die Tonaufnahme mit einer Nummer und nicht mit dem Namen versehen wird. Außerdem existiert eine Kodierliste, die Name und Nummer in Verbindung bringt. Diese ist nur dem Versuchsleiter zugänglich und wird nach Abschluss der Datenerhebung gelöscht.

Es besteht die sehr geringe Wahrscheinlichkeit, dass eine an der Datenauswertung beteiligte Person mich erkennt. Aus diesem Grund dürfen alle an der Auswertung beteiligten Personen unter keinen Umständen vertrauliche Informationen an Dritte weitergeben.

Mir ist bekannt, dass ich mein Einverständnis zur Aufbewahrung/Speicherung dieser Daten widerrufen kann, ohne dass mir daraus Nachteile entstehen. Die Tonaufnahme wird in einem verschlossenen Schrank aufbewahrt. Ich bin darüber informiert worden, dass ich jederzeit eine Löschung meiner Aufnahme verlangen kann, solange die Kodierliste existiert.
Mit der beschriebenen Handhabung der erhobenen Aufnahmen bin ich einverstanden.

Die Einverständniserklärung für die Tonaufnahme ist freiwillig. Ich kann diese Erklärung jederzeit widerrufen. Im Falle meiner Ablehnung oder meines Rücktritts entstehen für mich keinerlei Kosten oder anderweitige Nachteile. Allerdings ist eine Teilnahme an der Studie dann nicht möglich.
Ich hatte genügend Zeit für eine Entscheidung. Ich habe alles gelesen und verstanden und erkläre mich hiermit bereit, dass Tonaufnahmen gemacht werden.

Eine Ausfertigung dieser Einwilligungserklärung habe ich erhalten.

Ort, Datum & Unterschrift des Teilnehmers

Ort, Datum & Unterschrift des Versuchsleiters

A-1.4 Participant instructions

1. Herzlich Willkommen zum ersten Teil der Studie
2. Es beginnt nun der erste Teil der Studie „Informationsverarbeitung bei körperlichen Beschwerden“. Ihre Aufgabe besteht darin, verschiedene schriftliche und mündliche Fragen bzw. Aussagen zu bewantworten bzw. einzuschätzen.
3. Eingangsfragebogen
4. Im Folgenden werde ich Ihnen zunächst einige Fragen zu Ihrer Person sowie verschiedene Fragen zu möglichen aktuellen körperlichen Beschwerden geben. Bitte antworten Sie so genau und so wahrheitsgemäß wie möglich.
5. Mini-DIPS
6. Wir werden Ihnen nun einige Fragen in Form eines strukturierten und standardisierten Interviews stellen. Wir bitten Sie möglichst Angaben zu machen.
7. Versuchsleiter füllt CIDI-SOM am PC aus. Bitte dem Probanden gegenüber sitzen.
8. Diagnostische Einschätzung. Vom Versuchsleiter auszufüllen!
9. PHQ-15
10. PDI
11. mSHAI
12. HFERST
13. SEK-27
14. Herzlich Willkommen zum zweiten Teil der Studie
15. Bitte bleiben Sie ruhig sitzen und warten Sie auf weitere Anweisungen
16. Sachaufgabe 1
17. Es folgt nun ein kurzer Hörfunkbeitrag. Bitte hören Sie gut zu und beantworten Sie die Fragen am Ende des Beitrags.
18. Bitte beantworten Sie nun einige Fragen zum Inhalt des Beitrags („Knirps“). Sie haben 60 Sekunden Zeit für die Beantwortung der 6 Fragen
19. Ratingskalen
20. Abspielen Skript 1
21. Bitte bleiben Sie ruhig sitzen und warten Sie auf weitere Anweisungen
22. Ratingskalen
23. HFERST state
24. Bitte bleiben Sie ruhig sitzen und warten Sie auf weitere Anweisungen
25. Sachaufgabe 2
26. Es folgt nun ein kurzer Hörfunkbeitrag. Bitte hören Sie gut zu und beantworten Sie die Fragen am Ende des Beitrags.
27. Bitte beantworten Sie nun einige Fragen zum Inhalt des Beitrags („Biathlon“). Sie haben 60 Sekunden Zeit für die Beantwortung der 6 Fragen
28. Ratingskalen
29. Abspielen Skript 2
30. Bitte bleiben Sie ruhig sitzen und warten Sie auf weitere Anweisungen
31. Ratingskalen
32. HFERST state

A-1.5 Demographic questionnaire

I. Allgemeine Fragen zu Ihrer Person:

Alter: …….

Geschlecht: 1) Männlich
2) Weiblich

Familienstand: 1) Single
2) in einer Beziehung
3) verheiratet

Staatsangehörigkeit: 1) Deutsch
2) Andere
…….

Muttersprache: 1) Deutsch
2) Andere
…….

Höchster Bildungsabschluss: 1) Keiner
2) Hauptschule
3) Realschule
4) Abitur
5) Studium

II. Fragen zu Ihrer körperlichen Verfassung:

1. Leiden Sie derzeit unter körperlichen Beschwerden?
1) Ja
2) Nein

2. Wenn ja, wie lange leiden Sie unter Ihren Beschwerden?
…….

3. Konnten die Beschwerden nach ärztlicher Untersuchung organisch erklärt werden?
1) Ja
2) Nein

4. Wenn Ja, welche Krankheiten wurden bei Ihnen festgestellt? Wann wurde die jeweilige Diagnose gestellt?

……
……

5. Sind sie dauerhaft auf die Unterstützung eines medizinischen Gerätes angewiesen? (z.B. Herzschrittmacher)

1) Ja
2) Nein

Wenn ja, welches:
…….

6. Nehmen Sie regelmäßig Medikamente ein?
1) Ja
2) Nein

Wenn ja, welche?
……

7. Haben Sie heute Medikamente eingenommen? Wenn ja, welche?
……

III. Weitere Fragen zu Ihrer Person:

1. Sind Sie schwanger oder besteht bei Ihnen Verdacht auf eine Schwangerschaft?
1) Ja
2) Nein

2. Fühlen Sie sich derzeit von Alkohol, einem Medikament oder einer Droge abhängig?
1) Ja
2) Nein
Wenn ja, wovon?
……

3. Befinden Sie sich derzeit in Psychotherapie?
1) Ja
2) Nein

4. Befanden Sie sich in den letzten zwei Jahren aufgrund von körperlichen Beschwerden in Psychotherapie?

1) Ja
2) Nein

A-1.6 Diagnostic assessment

Diagnostische Einschätzung

1) Somatische Belastungsstörung - MUS
2) Somatische Belastungsstörung - MES
3) Gesund
4) Ausschluss

…….

