

**Psychosocial Determinants and Interventions in the
Context of Stress and Cardiovascular Health – Focus on
Stress Contagion, Appreciation, Anger, and Cognitive-
Behavioral Stress Management Trainings**

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To my family

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TABLE OF CONTENTS

SUMMARY.....	XVI
ZUSAMMENFASSUNG	XVIII
GENERAL INTRODUCTION	1
Stress.....	2
<i>Concepts and Definition of Stress</i>	<i>3</i>
<i>The Physiological Stress Response</i>	<i>6</i>
<i>Relevance and Implications of the Stress Response.....</i>	<i>11</i>
Stress and Cardiovascular Health	12
<i>Hypertension</i>	<i>13</i>
<i>Coronary Heart Disease</i>	<i>17</i>
<i>Stress as a Risk Factor for Hypertension and Coronary Heart Disease</i>	<i>24</i>
Psychosocial Determinants of Stress and Cardiovascular Health	25
<i>Stress Contagion.....</i>	<i>28</i>
<i>Appreciation</i>	<i>31</i>
<i>Anger</i>	<i>35</i>
<i>Relevance and Implications of Psychosocial Determinants of Stress and Cardiovascular Health.....</i>	<i>39</i>
Interventions in the Context of Stress and Cardiovascular Health	39
<i>Cognitive-Behavioral Stress Management Trainings</i>	<i>42</i>
SUMMARY OF THEORETICAL BACKGROUND, AIMS, AND HYPOTHESES.....	45
SUMMARY OF RESEARCH PAPERS	53
Research Paper 1	53
Research Paper 2.....	55
Research Paper 3.....	57
Research Paper 4.....	59
GENERAL DISCUSSION.....	61
Summary and Discussion of the Study Findings and Designs	61
Relevance and Implications of the Findings.....	67
Concluding Remarks and Directions for Future Research	71
LIST OF AUTHOR CONTRIBUTIONS.....	78
REFERENCES.....	80
APPENDIX A	123
APPENDIX B.....	175
APPENDIX C	211
APPENDIX D	251

LIST OF FIGURES

Figure 1. Simplified Framework of Psychosocial Determinants, Including Risk Factors and Resources, and Interventions in the Context of Stress and Cardiovascular Health Underlying the Present Thesis	47

LIST OF ABBREVIATIONS

A		G	
ACE	Angiotensin-converting enzyme	GR	Glucocorticoid receptor
ACS	Acute coronary syndrome	H	
ACTH	Adrenocorticotrophic hormone	HbA1c	glycated hemoglobin A1c
ANG	Angiotensin	HDL-C	High-density lipoprotein-cholesterol
APA	American Psychological Association	HPA	Hypothalamic-pituitary-adrenal
AT	Alternative control training	HR	Heart rate
AVP	Arginine vasopressin	I	
B		IL	Interleukin
BMI	Body mass index	IMT	Intima-media thickness
BP	Blood pressure	J	
C		JDC	Job demand-control
CCS	Chronic coronary syndrome	JD-R	Job demand-resources
CHD	Coronary heart disease	L	
CI	Confidence interval	LDL-C	Low-density lipoprotein-cholesterol
CRH	Corticotropin-releasing hormone	LL	Lower limit
CRP	C-reactive protein	M	
CVD	Cardiovascular disease	MAP	Mean arterial pressure
D		MI	Myocardial infarction
DBP	Diastolic blood pressure	MONICA	Monitoring of Trends and Determinants in Cardiovascular Disease
E		MR	Mineralocorticoid receptor
ECG	Electrocardiogram	N	
EHT	Essential hypertension	NMEC	Neurocognitive Model of Emotional Contagion
EP	Epinephrine	NEP	Norepinephrine
ERI	effort-reward imbalance		

O	
obsTSST	Observation TSST
obsPlacTSST	Observation placebo TSST
P	
PAM	Perception-Action Model for Empathy
PVN	Paraventricular nucleus
R	
RAAS	Renin-angiotensin-aldosterone system
S	
sAA	Salivary α -amylase
SAM	Sympathetic-adrenal-medullary
SBP	Systolic blood pressure
SIT	Stress Inoculation Training
SMT	Stress management training
SNS	Sympathetic nervous system
SOS	Stress-as-offense-to-self
T	
TC	Total cholesterol
TNF- α	Tumor necrosis factor- α
TSST	Trier Social Stress Test
U	
UL	Upper limits
W	
WHO	World Health Organization

SUMMARY

Tackling the largely preventable global burden of cardiovascular disease, particularly coronary heart disease (CHD), is a major challenge for the future. Hypertension is a major risk factor for CHD and therefore contributes substantially to the global burden of disease. To identify individuals at risk and to guide the development and implementation of interventions, a comprehensive understanding of health determinants is essential. Psychosocial determinants have been found to play an important role in the context of cardiovascular health, specifically with regard to hypertension and CHD. Among the psychosocial determinants, stress has emerged as a major health concern. Notably, stress is not only a psychosocial risk factor for adverse health consequences but is also influenced by various other psychosocial determinants. Given this, a comprehensive understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health is critical to effectively address the global burden of disease. Building on existing research, this thesis aimed to enhance the understanding of psychosocial determinants (research papers 1, 2, and 3) and interventions (research paper 4) in the context of stress, essential hypertension (EHT), and CHD.

In light of emerging evidence that the social environment may be an indirect source of stress in terms of physiological stress contagion, research paper 1 introduced a standardized, randomized-controlled paradigm to investigate stress contagion based on direct stress observation. Physiological stress contagion responses to stress observation compared to placebo-stress observation were found with regard to the sympathetic-adrenal-medullary axis and the hypothalamic-pituitary-adrenal axis in healthy young men. The physiological reactivity kinetics to stress observation were similar but less pronounced compared to direct stress exposure. Shifting the focus to a largely neglected resource, research paper 2 assessed appreciation at work and major biological CHD risk factors in a sample of apparently healthy, medication-free men in the normotensive to hypertensive range and medicated hypertensive and CHD patients. Appreciation at work was independently associated with lower biological

CHD risk, indicating that appreciation at work might be an independent health-promoting resource. Given the numerous but inconsistent findings on anger in EHT, research paper 3 investigated trait anger in apparently healthy, medication-free men in the normotensive to hypertensive range in a social context by considering anger recognition of mixed facial affect. With increasing blood pressure, men with EHT displayed an anger recognition bias in terms of overrating anger in facial expressions of mixed emotions compared to normotensive men. Moreover, higher trait anger predicted increases in blood pressure over time only in combination with an anger recognition bias, but none of them alone. This indicates that trait anger requires situational features to be elicited and to contribute to EHT. In the context of interventions, research paper 4 confirmed the beneficial effects of occupational cognitive-behavioral stress management training (SMT) compared to alternative control training on stress-related, work-related, and specific-context-related outcome measures in male and female employees. Focusing on the hitherto unclear mechanisms of change, SMT-induced improvements in perceived mastery of changing stress-relevant cognitions emerged as the most important mediator of the beneficial SMT effects and thus as a crucial stress management skill, highlighting the importance of training cognitive techniques.

Altogether, these findings further strengthen the important role of psychosocial determinants in the context of stress and cardiovascular health. In light of this, health care and clinical practices should move beyond the traditional biomedical approach and consider biological, psychological, and social aspects following a biopsychosocial, patient-centered approach. The preventive potential of interventions such as SMTs highlights the importance of preventive and health-promoting health services beyond clinical and curative health care in addressing the global burden of disease. Future research is needed to comprehensively understand the complex intertwined effects of health determinants and interventions to tackle the global burden of disease most effectively.

ZUSAMMENFASSUNG

Die Bewältigung der weitgehend vermeidbaren weltweiten Belastung durch Herz-Kreislauf-Erkrankungen, insbesondere durch koronare Herzerkrankungen (KHK), stellt eine große Herausforderung dar. Bluthochdruck ist ein wichtiger Risikofaktor für KHK und trägt daher wesentlich zur globalen Krankheitslast bei. Um Risikopersonen zu identifizieren und Interventionen zu steuern, ist ein umfassendes Verständnis von Gesundheitsdeterminanten erforderlich. Psychosoziale Determinanten spielen eine wichtige Rolle für die kardiovaskuläre Gesundheit, insbesondere bei Hypertonie und KHK. Unter den psychosozialen Determinanten hat sich Stress als eines der größten Gesundheitsrisiken erwiesen. Stress ist jedoch nicht nur ein psychosozialer Risikofaktor, sondern wird auch von zahlreichen anderen psychosozialen Determinanten beeinflusst. Daher ist ein umfassendes Verständnis von psychosozialen Determinanten und Interventionen im Rahmen von Stress und kardiovaskulärer Gesundheit entscheidend, um die globale Krankheitslast nachhaltig anzugehen. Das Ziel dieser Arbeit war es, das Verständnis von psychosozialen Determinanten (Research Papers 1, 2 und 3) und Interventionen (Research Paper 4) im Zusammenhang mit Stress, essentieller Hypertonie (EHT) und KHK zu vertiefen.

Ausgehend von Hinweisen, dass das soziale Umfeld eine indirekte Stressquelle im Sinne einer Stressübertragung sein kann, wurde in Research Paper 1 ein standardisiertes, randomisiert-kontrolliertes Paradigma zur Untersuchung von Stressübertragung eingeführt. Gesunde junge Männer zeigten physiologische Reaktionen der Sympathikus-Nebennierenmark-Achse und der Hypothalamus-Hypophysen-Nebennierenrinden-Achse auf Stressbeobachtung im Vergleich zur Placebo-Stressbeobachtung. Die physiologische Reaktivitätskinetik auf Stressbeobachtung war ähnlich, aber weniger ausgeprägt als bei direkter Stressbelastung. Research Paper 2 untersuchte eine bisher vernachlässigte Ressource, Wertschätzung am Arbeitsplatz, im Kontext von KHK bei ansonsten gesunden Männern mit Blutdruckwerten im Bereich von normotensiv bis hypertensiv sowie bei Patienten mit

Hypertonie und KHK. Da höhere Wertschätzung am Arbeitsplatz unabhängig mit einem geringeren biologischen KHK-Risiko assoziiert war, könnte sie eine unabhängige gesundheitsfördernde Ressource darstellen. Auf Grund zahlreicher, aber widersprüchlicher Befunde zu Ärger im Zusammenhang mit EHT untersuchte Research Paper 3 Eigenschaftsärger bei ansonsten gesunden Männern mit Blutdruckwerten im Bereich von normotensiv bis hypertensiv in einem sozialen Kontext, unter Berücksichtigung der Erkennung von Ärger in gemischt-emotionalen Gesichtsausdrücken. Mit steigenden Blutdruckwerten zeigten Hypertoniker im Vergleich zu Normotonikern eine höhere Neigung Ärger zu erkennen. Zudem sagte höherer Eigenschaftsärger nur in Kombination mit einer Überschätzung von Ärger Blutdruckanstiege über die Zeit vorher. Dies deutet darauf hin, dass Eigenschaftsärger situative Merkmale erfordert, um ausgelöst zu werden und um zu EHT beizutragen. Research Paper 4 bestätigte die positiven Effekte von kognitiv-behavioralen Stressbewältigungstrainings (SMTs) im Vergleich zu alternativen Kontrolltrainings auf stress-, arbeits- und kontextbezogene Variablen bei männlichen und weiblichen Arbeitnehmenden. Bezüglich bislang unklarer Veränderungsmechanismen erwies sich die wahrgenommene Fähigkeit zur Veränderung stressrelevanter Kognitionen als wichtigster Mediator der positiven SMT-Effekte und damit als zentrale Stressbewältigungsfähigkeit, was die Wichtigkeit kognitiver Techniken hervorhebt.

Insgesamt untermauern diese Ergebnisse die entscheidende Rolle psychosozialer Determinanten im Zusammenhang mit Stress und kardiovaskulärer Gesundheit. Daher sollten Gesundheitsversorgung und klinische Praxis über den traditionellen biomedizinischen Ansatz hinaus biologische, psychologische und soziale Aspekte im Rahmen eines biopsychosozialen, patientenzentrierten Ansatzes berücksichtigen. Das präventive Potenzial von Interventionen wie SMTs unterstreicht die Bedeutung präventiver und gesundheitsfördernder Leistungen über klinisch-kurative Gesundheitsleistungen hinaus. Weitere Forschung ist erforderlich, um die komplexen Auswirkungen von Gesundheitsdeterminanten und Interventionen umfassend zu verstehen und die globale Krankheitslast möglichst nachhaltig anzugehen.

GENERAL INTRODUCTION

Over the past decades, the global burden of disease has shifted from primarily infectious diseases to chronic noncommunicable diseases, driven by changing living conditions (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018; World Health Organization [WHO], 2009). Cardiovascular disease (CVD), most notably coronary heart disease (CHD), has become the leading cause of death and disability worldwide (Roth et al., 2020; Tsao et al., 2023; Vardas et al., 2022). As hypertension is a major risk factor for a variety of diseases, including CHD, it is another chronic noncommunicable condition that contributes substantially to the global burden of disease (Fuchs, 2017, Chapter 1; Liu et al., 2021; Victor, 2018). The evolving patterns in morbidity and mortality indicate that a considerable amount of today's global burden of disease is preventable (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018; Roth et al., 2020). In order to identify individuals at risk and to guide the development and implementation of interventions, a comprehensive understanding of the determinants of health is fundamental (Bonita et al., 2006, Chapter 10; Grundy, 1999; Ridker et al., 2018; WHO, 2009). From the old proverb “*Mens sana, in corpore sano*” (English: “A healthy mind in a healthy body”) to recent concepts of mind-heart-body connections (e.g., Labarthe et al., 2016; Levine et al., 2021), potential associations between psychosocial factors and physical health have been a topic of interest across time. Indeed, there is considerable evidence that psychosocial determinants play an important role in the context of cardiovascular health, particularly with regard to hypertension and CHD (e.g., Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Rozanski et al., 2005). Among the psychosocial determinants of health, stress has emerged as a major health concern due to its widespread prevalence (Daly & Macchia, 2023; Gallup, 2023a) and associated adverse health consequences (e.g., Brotman et al., 2007; Chida & Steptoe, 2010; Kivimäki & Steptoe, 2018; M.-Y. Liu et al., 2017). Remarkably, stress is not only a psychosocial risk factor. Like hypertension and CHD, stress is also influenced by various other psychosocial determinants (e.g., Chida & Hamer, 2008; Kiecolt-Glaser et al., 2020). In

light of this, a comprehensive understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health is critical to effectively address the global burden of disease.

Drawing on the current state of research, this thesis aimed to enhance the understanding of psychosocial determinants (research papers 1, 2, and 3) and interventions (research paper 4) in the context of stress, essential hypertension (EHT), and CHD based on four research papers. Research paper 1 (Auer, Walther, et al., 2024) investigated the social environment as an indirect source of stress in terms of physiological stress contagion in healthy participants, introducing and applying a standardized, randomized-controlled paradigm to study stress contagion based on direct stress observation. Research paper 2 (Auer, Semmer, et al., 2024) examined appreciation at work as a resource with regard to biological CHD risk. In the context of EHT, research paper 3 (Auer et al., 2022) investigated trait anger in a social context by considering anger recognition of mixed facial affect. Research paper 4 (Wirtz et al., 2024) examined the underlying mechanisms of occupational cognitive-behavioral stress management group trainings to reduce stress and the resulting burden, focusing on perceived mastery of functional stress management skills, particularly of changing cognitions.

The following outlines the theoretical background on stress and cardiovascular health (particularly hypertension and CHD) as well as psychosocial determinants and interventions in this context, which provides the theoretical basis for the four research papers. Afterward, the four research papers are briefly summarized, followed by a general discussion of the study findings and designs, implications, and directions for future research. The full-length research papers are provided in Appendices A–D.

Stress

Stress is a ubiquitous and inevitable part of our daily lives. In 2022, 40% of adults worldwide reported experiencing a lot of stress (Gallup, 2023a). Despite the widespread use of the term stress, Hans Selye (1973), known as the father of stress, rightly observed that

“everybody knows what stress is and nobody knows what it is” (p. 692). In general, stress is a natural human response to internal or external demands or threats that can affect mental and physical health and well-being (American Psychological Association [APA], n.d.; WHO, 2023). Based on the theoretical orientation to define stress, three stress concepts can be differentiated: stress as a response, stress as a stimulus, and stress as a transaction.

Concepts and Definition of Stress

The *response-based stress concept* focuses on the stress response. Based on Bernard’s (1865/1961) idea of the “milieu intérieur,” Cannon (1929, 1939) introduced the concept of homeostasis to describe self-regulating physiological processes that maintain internal stability, i.e., the physiological system within an optimal range, despite changing conditions. In addition, Cannon (1915) was the first to describe the fight-or-flight response, i.e., the acute physiological stress response that serves to prepare to deal with threats by mobilizing energy to maintain homeostasis (Cannon, 1929, 1939). Based on this, Selye (1936, 1956) defined stress as a nonspecific response of the body to any demand. Referred to as General Adaptation Syndrome, this nonspecific physiological response is characterized by the three stages of alarm, resistance, and exhaustion and thus considers potential health impairments due to severe, prolonged stress responses (Selye, 1936, 1950). Although Selye’s definition and concept of stress have remained partially controversial, particularly concerning the non-specificity of the stress response (e.g., Lazarus & Folkman, 1984; Mason, 1968, 1971, 1975), Selye’s understanding of stress continues to have a significant impact on our understanding of stress today.

Rather than focusing on the stress response, the less influential *stimulus-based stress concept* takes a closer look at the triggers of a stress response, i.e., the stressors. Particular attention is paid to (critical) life events (e.g., divorce, relocation, and loss of loved ones) that require major adaptations (Holmes & Rahe, 1967; Sarason et al., 1978). Daily hassles or micro-stressors (e.g., traffic jams and neighborhood disputes), which are important because of their

frequent occurrence in everyday life, are also considered (DeLongis et al., 1982; Kanner et al., 1981).

The most influential psychological stress model, namely the transactional model of stress (Lazarus, 1966; Lazarus & Folkman, 1984), combines response- and stimulus-oriented perspectives in a *transactional-based stress concept*. According to this model, stress results from a perceived imbalance between situational demands and personal resources based on cognitive appraisal processes (Lazarus, 1966; Lazarus & Folkman, 1984). More specifically, these cognitive appraisal processes address, on the one hand, the harm, threat, and challenge of a situation (primary appraisal) and, on the other hand, the person's coping options and resources (secondary appraisal). A stress response is elicited when a situation is perceived as threatening, challenging, and/or harmful, and the available coping resources are perceived as insufficient. In the case of a stress response, the success of the coping effort initiated is evaluated (reappraisal) to ensure a dynamic adaptation of cognitive appraisal processes with regard to future situations. Thus, in this approach, stress does not depend on objective characteristics but rather on subjective appraisal and evaluation. This explains interindividual differences in stress experiences and physiological stress responses in similar situations (e.g., Gaab et al., 2005; Wirtz, Ehlert, et al., 2006; Wirtz et al., 2007).

Given the high prevalence of stress in the workplace (Gallup, 2023b; Leclerc et al., 2022), several models and theories that explicitly relate to work, occupational, or job stress have emerged. Among the most influential models are the job demand-control (JDC) model (Karasek, 1979) with its extension of the job demand-resources (JD-R) model (Demerouti et al., 2001) and the effort-reward imbalance (ERI) model (Siegrist, 1996). According to the *JDC model* (Karasek, 1979; Karasek & Theorell, 1990), job strain results from the interaction between job demands in terms of workload and job control in terms of decision latitude when job demands are high and job control is low. The *JD-R model* (Demerouti et al., 2001) extends the JDC model with respect to several types of job demands and, in particular, job resources

beyond job control depending on job characteristics. The *ERI model* (Siegrist, 1996) proposes that job stress results from a perceived imbalance between a person's effort and the reward offered for that effort, i.e., high effort and low reward lead to stress. Rewards include not only money but also esteem, career opportunities, and job security. Effort refers to extrinsic job demands and obligations. However, the ERI model also considers intrinsic aspects such as motivation. Given this, the ERI model takes the considerable social aspects of stress into account by focusing on social exchange and reciprocity. Another approach that considers the social aspects of stress is the *stress-as-offense-to-self (SOS) theory* (Semmer et al., 2007; Semmer et al., 2019). The SOS theory focuses on the self or self-esteem and proposes threats to the self as a major source of stress while boosts to self-esteem are considered important resources. A distinction can be made between personal self-esteem (self-evaluation) and social self-esteem (evaluation by others). Thus, threats to the self either result from not meeting one's personal standards (stress through insufficiency) or messages of disrespect from others, including illegitimate tasks (stress as disrespect). Regarding boosts to self-esteem, appreciation is considered a core resource.

These different definitions and models of stress reflect the complexity and multidimensional nature of stress. Overall, stress consists of stress-triggering stimuli or environmental conditions, i.e., stressors, on the one hand, and stress responses, on the other hand. Whether a stimulus or situation becomes a stressor and elicits a stress response depends on the stimulus itself as well as on a variety of influencing and interacting factors such as subjective appraisal and evaluation, prevailing conditions, situational characteristics, the social environment, and finally, whether the demands exceed available resources. Characteristics that have been proposed to have a high potency to induce a stress response are novelty, uncertainty, unpredictability, uncontrollability, or social-evaluative threat (Dickerson & Kemeny, 2004; Koolhaas et al., 2011; Mason, 1968). Stressors can generally be physical or physiological (e.g., noise, heat, or injury), psychological (e.g., time pressure, overload, failure, frustration,

interpersonal conflicts, or isolation), or mixed (Bali & Jaggi, 2015; Lovallo, 2015, Chapter 4; McEwen, 2009). In stress research, various acute, time-limited, laboratory stressor tasks are used to investigate acute stress under controlled conditions (Bali & Jaggi, 2015; Dickerson & Kemeny, 2004; Skoluda et al., 2015). The gold standard among these stressor tasks is the psychosocial stress induction procedure, the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). The TSST includes a preparation period followed by an audio- and video-taped free speech in the form of a mock job interview (five minutes) and a mental arithmetic task (five minutes) in front of an evaluating panel. Due to the social-evaluative components combined with uncontrollability, this procedure has been shown to reliably induce a comprehensive acute stress response (Allen et al., 2014; Dickerson & Kemeny, 2004; Skoluda et al., 2015). The stress response is a complex coordinated pattern of changes that includes physiological, psychological, and behavioral responses (Dickerson & Kemeny, 2004; Schlebusch, 2004; Skoluda et al., 2015).

The Physiological Stress Response

The physiological stress response is the result of multiple, interrelated processes involving the central nervous system, the autonomic nervous system, the endocrine system, and other physiological systems (e.g., Charmandari et al., 2005; Chrousos & Gold, 1992; Fink, 2010; Godoy et al., 2018). This includes the activation of the major endocrine stress axes, i.e., the sympathetic-adrenal-medullary (SAM) axis, the hypothalamic-pituitary-adrenal (HPA) axis, and the renin-angiotensin-aldosterone system (RAAS).

The complex physiological stress response cascade starts with the perception of a stressful stimulus or situation in the brain (Ulrich-Lai & Herman, 2009). Depending on whether the stressor is physical or psychological, different neuronal circuitries, which partially overlap, are involved in stressor processing (Godoy et al., 2018; Ulrich-Lai & Herman, 2009). In either case, the hypothalamus, particularly the paraventricular nucleus (PVN) of the hypothalamus, plays an essential role in regulating the physiological stress response (Buijs & van Eden, 2000;

Herman et al., 2003). Depending on the stressor, the PVN receives direct projections from the brainstem and/or relayed input from limbic forebrain structures, including the amygdala, the hippocampus, and the prefrontal cortex (Buijs & van Eden, 2000; Herman et al., 2003). In turn, the PVN has substantial projections to several autonomic nuclei, including the sympathetic nuclei of the locus coeruleus, the ventrolateral medulla, and the nucleus of the solitary tract (Ulrich-Lai & Herman, 2009). In addition, the PVN secretes hormones that contribute to the regulation of the physiological stress response, such as corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP) (Ulrich-Lai & Herman, 2009). Thus, the PVN is involved in the regulation of autonomic responses to stressors, including the SAM axis, and the responses of the HPA axis to stressors.