Klassifikation Somatische Belastungsstörung

Schmerz:
1) mit überwiegendem Schmerz
2) andauernd

Schweregrad
1) leicht
2) mittel
3) schwer

A-1.7 Interview guideline to create a health-related script

- Unter welcher körperlichen Beschwerde leiden/litten Sie am meisten?

  → ***Beschwerde notieren***

_____

_____

_____

- Wie schätzen Sie die Belastung durch diese Beschwerde auf einer Skala von 0 bis 100 ein?

_____

- Können Sie mir genau beschreiben, wie sich das anfühlt/angefühlt hat? Wie fängt/fing das an? Was ist/war das Schlimmste?

  → **Sensorische Eindrücke notieren**

_____

_____

_____

_____

_____

- Können Sie mir genau beschreiben, was Sie dabei fühlen/fühlten?

  → ***Emotionen notieren***

_____

_____

_____

_____

_____

- Was geht/ging Ihnen durch den Kopf, wenn Sie jetzt daran denken?

  → ***Gedanken notieren***

_____

_____

_____

_____

_____

A-1.8 Interview guideline to create a script regarding a social conflict

- Um was für einen zwischenmenschlichen Konflikt handelt es sich? Unter welchem Aspekt des Konflikts leiden/litten Sie am meisten?

  → ***Konflikt notieren***

_____

_____

_____

- Wie schätzen Sie die Belastung durch diesen Konflikt auf einer Skala von 0 bis 100 ein?

_____

- Können Sie mir genau beschreiben, wie sich das angefühlt hat? Wie fing das an? Was war das Schlimmste?

  → ***Sensorische Eindrücke notieren***

_____

_____

_____

_____

- Können Sie mir genau beschreiben, was Sie dabei fühlen/fühlten?

  → ***Emotionen notieren***

_____

_____

_____

_____

- Was ging Ihnen damals durch den Kopf, wenn Sie jetzt an diese Situation denken?

  → ***Gedanken notieren***

_____

_____

_____

_____

A-1.9 Distraction task 1

Wer gilt als „Vater“ des Knirps?

Reiner Schulz
Holger Rohrschach
Detlef Karstens
Hans Haupt (X)

Was machte den Erfinder des Knirps erfinderisch?

Eine Kriegsverletzung (X)
Heftige Sturmböen
Starke Regenfälle
Ein Verkehrsunfall

Wann wurde der erste teleskopierbare Taschenschirm kontruiert?

1975
1928 (X)
1877
1923

Wann wurde mit der Produktion des Knirps begonnen?

Anfang der 40er Jahre
Ende der 20er Jahre
Anfang der 30er Jahre (X)
Ende der 30er Jahre

Wer freute sich besonders über einen Knirps im Krokodillederetui?

Queen Elizabeth II
Prinz Harry
King George
Prinzessin Beatrix (X)

Wen überholte der Taschenschirm in der Gunst der Kunden?

Stockschirm (X)
Kunstschirm
Satellitenschirm
Regenschirm

A-1.10 Distraction task 2

Worum geht es beim Biathlon?

Zusammenhang von Skilaufen und Skispringen
Zusammenhang von Schießen und Skilaufen (X)
Zusammenhang von Werfen und Skilaufen
Zusammenhang von Skifliegen und Skilaufen

Wo fand der Biathlonweltcup vor ein paar Wochen statt?

In Antholz (X)
In Genf
In Skandinavien
In Bremen

Wer waren berühmte Militärskiläufer?

Die Ostmarker
Die Uckermarker
Die Normarker
Die Finnmarker (X)

Wann maßen sich Soldaten an der norwegisch-schwedischen Grenze an einem legendären Wettkampf?

Anfang des 20. Jahrhunderts
Ende des 19. Jahrhunderts
Ende des 18. Jahrhunderts (X)
Anfang des 19. Jahrhunderts

Was galt als Vorläufer des Biathlons?

Militärpatrouillenlauf (X)
Turmtaubenschießen
Schützenfest
Militärstiefelmarsch

Wann wurde Biathlon erstmals offizielle Sportart bei olympischen Winterspielen?

1940
1960 (X)
1972
1955

A-1.11 Numeric rating scales

Dieser Fragebogen beschäftigt sich mit Ihrem momentanen Empfinden. Bitte wählen Sie mit den Tasten [1] bis [9] aus, wie sehr folgende Aussagen zutreffen.

Wie stark empfinden Sie Ihre körperlichen Beschwerden im Augenblick?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

überhaupt nicht — sehr stark

Wie sehr fühlen Sie sich durch Ihre körperlichen Beschwerden im Augenblick beeinträchtigt?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

überhaupt nicht — sehr stark

Wie sehr sind Sie davon überzeugt eine ernsthafte Krankheit zu haben?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

gar nicht — sehr stark

Wie besorgt sind Sie bezüglich Ihrer Gesundheit?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

überhaupt nicht — sehr stark

Wie ist Ihre derzeitige Anspannung?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

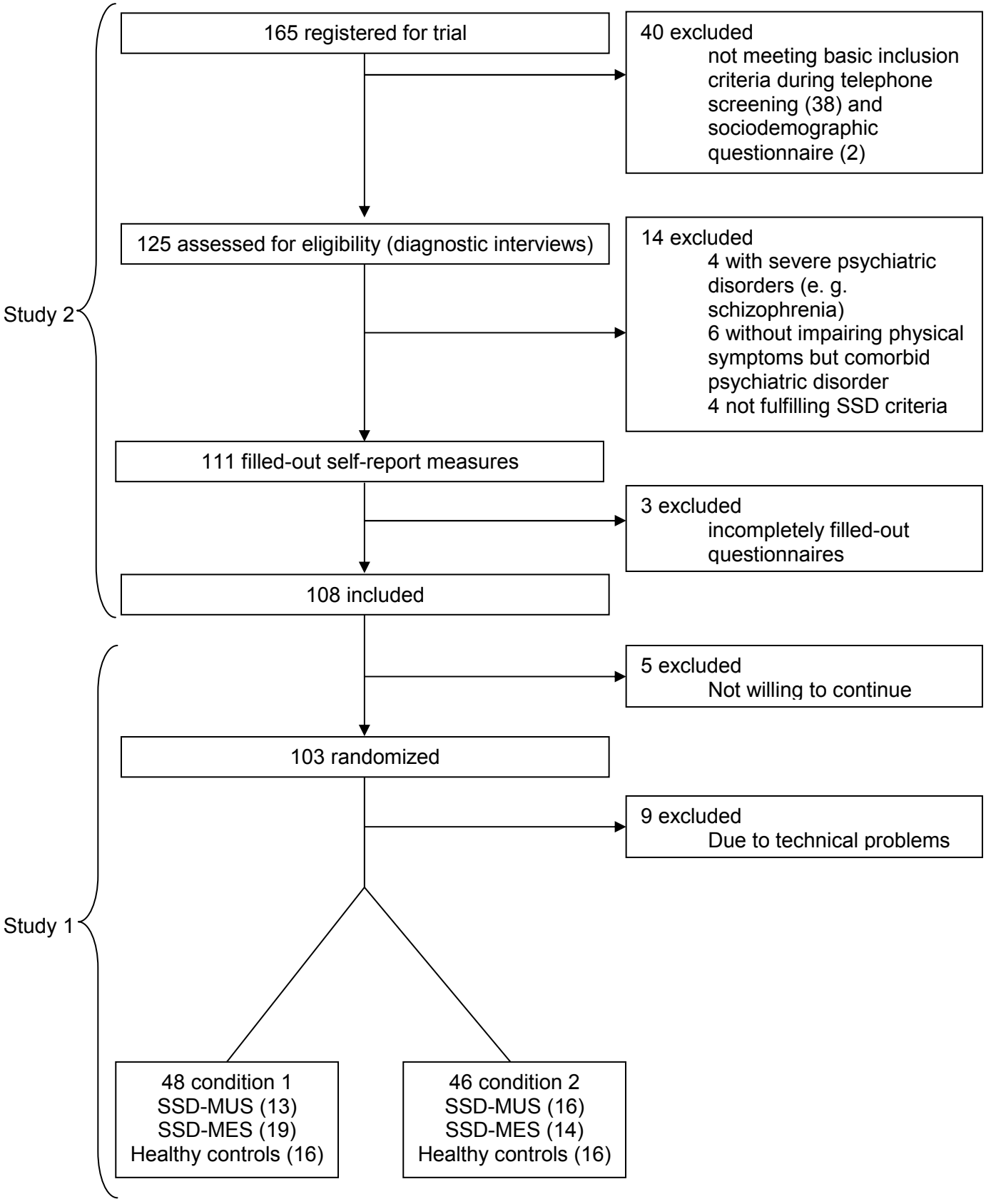
überhaupt nicht vorhanden — maximal

Wie ist Ihre derzeitige Stimmung?

[1] [2] [3] [4] [5] [6] [7] [8] [9]

sehr schlecht — sehr gut

A-1.12 Trial profile



A-1.13 Kolmogorov-Smirnov-tests of normal distribution, Levene´s-tests of variance homogeniety and Box-tests of equality of covariance matrices with respect to numeric rating scales and physiological data - study 1

Variable	Kolmogorov-Smirnov-test				Levene´s test				Box-test				
	N	*Z*	*df*	*p*	*F*	*df1*	*df2*	*p*	*M*	*F*	*df1*	*df2*	*p*
PC_int_pre	94				41.71	2	91	< .001	266.12	12.46	20	28973.28	< .001
MUS	29	.14	29	.167									
MES	33	.20	33	.001									
HC	32	.53	32	< .001									
PC_int_post	94				39.77	2	91	< .001					
MUS	29	.15	29	.120									
MES	33	.18	33	.011									
HC	32	.52	32	< .001									
SC_int_pre	94				42.81	2	91	< .001					
MUS	29	.18	29	.013									
MES	33	.24	33	< .001									
HC	32	.52	32	< .001									
SC_int_post	94				38.15	2	91	< .001					
MUS	29	.15	29	.099									
MES	33	.16	33	.033									
HC	32	.53	32	< .001									
PC_im_pre	94				57.55	2	91	< .001	11.23	1.04	10	16541.51	.405
MUS	29	.18	29	.015									
MES	33	.21	33	.001									
HC	32	.54	32	< .001									
PC_im_post	94				50.56	2	91	< .001					
MUS	29	.19	29	.009									
MES	33	.17	33	.020									
HC	32	.54	32	< .001									
SC_im_pre	94				45.53	2	91	< .001					
MUS	29	.21	29	.002									
MES	33	.18	33	.012									

HC	32	.54	32	< .001								
SC_im_post	94				44.04	2	91	< .001				
MUS	29	.23	29	<.001								
MES	33	.17	33	.017								
HC	32	.53	32	< .001								
PC_te_pre	94				3.56	2	91	.032	43.65	2.04	20	28973.28	.004
MUS	29	.14	29	.165								
MES	33	.14	33	.109								
HC	32	.26	32	< .001								
PC_te_post	94				4.03	2	91	.021				
MUS	29	.15	29	.115								
MES	33	.17	33	.012								
HC	32	.24	32	< .001								
SC_te_pre	94				9.99	2	91	< .001				
MUS	29	.19	29	.010								
MES	33	.16	33	.032								
HC	32	.23	32	< .001								
SC_te_post	94				3.94	2	91	.023				
MUS	29	.18	29	.022								
MES	33	.19	33	.005								
HC	32	.26	32	< .001								
KB_mo_pre	94				2.73	2	91	.071	81.47	3.81	20	28973.28	< .001
MUS	29	.19	29	.010								
MES	33	.18	33	.006								
HC	32	.18	32	.011								
KB_mo_post	94				3.90	2	91	.024				
MUS	29	.13	29	.200								
MES	33	.15	33	.060								
HC	32	.16	32	.050								
SK_mo_pre	94				5.41	2	91	.006				
MUS	29	.23	29	< .001								
MES	33	.14	33	.100								
HC	32	.22	32	< .001								

SK_mo_post	94				4.14	2	91	.019				
MUS	29	.13	29	.200								
MES	33	.13	33	.137								
HC	32	.23	32	< .001								
PC_hb_pre	94				31.25	2	91	< .001	146.24	6.84	20	28973.28	< .001
MUS	29	.26	29	< .001								
MES	33	.18	33	.011								
HC	32	.46	32	< .001								
PC_hb_post	94				41.11	2	91	< .001				
MUS	29	.20	29	.003								
MES	33	.17	33	.019								
HC	32	.39	32	< .001								
SC_hb_pre	94				30.86	2	91	< .001				
MUS	29	.19	29	.008								
MES	33	.18	33	.008								
HC	32	.44	32	< .001								
SC_hb_post	94				27.38	2	91	< .001				
MUS	29	.29	29	< .001								
MES	33	.16	33	.025								
HC	32	.39	32	< .001								
PC_hw_pre	94				19.15	2	91	< .001	143.15	6.70	20	28973.28	< .001
MUS	29	.20	29	.004								
MES	33	.10	33	.200								
HC	32	.36	32	< .001								
PC_hw_post	94				25.13	2	91	< .001				
MUS	29	.23	29	< .001								
MES	33	.14	33	.090								
HC	32	.38	32	< .001								
SC_hw_pre	94				23.59	2	91	< .001				
MUS	29	.20	29	.005								
MES	33	.15	33	.074								
HC	32	.35	32	< .001								
SC_hw_post	94				19.89	2	91	< .001				