Sympathetic-Adrenal-Medullary Axis. The SAM axis comprises the sympathetic nervous system (SNS) and the adrenal medulla (Godoy et al., 2018; Palkovits, 2009). Upon stimulation, SNS activation is characterized by sympathetic nerve endings releasing norepinephrine (NEP), which directly stimulates target organs (Palkovits, 2009; Tank & Wong, 2011). In addition, sympathetic neurons innervate chromaffin cells of the adrenal medulla to release additional NEP and primarily epinephrine (EP), which reach target organs through the circulation (Palkovits, 2009; Tank & Wong, 2011). By interacting with adrenergic receptors on target organs, NEP and EP evoke various physiological adaptations, including cardiovascular, vascular, metabolic, pulmonary, and gastrointestinal changes (Tank & Wong, 2011). These changes include increases in blood pressure (BP), heart rate (HR), skeletal muscle blood flow, blood glucose levels, and free fatty acids, as well as relaxation of bronchial and intestinal smooth muscles (Tank & Wong, 2011). In this way, activation of the SAM-axis, also referred to as the fight-or-flight response (Cannon, 1915), prepares the body for action within seconds. In human stress research, SAM axis (re)activity is usually assessed by measuring plasma catecholamine levels (i.e., NEP and EP) or cardiovascular (re)activity resulting from catecholamine release (e.g., assessing BP or HR) (Bali & Jaggi, 2015; Winters, 2012).

Moreover, the enzyme salivary α -amylase (sAA) is considered a surrogate marker of SNS activation (Nater & Rohleder, 2009). Regarding the stress reactivity kinetics, all of these parameters reach their peak either during stress or immediately after stress cessation and return to baseline between 5 and 20 minutes afterward (Allen et al., 2014; Nater et al., 2006; Nater et al., 2005). The rapid, immediate, and short-term effects of SAM axis activation are followed by and interact with the delayed but more sustained effects of HPA axis activation (Eriksen et al., 1999; Mueller et al., 2022).

Hypothalamic-Pituitary-Adrenal Axis. In case of stimulation, parvocellular neurons of the PVN synthesize and release CRH, the principal regulator of the HPA axis, into the hypophyseal portal system (Herman et al., 2016; Papadimitriou & Priftis, 2009; Smith & Vale, 2006). Reaching the anterior lobe of the pituitary, CRH stimulates the secretion of adrenocorticotrophic hormone (ACTH) into the systemic circulation (Rivier & Vale, 1983b; Vale et al., 1981). AVP, also released from the PVN, interacts synergistically with CRH on ACTH secretion (Antoni, 1993; Rivier & Vale, 1983a). Circulating ACTH primarily initiates the synthesis and release of the corticosteroid cortisol from the zona fasciculata of the adrenal cortex (Papadimitriou & Priftis, 2009; Selye, 1956; Smith & Vale, 2006). By binding to intracellular high-affinity mineralocorticoid receptors (MRs) and lower-affinity glucocorticoid receptors (GRs), which are widely distributed throughout the body and brain, cortisol exerts various complex effects that are essential for maintaining homeostasis during stress (Buckingham, 2006; de Kloet et al., 2005). In general, cortisol acts in a complementary manner: permissive or stimulatory, facilitating or enhancing physiological responses to prepare the body for action, and suppressive or protective, limiting these responses to prevent the body from overshooting and damaging the organism (Buckingham, 2007; Sapolsky et al., 2000). In line with this, the effects of stress-induced cortisol levels include various cardiovascular, metabolic, immunological, cognitive, and behavioral responses. These responses comprise increased cardiovascular activation, catabolic actions to mobilize energy, the impairment of non-essential

functions such as reproduction, and the regulation of immunological and inflammatory stress responses and learning and memory processes (Buckingham, 2007; Sapolsky et al., 2000). In addition, cortisol regulates its release through negative feedback loops that inhibit HPA axis activity, such as by suppressing the release of CRH and ACTH (de Kloet et al., 2005; Herman et al., 2016). In human stress research, HPA axis (re)activity is typically assessed by measuring salivary cortisol, which represents the amount of free, unbound, biologically active cortisol (Hellhammer et al., 2009; Levine et al., 2007). Regarding the stress reactivity kinetics, salivary cortisol levels reach their peak levels between 10 and 20 minutes after stress cessation and slowly return to baseline about 60 minutes afterward (Kirschbaum et al., 1993; Kudielka, Schommer, et al., 2004).

Renin-Angiotensin-Aldosterone System. The RAAS is another stress-reactive physiological system that interacts with both the SAM axis and the HPA axis (Gideon et al., 2020; Gideon, Sauter, Pruessner, et al., 2022; Kubzansky & Adler, 2010). In general, the RAAS plays an important role in cardiovascular homeostasis by regulating BP as well as fluid and electrolyte balance (Atlas, 2007; Patel et al., 2017). Activation of the RAAS begins with the release of renin from the juxtaglomerular cells of the kidney into the circulation (Atlas, 2007; Patel et al., 2017). Among other mechanisms, the binding of catecholamines (e.g., released in response to activation of the SAM axis) to β -adrenergic receptors initiates the release of renin (DiBona, 2001; Kurtz, 2011). Renin, in turn, hydrolyzes circulating angiotensinogen, which is primarily produced in the liver, to angiotensin (ANG)-I (Morgan et al., 1996). Subsequently, the inactive decapeptide ANG-I is hydrolyzed by angiotensin-converting enzyme (ACE) to the biologically active octapeptide ANG-II (Atlas, 2007; Patel et al., 2017). ANG-II is the primary effector hormone for many of the numerous effects induced by RAAS activation (Atlas, 2007). These include ANG-II actions on the cardiovascular system, the kidney, and the adrenal glands via ANG-II type 1 receptors (Mehta & Griendling, 2007; Timmermans et al., 1992). Concerning the adrenal glands, especially the adrenal cortex, ANG-II stimulates the synthesis and secretion

of the mineralocorticoid aldosterone from the zona glomerulosa (Bollag, 2011; Connell & Davies, 2005; Timmermans et al., 1992). In addition, circulating ACTH released in response to HPA axis activation stimulates the adrenal cortex to release not only cortisol from the zona fasciculata but also aldosterone from the zona glomerulosa (Bollag, 2011; Connell & Davies, 2005). Given its various actions, aldosterone is considered a key cardiovascular hormone that primarily regulates electrolyte and fluid homeostasis via MRs by stimulating sodium (re)absorption and potassium excretion in the kidney and colon, thereby affecting BP (Booth et al., 2002; Connell & Davies, 2005). In human stress research, RAAS (re)activity is assessed by measuring plasma renin, ANG-II, and aldosterone (Cartledge & Lawson, 2000; Schulz et al., 2014). In addition, aldosterone, particularly free, unbound, biologically active aldosterone (Few et al., 1986), can be reliably assessed from saliva (Gideon et al., 2020; Manolopoulou et al., 2009). With respect to the stress reactivity kinetics, salivary aldosterone levels have been found to peak 20 minutes after stress cessation and slowly return to baseline about 90 minutes afterward (Gideon et al., 2020).

Taken together, the physiological stress response, comprising activation of the SAM axis, the HPA axis, and the RAAS, can be non-invasively and reliably assessed by measuring changes in cardiovascular parameters such as BP or HR and by measuring changes in salivary biochemical parameters such as sAA, cortisol, and aldosterone (Bali & Jaggi, 2015; Gideon et al., 2020; Hellhammer et al., 2009; Nater & Rohleder, 2009; Winters, 2012). Depending on the parameter, confounding influences such as the circadian rhythm of some parameters (e.g., cortisol (Pruessner et al., 1997; Russell & Lightman, 2019) and aldosterone (Gideon, Sauter, Deuber, et al., 2022)) as well as sex, age, and/or body mass index (BMI) should be taken into account (Gideon, Sauter, Pruessner, et al., 2022; Kajantie & Phillips, 2006; Kudielka et al., 2009; Mennella et al., 2014; Strahler et al., 2010; Therrien et al., 2010). Moreover, in order to clearly attribute the observed effects of a stress induction procedure to stress, an appropriate control condition that controls for secondary effects of the setting and task (e.g., standing,

speaking, calculating) is required (Green et al., 2014; Shapiro et al., 1978). For example, the placebo-stress control procedure of the TSST, i.e., the Placebo TSST (Het et al., 2009), represents such a control condition.

Relevance and Implications of the Stress Response

Overall, the stress response comprises not only physiological changes but also psychological (i.e., emotional and cognitive) and behavioral responses (Jacoby et al., 2021; Schlebusch, 2004). The complex coordinated pattern of changes representing the stress response aims to adequately prepare for dealing with a stressor. The physiological stress response, for example, contributes by mobilizing energy (Charmandari et al., 2005). A short-term, acute physiological stress response is therefore considered adaptive and represents a survival mechanism (Charmandari et al., 2005; Dhabhar, 2018). However, inappropriate physiological stress responses, such as frequent or chronic as well as exaggerated, prolonged or blunted stress responses, are maladaptive and may entail negative health consequences (Carroll et al., 2017; Charmandari et al., 2005; McEwen, 1998a; O'Connor et al., 2021; O'Riordan et al., 2023; Turner et al., 2020). In the context of frequent or chronic stress, pervasive or ongoing stressors (e.g., caregiving, unemployment, or job or marital stress), stressful event sequences (e.g., bereavement), and distant severe stressors (e.g., traumatic events such as combat exposure or abuse) are considered chronic stressors (Dimsdale, 2008; Rohleder, 2019; Segerstrom & Miller, 2004). Regarding the magnitude of the stress response, the impact of the physiological stress response can be understood as an inverted U-shaped dose-response pattern (Calabrese, 2008; Chrousos, 2009). Adaptive or beneficial effects of stress occur in the middle, optimal range of the curve, while hypofunction and hyperfunction of the stress system have adverse effects (Chrousos, 2009; Chrousos & Gold, 1992).

Stress and Cardiovascular Health

There is a vast amount of literature linking stress to numerous somatic and mental health conditions (for reviews, see e.g., Aronsson et al., 2017; Brotman et al., 2007; Carroll et al., 2017; Dimsdale, 2008; Kivimäki & Steptoe, 2018; O'Connor et al., 2021; O'Riordan et al., 2023; Sparrenberger et al., 2009; Spruill, 2010; Stansfeld & Candy, 2006; Theorell et al., 2015; Turner et al., 2020). In the context of stress and somatic health, McEwen (1998a, 1998b, 2009) coined the terms allostasis and allostatic load. Under stress, various physiological systems are activated to deal with the stressor that challenges homeostasis (McEwen & Stellar, 1993). These adaptations are referred to as allostasis, which is the process of achieving stability, i.e., homeostasis, through change (McEwen, 1998b, 2009; McEwen & Stellar, 1993; Sterling, 1988). Allostatic states, resulting from allostatic responses with altered and sustained physiological activity, can be managed for limited periods of time (McEwen, 2009). However, the accumulation of allostatic states over time results in an allostatic (over)load (McEwen, 2009). Allostatic load is defined as “the wear and tear on the body and brain resulting from chronic overactivity or inactivity of physiological systems that are normally involved in adaption to environmental challenge” (McEwen, 1998b, p. 37). More specifically, frequent stress exposure, the failure to habituate to repeated similar stressors, the inability to shut down the allostatic response, and inadequate allostatic responses with compensatory effects on the activity of interacting physiological systems are proposed to contribute to allostatic load and related pathophysiological consequences (McEwen, 1998a, 2009). Indeed, there is evidence from several meta-analyses for associations between stress and CHD (Chida & Steptoe, 2010; Kivimäki et al., 2015; Kivimäki et al., 2012; Kivimäki et al., 2006; Li et al., 2014; Panaite et al., 2015; Richardson et al., 2012; Steptoe & Kivimäki, 2013; Xu et al., 2015). In addition, hypertension, which is a major risk factor for CHD (Fuchs & Whelton, 2020; Kjeldsen, 2018; Liu et al., 2021), has also been associated with stress (Babu et al., 2014; Chida & Steptoe, 2010; Eddy et al., 2017; M.-Y. Liu et al., 2017).

Hypertension

Hypertension is a chronic medical condition characterized by elevated BP levels (Brouwers et al., 2021; Victor, 2018). As a major risk factor for a variety of diseases, most notably CHD, hypertension contributes substantially to the global burden of disease (Fuchs, 2017, Chapter 1; Victor, 2018). Globally, the number of people between 30 and 97 years with hypertension has nearly doubled from about 331 million women and 317 million men in 1990 to about 626 million women and 652 million men in 2019 (Zhou et al., 2021). In 2019, about 41% of the women and 51% of the men with hypertension did not report a previous diagnosis (Zhou et al., 2021), leaving a high rate of individuals with a common and largely controllable major contributor to the global burden of disease untreated (Brouwers et al., 2021; Victor, 2018). Hypertension is divided into primary, also called EHT, and secondary hypertension (Mancia et al., 2023; Unger et al., 2020; Victor, 2018). Secondary hypertension represents sustained elevated BP caused by identifiable underlying medical conditions such as renal or renovascular disease, primary aldosteronism, pheochromocytoma, and others (Rimoldi et al., 2014; Taler, 2008). In contrast, primary hypertension, or EHT, represents sustained elevated BP without a clear secondary medical explanation and accounts for approximately 95% of all hypertension cases (Carretero & Oparil, 2000; Messerli et al., 2007). Although the specific causes of EHT remain incompletely understood, important progress has been made in understanding the pathophysiology of hypertension.

Pathophysiology. Given that multiple systems and processes are involved in the short-term and long-term regulation of BP, the pathophysiology of hypertension comprises various interacting mechanisms, entailing neural, renal, hormonal, vascular, and immune, including inflammatory mechanisms (Hall et al., 2012; Harrison et al., 2021; Ma & Chen, 2022; Saxena et al., 2018; Victor, 2018).

Neural mechanisms involve processes that contribute to sympathetic overactivity (Victor, 2018). Central and reflex mechanisms driving sympathetic overactivity in hypertension

comprise the activation of excitatory neural inputs, particularly sympathetic renal nerve activation, and the deactivation of inhibitory neural inputs, particularly baroreceptor resetting (Hall et al., 2012; Victor, 2018). In addition, among other *renal mechanisms*, renal-body fluid feedback mechanisms that control extracellular fluid volume and thus intravascular volume, one of the key determinants of BP, play a pivotal role in hypertension (Hall et al., 2012; Harrison et al., 2021; Victor, 2018). The balance between salt and water intake and excretion by the kidneys regulated by pressure natriuresis and diuresis determines the extracellular fluid (Hall et al., 2012). An increase in BP typically causes an increase in sodium and water excretion via pressure natriuresis to return BP to normal levels (Hall et al., 2012; Harrison et al., 2021). In hypertension, there is a shift in pressure natriuresis that prevents BP from returning to normal (Hall et al., 2012; Harrison et al., 2021; Victor, 2018). In terms of *hormonal mechanisms*, an inappropriate activity of the RAAS plays a particular important role in hypertension (Hall et al., 2012; Victor, 2018). In this context, ANG-II and aldosterone are relevant hormones involved in BP regulation, for example, by influencing renal pressure natriuresis (Hall et al., 2012). *Vascular mechanisms* include alterations in arterial structure and function (Victor, 2018). In general, arteries have a trilaminar wall structure, including the tunica intima (the innermost layer), the tunica media (the middle layer), and the tunica adventitia (the outermost layer) (Libby, 2018). The endothelial cells lining the lumen, known as the endothelium, which together with a subendothelial layer make up the intima, are of major importance for vascular homeostasis and thus BP regulation (Libby, 2018; Lüscher & Barton, 1997). Hypertension is characterized by an endothelial dysfunction with an impaired release of endothelium-derived relaxing factors (e.g., nitric oxide) and an enhanced release of endothelium-derived contracting factors (e.g., endothelin-1) (Hall et al., 2012; Victor, 2018). Notably, endothelial dysfunction also plays a pivotal role in atherosclerosis, which is the primary pathological process in CHD (Gimbrone Jr & García-Cardena, 2016; Ross, 1999; see Coronary Heart Disease, Atherosclerosis). Furthermore, increased *immune system and inflammatory activity* and

oxidative stress have been proposed to also play an important role in hypertension (Hall et al., 2012; Harrison et al., 2021; Victor, 2018). Taken together, hypertension is the result of a complex, multi-mechanistic, interactive pathophysiology that remains to be further elucidated. Different grades of hypertension are distinguished based on the magnitude of the resulting BP elevations.

Classification and Diagnostic Evaluation. The current European guidelines (Mancia et al., 2023), similar to the international guidelines (Unger et al., 2020), distinguish the following grades of hypertension: grade 1 hypertension (systolic BP (SBP) 140–159 mmHg and/or diastolic BP (DBP) 90–99 mmHg), grade 2 hypertension (SBP 160–179 mmHg and/or DBP 100–109 mmHg), and grade 3 hypertension (SBP $\geq$ 180 mmHg and/or DBP $\geq$ 110 mmHg).

To obtain a valid and reliable diagnosis, including the differentiation of EHT and secondary hypertension, the diagnostic evaluation of hypertension comprises BP measurements and a comprehensive patient work-up (Mancia et al., 2023; Unger et al., 2020). Given BP variability, *BP measurements* should include multiple BP readings in different settings, i.e., office BP measurements and out-of-office BP measurements (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). The office BP measurements provide the basis for BP status evaluation based on the cut-offs mentioned above (hypertension: SBP $\geq$ 140 mmHg and/or DBP $\geq$ 90 mmHg) (Mancia et al., 2023). Office BP measurements should be obtained on at least two to three visits within four weeks using a validated automated electronic upper arm cuff device (Stergiou et al., 2021). More specifically, after a seated resting period of three to five minutes, BP measurements should be taken in triplicate in a seated position in a quiet and comfortable setting (Stergiou et al., 2021). The average of the last two measurements is the representative office BP value (Stergiou et al., 2021). Because of potential between-arm differences, the first measurements should be taken in both arms at the initial visit (Stergiou et al., 2021). Out-of-office BP measurements may include home and ambulatory BP

measurements and are essential to validate the BP status determined by office BP measurement (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). For home BP measurements, patients are required to measure BP in duplicate in a seated position after a seated resting period of three to five minutes, once in the morning and once in the evening for three to seven days using a validated automated electronic upper arm cuff device after careful instruction (Stergiou et al., 2021). Based on average home BP values (excluding first-day measurements), patients are classified as hypertensive with average home SBP ≥ 135 mmHg and/or average home DBP ≥ 85 mmHg (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). For detailed practice guidelines for office and out-of-office BP measurements, please see Stergiou et al. (2021). Consistent classification of office and out-of-office BP measurements indicates sustained hypertension (i.e., consistently elevated hypertensive BP) or true normotension (i.e., consistently normal BP) (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). Inconsistent classification indicates white coat or masked hypertension (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). More specifically, elevated hypertensive office BP and normal out-of-office BP are referred to as white coat hypertension (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). Normal office BP and elevated hypertensive out-of-office BP are referred to as masked hypertension (Mancia et al., 2023; Stergiou et al., 2021; Unger et al., 2020). Both white coat and masked hypertension increase the risk of developing sustained hypertension (Mancia et al., 2009; Sívén et al., 2016).

In addition to the BP measurements, the *patient work-up* comprises an evaluation of personal and clinical history, physical examination, and laboratory investigations (Mancia et al., 2023; Unger et al., 2020). Laboratory investigations include, among others, blood tests to assess hemoglobin, hematocrit, blood glucose, glycated hemoglobin A1c (HbA1c), lipid profile, potassium, sodium, uric acid, creatinine, and calcium (Mancia et al., 2023; Unger et al., 2020).

Risk Factors. With an increasing understanding of the pathophysiology of hypertension, several risk factors, including non-modifiable and modifiable risk factors, have been identified (Hall et al., 2012; Saxena et al., 2018; Slama et al., 2002; Victor, 2018). *Non-modifiable risk* factors include age, sex, ethnicity, genetics, and family history (Hall et al., 2012; Saxena et al., 2018; Slama et al., 2002; Victor, 2018). More precisely, advancing age (Buford, 2016), specific ethnic origins (Ogunniyi et al., 2021), specific genetic factors (Padmanabhan & Dominiczak, 2021), and family history of hypertension (Wang et al., 2008) relate to a higher risk of hypertension. Sex differences in hypertension change or even reverse over the life course, with menopause playing a pivotal role in increasing the risk of hypertension in women (Di Giosia et al., 2018; Gerds et al., 2022). With regard to *modifiable risk factors*, smoking (Bowman et al., 2007; Gao et al., 2023; Halperin et al., 2008), unhealthy or unbalanced diet (e.g., insufficient fruit and vegetable intake and excessive sodium intake) including alcohol consumption (Roerecke et al., 2018; Schwingshackl et al., 2017; Zhao et al., 2011), and physical inactivity (Huai et al., 2013; X. Liu et al., 2017) have been identified as behavioral risk factors. In addition, overweight or obesity is another modifiable risk factor (Landi et al., 2018; Shariq & McKenzie, 2020; Shihab et al., 2012). Moreover, hypertension is often accompanied by other medical conditions, such as dyslipidemia or diabetes, which further increase the CHD risk associated with hypertension (Dalal et al., 2012; Lu et al., 2019; Sehestedt et al., 2011).

Coronary Heart Disease

CVD comprises a group of diseases that affect the heart and blood vessels and has become the leading cause of death and disability worldwide (Roth et al., 2020; Tsao et al., 2023; Vardas et al., 2022). In terms of the global burden of CVD, the prevalence nearly doubled from about 271 million cases in 1990 to about 523 million cases in 2019, with a steady increase in CVD deaths from about 12.1 million in 1990 to about 18.6 million in 2019 (Roth et al., 2020). Of these, approximately 37% of prevalence cases and approximately 49% of deaths in 2019 were due to CHD (Roth et al., 2020), the most common type of CVD. CHD, also known as

coronary artery disease or ischemic heart disease, is a disease of the blood vessels supplying the heart muscle, i.e., the coronary arteries, characterized by reduced blood flow resulting in an imbalance between myocardial oxygen supply and demand, i.e., myocardial ischemia (Jensen et al., 2020; Malakar et al., 2019). The primary pathological process affecting the coronary arteries that underlies CHD is atherosclerosis (Libby et al., 2019; Libby & Theroux, 2005).

Atherosclerosis. The progressive inflammatory process of arterial wall thickening, known as atherosclerosis, primarily affects large and medium-sized arteries, particularly at branches and curvatures (Libby, 2018; Libby, 2021; Ross, 1999). In doing so, atherosclerosis, which is still not fully understood despite extensive research, affects the structure and function of all three arterial wall layers, i.e., the tunica intima, media, and adventitia (Libby & Hansson, 2015; Milutinović et al., 2020).