MUS	29	.26	29	< .001									
MES	33	.13	33	.200									
HC	32	.36	32	< .001									
PC_hr_pre	94				1.61	2	91	.206	18.95	.89	20	28973.28	.604
MUS	29	.14	29	.169									
MES	33	.14	33	.103									
HC	32	.11	32	.200									
PC_hr_post	94				.55	2	91	.582					
MUS	29	.15	29	.107									
MES	33	.16	33	.025									
HC	32	.13	32	.174									
SC_hr_pre	94				.84	2	91	.436					
MUS	29	.12	29	.200									
MES	33	.14	33	.116									
HC	32	.13	32	.200									
SC_hr_post	94				1.13	2	91	.329					
MUS	29	.09	29	.200									
MES	33	.11	33	.200									
HC	32	.07	32	.200									
PC_hrv_pre	94				1.63	2	91	.201	52.60	2.46	20	28973.28	< .001
MUS	29	.13	29	.195									
MES	33	.09	33	.200									
HC	32	.08	32	.200									
PC_hrv_post	94				.62	2	91	.541					
MUS	29	.11	29	.200									
MES	33	.14	33	.108									
HC	32	.10	32	.200									
SC_hrv_pre	94				.79	2	91	.459					
MUS	29	.13	29	.200									
MES	33	.08	33	.200									
HC	32	.10	32	.200									
SC_hrv_post	94				2.91	2	91	.059					
MUS	29	.11	29	.200									

MES	33	.07	33	.200
HC	32	.11	32	.200

Note. PC_int_pre = state symptom intensity prior to physical complaint exposure; PC_int_post = state symptom intensity after physical complaint exposure; SC_int_pre = state symptom intensity prior to social conflict exposure; state SC_int_post = state symptom intensity after social conflict exposure; PC_im_pre = state symptom impairment prior to physical complaint exposure; PC_im_post = state symptom impairment after physical complaint exposure; SC_im_pre = state symptom impairment prior to social conflict exposure; SC_im_post = state symptom impairment after social conflict exposure; PC_te_pre = state tension prior to physical complaint exposure; PC_te_post = state tension after physical complaint exposure; SC_te_pre = state tension prior to social conflict exposure; SC_te_post = state tension after social conflict exposure; PC_mo_pre = state mood prior to physical complaint exposure; PC_mo_post = mood after physical complaint exposure; SC_mo_pre = mood prior to social conflict exposure; SC_mo_post = mood after social conflict exposure; PC_hb_pre = state health beliefs prior to physical complaint exposure; PC_hb_post = state health beliefs after physical complaint exposure; SC_hb_pre = state health beliefs prior to social conflict exposure; SC_hb_post = state health beliefs after social conflict exposure; PC_hw_pre = state health worries prior to physical complaint exposure; PC_hw_post = state health worries after physical complaint exposure; SC_hw_pre = state health worries prior to social conflict exposure; SC_hw_post = state health worries after social conflict exposure; PC_hr_pre = heart rate prior to physical complaint exposure; PC_hr_post = heart rate after physical complaint exposure; SC_hr_pre = heart rate prior to social conflict exposure; SC_hr_post = heart rate after social conflict exposure; PC_hrv_pre = heart rate variability prior to physical complaint exposure; PC_hrv_post = heart rate variability after physical complaint exposure; SC_hrv_pre = heart rate variability prior to social conflict exposure; SC_hrv_post = heart rate variability after social conflict exposure.

A-1.14 Group differences in subjective measures (with order as further between subjects factor) - study 1

Variable	Order	ST	Ex	SSD-MUS		SSD-MES		HC		Main effects	Interaction effects
				N	*M (SD)*	*N*	*M (SD)*	*N*	*M (SD)*		
Symptom intensity	1	PC	P	13	4.38 (2.14)	19	3.58 (2.32)	16	1.19 (.54)	$F_{Gr}(2,92) = 28.80^{***}$	$F_{Gr \times Ex}(2,92) = 0.74$
			E	13	4.08 (2.10)	19	3.58 (2.39)	16	1.19 (.54)	$F_{O}(1,93) = .49$	$F_{Gr \times ST}(2,92) = 0.63$
		SC	P	13	3.77 (1.83)	19	3.47 (2.20)	16	1.19 (.54)	$F_{Ex}(1,93) = 1.93$	$F_{Gr \times O}(2,92) = .24$
			E	13	3.85 (1.91)	19	3.74 (2.33)	16	1.06 (.25)	$F_{ST}(1,93) = 1.74$	$F_{O \times Ex}(2,92) = 2.82$
	2	PC	P	16	4.50 (2.39)	14	3.93 (2.23)	16	1.00 (.00)		$F_{O \times ST}(2,92) = 1.09$
			E	16	3.93 (2.23)	14	4.14 (2.32)	16	1.06 (.25)		$F_{Gr \times ST \times Ex}(2,92) = 1.73$
		SC	P	16	4.31 (2.30)	14	3.79 (2.55)	16	1.06 (.25)		$F_{O \times ST \times Ex}(2,92) = .02$
			E	16	4.81 (2.37)	14	3.93 (2.09)	16	1.13 (.34)		$F_{Gr \times O \times Ex}(2,92) = .78$
											$F_{Gr \times O \times ST}(2,92) = 2.32$
											$F_{Gr \times O \times ST \times Ex}(2,92) = .47$
											$F_{ST \times Ex}(1,93) = 2.02$
Symptom impairment	1	PC	P	13	4.46 (2.43)	19	3.00 (2.16)	16	1.06 (.25)	$F_{Gr}(2,92) = 25.97^{***}$	$F_{Gr \times Ex}(2,92) = .91$
			E	13	4.00 (2.20)	19	3.05 (2.22)	16	1.06 (.25)	$F_{O}(1,93) = .99$	$F_{Gr \times ST}(2,92) = 2.40$
		SC	P	13	3.23 (2.05)	19	3.00 (2.36)	16	1.06 (.25)	$F_{Ex}(1,93) = 7.58^{**}$	$F_{Gr \times O}(2,92) = .53$
			E	13	3.84 (1.95)	19	3.32 (2.43)	16	1.13 (.34)	$F_{ST}(1,93) = 2.30$	$F_{O \times Ex}(2,92) = .50$
	2	PC	P	16	4.19 (2.34)	14	3.86 (2.57)	16	1.00 (.00)		$F_{O \times ST}(2,92) = .34$
			E	16	4.25 (2.11)	14	4.14 (2.38)	16	1.06 (.25)		$F_{Gr \times ST \times Ex}(2,92) = 4.80^{*}$
		SC	P	16	3.94 (1.91)	14	3.79 (2.19)	16	1.00 (.00)		$F_{O \times ST \times Ex}(2,92) = 4.49^{*}$
			E	16	4.31 (1.99)	14	3.93 (2.20)	16	1.06 (.25)		$F_{Gr \times O \times Ex}(2,92) = .15$
											$F_{Gr \times O \times ST}(2,92) = 2.32$
											$F_{Gr \times O \times ST \times Ex}(2,92) = 1.07$
											$F_{ST \times Ex}(1,93) = 7.30^{**}$
Tension	1	PC	P	13	4.46 (1.76)	19	4.74 (2.05)	16	2.56 (1.15)	$F_{Gr}(2,92) = 16.32^{***}$	$F_{Gr \times Ex}(2,92) = .33$
			E	13	4.92 (1.50)	19	4.89 (2.08)	16	2.94 (1.65)	$F_{O}(1,93) = .58$	$F_{Gr \times ST}(2,92) = 1.47$
		SC	P	13	4.08 (1.66)	19	4.52 (1.90)	16	2.50 (1.41)	$F_{Ex}(1,93) = 9.27^{**}$	$F_{Gr \times O}(2,92) = .35$
			E	13	4.62 (1.76)	19	5.58 (2.14)	16	2.75 (1.65)	$F_{ST}(1,93) = .54$	$F_{O \times Ex}(2,92) = 2.55$
	2	PC	P	16	5.00 (1.93)	14	4.57 (2.28)	16	3.06 (1.73)		$F_{O \times ST}(2,92) = 2.00$