The endothelium is of major importance for vascular homeostasis (Libby, 2018; Lüscher & Barton, 1997). Various processes and circulating factors modulated by local anatomy and hemodynamics can injure the endothelium, leading to endothelial dysfunction with alterations in its normal homeostatic properties (Gimbrone Jr & García-Cardena, 2016; Ross, 1999). Dysfunctional changes associated with impaired barrier function may promote the accumulation of lipoproteins, particularly low-density lipoprotein-cholesterol (LDL-C), in the intima, which has been proposed to play an important role in the initiation of atherosclerosis (Hansson & Hermansson, 2011; Libby, 2018; Libby et al., 2019; Ross, 1999). Once trapped in the intima, LDL-C is prone to oxidative modification, with oxidized LDL-C initiating vascular inflammation (Hansson & Hermansson, 2011; Libby, 2018; Libby et al., 2019; Ross, 1999). Oxidized LDL-C activates the endothelial cells to produce adhesion molecules and chemokines to recruit, among others, monocytes and T cells into the intima (Hansson & Hermansson, 2011; Libby, 2018; Libby et al., 2019). Monocyte-derived macrophages take up oxidized LDL-C through scavenger receptors, which can transform these macrophages into foam cells (Hansson & Hermansson, 2011; Libby, 2018; Libby et al., 2019). This leads to the development of fatty

streaks, the earliest type of atherosclerotic lesions (Libby, 2018; Ross, 1999). These responses further stimulate local inflammation, for example, by involving inflammatory mediators (e.g., proinflammatory cytokines) (Hansson & Hermansson, 2011; Libby, 2018). Continued inflammation triggers the migration of smooth muscle cells from the tunica media into the intima (Libby & Theroux, 2005; Ross, 1999). Smooth muscle cells contribute to lesion progression by proliferating and elaborating extracellular matrix that accumulates in the growing atherosclerotic plaque (Libby, 2018; Libby & Theroux, 2005; Ross, 1999). The fatty streak evolves into an intermediate fibrofatty lesion (Libby, 2018; Ross, 1999). Over time, various processes, including persistent inflammation, fibrosis, angiogenesis, apoptosis, necrosis, and often calcification, lead to the development of more complex lesions with atherosclerotic plaques consisting of a lipid-rich, necrotic core that is covered by a fibrous cap (Bentzon et al., 2014; Falk, 2006; Libby, 2018; Libby & Theroux, 2005; Ross, 1999).

The progression of atherosclerosis typically extends over many years and often remains without symptoms (Libby, 2018). Initially, the plaque grows outward rather than inward, resulting in a compensatory enlargement of the artery, i.e., positive remodeling (Bentzon et al., 2014; Libby, 2018). However, when the plaque growth exceeds the capacity of the artery to remodel outward, narrowing of the arterial lumen begins, resulting in stenosis (Bentzon et al., 2014; Libby, 2018). In addition, mechanisms such as rupture of the fibrous cap or superficial erosion of plaques affecting vulnerable plaques provoke a thrombotic response involving activation of the coagulation cascade (Bentzon et al., 2014; Libby, 2018; von Känel & Bacon, 2022). The potentially resulting thrombus may impede blood flow locally or as a detached embolus that travels through the bloodstream distal to its point of origin (Bentzon et al., 2014; Hansson & Hermansson, 2011). Coronary artery stenosis and atherothrombosis cause myocardial ischemia and can manifest clinically in different ways depending on the severity (Bentzon et al., 2014; Libby, 2018; Libby & Theroux, 2005).

Clinical Manifestations and Diagnostic Evaluation. In line with the dynamic nature of CHD, the current European guidelines categorize the clinical manifestations of CHD into chronic coronary syndrome (CCS) and acute coronary syndrome (ACS) (Byrne et al., 2023; Knuuti et al., 2019). CCS refers to the stable periods of the chronic disease, which is most often progressive (Knuuti et al., 2019). In contrast, ACS refers to unstable states of the disease characterized by symptoms of sudden myocardial ischemia typically caused by plaque rupture or erosion, i.e., acute coronary events (Byrne et al., 2023; Makki et al., 2015; Overbaugh, 2009).

The most common symptom of CHD resulting from myocardial ischemia is angina pectoris (Bonaca & Sabatine, 2018; Gulati et al., 2021). Angina pectoris is typically characterized by chest discomfort or pain, often radiating to the arm, shoulder, back, or jaw, that may be accompanied by dyspnea and less specific symptoms such as nausea and sweating (Knuuti et al., 2019). The severity of angina pectoris is usually graded according to the Canadian Cardiovascular Society classification, which quantifies the occurrence of symptoms in relation to physical activity or exertion (Campeau, 1976; Knuuti et al., 2019). Stable and unstable angina pectoris are distinguished. Angina pectoris resulting from exertion or stress that resolves quickly at rest is called stable angina pectoris and is classified as CCS (Braunwald & Morrow, 2013; Knuuti et al., 2019). In contrast, unstable angina pectoris occurs more unexpectedly and suddenly at rest or with minimal exertion and is therefore classified as ACS. Accordingly, unstable angina pectoris presents as prolonged angina pectoris at rest, new onset of severe angina pectoris, and as angina pectoris that is increasing in frequency, longer in duration, or lower in threshold (Braunwald, 1989; Braunwald & Morrow, 2013; Byrne et al., 2023). In the spectrum of ACS, unstable angina pectoris precedes myocardial infarction (MI) (Braunwald, 1989; Braunwald & Morrow, 2013; Byrne et al., 2023). MI is defined as myocardial necrosis due to prolonged ischemia (Thygesen et al., 2018). Based on ST-segment deviation, MIs are subdivided into non-ST-elevation and ST-elevation MI (Thygesen et al., 2018). Overall, ACS includes unstable angina pectoris and non-ST-elevation MI, usually

resulting from partial or intermittent thrombotic occlusion of coronary arteries, and ST-elevation MI, usually resulting from complete thrombotic occlusion of coronary arteries (Libby, 2018; Makki et al., 2015; Overbaugh, 2009). ACS may be followed by sudden cardiac death (Fordyce et al., 2022; Koivunen et al., 2023; Myerburg & Goldberger, 2018).

The diagnostic approach to CHD differs in part depending on whether CCS or ACS is suspected (Byrne et al., 2023; Knuuti et al., 2019). The diagnosis of CCS involves the exclusion of ACS (Knuuti et al., 2019). The initial evaluation of ACS comprises an assessment of presenting symptoms and clinical history, physical examination, electrocardiogram (ECG), and measurement of biomarkers of myocardial injury or necrosis (Bergmark et al., 2022; Byrne et al., 2023). Based on ECG findings, ST-elevation MI can be differentiated from non-ST-elevation ACS, i.e., unstable angina pectoris and non-ST-elevation MI (Byrne et al., 2023). Biomarkers of myocardial injury or necrosis, such as high-sensitivity cardiac troponin, are used to differentiate non-ST-elevation MI from unstable angina pectoris (Byrne et al., 2023). The final diagnosis requires further diagnostic steps depending on whether CCS or ACS is suspected and the initial assessment of ACS (see Byrne et al., 2023; Knuuti et al., 2019).

Risk Factors. CHD is a multifactorial disease with various risk factors contributing to its development (Malakar et al., 2019; Zipes et al., 2018). Epidemiological studies such as the Monitoring of Trends and Determinants in Cardiovascular Disease (MONICA) (e.g., Kuulasmaa et al., 2000; WHO MONICA Project Principal Investigators, 1988), the INTERHEART study (e.g., Anand et al., 2008; Yusuf et al., 2004), and most notably the Framingham Heart Study (e.g., Dawber et al., 1951; Kannel et al., 1961; Mahmood et al., 2014; Wilson et al., 1998) have made important contributions to the identification of CHD risk factors (Wong, 2014).

Traditional, classical CHD risk factors partially overlap with hypertension risk factors and can also be divided into non-modifiable and modifiable risk factors (Flora & Nayak, 2019; Gaziano et al., 2018; Hajar, 2017; Poulter, 1999; Ridker et al., 2018; Visseren et al., 2021).

Non-modifiable risk factors include age, sex, genetics, and family history (Flora & Nayak, 2019; Gaziano et al., 2018; Hajar, 2017; Visseren et al., 2021). More precisely, advancing age (Forman et al., 2018; Tyrrell & Goldstein, 2021) as well as specific genetic factors and family history of CHD (Lloyd-Jones et al., 2004; McPherson & Tybjaerg-Hansen, 2016; Musunuru & Kathiresan, 2018) relate to CHD risk. Since men have a higher prevalence (Tsao et al., 2023) and an earlier onset (Maas & Appelman, 2010) of CHD than women, men are traditionally considered the higher-risk sex. However, given the underrepresentation of women in research and findings such as the adverse effects of menopause on CHD risk or a somewhat atypical presentation of symptoms in women that complicates diagnosis, sex differences remain to be further elucidated (e.g., Aggarwal et al., 2018; Gulati & Merz, 2018; Maas & Appelman, 2010; Yahagi et al., 2015). *Modifiable risk factors* include behavioral and metabolic risk factors (Flora & Nayak, 2019; Gaziano et al., 2018; Hajar, 2017; Ridker et al., 2018; Visseren et al., 2021; Yusuf et al., 2020). More specifically, smoking (Hackshaw et al., 2018; Huxley & Woodward, 2011), unhealthy or unbalanced diet (e.g., insufficient fruit and vegetable intake and excessive trans fats intake) (Mente et al., 2009; Mozaffarian, 2018), and physical inactivity (Li & Siegrist, 2012; Sattelmair et al., 2011) have been identified as behavioral risk factors. In addition, obesity (Després et al., 2018; Mandviwala et al., 2016), dyslipidemia (Genest & Libby, 2018; Leon & Bronas, 2009), diabetes (Low Wang et al., 2016; McGuire et al., 2018), and, particularly hypertension (Fuchs & Whelton, 2020; Kjeldsen, 2018; Liu et al., 2021) have been identified as metabolic risk factors.

In addition, several biological markers related to the pathological process of atherosclerosis have emerged as further independent CHD risk factors, namely *intermediate biological risk factors* (Frishman, 1998; Ridker et al., 2018; Wirtz & von Känel, 2017; Wood, 2001). Among others, the major intermediate biological risk factors include markers of dyslipidemia (also classified as a metabolic modifiable risk factor), inflammation, and coagulation and fibrinolysis. With regard to dyslipidemia, lipoproteins, particularly LDL-C, are

proposed to play an important role in the initiation of atherosclerosis (Hansson & Hermansson, 2011; Libby, 2018). In line with this, increased LDL-C and total cholesterol (TC) but also decreased high-density lipoprotein-cholesterol (HDL-C) and increased TC/HDL-C ratio, i.e., a shift in the lipid and lipoprotein composition characterizing dyslipidemia, have been associated with CHD (Calling et al., 2021; Genest & Libby, 2018; Leon & Bronas, 2009; Ridker, 2014). Furthermore, thrombotic responses, including activation of the coagulation cascade, characterize advanced atherosclerosis (Bentzon et al., 2014; Libby, 2018; von Känel & Bacon, 2022). Accordingly, markers reflecting a hypercoagulable state, i.e., an increased tendency to thrombosis (Senst et al., 2022), have been related to CHD (Lowe & Rumley, 2014; von Känel & Bacon, 2022). For example, the coagulation-related parameters fibrinogen (Fibrinogen Studies Collaboration, 2005; Surma & Banach, 2022) and D-dimer (Danesh et al., 2001; Simes et al., 2018), which represent the source material and thus the starting point (fibrinogen) and the net result (D-dimer) of coagulation, have been found to predict CHD risk and acute coronary events. Moreover, inflammation is a key driver of atherosclerosis in all stages (Libby, 2021). Accordingly, circulating inflammatory biomarkers indicative of inflammation have been associated with CHD (Aday & Ridker, 2019; Ridker, 2016; Wirtz & von Känel, 2017). Biomarkers of inflammation that are predictive of CHD risk and acute coronary events include the acute-phase protein C-reactive protein (CRP) (Ridker et al., 1998; Ridker, Hennekens, et al., 2000; The Emerging Risk Factors Collaboration, 2010) and cytokines that regulate the inflammatory cascade and thus CRP, such as tumor necrosis factor- α (TNF- α) or interleukin-6 (IL-6) (Danesh et al., 2008; Kaptoge et al., 2014; Ridker, Rifai, et al., 2000). These intermediate biological risk factors, i.e., lipids and lipoproteins (Stoney, 2022), coagulation-related parameters (Lewis et al., 2001; von Känel & Bacon, 2022), and markers of inflammation (Skogstrand et al., 2008; Suarez, 2022), can be reliably determined from blood samples.

Given that CHD is multifactorial in nature and results from the combined effect of multiple risk factors, multiple risk factors should be considered when assessing CHD risk, as realized in several cardiovascular risk assessment systems (for reviews, see Cooney et al., 2010; Cooney et al., 2009; Zhao et al., 2015). Furthermore, when investigating further potential risk factors for hypertension or CHD, other established risk factors should be considered to test whether the investigated factor is independent of established risk factors. Accordingly, in addition to the risk factors described above, environmental factors such as air pollution (Bhatnagar, 2018; Cosselman et al., 2015; Münzel et al., 2017; Yusuf et al., 2020) and a variety of psychosocial factors (Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Rozanski et al., 2005; Yusuf et al., 2004; Yusuf et al., 2020) have been identified as additional modifiable risk factors for both hypertension and CHD. Among the psychosocial risk factors, stress has emerged as a major risk factor for both hypertension and CHD (e.g., Brotman et al., 2007; Chida & Steptoe, 2010; Pickering, 2001; Rosengren et al., 2004; Treiber et al., 2003).

Stress as a Risk Factor for Hypertension and Coronary Heart Disease

In addition to activation of the SAM axis, the HPA axis, and the RAAS, the acute physiological stress response also includes increases in lipids and lipoproteins (Brindley et al., 1993; Stoney, 2022; Stoney et al., 1999), coagulation and fibrinolysis (Austin et al., 2013; von Känel, 2015; von Känel & Bacon, 2022), and inflammation (Marsland et al., 2017; Steptoe et al., 2007; Wirtz & von Känel, 2017), with interactions between the physiological systems (Juster et al., 2010; Kivimäki & Steptoe, 2018; Kubzansky & Adler, 2010). Thus, several biological parameters represent stress-reactive parameters and markers of hypertension or CHD (Stoney, 2022; von Känel & Bacon, 2022; Walther & Wirtz, 2023; Wirtz & von Känel, 2017). Given this, frequent and inappropriate physiological stress responses may accumulate over time, resulting in pathophysiological consequences in line with the allostatic load model, particularly contributing to the development and progression of hypertension and CHD (Juster et al., 2010; McEwen, 1998a, 1998b, 2009). In general, stress is supposed to affect

cardiovascular health directly via pathophysiological changes and indirectly via adverse behavioral changes (Chandola et al., 2008; Kivimäki & Steptoe, 2018; O'Connor et al., 2021; Sara et al., 2018). Indeed, among a vast amount of cross-sectional and prospective studies, meta-analyses confirm that chronic stress is associated with an increased risk of hypertension (Babu et al., 2014; Eddy et al., 2017; M.-Y. Liu et al., 2017) and an increased risk of CHD, including incident CHD and (recurrent) acute coronary events (Eddy et al., 2017; Kivimäki et al., 2015; Kivimäki et al., 2012; Kivimäki et al., 2006; Li et al., 2014; Richardson et al., 2012; Steptoe & Kivimäki, 2013; Xu et al., 2015). In addition, meta-analyses found that acute emotional stress triggers ACS in individuals with advanced CHD (Steptoe & Kivimäki, 2013), and that exaggerated cardiovascular stress reactivity and impaired recovery are associated with adverse cardiovascular outcomes, including increased risk of hypertension and CHD (Chida & Steptoe, 2010; Panaite et al., 2015). Moreover, the reported altered physiological stress reactivity in hypertension and CHD compared to controls (Degroote et al., 2021; Gideon et al., 2021; von Känel & Bacon, 2022; Walther, von Känel, Heimgartner, et al., 2022; Walther, von Känel, Zuccarella-Hackl, & Wirtz, 2022; Walther & Wirtz, 2023; Wirtz, 2022; Wirtz & von Känel, 2017) suggests a bidirectional relationship between stress and hypertension and CHD.

Besides stress, several other factors have been identified as psychosocial determinants of hypertension and CHD (e.g., Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Rozanski et al., 2005; Yusuf et al., 2004; Yusuf et al., 2020). These other psychosocial determinants may also influence stress, for example, by modulating the magnitude of the physiological stress response (e.g., Chida & Hamer, 2008; Kiecolt-Glaser et al., 2020; von Känel & Bacon, 2022; Walther & Wirtz, 2023; Wirtz, 2022; Wirtz & von Känel, 2017; Wirtz, von Känel, et al., 2006).

Psychosocial Determinants of Stress and Cardiovascular Health

In the context of Cartesian dualism, Descartes (1637/1956) once argued that the body follows mechanical laws and can therefore be studied like a machine, while the mind follows

different laws from those of the body. Based on this dualistic view, i.e., mind and body as distinct entities, the *biomedical model* has emerged (Lovallo, 2015, Chapter 1). More precisely, the biomedical model considers illness and disease as simple cause-and-effect relationships based purely on biological or physiological processes (Lovallo, 2015, Chapter 1; Wade & Halligan, 2004). Health is defined as the absence of disease (Wade & Halligan, 2004). This approach, which has shaped health care and clinical practice worldwide, has contributed substantially to our understanding of disease and has proven effective in treating various diseases (Lovallo, 2015, Chapter 1; Wade & Halligan, 2004). However, over the years, it has become apparent that a simple causal model with a narrow view is insufficient to describe the entire phenomenon of health, illness, and disease and limits our understanding, especially with regard to diseases without a clear medical explanation such as EHT (Engel, 1977; Farre & Rapley, 2017; Lovallo, 2015, Chapter 1; Wade & Halligan, 2004). Broadening the view of the biomedical model, Engel (1977) proposed the *biopsychosocial model*, which considers psychological and social aspects in addition to biological ones. In this view, health, illness, and disease result from biological, psychological, and social interactions (Engel, 1977, 1980; Farre & Rapley, 2017). Consistent with this multidimensional view, the WHO (1946) defined health as “a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity” (p. 1).

Indeed, a variety of psychosocial risk factors for the development and the progression of diseases have been identified (Adler & Matthews, 1994; Schneiderman et al., 2001), particularly in the context of cardiovascular health with regard to hypertension (for reviews, see e.g., Cuevas et al., 2017; Cuffee et al., 2014; Markovitz et al., 2001; Rutledge & Hogan, 2002; Steptoe, 2000; Trudel-Fitzgerald et al., 2015) and CHD (for reviews, see e.g., Everson-Rose & Lewis, 2005; Kop, 1999; Krantz & McCeney, 2002; Kuper et al., 2002; Powell-Wiley et al., 2022; Rozanski et al., 2005; Smith & Ruiz, 2002; Strike & Steptoe, 2004; von Känel, 2012). In general, psychosocial factors include a variety of psychological and social factors

such as stress, emotions, personality, qualitative and quantitative characteristics of social connections, and socio-environmental characteristics (Everson-Rose & Clark, 2010). Accordingly, psychosocial risk factors can be categorized into two main domains: (1) the social environment (e.g., job stress, low social support, and low socioeconomic status) and (2) individual dispositions, including personality (e.g., anger and hostility, type D personality, and neuroticism) and negative affect (e.g., depression, anxiety, and vital exhaustion) (Adler & Matthews, 1994; von Känel, 2012). Notably, the concept of stress represents an integrated model of psychosocial risk factors, as each can be assigned to a level of the stress cascade, ranging from stressors, to perception and evaluation of stressful situations, to stress responses, and, finally to the consequences of stress (see Cuffee et al., 2014; Kivimäki & Steptoe, 2018; von Känel, 2012). In line with this, stress is, on the one hand, a psychosocial risk factor (e.g., Brotman et al., 2007; Chida & Steptoe, 2010), that is, on the other hand, influenced by a variety of other psychosocial risk factors (e.g., Chida & Hamer, 2008; Kiecolt-Glaser et al., 2020).

Interested in the other side of the coin, Antonovsky (1979, 1987) explored how and why people stay healthy. More precisely, in the *salutogenic model*, he focused on the process of staying healthy despite exposure to adverse conditions such as stress, i.e., salutogenesis (Antonovsky, 1979, 1987; Horsburgh & Ferguson, 2012). In this context, generalized resistance resources and resistance deficits, i.e., resources and risk factors, are proposed to determine the individual's sense of coherence that is introduced as the primary determinant of health (Antonovsky, 1979, 1987; Horsburgh & Ferguson, 2012). Furthermore, the integration of positive psychology and preventive cardiology has led to the *concept of positive cardiovascular health* (Labarthe et al., 2016). Accumulating evidence suggests that positive psychological factors indicative of positive psychological health or well-being (e.g., positive affect, happiness, optimism, and purpose in life) are independently associated with cardiovascular health beyond the absence of negative psychological factors (e.g., stress, anger and hostility, and depression) (Boehm & Kubzansky, 2012; Levine et al., 2021). Based on this, it is proposed that positive

psychological health or well-being promotes cardiovascular health, i.e., positive cardiovascular health (Boehm, 2021; Boehm & Kubzansky, 2012; Kubzansky et al., 2018; Levine et al., 2021).

Taken together, stress and (cardiovascular) health are influenced by multidimensional factors. In this regard, psychosocial determinants, including risk factors that contribute to stress and the development and progression of disease, as well as resources that buffer stress and promote health, play a crucial role. The underlying pathways have been proposed to include direct biological or physiological and indirect behavioral mechanisms (Kubzansky et al., 2018; Levine et al., 2021; Rozanski et al., 2005; von Känel, 2012). Psychosocial determinants of stress and health are not necessarily either a risk factor or a resource. For example, low levels of social support are considered a risk factor, while high levels of social support are recognized as a resource (e.g., Barth et al., 2010; Cohen & Wills, 1985; Ditzen & Heinrichs, 2014; Lett et al., 2005; Rozanski et al., 2005).

Psychosocial determinants of stress and cardiovascular health, introduced in more detail below, are the social environment as an indirect source of stress in terms of stress contagion, appreciation, and anger.

Stress Contagion

Although stress is at its core a highly individual phenomenon, stress has considerable social aspects (e.g., ERI model (Siegrist, 1996); SOS theory (Semmer et al., 2007; Semmer et al., 2019)). For example, the social environment can be a direct source of stress, such as through social conflict (Aneshensel, 1992; Pearlin, 1989; Semmer et al., 2016). Since encountering others under stress is unavoidable in today's world (Gallup, 2023a, 2023b), the social environment has also been considered an indirect source of stress, inducing stress through interindividual stress contagion. Indeed, second-hand stress resulting from stress contagion has been found to elicit psychological, physiological, and behavioral responses (for reviews, see Engert et al., 2019; Westman, 2001; Wethington, 2000; White & Buchanan, 2016).

Definition. In the context of stress contagion, research initially focused on the interindividual contagion of stress at the psychological and behavioral levels, primarily between closely related individuals (e.g., spouses), by assessing stress experiences via self-report (e.g., Barnett et al., 1995; Bolger et al., 1989; Neff & Karney, 2007). This has been referred to as stress crossover (for reviews, see Westman, 2001; Wethington, 2000).

Over the past decade, there has been an increasing interest in the interindividual contagion of stress at the physiological level (for reviews, see Engert et al., 2019; White & Buchanan, 2016), which is also the focus of the present thesis. In this context, studies focus on physiological stress contagion effects by simultaneously assessing stress-reactive physiological parameters in stress observers and the observed stressed individuals (e.g., Buchanan et al., 2012; Engert et al., 2014). In most of the studies — as well as in the following — physiological stress contagion is simply captured by the term stress contagion (Dimitroff et al., 2017; Erkens et al., 2019; Schury et al., 2020; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014). In addition, this phenomenon has also been referred to as autonomic contagion (Ebisch et al., 2012), vicarious autonomic response or vicarious stress (Engert et al., 2014; Engert et al., 2018; Manini et al., 2013; Park et al., 2021), and stress resonance or physiological resonance (Blons et al., 2021; Buchanan et al., 2012; Engert et al., 2014; Engert et al., 2018), with slightly different implications. In general, stress contagion is assumed to be an empathic process of emotional contagion, i.e., “catching” and thus sharing the emotional state of another person (Engert et al., 2019; Nitschke & Bartz, 2023; White & Buchanan, 2016). Given this, models invoked to explain empathy, such as the Perception-Action Model for Empathy (PAM; Preston & De Waal, 2002) or the Neurocognitive Model of Emotional Contagion (NMEC; Prochazkova & Kret, 2017), may also apply to stress contagion (Engert et al., 2019; White & Buchanan, 2016). These models propose that shared neural representations for the perception and generation of actions or emotional states, as well as automatic mimicry, play an essential role in emotional contagion (Preston, 2007; Preston & De Waal, 2002; Prochazkova & Kret, 2017).

Stress Contagion in the Context of Stress and Cardiovascular Health. Overall, research on stress contagion is just emerging. To date, there is evidence for stress contagion between parents and children (Ebisch et al., 2012; Manini et al., 2013; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014), couples (Engert et al., 2014; Engert et al., 2018), and strangers (Blons et al., 2021; Buchanan et al., 2012; Dimitroff et al., 2017; Engert et al., 2014; Erkens et al., 2019; Manini et al., 2013; Park et al., 2021; Schury et al., 2020). Stress contagion has been observed with regard to the HPA axis (Buchanan et al., 2012; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Schury et al., 2020), the SAM axis (Buchanan et al., 2012; Engert et al., 2014; Park et al., 2021; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014), and sympathetic-parasympathetic interactions (Blons et al., 2021; Dimitroff et al., 2017; Ebisch et al., 2012; Manini et al., 2013; Waters et al., 2017). Of note, stress contagion has been investigated by examining physiological increases following the stress observation and/or by examining the covariation of physiological parameters between stress observers and stressed individuals. In addition, regarding the HPA axis, some studies have reported cortisol responder rates, i.e., the percentage of individuals showing a substantial increase in cortisol (≥ 1.5 nmol/l; Miller et al., 2013), with about 16–30% responders among stress observers (Engert et al., 2014; Erkens et al., 2019; Schury et al., 2020).

The existing studies show a high degree of methodological heterogeneity. More specifically, all studies that included only adults (Blons et al., 2021; Buchanan et al., 2012; Dimitroff et al., 2017; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Park et al., 2021; Schury et al., 2020) used the well-established psychosocial stress induction procedure TSST (Kirschbaum et al., 1993) or a variation of it to induce stress for stress observation, while different observation modalities were used to observe stress. In three studies, observers and the observed stressed individuals were in the same room during stress induction and observation (Blons et al., 2021; Buchanan et al., 2012; Schury et al., 2020). In two of these studies (Blons et al., 2021; Buchanan et al., 2012), the observers observed the stressed individuals directly,

i.e., face-to-face, but had additional potentially stress-inducing tasks, which may have confounded stress contagion effects. In the third study (Schury et al., 2020), direct observation was prevented because observers were placed behind the stressed individuals and observed the stress induction via video. In the other studies, the stress observation was realized in different rooms via video (Dimitroff et al., 2017; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Park et al., 2021) or via a one-way mirror (Engert et al., 2014; Engert et al., 2018), which limited the amount of information available during the observation. Moreover, only one of the studies included a control group in terms of a resting but non-observing control group (Blons et al., 2021), leaving questions as to whether the results obtained so far are due to stress contagion or due to the observation task and setting (Green et al., 2014; Shapiro et al., 1978).

Taken together, the hitherto existing methodologically heterogeneous studies provide initial evidence for physiological stress contagion. However, these results need to be verified and extended in future studies. For example, understanding the physiological reactivity kinetics of the stress contagion response is important to derive and anticipate possible consequences, such as potential adverse health consequences of accumulating stress contagion responses (see McEwen, 1998a, 1998b, 2009), which also remain to be elucidated. A standardized stress observation paradigm to increase the comparability across studies would be useful to investigate stress contagion systematically and comprehensively. In addition, an active control group that allows comparison of stress observation with placebo-stress observation and thus controls for secondary effects of the observation setting and task is required to specifically attribute observed effects to stress contagion (Green et al., 2014; Shapiro et al., 1978).

Appreciation

In addition to being recognized as a source of stress (e.g., Aneshensel, 1992; Engert et al., 2019; Semmer et al., 2016), the social environment has also been recognized as a stress-buffering resource. One of the most studied social resources is social support (e.g., Cobb, 1976; Cohen & Wills, 1985; Ditzen & Heinrichs, 2014). Another resource that has been considered

in this context but has received much less attention is appreciation. Esteem or appreciation has been proposed as one of the three reward factors in the ERI model (Siegrist, 1996), with higher rewards counterbalancing the amount of job stress. In addition, in the SOS theory (Semmer et al., 2007; Semmer et al., 2019), appreciation has been considered a core resource that boosts self-esteem and thus buffers stress resulting from threats to the self. Furthermore, appreciation has also been linked to other health-relevant concepts such as social support (Lett et al., 2005; Semmer et al., 2008; Thoits, 1995) or leadership (e.g., Judge et al., 2004; Vincent-Höper & Stein, 2019). Overall, this points to the relevance and importance of appreciation as a resource that should be studied as a concept in its own right (Semmer et al., 2019).

Definition. In general, appreciation refers to the recognition of a person's positive qualities by others, i.e., valuing someone (Semmer et al., 2007; Semmer et al., 2019; Stocker et al., 2019). Appreciation can be expressed about different aspects, such as acknowledging the person as an individual, specific behaviors, or achievements, which boosts self-esteem (Brun & Dugas, 2008; Leary & Baumeister, 2000; Semmer et al., 2007; Stocker et al., 2019). Remarkably, social support entails appreciation because of its implications on being accepted and cared for; however, in contrast to social support, appreciation can be expressed in any situation and is not restricted to difficult times (Pfister, Jacobshagen, Kälin, et al., 2020; Semmer et al., 2008; Semmer et al., 2007; Stocker et al., 2019). With a slightly different focus, appreciation has been captured by additional terms in research, such as respect (Rudolph et al., 2021; van Quaquebeke & Eckloff, 2010), esteem (Siegrist, 1996), acknowledgment (Cleary et al., 2012), or recognition (Wang et al., 2020; Yukl, 2013, Chapter 3). However, given that all these terms relate to the same basic idea of valuing someone, they are regarded as equivalent in the following, in line with Pfister, Jacobshagen, Kälin, and Semmer (2020).

Appreciation can be expressed through several behaviors (Brun & Dugas, 2008; Stocker et al., 2014; van Quaquebeke & Eckloff, 2010; Yukl, 2013, Chapter 3). In the context of work, Stocker et al. (2014) divided appreciative behaviors into six categories. These categories

include (1) praise and gratitude (e.g., compliments, saying thank you, and positive feedback), (2) trust and responsibility (e.g., expressing trust, asking for advice, and assigning interesting tasks or new responsibilities), (3) support and respect (e.g., emotional or instrumental support, respecting needs, and saying sorry), (4) motivated cooperation (e.g., motivated teamwork, joy, and finding solutions together), (5) tangible reward and promotion (e.g., presents, bonuses, and being invited), and (6) no specific behavior, i.e., indirectly implied appreciation (Stocker et al., 2014). From an interactional perspective, appreciation can be expressed through different levels of interaction, including various sources (Brun & Dugas, 2008; Stocker et al., 2014). Appreciation can be reciprocal, one-sided, or non-existent in human relationships, and in each case, it represents a message (Brun & Dugas, 2008). According to Brun and Dugas (2008), the different levels of interaction to express appreciation in the context of work include the organizational (interactions through, for example, policies and programs), vertical (interactions top-down and bottom-up), horizontal (interactions between team members), external (interactions, for example, with customers), and social (for example, the community's appreciation of the organization or its social value) level. In research, the different forms of appreciation have been assessed using various self-report measures, including questionnaires (e.g., Pohrt et al., 2022; Stocker et al., 2010; Wöhrmann et al., 2017) and diary or event-sampling methods (e.g., Cundiff et al., 2022; Stocker et al., 2014).

Appreciation in the Context of Stress and Cardiovascular Health. Although appreciation is a component of theoretical stress models (Semmer et al., 2007; Semmer et al., 2019; Siegrist, 1996) and other health-relevant concepts (e.g., Semmer et al., 2008; Vincent-Höper & Stein, 2019), there are comparatively few studies that have investigated appreciation as a concept in its own right in the context of stress and cardiovascular health.

The few existing findings in the context of stress suggest stress-buffering effects of appreciation. Specifically, appreciation at work has been found to buffer the negative effects of stressors in the form of illegitimate tasks (Pfister, Jacobshagen, Kälin, et al., 2020), long

working hours (Stocker et al., 2010), or work interruptions (Stocker et al., 2019) on self-reported indicators of health and well-being. Overall, hitherto existing studies in the context of health primarily examined associations between appreciation and self-reported indicators of health and well-being. For example, studies reported positive associations between appreciation and various indicators of well-being (Decker & Van Quaquebeke, 2015; Elfering, Gerhardt, et al., 2017; Gilbert & Kelloway, 2018; Merino & Privado, 2015; Pfister, Jacobshagen, Kälin, & Semmer, 2020; Pfister, Jacobshagen, Kälin, et al., 2020; Semmer et al., 2005; Stocker et al., 2014; Stocker et al., 2010; van Quaquebeke & Eckloff, 2010; Wöhrmann et al., 2017). Furthermore, higher appreciation is related to higher levels of subjective health (Wöhrmann et al., 2017) and lower levels of depression (Pfister, Jacobshagen, Kälin, et al., 2020; Pohrt et al., 2022), anxiety (Pfister, Jacobshagen, Kälin, et al., 2020), emotional exhaustion (Laschinger & Finegan, 2005), and lower-back-pain (Elfering, Häfliger, et al., 2017). In addition, higher appreciation has been reported to be associated with a reduced risk of sickness absence, as assessed by national register data (Hinkka et al., 2013), and less early retirement, as assessed by self-report (non-disability pension; De Wind et al., 2014) or register data (disability pension; Sundstrup et al., 2018). In the context of cardiovascular health, appreciation has been largely neglected so far. However, appreciation induces a wide range of positive feelings (Basch & Fisher, 2000; Ohly & Schmitt, 2015; Pfister, Jacobshagen, Kälin, et al., 2020). In light of this, the concept of positive cardiovascular health (Labarthe et al., 2016; Levine et al., 2021), in which positive psychological well-being, including positive affect, has been proposed to relate to better cardiovascular health, provides a theoretical rationale for associations between appreciation and cardiovascular health. In addition, the ERI model (Eddy et al., 2017; Kivimäki & Siegrist, 2016) and social support (Barth et al., 2010), both of which are related to appreciation, have been reported to be associated with cardiovascular health, thus also suggesting associations between appreciation and cardiovascular health. Despite these indications, only one study has examined associations between appreciation, specifically the

average situation-specific expectation of appreciation and respect for one's effort, and cardiovascular health (Cundiff et al., 2022). This study found a negative association between appreciation for one's effort and a marker indicative of the severity of atherosclerosis, i.e., lower intima-media thickness (IMT) of the carotid artery (Cundiff et al., 2022).

Taken together, research indicates that appreciation can be considered a resource in the context of stress and health. However, further studies are needed to confirm existing evidence. In particular, there is a lack of studies examining associations between appreciation and biological or physiological markers of stress and cardiovascular health. In addition, related constructs such as social support, ERI esteem, or self-esteem (see Leary & Baumeister, 2000; Semmer et al., 2008; Semmer et al., 2007; Semmer et al., 2019; Siegrist, 1996) should be controlled. In this way, the specificity of associations with appreciation can be tested, i.e., whether appreciation can be considered a resource in its own right.

Anger

Anger is a negative emotion typically experienced in response to being treated badly or unfairly (Novaco, 2016; Spielberger & Reheiser, 2010). Thus, triggers of anger include provocations such as intentional harm or insult to oneself or intimates, a sense of injustice, frustration, or goal blockage (Novaco, 2016). Therefore, the activation of anger is related to the perception of threat and represents a survival mechanism associated with the mobilization of resources that prepare the organism to attack (Novaco, 2016). In addition to these adaptive functions, maladaptive effects of anger and related dimensions have been extensively studied, for example, in the context of stress and cardiovascular health (e.g., Diamond, 1982; Novaco, 2016; Suls, 2013; Williams, 2010). The terms anger, hostility, and aggression have been used interchangeably in research without a clear conceptual distinction, leading to definitional ambiguity (Martin et al., 2000; Spielberger & Reheiser, 2010).

Definition. Among the various proposed definitions of anger, Spielberger et al. (1985) collectively referred to anger, hostility, and aggression as the AHA! Syndrome. Consistent with

this, a three-factor model has been proposed to provide the best conceptual structure for trait manifestations of anger, i.e., trait anger (Martin et al., 2000). This model considers trait anger to have distinct affective, behavioral, and cognitive dimensions (Martin et al., 2000). The *affective dimension*, usually referred to simply as anger, relates to feelings of anger (Martin et al., 2000) and is considered the core of the AHA! Syndrome (Spielberger & Reheiser, 2010). According to the AHA! Syndrome, anger refers to an emotional state in which the intensity of feelings ranges from mild irritation or annoyance to intense fury or rage (Spielberger et al., 1983; Spielberger & Reheiser, 2010). In addition, individual differences in the frequency of experiencing emotional states of anger over time are distinguished and referred to as trait anger (Spielberger, 1988; Spielberger & Reheiser, 2010). In this context, trait anger relates primarily to the affective dimension. Individuals high in trait anger experience emotional states of anger more frequently and intensively across a range of situations than individuals low in trait anger (Deffenbacher et al., 1996; Spielberger, 1988; Spielberger & Reheiser, 2010). The *behavioral dimension* of the three-factor model of trait anger relates to aggression and comprises tendencies to be verbally or physically antagonistic, confrontational, or destructive (Martin et al., 2000). The *cognitive dimension* relates to hostility, which reflects a negative, cynical attitude toward the world and others (Martin et al., 2000). The different dimensions of trait anger considered in the three-factor model are proposed to be interrelated, although they may also arise separately from each other (Martin et al., 2000). Overall, this conceptualization captures the multidimensional nature of anger that is also reflected in the various measures, including self-report questionnaires and structured interviews, to assess anger and related dimensions in research (Eckhardt et al., 2004; Martin et al., 2000).

Anger in the Context of Stress and Cardiovascular Health. More than eight decades ago, Alexander (1939) proposed associations between anger suppression and EHT. In the 1950s, Friedman and Rosenman (1959) introduced the type A behavior pattern, which is characterized by being highly ambitious, competitive, constantly under time pressure,

impatient, aggressive, and hostile, as a risk factor for CHD (Friedman & Rosenman, 1959; Rosenman et al., 1976). Since then, numerous studies have examined anger and related dimensions in the context of cardiovascular health, particularly with regard to hypertension and CHD (for reviews, see e.g., Diamond, 1982; Suls, 2013; Williams, 2010). Indeed, considering studies using various measures of anger and related dimensions, meta-analyses indicate associations between anger and both hypertension (Rutledge & Hogan, 2002; Schum et al., 2003; Suls et al., 1995) and CHD (Chida & Steptoe, 2009; Miller et al., 1996; Mostofsky et al., 2014). More specifically, in the context of hypertension, meta-analyses found associations between anger and BP (Schum et al., 2003; Suls et al., 1995). In addition, prospective relationships between anger and hypertension development have been reported across studies (Rutledge & Hogan, 2002). In the context of CHD, the original findings regarding associations between type A behavior pattern and CHD (Rosenman et al., 1976) could not be confirmed (Myrtek, 2001). However, anger was found to be an independent risk factor for CHD across studies (Miller et al., 1996). In addition, Chida and Steptoe (2009) focused on prospective studies and observed associations between anger and increased incident CHD in initially healthy populations as well as a poor prognosis in patients with existing CHD. Furthermore, across studies, anger outbursts were found to increase the risk of acute coronary events in the two hours following the anger outburst compared to other times (Mostofsky et al., 2014). Notably, although these findings across studies point to associations between anger and both hypertension and CHD, the results of individual studies remain inconsistent with mixed findings (see Chida & Steptoe, 2009; Miller et al., 1996; Myrtek, 2001; Rutledge & Hogan, 2002; Schum et al., 2003; Suls, 2013; Suls et al., 1995).

Proposed mechanisms underlying the association between anger and cardiovascular health include indirect behavioral pathways via adverse health behaviors and direct pathophysiological pathways that place anger in the context of stress (Kubzansky & Kawachi, 2000; Suls, 2013; Williams, 2010). Emotions, including anger, can arise in response to stressors

(Kubzansky & Kawachi, 2000; Lazarus, 1991, Chapter 1). Feelings of anger have been described as the emotional component of the fight-or-flight response, which includes the urge to fight or attack, associated with a heightened physiological activation to mobilize resources for attack (Cacioppo et al., 2000; Kreibig, 2010; Novaco, 2016). In addition, contrary to an earlier meta-analysis (Suls & Wan, 1993), a more recent meta-analysis found that anger and related dimensions are associated with increased cardiovascular stress reactivity (Chida & Hamer, 2008). Given this, frequent and inappropriate physiological reactivity associated with anger and related dimensions may accumulate over time and contribute to the development and progression of disease in line with the allostatic load model (McEwen, 1998a, 1998b, 2009).

Taken together, associations between anger, stress, and cardiovascular health seem plausible. Indeed, findings from meta-analyses indicate respective associations; however, the findings are far from consistent. The inconsistent findings may be explained by the multidimensional nature of anger, with potentially different effects depending on the dimension of anger (see Kop, 1999; Martin et al., 2000). In addition, anger and related dimensions have been assessed using different measures based on various ambiguous definitions of anger with partly poor psychometric properties (Eckhardt et al., 2004; Martin et al., 2000), which limits the validity and comparability of existing studies. Moreover, the inconsistent findings may be due to the neglect of important moderators and mediators. For example, personality in context approaches emphasize the importance of contexts, including interpersonal or social contexts, in understanding personality (Mischel & Shoda, 1995; Zayas et al., 2002). It is proposed that personality traits, such as trait anger, require situational features to be elicited (Mischel & Shoda, 1995; Zayas et al., 2002). In line with this, emotions, including anger, are proposed to arise from person-environment interactions (Lazarus, 1991, Chapter 3). In light of this, future research should systematically examine the effects of the different dimensions of anger using reliable, validated, and comparable measures. Furthermore, potential moderators and

mediators, such as situational features that elicit anger, should be considered to fully understand the effects of anger and related dimensions.

Relevance and Implications of Psychosocial Determinants of Stress and Cardiovascular Health

Overall, a growing body of research provides evidence that psychosocial determinants play an important role in stress and cardiovascular health (e.g., Chida & Hamer, 2008; Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Kiecolt-Glaser et al., 2020; Labarthe et al., 2016; Levine et al., 2021; Rozanski et al., 2005). A comprehensive understanding of the determinants of health is essential for health decision-making and planning (Bonita et al., 2006, Chapter 10; WHO, 2002, 2009). According to the health planning cycle, a framework outlining the necessary steps in the health planning process, the identification of preventable or modifiable causes, including risk factors for disease, provides the starting point for developing health interventions after burden assessment (Bonita et al., 2006, Chapter 10). Here, psychosocial determinants of stress and health offer a promising starting point (Labarthe et al., 2016; Rozanski et al., 2005; Schneiderman et al., 2001). The next step in the health planning cycle is to test whether the derived interventions effectively and efficiently prevent diseases (Bonita et al., 2006, Chapter 10). Based on the evaluation of the interventions, the implementation, monitoring, and measurement of progress follow (Bonita et al., 2006, Chapter 10). Consequently, a comprehensive understanding of the (psychosocial) determinants of stress and health is fundamental to identifying individuals at risk and to guiding the development and implementation of interventions (Bonita et al., 2006, Chapter 10; Grundy, 1999; Ridker et al., 2018; Rozanski et al., 2005; WHO, 2009).

Interventions in the Context of Stress and Cardiovascular Health

Evolving patterns in mortality and morbidity indicate that a considerable amount of today's global burden of disease is preventable (Bonita et al., 2006, Chapter 6; Gaziano et al.,

2018). This substantially increases the importance of early prevention and health promotion in healthcare. Based on the different stages of disease development, there are four levels of prevention, namely primordial, primary, secondary, and tertiary prevention (Bonita et al., 2006, Chapter 6). While primordial and primary prevention are proactive, secondary and tertiary prevention are reactive (Bonita et al., 2006, Chapter 6). More specifically, *primordial prevention* aims to prevent the emergence and establishment of health risks within environmental, economic, social, and behavioral conditions through public health policies and programs targeting, for example, unhealthy lifestyles and consumption patterns (Bonita et al., 2006, Chapter 6). *Primary prevention* aims to reduce existing health risks and thus the incidence of diseases through public health programs, such as vaccinations, health education, and behavioral counseling (Bonita et al., 2006, Chapter 6). *Secondary prevention* aims to avoid the progression of diseases at an early stage through early diagnosis and treatment, including health check-ups and screening programs (Bonita et al., 2006, Chapter 6). *Tertiary prevention* aims to avoid the progression of established diseases and the occurrence of complications through rehabilitation programs (Bonita et al., 2006, Chapter 6). Thus, primordial and primary prevention are directed at the entire population, with primary prevention additionally targeting individuals at high risk (Bonita et al., 2006, Chapter 6). In contrast, secondary prevention targets individuals with established diseases at an early stage, and tertiary prevention targets patients at a later stage of diseases (Bonita et al., 2006, Chapter 6). In line with the biomedical model (Lovallo, 2015, Chapter 1; Wade & Halligan, 2004), interventions in the context of prevention focus primarily on health risks and diseases. Health promotion, on the other hand, shifts the focus from disease to health and aims to empower people to increase control over their health and to improve their health (WHO, 1986), in line with the salutogenetic model (Antonovsky, 1979, 1987) and the concept of positive cardiovascular health (Labarthe et al., 2016). According to the Ottawa Charter for Health Promotion (WHO, 1986), efforts to promote health literacy through multisectoral actions should be at the core of health promotion. Interventions in the

context of health promotion include fiscal measures, creating health-promoting living and working conditions, reorienting health services beyond clinical and curative services, supporting health-promoting community actions, and developing personal and social skills through information and education (WHO, 1986). Thus, prevention and health promotion include interventions that are directed at the individual (e.g., health education and behavioral counseling) and interventions that are directed at the environment or living conditions (e.g., creating health-promoting conditions).