			E	16	4.88 (1.74)	14	4.64 (2.50)	16	2.94 (1.34)		$F_{Gr \times ST \times Ex}(2,92) = .37$
		SC	P	16	4.93 (2.08)	14	5.07 (2.43)	16	2.75 (1.00)		$F_{O \times ST \times Ex}(2,92) = .17$
			E	16	5.69 (1.58)	14	5.07 (2.40)	16	3.06 (1.34)		$F_{Gr \times O \times Ex}(2,92) = .37$
											$F_{Gr \times O \times ST}(2,92) = .76$
											$F_{Gr \times O \times ST \times Ex}(2,92) = 2.97$
											$F_{ST \times Ex}(1,93) = 4.63^{*}$
Mood	1	PC	P	13	6.00 (2.16)	19	5.79 (1.47)	16	7.06 (1.34)	$F_{Gr}(2,92) = 8.38^{***}$	$F_{Gr \times Ex}(2,92) = 1.37$
			E	13	5.85 (2.12)	19	5.37 (2.03)	16	6.88 (1.50)	$F_{O}(1,93) = .62$	$F_{Gr \times ST}(2,92) = 1.63$
		SC	P	13	5.62 (2.18)	19	5.63 (1.42)	16	7.13 (1.26)	$F_{Ex}(1,93) = 9.34^{**}$	$F_{Gr \times O}(2,92) = .17$
			E	13	5.23 (2.05)	19	5.37 (1.57)	16	6.81 (1.42)	$F_{ST}(1,93) = 1.89$	$F_{O \times Ex}(2,92) = .00$
	2	PC	P	16	5.94 (1.77)	14	6.07 (2.20)	16	7.00 (1.46)		$F_{O \times ST}(2,92) = .43$
			E	16	5.50 (1.51)	14	6.36 (1.95)	16	7.19 (.98)		$F_{Gr \times ST \times Ex}(2,92) = .11$
		SC	P	16	6.19 (1.72)	14	6.14 (2.21)	16	7.38 (.96)		$F_{O \times ST \times Ex}(2,92) = 2.31$
			E	16	5.19 (1.64)	14	5.50 (2.38)	16	7.25 (.93)		$F_{Gr \times O \times Ex}(2,92) = 1.39$
											$F_{Gr \times O \times ST}(2,92) = 1.47$
											$F_{Gr \times O \times ST \times Ex}(2,92) = .65$
											$F_{ST \times Ex}(1,93) = 3.59$
Health beliefs	1	PC	P	13	1.92 (1.04)	19	3.47 (3.06)	16	1.50 (1.51)	$F_{Gr}(2,92) = 19.06^{***}$	$F_{Gr \times Ex}(2,92) = 1.39$
			E	13	2.00 (1.15)	19	3.58 (3.02)	16	1.63 (1.50)	$F_{O}(1,93) = .62$	$F_{Gr \times ST}(2,92) = .19$
		SC	P	13	2.23 (1.36)	19	3.32 (2.85)	16	1.69 (1.49)	$F_{Ex}(1,93) = 3.85$	$F_{Gr \times O}(2,92) = 2.20$
			E	13	2.00 (1.35)	19	3.42 (2.91)	16	1.69 (1.49)	$F_{ST}(1,93) = .43$	$F_{O \times Ex}(2,92) = 1.87$
	2	PC	P	16	2.06 (.93)	14	4.57 (2.71)	16	1.13 (.34)		$F_{O \times ST}(2,92) = .00$
			E	16	2.06 (.85)	14	4.93 (2.70)	16	1.19 (.40)		$F_{Gr \times ST \times Ex}(2,92) = .02$
		SC	P	16	2.00 (.89)	14	4.79 (2.78)	16	1.00 (.00)		$F_{O \times ST \times Ex}(2,92) = 2.18$
			E	16	2.19 (1.17)	14	5.07 (2.79)	16	1.13 (.34)		$F_{Gr \times O \times Ex}(2,92) = .31$
											$F_{Gr \times O \times ST}(2,92) = 2.08$
											$F_{Gr \times O \times ST \times Ex}(2,92) = 1.38$
											$F_{ST \times Ex}(1,93) = .38$
Health worries	1	PC	P	13	3.62 (1.50)	19	4.00 (2.29)	16	1.63 (.81)	$F_{Gr}(2,92) = 27.04^{***}$	$F_{Gr \times Ex}(2,92) = .96$
			E	13	3.38 (1.56)	19	3.63 (2.33)	16	1.56 (.81)	$F_{O}(1,93) = 4.30^{*}$	$F_{Gr \times ST}(2,92) = 1.01$
		SC	P	13	3.00 (1.68)	19	3.63 (2.45)	16	1.56 (.73)	$F_{Ex}(1,93) = 2.07$	$F_{Gr \times O}(2,92) = 3.11^{*}$
			E	13	2.69 (1.65)	19	3.47 (2.41)	16	1.56 (.63)	$F_{ST}(1,93) = 3.06$	$F_{O \times Ex}(2,92) = 1.06$
	2	PC	P	16	3.44 (2.00)	14	5.86 (2.80)	16	1.44 (.63)		$F_{O \times ST}(2,92) = 2.39$