Given that stress is a major contributor to the global burden of disease (e.g., Brotman et al., 2007; Chida & Steptoe, 2010; O'Connor et al., 2021), considerable efforts have been made to develop stress management interventions, particularly in the context of work, to reduce stress and the resulting burden. In line with the categorization of prevention, stress management interventions can be classified as primary, secondary, or tertiary interventions (e.g., Bhui et al., 2012; Lamontagne et al., 2007; Tetrick & Winslow, 2015). According to Lamontagne et al. (2007), primary interventions related to stress aim to prevent or reduce exposure to stressors, such as through organizational restructuring or conflict management training. Secondary interventions target an individual's ability to cope with existing stress by providing individuals with knowledge, skills, and resources to manage stressful situations successfully (Lamontagne et al., 2007). Tertiary interventions aim to minimize existing stress-related symptoms or diseases, such as through return-to-work and other rehabilitation programs (Lamontagne et al., 2007). Furthermore, stress management interventions in the context of work are often categorized based on the level of intervention into organizational (e.g., organizational restructuring), the individual-organizational interface (e.g., participation and autonomy and peer support groups), and the individual (e.g., coping or stress management skills) level (e.g., Bhui et al., 2012; DeFrank & Cooper, 1987; Lamontagne et al., 2007; Tetrick & Winslow, 2015). Meta-analyses confirm beneficial effects across different types of stress management interventions and outcome measures in the non-clinical context, particularly with regard to

individual-level stress management interventions in the context of work (Amanvermez et al., 2020; de Wijn & van der Doef, 2022; Kröll et al., 2017; Paudel et al., 2022; Regehr et al., 2013; Regehr et al., 2014; Richardson & Rothstein, 2008; van der Klink et al., 2001). In addition, individual-level stress management interventions have also been shown to yield beneficial effects in the clinical context, particularly in the context of cardiovascular health in patients with CHD (for meta-analyses, see e.g., Dusseldorp et al., 1999; Linden et al., 2007; Magán et al., 2022; Richards et al., 2018; Tang et al., 2020). Stress management interventions targeting the individual, include various types of stress management trainings (SMTs) that teach stress management or coping skills based on different components and stress management techniques (e.g., Murphy, 1996; Richardson & Rothstein, 2008; van der Klink et al., 2001). Established stress management techniques include muscle relaxation, biofeedback, meditation, mindfulness strategies, hypnotic methods including autogenic training, breathing techniques, physical exercise, and cognitive-behavioral techniques (e.g., Everly & Lating, 2019; Lehrer et al., 2007). Among the different types of SMTs, meta-analyses indicate that cognitive-behavioral SMTs are particularly promising (Amanvermez et al., 2020; Paudel et al., 2022; Richardson & Rothstein, 2008; Saunders et al., 1996; van der Klink et al., 2001).

Cognitive-Behavioral Stress Management Trainings

According to the transactional model of stress, stress results from a perceived imbalance of situational demands exceeding personal resources, including coping abilities, based on cognitive appraisal processes (Lazarus, 1966; Lazarus & Folkman, 1984). Accordingly, cognitive-behavioral SMTs teach cognitive techniques for modifying stress-relevant cognitive appraisal processes and behavioral coping skills for managing stress (Forman, 1983; Murphy, 1996). Meichenbaum's (1985) Stress Inoculation Training (SIT) is one of the best-established cognitive-behavioral SMTs with beneficial effects in a variety of stressed populations, in both non-clinical and clinical contexts (e.g., Maag & Kotlash, 1994; Meichenbaum, 2017; Saunders et al., 1996). The SIT provides general principles and guidelines for cognitive-behavioral SMTs

that can be flexibly adapted to the needs of the target population (Meichenbaum, 1985, 2017). In general, the SIT comprises three interlocking and overlapping phases (Meichenbaum, 1985, 2017). These phases include (1) a conceptual education phase, which aims to establish a collaborative working relationship or therapeutic alliance between the participants and the trainer and to increase the understanding and awareness of the nature and impact of stress and coping, (2) a skill acquisition and consolidation phase about coping skills, and (3) an application and flow-through phase, in which the acquired coping skills are applied to increasingly demanding levels of stressors (Meichenbaum, 1985, 2017). The core elements of cognitive-behavioral SMTs are cognitive techniques such as cognitive restructuring, self-instruction, and systematic problem-solving (Meichenbaum, 1985). Cognitive restructuring aims to identify, explore, and evaluate irrational, maladaptive thoughts and replace them with rational, adaptive ones (Beck, 1967; Ellis, 1962; Meichenbaum, 1985). Self-instruction aims to change self-statements that exacerbate or worsen stress into adaptive coping self-statements to manage stressful situations successfully (Meichenbaum, 1985). Systematic problem-solving aims to recognize stress as a solvable problem and to identify, analyze, and solve stress-related problems in a systematic way (D’Zurilla & Nezu, 1982; Meichenbaum, 1985). As in the SIT (Meichenbaum, 1985), these cognitive techniques are often combined with behavioral techniques, most often relaxation techniques such as progressive muscle relaxation (Jacobsen, 1929). Depending on the needs of the target population, these techniques can be trained in specific stress-relevant contexts such as anger, social context, perfectionism, or work-life balance (e.g., Kaluza, 2018a, 2018b; Reschke & Schröder, 2000; Siegrist & Silberhorn, 1998; Wiegard et al., 2000; Williams & Williams, 2010).

Overall, meta-analyses confirm the beneficial effects of cognitive-behavioral SMTs in the non-clinical and clinical context for a wide range of outcome measures, including physiological (e.g., cortisol), organizational (e.g., performance), and, in particular, psychological (e.g., stress, anxiety, and general mental health) outcome measures (e.g., Kröll

et al., 2017; Magán et al., 2022; Regehr et al., 2013; Richardson & Rothstein, 2008; Saunders et al., 1996; Tang et al., 2020; van der Klink et al., 2001). Compared to the beneficial effects of cognitive-behavioral SMTs, the underlying psychological mechanisms have been previously neglected in research. According to the transactional model of stress (Lazarus, 1966; Lazarus & Folkman, 1984), changing the cognitive appraisal of stressful situations should alter the resulting stress experience. Given that cognitive-behavioral SMTs aim to modify stress-relevant cognitive appraisal processes by teaching cognitive techniques, improving coping in terms of changing stress-relevant cognitions may explain beneficial effects. Indeed, based on quantitative assessment such as the frequency of use of a particular coping strategy, research points to a mediating role of coping in terms of changing stress-relevant cognitions with regard to beneficial effects of cognitive-behavioral SMTs (Bond & Bunce, 2000; Brinkborg et al., 2011; Flaxman & Bond, 2010; Gaab et al., 2003; Hammerfald et al., 2006; Keogh et al., 2006; Leung et al., 2010; Lloyd et al., 2013). However, besides or even above the frequency of use of a specific coping strategy, particularly the quality of use, i.e., mastery, may be relevant for beneficial SMT effects (Bunce, 1997). So far, only one study has investigated the mediating effects of perceived mastery of changing stress-relevant cognitions using a cognitive-behavioral SMT in an individual therapeutic setting for mothers of children who were newly diagnosed with cancer (Marsland et al., 2020). This study found that improvements in perceived mastery of changing stress-relevant cognitions mediated beneficial effects on perceived stress and anxiety (Marsland et al., 2020). In addition, given that cognitive-behavioral SMTs usually teach cognitive techniques in combination with behavioral techniques (e.g., Meichenbaum, 1985; Wagner-Link, 2001; Wiegard et al., 2000), improvements in coping or stress management skills in general may also explain beneficial effects, as indicated by existing studies (Gardiner et al., 2004; Leung et al., 2010; Marsland et al., 2020; Penedo et al., 2004; Penedo et al., 2006; West et al., 1984).

Taken together, there is evidence for the beneficial effects of cognitive-behavioral SMTs in the context of stress and cardiovascular health in non-clinical and clinical populations. However, in order to realize the full preventive potential of interventions, it is essential to not only focus on their effectiveness but also to understand what makes them effective (Bunce, 1997; Nielsen & Miraglia, 2017). In light of this, future studies should systematically examine moderators and mediators of effective cognitive-behavioral SMTs to better understand the circumstances under which SMTs are maximally effective and the underlying mechanisms of change.

SUMMARY OF THEORETICAL BACKGROUND, AIMS, AND HYPOTHESES

As outlined above, psychosocial determinants play an important role in stress and cardiovascular health (e.g., Chida & Hamer, 2008; Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Kiecolt-Glaser et al., 2020; Labarthe et al., 2016; Levine et al., 2021; Rozanski et al., 2005). In general, psychosocial determinants can be categorized into the domains of the social environment (e.g., job stress and social support) and individual dispositions, including personality (e.g., trait anger and optimism) and affect (e.g., depression and positive affect) (Adler & Matthews, 1994; Everson-Rose & Clark, 2010; Levine et al., 2021; von Känel, 2012). Psychosocial determinants can have a negative impact and contribute to stress and the development and progression of disease, i.e., risk factors, or have a positive impact and buffer stress and promote health, i.e., resources. Of note, the concept of stress represents an integrated model of psychosocial risk factors (see Cuffee et al., 2014; Kivimäki & Steptoe, 2018; von Känel, 2012), with stress itself being a major psychosocial risk factor (e.g., Brotman et al., 2007; Chida & Steptoe, 2010) that is also influenced by various other psychosocial determinants (e.g., Chida & Hamer, 2008; Kiecolt-Glaser et al., 2020). Despite considerable research on psychosocial determinants in the context of stress and cardiovascular health, many questions remain to be addressed, including inconsistent findings regarding

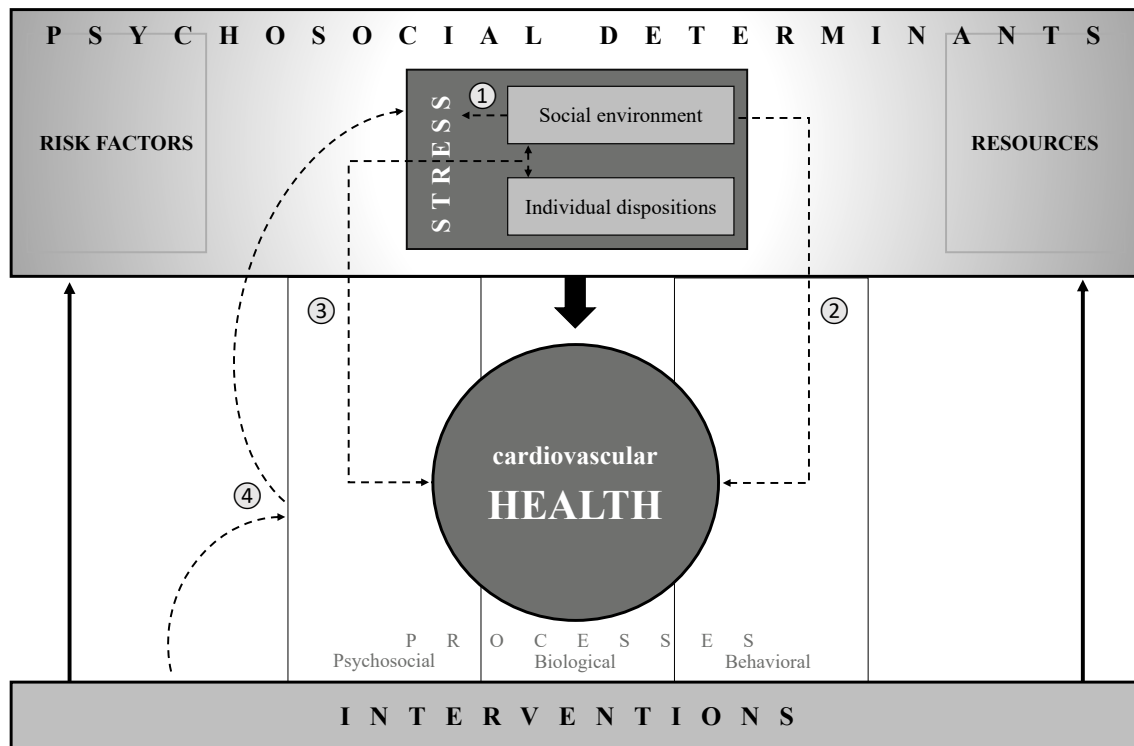
specific determinants, previously neglected variables, modulating and mediating variables, and underlying mechanisms. However, a comprehensive understanding of the (psychosocial) determinants of stress and cardiovascular health is essential to identify individuals at risk and to guide the development and implementation of interventions (Bonita et al., 2006., Chapter 10; Cuffee et al., 2014; Grundy, 1999; Kiecolt-Glaser et al., 2020; Ridker et al., 2018; Rozanski et al., 2005; WHO, 2009). With the transition from primarily infectious diseases to chronic noncommunicable, preventable diseases such as hypertension and CHD as the major global disease burden, the importance of preventive and health-promoting interventions has increased considerably (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018). In this regard, understanding the effectiveness of interventions and what makes them effective, including the mechanisms of change, is critical to realizing the full preventive potential to address the global burden of disease.

Based on the current state of research, the overarching aim of the present thesis was to enhance the understanding of (1) psychosocial determinants and (2) interventions in the context of stress and cardiovascular health with respect to EHT and CHD. Figure 1 depicts a simplified framework of psychosocial determinants and interventions in the context of stress and cardiovascular health, incorporating the four research papers that form the core of this thesis.

The *first main aim* was to continue investigating the psychosocial determinants of stress and cardiovascular health. In this regard, (1) the social environment was investigated as an indirect source of stress in terms of stress contagion (research paper 1; Auer, Walther, et al., 2024), (2) appreciation at work was examined as an independent resource with regard to CHD risk (research paper 2; Auer, Semmer, et al., 2024), and (3) trait anger, an established but equivocal psychosocial risk factor in EHT, was investigated in a social context by considering anger recognition of mixed facial affect (research paper 3; Auer et al., 2022).

Figure 1

Simplified Framework of Psychosocial Determinants, Including Risk Factors and Resources, and Interventions in the Context of Stress and Cardiovascular Health Underlying the Present Thesis



Note. For clarity, this framework is oversimplified and is not intended to be exhaustive; it represents only the pathways relevant to this thesis. The numbered dashed lines refer to the research papers that form the core of this thesis:

- ① Research paper 1: Focus on the social environment as an indirect source of stress in terms of stress contagion, introducing a standardized, randomized-controlled paradigm to investigate stress contagion based on direct stress observation.
- ② Research paper 2: Focus on appreciation at work as a resource in the context of coronary heart disease.
- ③ Research paper 3: Focus on investigating trait anger in a social context by considering anger recognition of mixed facial affect in essential hypertension.
- ④ Research paper 4: Focus on the underlying mechanisms of occupational cognitive-behavioral stress management training, emphasizing perceived mastery of functional stress management skills, particularly of changing cognitions.

First, while existing studies provide evidence for physiological stress contagion with respect to the HPA axis, the SAM axis, and sympathetic-parasympathetic interactions (e.g., Blons et al., 2021; Buchanan et al., 2012; Engert et al., 2014), other stress-reactive systems such as the RAAS have been neglected and the specific reactivity kinetics remain unknown. In addition, the observation modalities varied considerably among the existing studies (see Blons

et al., 2021; Buchanan et al., 2012; Engert et al., 2014; Schury et al., 2020). A standardized paradigm to reliably investigate physiological reactivity to stress observation compared to placebo-stress observation, controlling for secondary effects of the observation setting and task (Green et al., 2014; Shapiro et al., 1978), is lacking. Given this, research paper 1 (Auer, Walther, et al., 2024) aimed to introduce a standardized, randomized-controlled paradigm to investigate stress contagion in terms of the physiological reactivity to direct face-to-face stress observation compared to placebo-stress observation. In addition, by using this paradigm, we intended to further characterize the physiological reactivity to stress observation, including the full reactivity kinetics of stress axes sensitive to stress contagion. To this end, we adapted the well-established stress induction procedure, the TSST (Kirschbaum et al., 1993), and its placebo control procedure, the Placebo TSST (Het et al., 2009). In our study, healthy young male participants either underwent our adapted version of the TSST, observed our adapted version of the TSST, or observed our adapted version of the placebo TSST. We repeatedly assessed the SAM axis parameters HR and sAA, the HPA axis parameter salivary cortisol, and the RAAS parameter salivary aldosterone before, during, and after the experimental manipulation. We expected a higher physiological reactivity to direct stress observation compared to placebo-stress observation. In addition, we expected the stress contagion reactivity of the different stress axes to be similar to the first-hand stress reactivity but less pronounced.

Second, associations between appreciation and biological and physiological markers of cardiovascular health remain to be elucidated. In addition to the indications from the concept of cardiovascular health (Labarthe et al., 2016; Levine et al., 2021) and the findings on theoretical models and health-relevant concepts related to appreciation (Barth et al., 2010; Eddy et al., 2017; Kivimäki & Siegrist, 2016), only one study investigated associations between appreciation and cardiovascular health (Cundiff et al., 2022). This study assessed the average situation-specific expectation of appreciation and respect for one's effort and the IMT of the carotid artery and found negative associations (Cundiff et al., 2022). Based on this,

research paper 2 (Auer, Semmer, et al., 2024) aimed to investigate whether appreciation at work, notably not only for one's effort (Cundiff et al., 2022) but in a broader sense, would be related to CHD risk as assessed by major biological risk factors. In addition, we were also interested in the specificity of this proposed association, i.e., whether appreciation at work represents an independent resource in the context of CHD. For this purpose, we recruited apparently healthy, medication-free, non-smoking men in the normotensive to hypertensive range as well as medicated hypertensive and CHD patients. CHD risk was assessed by the major intermediate biological CHD risk factor categories of blood lipids (TC/HDL-C ratio), coagulation (D-dimer and fibrinogen), and inflammation (IL-6, TNF- α , and CRP) as well as by the classical risk factors of diabetes (HbA1c) and hypertension (mean arterial pressure (MAP)) under resting conditions. Appreciation at work and related, potentially confounding psychological factors (social support, self-esteem, work strain due to lack of appreciation (ERI esteem)) were assessed using self-report questionnaires. We hypothesized that higher appreciation at work would be independently associated with lower CHD risk.

Third, overall research points to associations between anger and cardiovascular health; however, the findings are far from consistent (e.g., Rutledge & Hogan, 2002; Schum et al., 2003; Suls et al., 1995). Considering the multidimensional nature of trait anger (Martin et al., 2000), the affective dimension of trait anger in particular may be associated with cardiovascular health. The affective dimension of trait anger — hereafter simply referred to as trait anger — relates to the disposition to frequently and intensively experience anger over a range of situations (Spielberger, 1988). Given that feelings of anger are associated with increased physiological activation (Cacioppo et al., 2000; Kreibig, 2010; Novaco, 2016), trait anger may accumulate over time and, for example, result in chronic BP elevations, in line with the allostatic load model (McEwen, 1998a, 1998b, 2009). In addition, based on personality in context approaches (Mischel & Shoda, 1995; Zayas et al., 2002) and the assumption that emotions arise from person-environment interactions (Lazarus, 1991, Chapter 3), trait anger

may require situational features to be elicited and to contribute to disease. Given that a person's own emotions strongly interact with the perception and recognition of the emotions of others (Preston, 2007; Preston & De Waal, 2002; Prochazkova & Kret, 2017), facial affect recognition, particularly the recognition of other persons' anger, may interact with trait anger. Based on this, research paper 3 (Auer et al., 2022) aimed to investigate trait anger in a social context by considering anger recognition, including prospective associations with BP changes, in EHT. In pursuing this goal, we recruited apparently healthy, medication-free essential hypertensive and normotensive men. In addition to a comprehensive hypertension assessment including BP measurements, we assessed emotion recognition accuracy of facial affect with a paradigm displaying two blended basic emotions including anger, fear, sadness, and happiness, and trait anger with the Spielberger trait anger scale (Schwenkmezger et al., 1992; Spielberger, 1988). BP measurements were repeated at a follow-up assessment about three years later to examine prospective associations with BP changes. Cross-sectionally, we expected that hypertensive and normotensive participants would differ in anger recognition in that hypertensive participants would overrate anger in pictures with mixed facial affect. Moreover, we expected prospective associations between anger recognition, trait anger, and BP increases over time.

The *second main aim* was related to interventions in the context of stress and cardiovascular health. Specifically, the emphasis was on investigating the underlying mechanisms of a cognitive-behavioral SMT to reduce stress and the resulting burden, with a focus on perceived mastery of functional stress management skills, particularly of changing cognitions (research paper 4; Wirtz et al., 2024).

While the effectiveness of cognitive-behavioral SMTs is well documented (e.g., Kröll et al., 2017; Richardson & Rothstein, 2008; van der Klink et al., 2001), the underlying mechanisms are not fully understood yet. Existing studies suggest that improvements in coping or stress management skills in general (Leung et al., 2010; Penedo et al., 2006; West et al., 1984) and, more specifically, improvements in coping in terms of changing stress-relevant

cognitions (Bond & Bunce, 2000; Keogh et al., 2006; Marsland et al., 2020) may explain the beneficial effects of cognitive-behavioral SMTs. So far, only one study (Marsland et al., 2020) examined perceived mastery of changing stress-relevant cognitions, i.e., the quality of use, which may be particularly important for beneficial SMT effects, as a mediator of change (see Bunce, 1997). Marsland et al. (2020) found that improvements in perceived mastery of changing stress-relevant cognitions mediated the beneficial effects of a cognitive-behavioral SMT on perceived stress and anxiety in an individual therapeutic setting specifically tailored for cancer caregivers. However, several questions regarding the generalization of these findings, such as to a broader range of outcome measures, non-cancer-specific contexts, or a group setting, remain to be addressed. In view of this, research paper 4 (Wirtz et al., 2024) aimed to investigate the effects of an occupational cognitive-behavioral group SMT, including the underlying mechanisms of change. To this end, we conducted a non-randomized controlled trial with male and female employees participating in either an occupational cognitive-behavioral group SMT or an alternative control training (AT). Based on the SIT (Meichenbaum, 1985), our occupational cognitive-behavioral SMT included the basic stress reduction techniques of cognitive restructuring, self-instruction, systematic problem-solving, and relaxation techniques. These techniques were applied to and trained in stress-relevant specific contexts, including anger management and assertiveness (Siegrist & Silberhorn, 1998; Wiegard et al., 2000), work-life balance (Kaluza, 1996; Reschke & Schröder, 2000; Wagner-Link, 2001), identification and activation of resources (Siegrist & Silberhorn, 1998), social support (Reschke & Schröder, 2000; Wagner-Link, 2001), and perfectionism (Siegrist & Silberhorn, 1998; Wiegard et al., 2000), as implemented in other SMT manuals. As outcome variables, we repeatedly assessed stress-related (perceived mastery of functional stress management skills (including a subscale assessing perceived mastery of changing cognitions), relaxation, stress reactivity, and exhaustion), work-related (job dissatisfaction), and specific-context-related (social support and trait anger) measures at baseline, two weeks, and three

months after the training. We hypothesized that the SMT would induce improvements in our stress-related, work-related, and specific-context-related outcome measures compared to AT. Moreover, we expected that SMT-induced improvements in perceived mastery of stress management skills, particularly in changing cognitions, would mediate the beneficial effects of our cognitive-behavioral SMT.

Summaries of the four research papers examining these aims and hypotheses are presented in the following chapter.

SUMMARY OF RESEARCH PAPERS

For reasons of readability and clarity, summaries of the research papers are presented in the main text of the present thesis. For full-length research papers, see Appendices A–D.

Research Paper 1

Is Your Stress My Stress? A Standardized, Randomized-Controlled Paradigm to Study Physiological Stress Contagion Based on Direct Stress Observation (Auer, Walther, et al., 2024)

Background & Objectives: Existing research indicates that not only own stress leads to physiological stress reactions, but also observing stress in others. So far, a standardized paradigm to reliably induce physiological stress contagion based on direct face-to-face stress observation compared to an active placebo-stress observing control condition is lacking. Here, we tested a standardized randomized placebo-controlled experimental paradigm to investigate physiological reactivity to direct stress observation and characterized the stress contagion response of the major endocrine stress systems, including full reactivity kinetics.

Methods: For our experimental paradigm, we adapted the well-established stress induction procedure, the TSST, and its placebo control procedure, the placebo TSST. While in the regular TSST all panel members are confederates, the panel in our modified observation TSST (obsTSST) consisted of up to three stress observer participants disguised as panel members in addition to one panel confederate who led the obsTSST. The observed TSST participant underwent the original TSST procedure. To control for the observation setting and task, we implemented an observation control condition, the observation placebo TSST (obsPlacTSST). Instead of observing the obsTSST procedure, placebo-stress observers observed a male confederate (reading confederate) reading out an emotionally neutral story followed by an easy mental arithmetic task disguised as panel members as the obsTSST observers. Thus, healthy young male participants were randomly assigned to (1) undergo the

obsTSST (“TSST participants,” $n = 20$), (2) observe it (“stress observers,” $n = 36$), or (3) observe the obsPlacTSST (“placebo-stress observers,” $n = 30$). We repeatedly assessed HR, sAA, salivary cortisol, and salivary aldosterone in all participants and reading confederates before and after the (placebo) stress (observation). The measurement time points included baseline and + 1, + 10, + 20, + 30, + 45, and + 120 min post-experimental manipulation. Established confounders, i.e., age and BMI, were controlled where appropriate.

Results: TSST participants showed higher reactivity to the obsTSST compared to reading confederates to the obsPlacTSST in all physiological parameters (p 's $\leq .085$), indicating successful stress induction. Stress observers exhibited greater physiological reactivity to stress observation than placebo-stress observers to placebo-stress observation in HR, sAA, and cortisol (p 's $\leq .027$), but not in aldosterone. We observed similar reactivity kinetics in TSST participants and stress observers but less pronounced in stress observers (p 's $\leq .002$).

Discussion: Extending previous literature, our findings indicate that independent of secondary effects of the observation setting and task, direct observation of stress in other individuals induces activation of the HPA axis and SAM axis. Moreover, the physiological stress contagion response resembles the physiological reactivity to first-hand stress but is less pronounced. Potential implications of physiological stress contagion regarding health, cognition, or behavior, as well as modulating factors need to be further elucidated.

The full manuscript of this study, including detailed information on theoretical background and objectives, methods, results, and their discussion, is provided in Appendix A. The manuscript was published as an original research article in the peer-reviewed journal *Psychoneuroendocrinology*:

Auer, A.*, Walther, L.-M.*, Jendryczko, D., Auer, L., & Wirtz, P. H. (2024). Is your stress my stress? A standardized, randomized-controlled paradigm to study physiological stress contagion based on direct stress observation. *Psychoneuroendocrinology*, 162, 106964. <https://doi.org/10.1016/j.psyneuen.2024.106964> (* equally contributing)

Research Paper 2

Taking Appreciation to Heart: Appreciation at Work and Cardiovascular Risk in Male Employees (Auer, Semmer, et al., 2024)

Background & Objective: While perceived appreciation at work has been associated with self-reported health and well-being, studies considering biological health markers are lacking. Here, we investigated whether appreciation at work would relate to CHD risk as well as the specificity of this proposed association.

Methods: Our study comprised a total of 103 male participants, including apparently healthy, medication-free, non-smoking men in the normotensive to hypertensive range ($n = 70$), as well as medicated hypertensive and CHD patients ($n = 33$). CHD risk was assessed by biological risk factors under resting conditions, i.e., MAP, the diabetes marker HbA1c, blood lipids (TC/HDL-C ratio), coagulation activity (D-dimer and fibrinogen), and inflammation (IL-6, TNF- α , and CRP). Coagulation and inflammatory markers were aggregated to respective indices. Perceived appreciation at work by supervisors and colleagues, as well as potentially confounding psychological factors, namely social support, self-esteem, and work strain due to lack of appreciation (ERI esteem), were measured by self-report questionnaires.

Results: We found higher appreciation at work to relate to lower overall CHD risk (p 's $\leq .011$) and, in particular, to lower MAP (p 's $\leq .007$) and lower blood lipids (p 's $\leq .031$) in medication-free participants as well as all participants. This overall association was independent of confounding factors (age and BMI as well as medication intake and smoking where appropriate), including related psychological factors (p 's $\leq .049$).

Discussion: Our findings indicate that appreciation at work might be an independent health-promoting resource in terms of CHD risk. Implications include that encouraging appreciation at work may help to reduce development and progression of CHD.

The full manuscript of this study, including detailed information on theoretical background and objectives, methods, results, and their discussion, is provided in Appendix B. The manuscript was published as an original research article in the peer-reviewed journal *Frontiers in Public Health*:

Auer, A., Semmer, N. K., von Känel, R., Thomas, L., Zuccarella-Hackl, C., Wiest, R., & Wirtz, P. H. (2024). Taking appreciation to heart: Appreciation at work and cardiovascular risk in male employees. *Frontiers in Public Health*, 12, 1284431. <https://doi.org/10.3389/fpubh.2024.1284431>

Research Paper 3**Do Hypertensive Men Spy with An Angry Little Eye? Anger Recognition in Men with Essential Hypertension – Cross-Sectional and Prospective Findings (Auer et al., 2022)**

Background & Objective: Higher trait anger has inconsistently been associated with hypertension and hypertension development, but the social context in terms of recognition of other persons' anger has been neglected in this context. Here, we investigated anger recognition of facial affect and trait anger in essential hypertensive and normotensive men in addition to prospective associations with BP increases.

Methods: The baseline assessment comprised a total of 145 participants, including 57 essential hypertensive and 65 normotensive men who were otherwise healthy and medication-free. Classification of EHT or normotension was based on a two-step procedure with home and study BP measurements, allowing the detection of white coat and masked hypertension and, thus, ensuring EHT status validity. Seventy-two eligible participants additionally completed a follow-up assessment 3.1 (± 0.08 SEM) years later to analyze BP changes over time. We assessed emotion recognition of facial affect with a paradigm displaying mixed facial affect of two morphed basic emotions, including anger, fear, sadness, and happiness. This paradigm allowed us to assess emotion recognition accuracy in terms of underrating or overrating the respective emotion. Trait anger was assessed via self-report with the Spielberger trait anger scale.

Results: Cross-sectionally, we found that with increasing BP, hypertensive men overrated anger displayed in facial expressions of mixed emotions as compared to normotensive men without and with controlling for age (p 's $\leq .019$) while there were no differences in trait anger ($p = .16$). Prospectively, the interaction between mean anger recognition and trait anger independently (i.e., controlling for baseline age, baseline MAP, BMI change, days between baseline and follow-up assessment, and medication intake) predicted BP increases from

baseline to follow-up (p 's $\leq .043$), in that overrating displayed anger predicted future BP increases only if trait anger was high.

Discussion: Our findings indicate an anger recognition bias in men with EHT and that overrating displayed anger in combination with higher trait anger seems to predict future BP increases. This might be of clinical relevance for the development and progression of hypertension and CVD.

The full manuscript of this study, including detailed information on theoretical background and objectives, methods, results, and their discussion, is provided in Appendix C. The manuscript was published as an original research article in the peer-reviewed journal *Annals of Behavioral Medicine*:

Auer, A., von Känel, R., Lang, I., Thomas, L., Zuccarella-Hackl, C., Degroote, C., Gideon, A., Wiest, R., & Wirtz, P. H. (2022). Do hypertensive men spy with an angry little eye? Anger recognition in men with essential hypertension – Cross-sectional and prospective findings. *Annals of Behavioral Medicine*, 56(9), 875–889.
<https://doi.org/10.1093/abm/kaab108>

Research Paper 4**Beneficial Effects of a Cognitive-Behavioral Occupational Stress Management Group Training: The Mediating Role of Changing Cognitions (Wirtz et al., 2024)**

Background & Objective: While the effectiveness of cognitive-behavioral SMTs is well documented, the underlying mechanisms, especially in an occupational context, are not fully understood. We tested whether SMT-induced improvements in stress management skills, particularly in the mastery of changing cognitions, may explain beneficial SMT effects.

Methods: Our non-randomized controlled trial comprised 108 male and female employees of a German health insurance company, with 65 of them participating in a cognitive-behavioral SMT and 43 participating in an AT. Our group-oriented cognitive-behavioral SMT comprised the basic stress reduction techniques of cognitive restructuring, self-instructions, systematic problem-solving, and relaxation techniques applied to and trained in stress-relevant specific contexts (i.e., anger management and assertiveness, work-life balance, identification and activation of resources, social support, and perfectionism). The SMT consisted of two blocks conducted over two consecutive workdays within a four-week time interval between the blocks. Participants in our control group received one of two ATs, either on the structure and organization of the company and its corporate guidelines (“Basic Training”) or on working strategies with respect to communication and cooperation (“Working efficiently”). As outcome variables, we repeatedly assessed stress-related (perceived mastery of functional stress management skills, relaxation after work, stress reactivity, vital exhaustion), work-related (job dissatisfaction), and specific-context-related (perceived social support, trait anger) measures at baseline, two weeks, and three months after the training via self-report questionnaires. Perceived mastery of functional stress management skills, particularly a subscale assessing perceived mastery of changing cognitions (“cognitive-strategies-and-problem-solving”) were tested as mediators of change.

Results: Repeated measures (M)AN(C)OVAs (without and with controlling for gender) and complementary multigroup latent difference models confirmed improvements in all outcomes in the SMT-group compared to the AT-group (p 's $\leq .015$). Regarding mechanisms of change, multivariate mediation path analyses revealed indirect effects via perceived mastery of functional stress management skills in general for all outcomes except for job dissatisfaction and social support (95% confidence interval (CI) for the expected increase in SMT- vs. AT-group = [lower limit (LL) = .033]; 95%CIs for expected decreases in SMT- vs. AT-group = [upper limits (ULs) $\leq -.045$]). Moreover, the subscale cognitive-strategies-and-problem-solving emerged as the most important mediator, as we found indirect effects via this subscale for all outcomes except for job dissatisfaction (95%CIs for expected increases in SMT- vs. AT-group = [LLs $\geq .004$]; 95%CIs for expected decreases in SMT- vs. AT-group = [ULs $\leq -.078$]).

Discussion: Our findings confirm that employees can effectively learn to master stress reduction techniques and consequently lower the resulting burden. Moreover, beneficial SMT effects seem to result from improvements in functional stress management skills, particularly in the ability to change cognitions. This points to the importance of training cognitive techniques.

The full manuscript of this study, including detailed information on theoretical background and objectives, methods, results, and their discussion, is provided in Appendix D. The manuscript was published as an original research article in the peer-reviewed journal *Frontiers in Psychology*:

Wirtz, P. H., Auer, A., Semmer, N. K., Ehler, U., & Nussbeck, F. W. (2024). Beneficial effects of a cognitive-behavioral occupational stress management group training: The mediating role of changing cognitions. *Frontiers in Psychology*, 14, 1232172. <https://doi.org/10.3389/fpsyg.2023.1232172>

GENERAL DISCUSSION

Effectively addressing the global burden of disease requires a comprehensive understanding of health determinants to identify individuals at risk and guide the development and implementation of interventions (Bonita et al., 2006, Chapter 10; Grundy, 1999; Ridker et al., 2018; WHO, 2009). This thesis aimed to enhance the understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health with regard to EHT and CHD. The focus was on the social environment as an indirect source of stress in terms of stress contagion (research paper 1), appreciation (research paper 2), anger (research paper 3), and cognitive-behavioral SMTs (research paper 4) (see Figure 1). In the following, the study findings and designs, implications, and directions for future research are discussed.

Summary and Discussion of the Study Findings and Designs

A growing body of research points to the pivotal role of *psychosocial determinants* in stress and cardiovascular health (e.g., Chida & Hamer, 2008; Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Kiecolt-Glaser et al., 2020; Labarthe et al., 2016; Levine et al., 2021; Rozanski et al., 2005). Building on emerging evidence, research paper 1 (Auer, Walther, et al., 2024) investigated the social environment as an indirect source of stress in terms of *stress contagion* in healthy young male participants. Specifically, we introduced a standardized, randomized-controlled paradigm to investigate stress contagion in terms of the physiological reactivity to direct face-to-face stress observation compared to placebo-stress observation. Stress contagion responses were examined in terms of the full reactivity kinetics of the major endocrine stress axes, i.e., the SAM axis (HR and sAA), the HPA axis (salivary cortisol), and the RAAS (salivary aldosterone). Our results suggest that observing stress in others activates the SAM axis and the HPA axis compared to placebo-stress observation. Given that previous studies have lacked an active observer control group, this is the first study to allow attribution of the physiological responses of the stress observers to stress observation, i.e., stress contagion, as our placebo-stress observation control condition controls for secondary effects of

the observation setting and task (Green et al., 2014; Shapiro et al., 1978). Our findings on stress contagion responsiveness of the SAM axis and the HPA axis are consistent with previous findings (e.g., Buchanan et al., 2012; Engert et al., 2014) and extend previous findings by demonstrating similar but less pronounced reactivity kinetics to stress observation compared to direct stress exposure. In addition, this is the first study to explore the stress contagion responsiveness of the RAAS, finding no stress contagion responses with respect to the RAAS. In summary, our findings indicate stress contagion effects on the SAM and HPA axes but not on the RAAS. In line with the PAM (Preston & De Waal, 2002) and the NMEC (Prochazkova & Kret, 2017), shared neural representations for the perception and generation of actions or emotional states and automatic mimicry could explain the stress contagion responses (Engert et al., 2019; White & Buchanan, 2016). In general, the stress contagion response might serve as nonverbal communication that signals imminent danger, as the source of stress could also pose a threat to oneself, or involve a silent request for help that mobilizes resources to fight, flight, or help (Engert et al., 2019). The effects and consequences of stress contagion at the physiological, cognitive, and behavioral levels require further elucidation. Presumably, the observed lower magnitude of the stress contagion responses compared to the first-hand stress responses makes negative health consequences such as those associated with first-hand stress (e.g., Brotman et al., 2007; McEwen, 1998a; O'Connor et al., 2021) unlikely.

Shifting the focus from the social environment as a source of stress to the social environment as a resource, research paper 2 (Auer, Semmer, et al., 2024) examined whether *appreciation* at work is associated with CHD risk. CHD risk was assessed by major biological risk factors (i.e., MAP, HbA1c, TC/HDL-C ratio, coagulation, and inflammation) in apparently healthy, medication-free men in the normotensive to hypertensive range and in medicated hypertensive and CHD patients. In addition, potentially confounding psychosocial factors in terms of social support, self-esteem, and work strain due to a lack of appreciation (ERI esteem) were controlled to test for the specificity of the proposed association. In line with the indications

from the concept of cardiovascular health (Labarthe et al., 2016; Levine et al., 2021) and the findings on theoretical models and health-relevant concepts related to appreciation (Barth et al., 2010; Eddy et al., 2017; Kivimäki & Siegrist, 2016), we found that higher appreciation at work was independently associated with lower biological CHD risk, especially with lower MAP and lower blood lipid levels. This finding is also consistent with the study that found average situation-specific expectation of appreciation and respect for one's effort to be negatively associated with carotid IMT (Cundiff et al., 2022). Given that the association between appreciation at work and CHD risk was independent of social support, self-esteem, and work strain due to a lack of appreciation (ERI esteem), appreciation seems to be a resource in its own right with respect to CHD risk. Concerning underlying mechanisms of the observed association between appreciation at work and CHD risk, stress-buffering effects of appreciation (Pfister, Jacobshagen, Kälin, et al., 2020; Stocker et al., 2010; Stocker et al., 2019), counteracting or preventing adverse health consequences associated with stress (e.g., Brotman et al., 2007; McEwen, 1998a; O'Connor et al., 2021), are conceivable. This potential pathway would be consistent with the ERI model (Siegrist, 1996), the SOS theory (Semmer et al., 2007; Semmer et al., 2019), and the JD-R model (Demerouti et al., 2001). Furthermore, appreciation induces a wide range of positive feelings (Basch & Fisher, 2000; Ohly & Schmitt, 2015; Pfister, Jacobshagen, Kälin, et al., 2020). The concept of positive cardiovascular health (Labarthe et al., 2016) proposes biological, behavioral, and psychosocial pathways underlying the association between positive psychological well-being, including positive affect, and better cardiovascular health (Kubzansky et al., 2018; Levine et al., 2021). These (positive affect) pathways may also apply to appreciation. Given this, encouraging appreciation in the workplace may help to reduce the development and progression of CHD and actively contribute to better cardiovascular health.

Based on the numerous but inconsistent findings on *anger* in EHT, research paper 3 (Auer et al., 2022) investigated trait anger in a social context by considering anger recognition

of mixed facial affect. We tested whether hypertensive and normotensive men, who were otherwise healthy and medication-free, differed from each other in anger recognition and trait anger. Moreover, we were interested in prospective associations between anger recognition, trait anger, and BP changes over time. Cross-sectionally, there were no group differences in trait anger. However, hypertensive men overrated anger displayed in facial expressions of mixed emotions with increasing BP compared to normotensive men, suggesting an overestimation of anger in facial expressions of non-anger emotions in EHT. This would lead to impaired emotion recognition across all emotions in hypertensive individuals (e.g., McCubbin et al., 2011; McCubbin et al., 2018; Shukla et al., 2018), but not when anger is considered separately (Rossi et al., 1986; Yoris et al., 2020), as indicated by previous studies. With regard to underlying mechanisms, the anger recognition bias observed in individuals with EHT may be predisposing to elevated BP and EHT. Perceiving anger in interaction partners is likely to induce own feelings of anger (Preston, 2007; Preston & De Waal, 2002; Prochazkova & Kret, 2017), accompanied by increased sympathetic activation (Cacioppo et al., 2000; Kreibig, 2010; Novaco, 2016), which may accumulate over time (McEwen, 1998a, 1998b, 2009). In addition, impaired emotion recognition, including an anger recognition bias, could lead to inappropriate social behavior (van Kleef, 2009) and contribute to a lower quality of social relationships, including less social support, and thus less stress-buffering effects (e.g., Cohen & Wills, 1985; Ditzen & Heinrichs, 2014). On the other hand, an anger recognition bias might result from elevated BP or hypertension. In line with the anger superiority effect (Horstmann & Bauland, 2006; Pinkham et al., 2010), hypertensive individuals might be on higher alert due to their BP hyperactivity and thus be even more sensitive to threatening stimuli. The non-existing group differences in trait anger between hypertensive and normotensive men are consistent with some of the previous studies (e.g., Friedman et al., 2001; Larkin & Zayfert, 2004) and question anger as an independent risk factor for EHT. Prospectively, only the interaction between anger recognition accuracy and trait anger, but none of them alone,

predicted MAP changes from baseline to follow-up. More specifically, an anger recognition bias in terms of overrating anger combined with higher trait anger predicted increases in MAP over time. This finding is consistent with personality in context approaches (Mischel & Shoda, 1995; Zayas et al., 2002) and the assumption that emotions arise from person-environment interactions (Lazarus, 1991, Chapter 3), suggesting that trait anger requires situational features to be elicited and to contribute to EHT. This highlights the importance of considering situational features and contexts when examining psychosocial determinants of stress and cardiovascular health, particularly with regard to personality traits such as trait anger. Overall, these findings may contribute to a better understanding of the clinical relevance of trait anger in the development and progression of EHT and CHD.

Understanding the determinants of health offers a promising starting point for the development of *interventions* (Bonita et al., 2006, Chapter 10). In the context of interventions, research paper 4 (Wirtz et al., 2024) examined the underlying mechanisms of *occupational cognitive-behavioral group SMT* to reduce stress and the resulting burden. We investigated the effects of our occupational cognitive-behavioral SMT compared to AT on stress-related, work-related, and specific-context-related outcome measures assessed in male and female employees at baseline, two weeks, and three months after the training. Moreover, we tested whether SMT-induced improvements in perceived mastery of stress management skills, particularly in changing cognitions, would mediate the beneficial SMT effects. Consistent with meta-analyses reporting beneficial effects of occupational cognitive-behavioral SMTs, particularly with regard to psychological outcome measures (Kröll et al., 2017; Richardson & Rothstein, 2008; van der Klink et al., 2001), we found improvements in all of our psychological outcome measures (i.e., stress-related, work-related, and specific-context-related outcome measures) in the SMT-group compared to the AT-group. Regarding mechanisms of change, we detected mediating effects of improvements in perceived mastery of functional stress management skills overall and specifically of improvements in the subscale assessing perceived mastery of

changing cognitions for all outcome measures except job dissatisfaction. These findings support the proposed framework of moderators, mediators, and mechanisms of change of occupational SMTs, which includes mastery as a mediator of change (Bunce, 1997). In addition, the findings emphasize the pivotal role of cognitions in stress in line with the transactional model of stress (Lazarus, 1966; Lazarus & Folkman, 1984) as perceived mastery of changing stress-relevant cognitions was found to be the most important mediator and thus a crucial stress management skill. In addition, the findings extend the results observed in clinical studies related to cancer (Marsland et al., 2020; Penedo et al., 2004; Penedo et al., 2006) to a non-clinical occupational context. Thus, our findings suggest that employees can successfully acquire stress management skills that reduce stress. Moreover, the beneficial SMT effects appear to result from improvements in the mastery of functional stress management skills, particularly of changing cognitions, highlighting the importance of cognitive techniques in SMTs. However, even optimal individual coping has limits, and a stressful environment with many stress sources is likely to induce stress in everyone. Therefore, to achieve optimal effects in stress reduction, interventions should not only target the individual but also further levels, such as the organizational level (Lamontagne et al., 2007; Nielsen & Christensen, 2021; Nielsen et al., 2017).

Overall, the four research papers contribute to a better understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health within the boundaries of their strengths and limitations, including their *study designs*. The studies described in research papers 1, 2, and 3 were conducted under highly standardized conditions, including strict inclusion and exclusion criteria. While the study of research paper 1 followed an experimental, placebo-controlled, between-subject study design with random group assignment, the studies of research paper 2 and research paper 3 followed an observational study design. In contrast, the field study of research paper 4 was a non-randomized controlled trial without inclusion or exclusion criteria and was conducted under naturalistic, real-life

conditions. All studies considered established confounders. Research papers 1 and 2 were based on cross-sectional data, while research papers 3 and 4 also included longitudinal data. Experimental randomized-controlled study designs (research paper 1) offer high internal validity and are close to demonstrating cause-and-effect relationships (Bonita et al., 2006, Chapter 5; Grimes & Schulz, 2002). By comparison, observational study designs (research papers 2 and 3) are more prone to some types of bias, such as the potential confounding of the observed effects by third variables, because confounding cannot be controlled as in experimental randomized-controlled study designs (Bonita et al., 2006, Chapter 3; Chalmers et al., 1983; Grimes & Schulz, 2002). However, for example, restriction in the form of strict inclusion and exclusion criteria and statistical control for established confounders (research papers 1, 2, and 3) can increase internal validity by considering a variety of potential confounding variables this way (Bonita et al., 2006, Chapter 3; Grimes & Schulz, 2002). At the same time, restrictions limit the generalizability of the study findings beyond the specific study setting and population, thus raising questions about external validity (Grimes & Schulz, 2002). Studies without inclusion and exclusion criteria, conducted under naturalistic, real-life conditions (research paper 4), may help to overcome limited external validity but are again limited in internal validity. In addition, the longitudinal study design of research papers 3 and 4, and thus the consideration of temporal sequence, is also conducive for establishing cause-and-effect relationships compared to the cross-sectional study design of research papers 1 and 2 (Bonita et al., 2006, Chapter 5; Grimes & Schulz, 2002). Additional strengths and limitations that are more study-specific are discussed in the research papers (see Appendices A–D). Overall, the study findings provide important implications and directions for future research within the boundaries of their strengths and limitations.