	E	16	3.88 (2.00)	14	5.36 (3.13)	16	1.44 (.63)	$F_{Gr\ x\ ST\ x\ Ex}(2,92) = 2.13$
SC	P	16	3.63 (1.86)	14	5.64 (2.62)	16	1.44 (.51)	$F_{O\ x\ ST\ x\ Ex}(2,92) = .12$
	E	16	3.56 (1.97)	14	5.64 (2.47)	16	1.38 (.50)	$F_{Gr\ x\ O\ x\ Ex}(2,92) = .94$
								$F_{Gr\ x\ O\ x\ ST}(2,92) = .78$
								$F_{Gr\ x\ O\ x\ ST\ x\ Ex}(2,92) = .66$
								$F_{ST\ x\ Ex}(1,93) = .03$

Note. ST = stimulus type; Ex = exposure; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; P = pre-exposure; E = exposure period; O = Order of stimulus presentation; *** $p < .001$; ** $p < .01$; * $p < .05$

With respect to state symptom impairment, a chronological order x stimulus type x exposure interaction was found, $F(1,93) = 4.49$, $p = .037$, $\eta^2 = .05$. Further exploration revealed a stimulus type x exposure interaction within chronological order “PC-SC”, $F(1,93) = 11.66$, $p < .001$, $\eta^2 = .21$, but not within chronological order “SC-PC”, $F(1,93) = .04$, $p = .683$. Within “PC-SC”, impairment levels before and after health-related stressor exposure did not differ $T(47) = 1.09$, $p = .280$. Contrarily, disability levels after social stressor exposure were higher compared to pre-exposure, $T(47) = -2.89$, $p = .006$, $r = -.39$. Concerning health worries, a main effect of chronological order was found, $F(1,93) = 4.30$, $p = .041$, $\eta^2 = .05$, outlining higher health worry levels when participants were initially exposed with the social conflict, $p = .041$. Further, a group x chronological order interaction effect was found, $F(2,92) = 3.11$, $p = .049$, $\eta^2 = .07$. Group differences with respect to health worries could be found both when participants were either intially exposed with health-related, $p = .002$, and social stressors, $p < .001$. Only within patients with SSD-MES, health worry levels between both conditions differed significantly. Health worries within SSD-MES were signficantly higher when participants were initially exposed with social stressors in comparison with the other condition, $p = .015$.

A-1.15 Group differences in physiological measures (with order as additional between subjects factor) – study 1

Variable	Order	ST	Ex	SSD-MUS		SSD-MES		HC		Main effects	Interaction effects
				N	*M (SD)*	*N*	*M (SD)*	*N*	*M (SD)*		
Heart rate	1	PC	P	13	71.48 (10.75)	19	74.73 (13.57)	16	67.20 (8.68)	$F_{Gr}(2,92) = 4.15$	$F_{Gr \times Ex}(2,92) = .16$
			E	13	73.80 (10.13)	19	77.43 (14.56)	16	71.61 (11.17)	$F_{O}(1,93) = .21$	$F_{Gr \times ST}(2,92) = .00$
		SC	P	13	72.81 (11.25)	19	76.81 (12.55)	16	68.51 (9.25)	$F_{Ex}(1,93) = 26.73$***	$F_{Gr \times O}(2,92) = 2.59$
			E	13	74.71 (11.99)	19	77.72 (14.27)	16	72.26 (9.79)	$F_{ST}(1,93) = 4.82$*	$F_{O \times Ex}(2,92) = 2.70$
	2	PC	P	16	81.90 (14.29)	14	71.67 (11.60)	16	67.92 (11.70)		$F_{O \times ST}(2,92) = 3.59$
			E	16	82.90 (15.31)	14	73.46 (10.36)	16	68.28 (11.72)		$F_{Gr \times ST \times Ex}(2,92) = .31$
		SC	P	16	81.42 (14.74)	14	71.47 (13.36)	16	67.85 (12.99)		$F_{O \times ST \times Ex}(2,92) = 2.30$
			E	16	83.56 (14.13)	14	73.68 (11.42)	16	68.64 (11.35)		$F_{Gr \times O \times Ex}(2,92) = 2.15$
											$F_{Gr \times O \times ST}(2,92) = .04$
											$F_{Gr \times O \times ST \times Ex}(2,92) = .10$
											$F_{ST \times Ex}(1,93) = .08$
RMSSD	1	PC	P	13	2.35 (1.52)	19	2.49 (1.13)	16	3.53 (1.36)	$F_{Gr}(2,92) = 2.30$	$F_{Gr \times Ex}(2,92) = 3.04$
			E	13	2.63 (1.61)	19	2.60 (1.21)	16	3.18 (1.46)	$F_{O}(1,93) = 3.35$	$F_{Gr \times ST}(2,92) = .09$
		SC	P	13	2.45 (1.78)	19	2.39 (1.10)	16	3.28 (1.56)	$F_{Ex}(1,93) = 3.25$	$F_{Gr \times O}(2,92) = .43$
			E	13	2.24 (1.26)	19	2.62 (1.09)	16	3.27 (1.45)	$F_{ST}(1,93) = .05$	$F_{O \times Ex}(2,92) = 3.61$
	2	PC	P	16	2.03 (1.20)	14	2.24 (1.10)	16	2.82 (1.73)		$F_{O \times ST}(2,92) = 3.04$
			E	16	2.01 (1.00)	14	2.10 (1.20)	16	2.10 (1.41)		$F_{Gr \times ST \times Ex}(2,92) = 2.65$
		SC	P	16	2.29 (1.36)	14	2.31 (1.45)	16	2.74 (1.68)		$F_{O \times ST \times Ex}(2,92) = .10$
			E	16	2.22 (1.19)	14	2.09 (1.05)	16	2.32 (1.64)		$F_{Gr \times O \times Ex}(2,92) = .47$
											$F_{Gr \times O \times ST}(2,92) = .65$
											$F_{Gr \times O \times ST \times Ex}(2,92) = .85$
											$F_{ST \times Ex}(1,93) = .06$
SDNN	1	PC	P	13	2.36 (1.14)	19	2.67 (1.29)	16	3.35 (1.41)	$F_{Gr}(2,92) = .88$	$F_{Gr \times Ex}(2,92) = 3.32$*
			E	13	2.93 (1.70)	19	3.21 (2.15)	16	3.61 (1.82)	$F_{O}(1,93) = 1,77$	$F_{Gr \times ST}(2,92) = .04$
		SC	P	13	2.45 (1.41)	19	2.94 (1.53)	16	3.26 (1.21)	$F_{Ex}(1,93) = .82$	$F_{Gr \times O}(2,92) = .67$
			E	13	2.65 (1.18)	19	3.23 (1.57)	16	3.50 (1.41)	$F_{ST}(1,93) = .69$	$F_{O \times Ex}(2,92) = 5.53$*
	2	PC	P	16	2.61 (1.61)	14	2.46 (1.54)	16	2.96 (1.77)		$F_{O \times ST}(2,92) = .99$
			E	16	2.53 (1.18)	14	2.53 (1.45)	16	2.18 (1.36)		$F_{Gr \times ST \times Ex}(2,92) =$
		SC	P	16	2.50 (1.39)	14	2.39 (1.28)	16	3.34 (2.60)		$F_{O \times ST \times Ex}(2,92) = 1.59$