Relevance and Implications of the Findings

In view of the research papers 1, 2, and 3, the present thesis indicates that psychosocial determinants have an impact on stress, EHT, and CHD and thus contribute to the global burden

of disease. Various psychosocial determinants, such as the social environment in terms of stress contagion, appreciation, or anger, appear to play an important role in contributing to stress and disease (i.e., risk factors) or in buffering stress and promoting health (i.e., resources). In the non-clinical context, this implies that proactive psychosocial interventions could prevent the development of diseases such as EHT and CHD and promote cardiovascular health. This includes primordial or primary prevention, which aims to avoid or counteract psychosocial risk factors, and health promotion, which aims to enhance psychosocial resources. In the clinical context, this suggests the need for a systematic screening of psychosocial determinants, especially risk factors, during the diagnostic process in the context of cardiovascular health in order to provide a comprehensive disease treatment (Albus et al., 2004; Rozanski et al., 2005; von Känel, 2012) in terms of secondary or tertiary prevention. According to Albus et al. (2004), a two-step evaluation procedure seems well-suited to detect psychosocial risk factors in clinical practice. First, incorporating single-item questions (e.g., job stress: “Do you have enough control over how to meet the demands at work? Is your reward appropriate for your effort?”; anger: “Do you frequently feel angry over little things? If someone annoys you, do you regularly let him know? Do you often feel annoyed about habits other people have?”) into the routine diagnostic interview offers a practical starting point for screening potential risk factors (Albus et al., 2004; von Känel, 2012). Second, if potential risk factors are identified (i.e., affirmative response to any single item question), a more detailed professional psychosocial evaluation is required, including validated self-report questionnaires and structured interviews (Albus et al., 2004; von Känel, 2012). On this basis, further therapeutic steps can be recommended, including appropriate psychosocial interventions (Albus et al., 2004; Rozanski et al., 2005). Notably, such screenings should aim to specifically identify relevant psychosocial determinants based on the current state of research rather than collecting all available information (see Smith et al., 2013). So far, evidence from meta-analyses confirms beneficial effects of psychosocial interventions, particularly of SMTs, both in non-clinical contexts (e.g., de Wijn & van der Doef, 2022; Kröll

et al., 2017; Paudel et al., 2022; Richardson & Rothstein, 2008; van der Klink et al., 2001) and clinical contexts, specifically with regard to cardiovascular health, (e.g., Dusseldorp et al., 1999; Linden et al., 2007; Magán et al., 2022; Richards et al., 2018; Tang et al., 2020). These findings underscore the substantial preventive and health-promoting potential of interventions derived from the understanding of psychosocial determinants. In addition, the beneficial effects of psychosocial interventions, as also confirmed in research paper 4, strengthen the evidence on the impact of psychosocial determinants in the context of stress and cardiovascular health.

Remarkably, it has been reported that psychosocial risk factors seldom occur independently but often cluster in individuals (e.g., job stress, exhaustion, and depression) (e.g., Kop, 1999; Rosengren et al., 2004; Rozanski et al., 2005). The clustering of psychosocial risk factors suggests an interrelationship among psychosocial determinants that could be leveraged to target multiple psychosocial determinants simultaneously in interventions. In this sense, it is argued that the concepts of causality should be understood in a broader context in epidemiology (Bonita et al., 2006, Chapter 5). More specifically, rather than having one single cause, diseases commonly have multiple causes and risk factors (Bonita et al., 2006, Chapter 5). These multiple causes and risk factors can often be connected in a causal pathway or structured in a hierarchy of causes, with one factor leading to another (Bonita et al., 2006, Chapter 5). This approach allows the identification of more remote or “upstream” causes that, if targeted by interventions, can lead to substantial progress in prevention (Bonita et al., 2006, Chapter 5). Thus, identifying the causal pathway or hierarchy of psychosocial determinants in stress and cardiovascular health, including “upstream” psychosocial determinants, may enable the development of highly effective interventions. Presumably, there are “upstream” factors in terms of both risk factors and resources. In addition, identifying coping techniques that are beneficial for various psychosocial determinants could also be instrumental in the development of highly effective interventions. In this context, we found that SMT-induced improvements in several stress-related and specific-context-related outcome measures resulted particularly from improvements

in the perceived mastery of changing stress-relevant cognitions (research paper 4; Wirtz et al., 2024). Given this, cognitive techniques may be well suited to address multiple psychosocial determinants simultaneously, further highlighting the importance of teaching cognitive techniques.

Besides clustering in individuals, the prevalence of psychosocial determinants varies across demographic and socioeconomic groups, contributing to health disparities (Peterson, 2020; Powell-Wiley et al., 2022; Thoits, 2010). For example, low socioeconomic status is associated with a high prevalence of other psychosocial risk factors (e.g., Clark et al., 2009; Matthews et al., 2010). In view of this, targeting psychosocial determinants could contribute to reducing the global burden of disease, promoting health, and reducing health disparities.

Altogether, these insights, along with the findings of the present thesis, call for an orientation of health care and clinical practice beyond the traditional biomedical model, which proposes simple cause-and-effect relationships of illness and disease based purely on biological or physiological processes (Lovallo, 2015, Chapter 1; Wade & Halligan, 2004). Instead, following the biopsychosocial model (Engel, 1977), health care and clinical practice should consider biological, psychological, and social aspects, including psychosocial determinants. This paradigm shift also involves patients playing a more active role in the clinical process, moving away from the traditional biomedical approach in which physicians diagnose and treat patients as passive objects (Borrell-Carrió et al., 2004; Smith et al., 2013). Here, the communication between physician and patient is essential in order to collaboratively identify and integrate an individualized, specific representation of the general biopsychosocial model of the patient that guides the clinical process (Borrell-Carrió et al., 2004; Farre & Rapley, 2017; Smith et al., 2013). This approach holds the potential to promote a more patient-centered orientation of health care and clinical practice (Farre & Rapley, 2017; Wade & Halligan, 2017). Although more research is needed, there are indications of beneficial effects associated with a patient-centered approach in terms of patient outcomes and cost-effectiveness (e.g., Janerka et

al., 2023; Olsson et al., 2013; Pirhonen et al., 2020; Rathert et al., 2013). In addition, a biopsychosocial, more patient-centered approach could also encourage a reorientation of health services beyond clinical and curative services to health-promoting services, as called for in the Ottawa Charter for Health Promotion (WHO, 1986). Notably, the current European guidelines on CVD prevention in clinical practice recommend not only pharmacological treatment but also lifestyle changes and psychosocial interventions, considering the patients' preferences (Visseren et al., 2021). Overall, this marks steps already taken to integrate biomedical and psychosocial aspects in the context of cardiovascular health. However, bridging the gaps in and between theory, research, and practice remains a challenging but critical task to effectively address the global burden of disease.

Concluding Remarks and Directions for Future Research

Changing living conditions, including changing health risks, have shifted the global burden of disease over time with different patterns across countries (Bonita et al., 2006., Chapter 6; Gaziano et al., 2018; WHO, 2009). More specifically, with the transition from a developing country to an industrialized country, the predominant diseases affecting a country's population shift from infectious diseases (e.g., tuberculosis, pneumonia, and diarrheal diseases) to chronic noncommunicable diseases such as EHT and CHD (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018; WHO, 2009). On the one hand, this shift can be attributed to improvements in medical care, clean water supply, and sanitation that reduce the burden of infectious diseases (Gaziano et al., 2018; WHO, 2009). On the other hand, continued improvements in economic conditions and increases in life expectancy, along with lifestyle changes, give rise to new health risks, such as unhealthy or unbalanced diets, physical inactivity, and other adverse health behaviors (Gaziano et al., 2018; WHO, 2009). These health risks increase the burden of chronic noncommunicable diseases (Gaziano et al., 2018; WHO, 2009). Changes in living conditions are also accompanied by changes in stressors, from stressors of a primarily physiological nature to stressors of a primarily psychological nature (Katsampouris et al., 2022; Kiecolt-Glaser et

al., 2020; Nesse et al., 2016). From an evolutionary perspective, the physiological stress response in terms of the fight-or-flight response was critical for surviving environmental threats, such as encountering a dangerous animal (Nesse et al., 2016). However, modern, mainly psychological, stressors are not life-threatening and do not require fighting or running for one's life (Almeida et al., 2002; Katsampouris et al., 2022; Kiecolt-Glaser et al., 2020). Thus, modern stressors might induce a physiological stress response of inappropriate magnitude, mobilizing more energy than is needed to face the stressor (Katsampouris et al., 2022; Kiecolt-Glaser et al., 2020). The accumulation of inappropriate physiological stress responses to modern stressors over time may contribute to adverse health consequences in line with the allostatic load model (McEwen, 1998a, 1998b, 2009), making stress a major health risk today.

Overall, the global burden of chronic noncommunicable diseases in terms of CVD continues to increase (Gaziano et al., 2018; Roth et al., 2020). These increases are primarily driven by a growing burden of CVD in low- and middle-income countries, where about 85% of the world's population lives (Gaziano et al., 2018; Roth et al., 2020). High-income countries have seen improvements in the burden of CVD, although progress has stalled and the age-standardized CVD rates have started to rise again in some regions (Gaziano et al., 2018; Roth et al., 2020). The observed improvements can be traced back to significant advances in diagnostic and therapeutic technologies combined with preventive measures (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018; Roth et al., 2020). Although this indicates that today's global burden of disease is preventable and that we are already on the right track in addressing it, dealing with this burden remains a major challenge for the future (Bonita et al., 2006, Chapter 6; Gaziano et al., 2018; Roth et al., 2020). Compared to the health risks that predominate in developing countries (e.g., unsafe water or poor sanitation), the health risks that emerge as a country develops into an industrialized country (e.g., adverse changes in diet or activity levels), including non-life-threatening modern psychological stressors, reflect more individual risks (Almeida et al., 2002; Gaziano et al., 2018; WHO, 2009). Given this, more

objective physiological health risks fade into the background, and more subjective psychosocial health risks become apparent. Accordingly, psychosocial determinants, including and extending beyond stress, have been reported to play an important role in chronic noncommunicable diseases, particularly in the context of cardiovascular health in terms of hypertension and CHD (e.g., Cuevas et al., 2017; Cuffee et al., 2014; Everson-Rose & Lewis, 2005; Rozanski et al., 2005), which is also supported by the present thesis.

Building on the path paved by previous studies, the present thesis represents another step forward in understanding psychosocial determinants and interventions in the context of stress and cardiovascular health. However, further steps need to be taken to achieve a comprehensive understanding. Overall, the focus should be extended even further beyond pathogenesis, i.e., risk factors, to salutogenesis, i.e., resources (e.g., Antonovsky, 1979, 1987; Horsburgh & Ferguson, 2012; Kubzansky et al., 2018; Labarthe et al., 2016; Levine et al., 2021). In addition, further studies are needed to examine individual psychosocial determinants in more detail. Specifically, many psychosocial determinants, such as anger (Martin et al., 2000), are multidimensional. Consequently, a clear conceptual clarification of multidimensional determinants with the development of reliable and valid measures should be followed by a systematic investigation, including the exploration of potential differences in effects between the different dimensions (see Martin et al., 2000). Furthermore, possible differential associations with specific phenotypes or manifestations of disease such as CHD (e.g., CCS, ACS), intermediate endpoints (e.g., carotid IMT, inflammatory markers), and hard endpoints (e.g., MI, cardiac death) should be considered (Rozanski et al., 2005; von Känel, 2012). Additionally, relationships should be explored in diverse populations to detect, among others, sex, gender, racial, ethnic, or cultural differences (e.g., Low et al., 2010; Peterson, 2020; Thoits, 2010). In addition, the different contexts in which psychosocial determinants are experienced should not be overlooked, as emphasized, for example, by personality in context approaches (Mischel & Shoda, 1995; Zayas et al., 2002). The consideration of different

moderators and mediators can also help to understand the underlying mechanisms of psychosocial determinants related to stress and cardiovascular health. Although plausible underlying biobehavioral mechanisms have been proposed (Kubzansky et al., 2018; Levine et al., 2021; Rozanski et al., 2005; von Känel, 2012), further studies are needed to confirm these mechanisms. Established risk factors or determinants (e.g., obesity in terms of BMI or smoking) should be considered not only as statistical control variables to reduce confounding and test for the independence of the psychosocial determinants under study but also as mediators (Christenfeld et al., 2004). This is particularly important in order to not overlook important determinants (i.e., “upstream” factors) that have not yet been identified (Christenfeld et al., 2004). Furthermore, temporal aspects, i.e., synchronous, lagged, and reverse causation effects, should be examined to understand the dynamics of how psychosocial determinants influence stress and cardiovascular health (Ford et al., 2014). However, a detailed understanding of each individual psychosocial determinant is not sufficient. Health in terms of the biopsychosocial model involves biological, psychological, and social interactions (Engel, 1977, 1980; Farre & Rapley, 2017). Therefore, research should consider the interrelationships among these factors, including those among psychosocial determinants, to fully understand the complex intertwined effects of psychosocial determinants. This includes disentangling distinct and overlapping psychosocial determinants (e.g., Kudiela, von Känel, et al., 2004; Suls, 2018; Suls & Bunde, 2005). In addition, it should be investigated whether psychosocial determinants contribute synergistically to exacerbating health risks or to amplifying resources beyond the sum of their individual effects (Bonita et al., 2006, Chapter 5). Moreover, the causal pathways of psychosocial determinants, or the hierarchy of psychosocial determinants, should be determined in order to identify “upstream” factors that may be instrumental in realizing the full preventive potential when developing interventions (Bonita et al., 2006, Chapter 5). In evaluating causality, considerations such as temporal relation (i.e., cause preceding effect), plausibility (i.e., consistency with other knowledge), consistency (i.e., unambiguous findings),

strength of the association, dose-response relationship (i.e., increased exposure is associated with increased effect), reversibility (i.e., removal of cause results in decreased risk), and study design (i.e., strength and limitations of different study designs; see above) should be taken into account (Bonita et al., 2006, Chapter 5). Notably, intervention studies that address risk factors and test for beneficial effects can provide information on reversibility (Bonita et al., 2006, Chapter 3). Given this, the investigation of psychosocial determinants should include the examination of interventions targeting these factors to confirm their effects. In general, interventions should also be analyzed in detail (e.g., individual coping techniques) and in a broader context (e.g., multimodal interventions and combinations of interventions targeting different levels such as the individual and the organization). Overall, to comprehensively understand the complex intertwined effects of psychosocial determinants and interventions in the context of stress and cardiovascular health, it is essential to thoughtfully employ diverse approaches, including different study designs and statistical methods to build on their respective strengths and compensate for their limitations (Hammerton & Munafò, 2021).

In conclusion, the path to a comprehensive understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health has been paved. The present thesis has contributed to further paving this path with four research papers. Research paper 1 investigated the social environment as an indirect source of stress in terms of stress contagion and found that the observed stress contagion responses with regard to the SAM and HPA axes were similar but less pronounced compared to first-hand stress reactivity. Research paper 2 found that appreciation at work might be an independent health-promoting resource with respect to CHD risk. Research paper 3 found an anger recognition bias in terms of overrating anger in facial expressions of mixed emotions with increasing BP in hypertensive compared to normotensive participants and indicates that trait anger requires situational features such as an anger recognition bias to be elicited and to contribute to EHT. Research paper 4 confirmed the beneficial effects of cognitive-behavioral SMTs and highlights the importance of training

cognitive techniques, as improvements in perceived mastery of changing stress-relevant cognitions appeared to be the most important mediator of the beneficial SMT effects. Hence, while progress is being made, achieving a comprehensive understanding of psychosocial determinants and interventions in the context of stress and cardiovascular health will require continued scientific endeavors, including interdisciplinary collaborations across fields such as epidemiology, cardiology, psychosomatics, and psychology. Moreover, research findings need to be translated into implications and recommendations that can be implemented in practice, with practitioners feeding back experiences to researchers. If we succeed on this path, we can make a meaningful contribution to reducing the global burden of disease, including health disparities. This would also get us closer to the definition of health by the WHO (1946), which states that “Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity” (p. 1). However, no matter what effort we make, this definition will likely remain ideal. Nevertheless, consistent with this multidimensional view of health, the shift in the global burden of disease, combined with the shift in health risks from more objective to more individualized risks, challenges the biomedical approach to health. Instead, it calls for a more patient-centered approach according to the biopsychosocial model and emphasizes the importance of preventive and health-promoting health services beyond clinical and curative health care. Finally, in light of the impact and preventive potential of psychosocial determinants in the context of stress and cardiovascular health, I would like to conclude this thesis with the words of Hippocrates (ca. 460 to 377 B.C.E.), acknowledged as the father of medicine, who early recognized that “It is more important to know what sort of person has a disease than to know what sort of disease a person has.”

LIST OF AUTHOR CONTRIBUTIONS

In the following, contributions are detailed based on the Contributor Roles Taxonomy (CRediT; <https://credit.niso.org>).

Research Paper 1

Auer, A.*, Walther, L.-M.*, Jendryczko, D., Auer, L., & Wirtz, P. H. (2024). Is your stress my stress? A standardized, randomized-controlled paradigm to study physiological stress contagion based on direct stress observation. *Psychoneuroendocrinology*, 162, 106964. <https://doi.org/10.1016/j.psyneuen.2024.106964>

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Alisa Auer: Conceptualization, Methodology, Resources, Investigation, Data Curation, Formal Analysis, Visualization, Writing – Original Draft

Lisa-Marie Walther: Conceptualization, Methodology, Resources, Investigation, Data Curation, Formal Analysis, Visualization, Writing – Original Draft

David Jendryczko: Formal Analysis, Writing – Review and Editing

Leon Auer: Resources, Writing – Review and Editing

Petra H Wirtz: Conceptualization, Methodology, Resources, Formal Analysis, Funding Acquisition, Supervision, Writing – Original Draft

Research Paper 2

Auer, A., Semmer, N. K., von Känel, R., Thomas, L., Zuccarella-Hackl, C., Wiest, R., & Wirtz, P. H. (2024). Taking appreciation to heart: Appreciation at work and cardiovascular risk in male employees. *Frontiers in Public Health*, 12, 1284431. <https://doi.org/10.3389/fpubh.2024.1284431>

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Research Paper 3

Auer, A., von Känel, R., Lang, I., Thomas, L., Zuccarella-Hackl, C., Degroote, C., Gideon, A., Wiest, R., & Wirtz, P. H. (2022). Do hypertensive men spy with an angry little eye? Anger recognition in men with essential hypertension – Cross-sectional and prospective findings. *Annals of Behavioral Medicine*, 56(9), 875–889. <https://doi.org/10.1093/abm/kaab108>

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Research Paper 4

Wirtz, P. H., Auer, A., Semmer, N. K., Ehlert, U., & Nussbeck, F. W. (2024). Beneficial effects of a cognitive-behavioral occupational stress management group training: The mediating role of changing cognitions. *Frontiers in Psychology*, 14, 1232172. <https://doi.org/10.3389/fpsyg.2023.1232172>

Petra H Wirtz: Conceptualization, Investigation, Data Curation, Formal Analysis, Visualization, Funding Acquisition, Supervision, Writing – Original Draft

Alisa Auer: Data Curation, Formal Analysis, Visualization, Writing – Original Draft

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APPENDIX A**Research Paper 1****Is Your Stress My Stress?****A Standardized, Randomized-Controlled Paradigm to Study Physiological Stress
Contagion Based on Direct Stress Observation**

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Auer, A.*, Walther, L.-M.*, Jendryczko, D., Auer, L., & Wirtz, P. H. (2024). Is your stress my stress? A standardized, randomized-controlled paradigm to study physiological stress contagion based on direct stress observation. *Psychoneuroendocrinology*, 162, 106964.

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Abstract

Background & Objectives: Existing research indicates that not only own stress leads to physiological stress reactions, but also observing stress in others. So far, a standardized paradigm to reliably induce physiological stress contagion based on direct face-to-face stress observation compared to an active placebo-stress observing control condition is lacking. Here, we tested a standardized randomized placebo-controlled experimental paradigm to investigate physiological reactivity to direct stress observation and characterized the stress contagion response of the major endocrine stress systems, including full reactivity kinetics.

Methods: Healthy young male participants were randomly assigned to (1) undergo an adapted version of the Trier Social Stress Test (“TSST participants”, $n=20$), (2) observe it (“stress observers”, $n=36$), or (3) observe a corresponding placebo-stress control condition (“placebo-stress observers”, $n=30$). We repeatedly assessed heart rate, salivary alpha-amylase, salivary cortisol, and salivary aldosterone.

Results: Stress observers exhibited greater physiological reactivity to stress observation as compared to placebo-stress observers to placebo-stress observation in heart rate, salivary alpha-amylase, and cortisol ($p's \leq .027$), but not in aldosterone. We observed similar reactivity kinetics in TSST participants and stress observers but less pronounced in stress observers.

Discussion: Extending previous literature, our findings indicate that independent of secondary effects of the observation setting, direct observation of stress in other individuals induces activation of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary axis. Moreover, the physiological stress contagion response resembles the physiological reactivity to first-hand stress but is less pronounced. Potential implications of physiological stress contagion regarding health, cognition, or behavior, as well as modulating factors need to be further elucidated.

Keywords: stress, stress contagion, Trier Social Stress Test, physiological reactivity

Introduction

While the individual experience of and reactivity to direct stress exposure has been extensively studied over decades, comparably little is known about the interindividual contagion effects of such stress exposure. Given the increasing stress burden on the one hand (Techniker Krankenkasse, 2021) and increasing population numbers, especially in metropolitan areas, on the other hand (Moreno-Monroy et al., 2021; United Nations, 2022), a better understanding of the contagion of stress is of importance.

In research, the interindividual contagion of stress, or stress contagion, respectively (Dimitroff et al., 2017; Erkens et al., 2019; Schury et al., 2020; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014), is captured by additional different terms such as crossover effects of stress (for review see: Wethington, 2000), autonomic contagion (Ebisch et al., 2012), vicarious autonomic response (Manini et al., 2013), empathic physiological resonance (Blons et al., 2021; Buchanan et al., 2012), and vicarious or resonant empathic stress (Blons et al., 2021; Engert et al., 2019; Engert et al., 2014; Engert et al., 2018; Park et al., 2021) that in part differ slightly with respect to implications. Stress contagion research started in the 1990s, with a focus on stress contagion at the cognitive and emotional level between close individuals (Wethington, 2000). Within the last 10 years, the investigation of stress contagion has been extended to the physiological level, with studies assessing physiological responses in reaction to the observation of stressed individuals.

So far, studies have investigated physiological stress contagion in terms of significant physiological increases following the observation of other stressed individuals and/or of covariation of physiological parameters between observers and stressed individuals (for reviews see: Engert et al., 2019; White & Buchanan, 2016). Hitherto, the physiological contagion of stress was examined between parents (primarily mothers) or stranger women and children (Ebisch et al., 2012; Manini et al., 2013; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014), couples (Engert et al., 2014; Engert et al., 2018), or strangers (Blons et al.,

2021; Buchanan et al., 2012; Dimitroff et al., 2017; Engert et al., 2014; Erkens et al., 2019; Park et al., 2021; Schury et al., 2020) in various settings.

Notably, all hitherto published stress contagion studies used within-group comparisons with resting baseline measurements to investigate stress contagion, with only one study (Blons et al., 2021) comparing contagion effects of stress observation with a resting but notably non-observing control group. To the best of our knowledge, an experimental approach with an observer control group that observes a comparable but placebo-stress control condition controlling for secondary effects of the stress observation setting and task (Green et al., 2014; Shapiro et al., 1978) is lacking so far. In our study, we, therefore, set out to test a standardized experimental paradigm to investigate physiological reactivity to stress observation, i.e., stress contagion, that includes a placebo-stress observation control condition.