	E	16	3.08 (1.88)	14	2.54 (1.53)	16	2.46 (1.62)

$F_{Gr\ x\ O\ x\ Ex}(2,92) = 1.86$
$F_{Gr\ x\ O\ x\ ST}(2,92) = .93$
$F_{Gr\ x\ O\ x\ ST\ x\ Ex}(2,92) = .88$
$F_{ST\ x\ Ex}(1,93) = .00$

Note. ST = stimulus type; Ex = exposure; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; P = pre-exposure; E = exposure period; O = Order of stimulus presentation; *** $p < .001$; ** $p < .01$; * $p < .05$

With respect to SDNN, a chronological order x exposure interaction was found, $F(1,93) = 5.53$, $p = .021$, $\eta^2 = .06$. In chronological order "PC-SC" SDNN levels increased during exposure compared to pre-exposure, $T(47) = -2.25$, $p = .029$, $r = .-32$. Contrarily, within chronological order "SC-PC" SDNN levels during exposure and pre-exposure did not differ, $T(45) = 1.07$, $p = .289$.

A-1.16 Kolmogorov-Smirnov-tests of normal distribution, Levene´s-tests of variance homogeniety and Box-tests of equality of covariance matrices with respect to state emotion regulation – additional analyses

Variable	Kolmogorov-Smirnov-test				Levene´s test				Box-test				
	N	*Z*	*df*	*p*	*F*	*df1*	*df2*	*p*	*M*	*F*	*df1*	*df2*	*p*
PC_reappr	93				1.18	2	90	.314	347.37	1.28	210	20550.10	.004
MUS	28	.21	28	.002									
MES	33	.30	33	< .001									
HC	32	.21	32	.001									
SC_reappr	93				3.61	2	90	.031					
MUS	28	.29	28	< .001									
MES	33	.32	33	< .001									
HC	32	.23	32	< .001									
PC_acc	93				1.95	2	90	.149					
MUS	28	.21	29	.002									
MES	33	.18	33	.007									
HC	32	.18	32	.008									
SC_acc	93				.99	2	90	.376					
MUS	28	.20	29	.005									
MES	33	.23	33	< .001									
HC	32	.20	32	.003									
PC_probl	93				4.26	2	90	.017					
MUS	28	.24	28	< .001									
MES	33	.21	33	.001									
HC	32	.20	32	.003									
SC_probl	93				2.92	2	90	.059					
MUS	28	.16	28	.073									
MES	33	.18	33	.008									
HC	32	.27	32	< .001									
PC_rum	93				.14	2	90	.870					
MUS	28	.20	29	.005									

MES	33	.24	33	< .001			
HC	32	.23	32	< .001			
SC_rum	93				.86	2	90	.427
MUS	28	.26	29	< .001			
MES	33	.23	33	< .001			
HC	32	.20	32	.002			
PC_av	93				1.45	2	90	.241
MUS	28	.22	28	.001			
MES	33	.24	33	<.001			
HC	32	.21	32	.001			
SC_av	93				1.29	2	90	.281
MUS	28	.21	28	.003			
MES	33	.18	33	.008			
HC	32	.24	32	< .001			
PC_exp.sup.	93				1.13	2	90	.329
MUS	28	.24	29	< .001			
MES	33	.22	33	< .001			
HC	32	.23	32	< .001			
SC_exp.sup.	93				.05	2	90	.953
MUS	28	.23	29	< .001			
MES	33	.19	33	.005			
HC	32	.22	32	< .001			
PC_expr.sup.	93				.17	2	90	.847
MUS	28	.21	29	.002			
MES	33	.29	33	< .001			
HC	32	.20	32	.002			
SC_expr.sup.	93				.00	2	90	.995
MUS	28	.22	29	.001			
MES	33	.24	33	< .001			
HC	32	.26	32	< .001			

Note. PC_reappr = reappraisal use after physical complaint exposure; SC_reappr = reappraisal use after social conflict exposure; PC_acc = acceptance use after physical complaint exposure; SC_acc = acceptance use after social conflict exposure; PC_probl = use of problem solving after

physical complaint exposure; SC_probl = use of problem solving after social conflict exposure; PC_rum = rumination use after physical complaint exposure; SC_rum = rumination use after social conflict exposure; PC_av = avoidance use after physical complaint exposure; SC_av = avoidance use after social conflict exposure; SC_exp.sup. = use of experience suppression after physical complaint exposure; SC_exp.sup. = use of experience suppression after social conflict exposure; PC_expr.sup. = use of expression suppression after physical complaint exposure; PC_expr.supr. = use of expression suppression after social conflict exposure.

A-1.17 Group differences in state emotion regulation (with order as additional between subjects factor) – additional analyses

									Multivariate ME	Multivariate IE
									λ_{Gr} = .74, F(14,162) = 1.86*	$\lambda_{Gr\,x\,O}$ = .88, F(14,162) = 0.78
									λ_{O} = .90, F(7,81) = 1.23	$\lambda_{Gr\,x\,ST}$ = .83, F(14,162) = 1.13
									λ_{ST} = .92, F(7,81) = 1.08	$\lambda_{ST\,x\,O}$ = .89, F(7,81) = 1.45
										$\lambda_{Gr\,x\,ST\,x\,O}$ = .81, F(14,162) = 1.31

Variable	Order	ST	SSD-MUS		SSD-MES		HC		Univariate ME
			N	*M (SD)*	*N*	*M (SD)*	*N*	*M (SD)*	
Reappr	1	PC	12	2.17 (.94)	19	2.16 (.83)	16	2.13 (1.09)	F_{Gr}(2,91) = .13
		SC	12	2.33 (1.15)	19	2.21 (.98)	16	3.00 (1.37)	
	2	PC	16	2.69 (.95)	14	2.57 (1.16)	16	2.38 (1.31)	
		SC	16	2.56 (1.31)	14	2.50 (1.16)	16	2.44 (1.36)	
Acceptance	1	PC	12	2.83 (.94)	19	2.84 (1.42)	16	2.81 (1.38)	F_{Gr}(2,91) = .11
		SC	12	2.83 (1.19)	19	2.58 (1.12)	16	2.81 (1.38)	
	2	PC	16	3.31 (1.25)	14	3.07 (1.21)	16	2.94 (1.57)	
		SC	16	3.19 (1.05)	14	3.36 (1.08)	16	3.06 (1.29)	
Probl	1	PC	12	2.92 (.79)	19	2.58 (1.30)	16	2.81 (1.38)	F_{Gr}(2,91) = .28
		SC	12	2.75 (1.06)	19	2.84 (1.07)	16	2.88 (1.45)	
	2	PC	16	3.13 (1.20)	14	3.21 (1.19)	16	2.63 (1.45)	
		SC	16	3.19 (1.28)	14	2.93 (1.21)	16	2.81 (1.33)	
Rumination	1	PC	12	3.00 (1.35)	19	2.53 (1.22)	16	2.19 (1.17)	F_{Gr}(2,91) = 4.59*
		SC	12	3.17 (1.40)	19	2.84 (1.21)	16	2.44 (1.21)	
	2	PC	16	3.44 (1.03)	14	2.79 (1.25)	16	2.44 (1.26)	
		SC	16	3.63 (.96)	14	3.29 (.99)	16	2.88 (1.31)	

Avoidance	1	PC	12	2.33 (1.07)	19	2.58 (1.22)	16	2.00 (.97)	$F_{Gr}(2,91) = 1.57$
		SC	12	2.25 (1.36)	19	2.37 (1.07)	16	1.69 (.79)	
	2	PC	16	2.63 (1.02)	14	2.79 (1.48)	16	2.50 (1.41)	
		SC	16	3.19 (1.22)	14	2.43 (1.28)	16	2.50 (1.32)	
Exp.supp.	1	PC	12	2.58 (1.00)	19	2.42 (1.35)	16	1.88 (.81)	$F_{Gr}(2,91) = .48$
		SC	12	1.75 (1.06)	19	2.58 (.96)	16	1.94 (.93)	
	2	PC	16	2.19 (1.11)	14	2.29 (1.27)	16	2.50 (1.63)	
		SC	16	2.44 (1.15)	14	2.43 (1.28)	16	2.44 (1.31)	
Expr.supp.	1	PC	12	2.67 (1.30)	19	2.95 (1.39)	16	2.44 (1.21)	$F_{Gr}(2,91) = 2.18$
		SC	12	1.75 (1.06)	19	2.95 (1.22)	16	2.44 (1.26)	
	2	PC	16	2.69 (1.20)	14	3.14 (1.10)	16	2.67 (1.25)	
		SC	16	3.06 (1.24)	14	3.00 (1.47)	16	2.89 (1.32)	

Note. Reappr = reappraisal; Probl = Problem solving; Exp.supp. = Experience suppression; Expr.supp. = Expression suppression; ST = stressor type; Multivariate ME = Multivariate main effects; Multivariate IE = Multivariate interaction effects; Univariate ME = Univariate main effects; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; O = Order of stimulus presentation; *** $p < .001$; ** $p < .01$; * $p < .05$

A-1.18 Group differences with respect to subjective reduction of negative emotion after state ER strategy use (with order as further between subjects factor) – additional analyses

	Multivariate IE
	$\lambda_{Gr \times O}$ = .80, F(14,162) = 1.37
	$\lambda_{Gr \times ST}$ = .84, F(14,162) = 1.07
	$\lambda_{ST \times O}$ = .89, F(7,81) = 1.38
	$\lambda_{Gr \times ST \times O}$ = .92, F(14,162) = 0.49

Variable	Order	ST	SSD-MUS		SSD-MES		HC	
			N	*M (SD)*	*N*	*M (SD)*	*N*	*M (SD)*
Reappr	1	PC	12	1.83 (.72)	19	1.68 (1.06)	16	2.50 (1.32)
		SC	12	1.67 (.89)	19	1.94 (1.13)	16	2.63 (1.31)
	2	PC	16	2.19 (1.22)	14	2.00 (1.04)	16	2.25 (1.29)
		SC	16	2.50 (1.21)	14	2.29 (1.20)	16	2.25 (1.06)
Acceptance	1	PC	12	1.92 (.79)	19	1.95 (1.03)	16	2.44 (1.26)
		SC	12	1.75 (.75)	19	1.84 (1.01)	16	2.44 (1.21)
	2	PC	16	2.13 (1.09)	14	1.86 (1.03)	16	2.31 (1.14)
		SC	16	2.31 (.95)	14	2.07 (1.07)	16	2.50 (1.26)
Probl	1	PC	12	2.17 (.83)	19	2.32 (1.11)	16	2.56 (1.31)
		SC	12	1.92 (1.00)	19	2.37 (1.30)	16	2.75 (1.34)
	2	PC	16	2.50 (1.32)	14	2.14 (1.03)	16	2.50 (1.46)
		SC	16	2.56 (1.21)	14	1.93 (1.07)	16	2.38 (1.15)
Rumination	1	PC	12	1.92 (1.08)	19	2.37 (.90)	16	2.50 (1.26)
		SC	12	1.83 (1.03)	19	2.11 (1.05)	16	2.00 (.89)
	2	PC	16	2.13 (.96)	14	1.71 (.99)	16	2.06 (1.29)
		SC	16	2.63 (1.20)	14	2.14 (1.10)	16	2.25 (.77)

Avoidance	1	PC	12	1.92 (.90)	19	1.74 (.99)	16	2.19 (1.17)
		SC	12	1.67 (.78)	19	2.00 (1.11)	16	2.00 (1.10)
	2	PC	16	2.25 (1.13)	14	1.93 (1.14)	16	2.75 (1.48)
		SC	16	2.25 (1.00)	14	2.21 (1.19)	16	2.25 (1.18)
Exp.supp.	1	PC	12	2.25 (1.14)	19	1.95 (1.31)	16	1.75 (.86)
		SC	12	1.92 (.79)	19	1.84 (1.01)	16	2.00 (1.10)
	2	PC	16	2.19 (1.22)	14	1.93 (1.00)	16	2.19 (1.52)
		SC	16	2.19 (1.05)	14	2.14 (1.10)	16	1.94 (1.00)
Expr.supp.	1	PC	12	2.08 (.79)	19	1.74 (1.19)	16	2.00 (1.10)
		SC	12	1.58 (.79)	19	1.53 (.90)	16	1.88 (1.02)
	2	PC	16	2.44 (1.21)	14	1.93 (1.14)	16	2.38 (1.50)
		SC	16	2.06 (.93)	14	2.00 (.96)	16	2.19 (.91)

Note. Reappr = reappraisal; Probl = Problem solving; Exp.supp. = Experience suppression; Expr.supp. = Expression suppression; ST = stressor type; Multivariate ME = Multivariate main effects; Multivariate IE = Multivariate interaction effects; Univariate ME = Univariate main effects; Gr = group; PC = physical complaint stressor; SC= social conflict stressor; O = Order of stimulus presentation; *** $p < .001$; ** $p < .01$; * $p < .05$