Moreover, the observation modalities varied considerably between studies. In all studies investigating stress contagion in adult participants (Blons et al., 2021; Buchanan et al., 2012; Dimitroff et al., 2017; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Park et al., 2021; Schury et al., 2020), the well-established psychosocial stress induction procedure Trier Social Stress Test (TSST; Kirschbaum et al., 1993) or a variation of it was used to induce stress for stress observation. In two studies, observers had additional potentially first-hand stress-inducing tasks that may have affected the physiological stress contagion reaction such as active stress induction in others as TSST committee member (Buchanan et al., 2012) or preparing for their own stress exposure (Blons et al., 2021). In other studies, the observation was realized with observers placed behind the stressed individuals in the same room (Schury et al., 2020), in different rooms via video (Dimitroff et al., 2017; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Park et al., 2021), or via one-way mirror (Engert et al., 2014; Engert et al., 2018). These observation modalities do not allow for direct observation, and consequently, accessible interactive face-to-face information is limited (Daft & Lengel, 1986). Unlike previous stress contagion studies, with the standardized experimental paradigm tested in this

study, we aimed for full access to interactive face-to-face information by direct stress observation, notably without additional potentially first-hand stress-inducing tasks.

Indications for stress contagion effects were found when assessing parameters of the hypothalamic-pituitary-adrenal (HPA) axis (Buchanan et al., 2012; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Schury et al., 2020), the sympathetic-adrenal-medullary (SAM) axis (Buchanan et al., 2012; Engert et al., 2014; Park et al., 2021; Waters et al., 2020; Waters et al., 2017; Waters et al., 2014), as well as sympathetic-parasympathetic interactions (Blons et al., 2021; Dimitroff et al., 2017; Ebisch et al., 2012; Manini et al., 2013; Waters et al., 2017). The renin-angiotensin-aldosterone system (RAAS) represents another stress-reactive physiological system, closely interrelated with the SAM axis and the HPA axis (Gideon et al., 2020; Gideon, Sauter, Pruessner, et al., 2022; Kubzansky & Adler, 2010). More precisely, SAM axis activation is associated with RAAS activation as stress-induced release of norepinephrine induces the synthesis and secretion of renin from juxtaglomerular cells in the kidney (Connell & Davies, 2005; DiBona, 2001). Furthermore, the HPA axis hormone adrenocorticotrophic hormone (ACTH) stimulates the release of aldosterone by the zona glomerulosa of the adrenal cortex (Bollag, 2011; Connell & Davies, 2005). Given these interactions between the SAM axis, the HPA axis, and the RAAS, it seems reasonable to assume that physiological stress contagion also extends to the RAAS. We, therefore, set out to investigate whether the RAAS is a stress contagion-responsive system.

Previous studies focused on the mere presence of stress contagion effects but did not depict and investigate the full reactivity kinetics of physiological stress contagion responses. However, the understanding of the full reactivity kinetics, including peak levels and recovery, is important for the comprehensive investigation of stress contagion, associated factors, underlying mechanisms, and potential implications (Geurts & Sonnentag, 2006; Lavallo, 2015). Therefore, we aimed at characterizing the full reactivity kinetics of the physiological stress contagion responses.

Taken together, the first aim of this proof-of-concept study was to test for the first time a standardized experimental paradigm to investigate physiological reactivity to direct stress observation compared to direct placebo-stress observation. In order to establish a standardized, controlled paradigm, we adapted the well-established stress induction procedure TSST (Kirschbaum et al., 1993) and its control procedure, the Placebo-TSST (PlacTSST; Het et al., 2009) to allow for stress contagion testing while actively controlling for secondary physical and cognitive demands of the observation task. Our second aim was to characterize the physiological reactivity to direct stress observation by (1) identifying which human stress axes are responsive to stress observation and thus stress contagion and (2) investigating the full reactivity kinetics of stress observation-responsive stress axes parameters. We repeatedly assessed the SAM axis parameters heart rate (HR) and sAA, the HPA axis parameter cortisol, as well as the RAAS parameter aldosterone before, during, and after (placebo-)stress (observation). We hypothesized higher physiological reactivity to direct stress observation compared to placebo-stress observation. Moreover, we expected stress contagion reactivity of the different stress axes to resemble first-hand stress reactivity, but of minor extent.

Methods

Study Participants

We recruited healthy, i.e., disease- and medication-free, non-smoking young men. To rule out potential interference with physiological stress reactivity, we excluded individuals with any of the following self-reported characteristics: age over 30 years, female sex, smoking, BMI over 30 kg/m², any occasional or acute intake of prescribed or non-prescribed medication, any psychiatric or somatic diseases (including allergies), regular excessive physical exercise, smoking, and illicit drug abuse. Recruitment was carried out through online and offline advertisements at the University of Konstanz as well as at the University of Applied Sciences Konstanz. All participants received financial compensation (10€/hour).

Study Design and Experimental Procedure

Study Design

We applied a placebo-controlled, single-blind, between-subject design. Participants were randomly assigned to either the experimental condition (“stress observation condition”) or the placebo-stress control condition (“placebo-stress observation condition”). Each data assessment comprised two to four individuals assigned to the same condition, i.e., a study group. In the stress observation condition, one participant was randomly selected to undergo a version of the TSST (Kirschbaum et al., 1993) adapted for observer stress (observation TSST, obsTSST) (“TSST participant”) with the other participant(s) observing the obsTSST disguised as panel member(s) (“stress observers”) (for a detailed description of the experimental procedure in the stress observation condition, see *2.3 Stress contagion paradigm – Stress observation condition*). In the placebo-stress observation condition, participants (“placebo-stress observers”) observed a confederate undergoing a version of the PlacTSST (Het et al., 2009) adapted for observation (observation Placebo TSST, obsPlacTSST; for a detailed description of the experimental procedure in the placebo-stress condition, see *2.3 Stress contagion paradigm – Placebo-stress observation condition*). Notably, to avoid confounding influences on our experimental manipulation, we verified that the observed individual (TSST participant or reading confederate, respectively) and the observers were strangers to each other.

Study Preparation

Participants abstained from any kind of sports and the consumption of alcohol 24h prior to study participation. Moreover, they were instructed to avoid caffeinated beverages and flavonoid containing food on the study day.

Study Day

To avoid an encounter between participants prior to the experimental procedure, participants were invited to individual meeting points at individual times between 11:30 a.m. and 12:00 p.m. Upon arrival, they were accompanied to the facilities of the laboratory of the

Biological Work and Health Psychology group at the University of Konstanz, where they were seated in individual rooms and provided written informed consent. Body weight and height were measured prior to a resting period until the start of the experimental procedure at 1:00 p.m.. Notably, each experimental procedure started at 1:00 p.m. to rule out potential confounding due to the circadian rhythm of stress hormones (Gideon, Sauter, Deuber, et al., 2022; Nater et al., 2007). To facilitate HPA axis reactivity and to minimize confounding effects due to interindividual differences in energy availability, all participants were asked to drink 200 ml of grape juice 45 min prior to the start of the experimental procedure (Zänkert et al., 2020). Seven min prior to the start of the experimental procedure, observer participants were guided into a second room nearby for the experimental procedure. After the experimental procedure, participants returned to their individual rooms and remained seated for another 120 min.

Stress Contagion Paradigm

Stress Observation Condition (Experimental Condition of the Stress Contagion Paradigm)

For a potential contagion of stress, it requires a stressed individual encountering other non-stressed individual. In our experimental condition, we, therefore, confronted one randomly selected participant in each study group, the TSST participant, with a version of the well-established psychosocial stress induction procedure the TSST (Kirschbaum et al., 1993) adapted for observer stress (obsTSST). The TSST has been shown to reliably elicit physiological stress responses (Dickerson & Kemeny, 2004). The TSST procedure includes an audio- and video-taped mock job interview (5 min) followed by a mental arithmetic task (5 min) in front of an evaluating panel wearing white coats (Kirschbaum et al., 1993). In the original TSST, all panel members are confederates. To allow for direct stress observation, notably without first-hand stress-inducing tasks, the panel in the obsTSST consisted of up to three stress observers disguised as panel members in addition to one panel confederate who led the obsTSST. The stress observers were guided to the TSST room a few minutes before the TSST

participant. The stress observers had to put on a white lab coat and sit down in the panel next to the panel confederate. They were instructed to observe the following situation carefully while maintaining a neutral facial expression during the whole procedure. Additionally, they were asked to write down their own feelings, thoughts, and physical experiences during the observation and to note the TSST participant's eye color to ensure eye contact. To prevent potential anticipation stress arising from the fear of getting in the TSST situation themselves and in order to allow for focusing on the observation only, the stress observers were explicitly informed that they would not end up in the situation of the TSST participant themselves.

Placebo-Stress Observation Condition (Control Condition of the Stress Contagion Paradigm)

To control for the observation setting and task, and thus for potential (social-evaluative) stress arising from the setting and/or the task, we developed an observation control condition based on the PlacTSST (Het et al., 2009). Instead of observing the obsTSST procedure, placebo-stress observers observed a male confederate ("reading confederate") reading out an emotionally neutral story (5 min) followed by an easy mental arithmetic task, namely adding up five (5 min). Comparable to the experimental condition, the placebo-stress observers were disguised as panel members wearing white coats and received instructions similar to those of the stress observers. Differing from the TSST/obsTSST, there was no audio- and video-taping in the obsPlacTSST. Notably, we had a total of two reading confederates who practiced reading out the story and adding up five several times in front of an audience to prevent them from being stressed by the placebo-stress observer panel. Comparable to the obsTSST, the obsPlacTSST was also led by a confederate. Notably, the instructing confederate was seated in the same room but out of direct sight of the reading confederate and the placebo-stress observers. Moreover, in both conditions, instructing confederates explicitly focused on the speaking individuals and intentionally avoided paying attention to the observers. To allow for monitoring, we also assessed the physiological reactions of the reading confederate intended to be as low as possible. To allow for comparability with the TSST participants, the reading

confederates came in 45 min before the beginning of the obsPlacTSST and drank 200 ml of grape juice at that time.

Taken together, the only major difference between obsTSST and obsPlacTSST was the extent of psychosocial stress induced by the respective task in the observed individual. The obsPlacTSST, therefore, allows to control for potential stress induction due to the observation setting and task in the observers. Our paradigm thus allows to attribute the expected difference in the physiological reactions between the stress observers and the placebo-stress observers to result explicitly from stress observation, i.e., stress contagion, and not from observation setting or task.

Physiological Assessment

Parameters Assessed from Saliva

Saliva samples were collected using Salivettes (Sarstedt, Rommelsdorf, Germany) with participants chewing on the synthetic swab for exactly 1 min. After each study day, Salivettes were centrifugated at 2500 rpm at room temperature for 10 min (Megafuge 40 R, Heraeus, Thermo Fisher Scientific, Langenselbold, Germany), aliquoted, and frozen at – 80°C until analysis. Saliva samples were taken at seven sampling time points in all participants (TSST participants, stress observers, and placebo-stress observers): 10 min before the start of the experimental procedure as well as + 1, + 10, + 20, + 30, + 45, and + 120 min after the end of the experimental procedure. Notably, reading confederates provided saliva samples in a similar manner, but only up to + 30 min after the end of the experimental procedure (rendering a total of five saliva samples). All analyses of saliva samples were performed in the biochemical laboratory of the Biological Work and Health Psychology group at the University of Konstanz and run in duplicates.

Salivary Alpha-Amylase (sAA). sAA was measured at five saliva sampling time points in participants (– 10, + 1, + 10, + 20, + 120 min) and at four sampling time points in reading confederates (– 10, + 1, + 10, + 20). For the determination of sAA, we used an enzymatic

colorimetric assay (alpha Amylase Saliva Assay, RE80111, IBL International GmbH, Hamburg, Germany) following the manufacturer's instructions. Amylase activity was expressed in units per milliliter (U/ml). Inter-assay coefficient of variability (CV) was 12.7%, and intra-assay CV was 1.9% in our sample. sAA data of three stress observers and of one placebo-stress observer are missing because of technical problems.

Salivary Cortisol. Salivary cortisol was measured at seven saliva sampling time points in all participants (− 10, + 1, + 10, + 20, + 30, + 45, + 120 min) and at five saliva sampling time points in the reading confederates (− 10, + 1, + 10, + 20, + 30 min). To measure salivary free cortisol levels, we used enzyme-linked-immunosorbent assays (ELISAs) according to the manufacturer's instructions (Cortisol Saliva ELISA, RE52611, IBL International GmbH, Hamburg, Germany). Inter- and intra-assay CVs were 9.2 % and 5.7 % in our sample. The detection limit was 0.003 µg/dl.

Salivary Aldosterone. Salivary aldosterone was measured at seven saliva sampling time points in all participants (− 10, + 1, + 10, + 20, + 30, + 45, + 120 min) and at five saliva sampling time points in the reading confederates (− 10, + 1, + 10, + 20, + 30 min). Salivary aldosterone levels were measured using a commercial ELISA (Aldosterone ELISA, RE52301, IBL International GmbH, Hamburg, Germany) according to the manufacturer's specifications, using 50 µL of saliva instead of plasma. Inter- and intra-assay CVs were 6.4% and 3.6% in our sample. The detection limit was 12.07 pg/ml. Due to technical problems, salivary aldosterone data are missing for two placebo-stress observers and one reading confederate in one obsPlacTSST.

Heart Rate Assessment

To obtain information on physiological reactivity *during* (placebo-)stress observation, participants were equipped with HR recording chest belts and sensors (Polar H10, Polar Electro GmbH, Büttelbronn, Germany), and we continuously recorded HR with the application Heart Rate Variability Logger (A.S.M.A. B.V., Amsterdam, The Netherlands). For analyses of HR

data in TSST and observer participants, means were calculated for six time intervals of three min duration: resting HR (– 16 to – 14 mins prior to the experimental manipulation), mock job interview (mins 2–4 of the mock job interview), mental arithmetic task (mins 2–4 of the mental arithmetic task), saliva sampling time points + 1 min (0–2 min after the experimental manipulation), + 10 min (9–11 min after the experimental manipulation), + 20 min (19–21 min after the experimental manipulation), and + 120 min (119–121 min after the experimental manipulation). Due to recording problems, HR data of seven stress observers and two TSST participants are missing.

In TSST participants and control condition reading confederates, HR was assessed via sphygmomanometry (Omron, M300, Nufringen, Germany) following the standard protocols of the TSST and PlacTSST (Het et al., 2009; Kirschbaum et al., 1993). For the comparison of HR reactivity between TSST participants and confederates, we considered a total of six HR measurement time points: a baseline measurement comprising the mean of two measures obtained – 15 min and – 10 min prior to the start of the experimental procedure, one measurement 2 min after the beginning of the mock job interview, one measurement 2 min after the beginning of the mental arithmetic task, as well as three measurements after cessation of the experimental procedure, i.e., + 1, + 10, and + 20 min. HR assessment during the experimental manipulation failed in four TSST participants.

Statistical Analyses

Data were analyzed using SPSS (Version 29.0) statistical software packages for Macintosh (IBM SPSS Statistics, Chicago IL, USA) and are presented as mean $\pm$ standard error of the mean (SE). All tests were two-tailed with the significance level set at $\alpha = .05$. Missing data were listwise excluded for the respective parameter.

We a-priori calculated power analyses using the statistical software G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany) based on previous literature on stress contagion effects for cortisol (Buchanan et al., 2012; Engert et al.,

2014; Erkens et al., 2019; Schury et al., 2020). To allow for the detection of conservatively expected small effect sizes of $f = 0.1$ with a power of $(1 - \beta) = .80$ in repeated measures analysis of variance (ANOVA) with two groups and seven repeated measures given the presumed lowest average correlation of $r = 0.7$ for cortisol, the required total sample size is $N = 60$.

Prior to statistical analyses, all data were tested for normal distribution using Kolmogorov-Smirnov and for homogeneity of variance using Levene's tests. As the assumption of normality was not met for cortisol and aldosterone, these data were transformed using the natural logarithm. To protect against violations of sphericity, we applied Huynh-Feld correction where appropriate. Effect size parameters f were calculated from partial η^2 (η_p^2) using G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany) and are reported where appropriate (effect size conventions f : .10 = small; .25 = medium; .40 = large (Cohen, 1988)). Body mass index (BMI) was calculated using the formula $\text{BMI} = \text{kg/m}^2$. For the graphical illustration of our findings, we used absolute changes from baseline of original data for reasons of clarity.

To test for differences in participant characteristics, we compared our participant groups in terms of demographic measures and baseline levels of physiological measures using univariate analyses of variance (ANOVA). Moreover, we tested for potential differences in panel size between experimental and control condition calculating an independent t-test.

To test for successful stress induction by the obsTSST as well as successful realization of the obsPlacTSST, we calculated repeated measures ANOVAs with group (TSST participants vs. reading confederates) as independent variable and repeated measurement time points of HR, sAA, cortisol, or aldosterone levels as repeated dependent variables. Post-hoc t -tests comprised testing for group differences in changes from baseline in terms of univariate ANOVAs.

As main analyses, we tested for differences in physiological reactivity in reaction to direct stress observation as compared to placebo-stress observation by calculating repeated measures ANOVAs with group (stress observers vs. placebo-stress observers) as independent

variable and repeated measurement of HR, sAA, cortisol, or aldosterone levels as repeated dependent variables. Due to potentially confounding effects of age and BMI on cortisol, sAA, and aldosterone reactivity (Gideon, Sauter, Pruessner, et al., 2022; Mennella et al., 2014; Strahler et al., 2010; Therrien et al., 2010), main analyses of these parameters were performed with age and BMI as covariates. Post-hoc testing of significant interaction effects comprised testing for group differences in changes from baseline in terms of univariate ANOVAs, as well as analyses for each group separately in terms of repeated measures ANOVAs with baseline and every later measurement time point. To further characterize direct stress observation reactivity as compared to a first-hand stress reaction, we accordingly performed analyses comparing TSST participants and stress observers (TSST participants vs. stress observers) for those measures that yielded significant interaction effects for the comparison between stress observers and placebo-stress observers.

Our experimental design may entail potential violations of the assumptions of the applied inferential-statistical procedures (ANOVAs and post-hoc *t*-tests). Namely, dependent variable values from observer participants in the stress observation condition and the placebo-stress observation condition were not necessarily independent from one another as these participants were nested in the respective study groups. It follows that a study group serves as a potential additional source of variance that is not considered in the inferential-statistical procedures and might cause an underestimation of the standard errors. Therefore, we computed intraclass-correlation coefficients (ICCs) for all dependent variables (that is all direct measures and all changes from baseline) at every measurement time point in order to quantify the proportion of variance due to study group. Moreover, we computed complementary structural equation models (SEMs) in which the study group is considered as an additional source of variance and in which the interdependence between the TSST participants and their respective study group is considered.

Results

Participant Characteristics

Our final sample comprised 20 TSST participants, 36 stress observers, and 30 placebo-stress observers. Participant characteristics are depicted in Table 1. As expected, participants did not significantly differ in terms of age and BMI as well as in terms of physiological measures at baseline (p 's $\geq .074$). Moreover, panel size did not differ between stress observation ($2.85 \pm .17$) and placebo-stress observation condition ($2.67 \pm .26$) ($t(30) = .63, p = .54$).

Intraclass-Correlation Coefficients

Table 2 shows the ICCs for all dependent variables at every measurement time point. With the exception of aldosterone and changes from baseline in aldosterone and HR, the proportion of variance that can be explained by study group was generally rather low (both for stress observers and placebo-stress observers), which suggests that ANOVAs and t -tests should be rather robust. Nevertheless, alternatives for the inferential-statistical procedures are reported in Supplemental Material 1. These are SEM-analyses that consider potential between-study group differences in accordance with the ICCs. No substantially different conclusions were yielded with the alternative analyses.

Manipulation Check: Comparison between TSST Participants and Reading Confederates

We intended the reading confederates to show very little stress reactions in absolute terms and especially in comparison to TSST participants. We also intended them to show as little variation as possible across the study groups they participated in. Since the two reading confederates knew about these intentions, they trained the situation several times in advance of the study. Figure 1 depicts changes from baseline over time in physiological parameters in TSST participants and reading confederates. Generally, the variation of changes from baseline across the time points and within a specific time point (indicated by the SE bars) was substantially smaller for reading confederates than for the TSST participants. This indicates that, as intended, our reading confederates were not (or not noteworthy) stressed. More

specifically, TSST participants exhibited greater increases with respect to salivary cortisol, salivary aldosterone, sAA, and HR in reaction to the obsTSST as compared to the reading confederates in reaction to the obsPlacTSST (interactions group-by-time: cortisol: $F(1.84, 55.20) = 9.62, p < .001, \eta_p^2 = .24, f = .56$; aldosterone: $F(4.00, 116.00) = 2.11, p = .085, \eta_p^2 = .07, f = .27$; sAA: $F(1.89, 56.56) = 11.89, p < .001, \eta_p^2 = .28, f = .62$; HR: $F(2.57, 66.79) = 10.84, p < .001, \eta_p^2 = .29, f = .64$). Post-hoc analyses revealed that for cortisol, sAA, and HR, TSST participants and reading confederates differed in all changes from baseline to separate post-manipulation time points (p 's $\leq .009$) with higher increases in the TSST participants. For salivary aldosterone, higher changes from baseline to separate post-manipulation time points in TSST participants compared to reading confederates were only observed + 20 min after obsTSST/obsPlacTSST cessation ($p = .035$).

Observer Reactivity: Comparison between Stress Observers and Placebo-Stress Observers

As expected, stress observers exhibited greater increases with respect to salivary cortisol, sAA, and HR in reaction to the observation of the obsTSST as compared to placebo-stress observers in reaction to the observation of the obsPlacTSST (see Figure 2; interactions group-by-time: cortisol: $F(2.67, 165.77) = 4.87, p = .004, \eta_p^2 = .07, f = .27$; sAA: $F(3.59, 208.30) = 3.47, p = .012, \eta_p^2 = .06, f = .25$; HR: $F(4.34, 246.91) = 2.72, p = .027, \eta_p^2 = .05, f = .23$). There were no group differences in salivary aldosterone reactivity ($F(4.98, 298.65) = 1.21, p = .31$). Post-hoc analyses of significant group-by-time interactions, i.e., for cortisol, sAA, and HR are detailed in Table 3 (see Supplemental Material 2, Table S4 for detailed aldosterone data). We found significant group differences in changes from baseline between stress and placebo-stress observers at + 1 min in all parameters and at + 10 min in cortisol and sAA (p 's $\leq .036$) with greater increases from baseline in stress observers. Further post-hoc

testing comprised analyses of changes from baseline over time in the groups separately (see Table 3).

ObsTSST: Comparison between TSST Participants and Stress Observers

To characterize the physiological reactivity to direct stress observation compared to a first-hand physiological stress reaction, we compared the identified stress observation reactive parameters cortisol, sAA, and HR between TSST participants and stress observers. TSST participants exhibited greater increases with respect to salivary cortisol, sAA, and HR in reaction to undergoing the obsTSST as compared to stress observers in reaction to observation of the obsTSST (interactions group-by-time: cortisol: $F(2.72, 141.58) = 22.32, p < .001, \eta_p^2 = .30, f = .65$; sAA: $F(3.05, 149.64) = 4.95, p = .002, \eta_p^2 = .09, f = .31$; HR: $F(2.26, 101.64) = 38.75, p < .001, \eta_p^2 = .46, f = .92$). Post-hoc analyses regarding group differences in changes from baseline between TSST participants and stress observers revealed significant differences in changes from baseline at post obsTSST measurement time points for cortisol until + 45 min (p 's $< .001$), for sAA at + 1 min ($p = .005$) as well as for HR at measurement time points during and after the obsTSST until + 20 min (p 's $\leq .006$), with TSST participants exhibiting greater increases. To allow for further characterization of the reactivity kinetics in each group, additional post-hoc testing comprised analyses of changes from baseline over time in TSST participants and stress observers separately (see Table 3). Except for decreases in cortisol and HR and no changes in sAA at + 120 min, TSST participants showed the regular reactivity kinetics with significant increases from baseline at all other measurement time points in cortisol, sAA, and HR (p 's $\leq .006$). In stress observers, cortisol levels significantly increased from baseline to + 1 min post obsTSST ($p = .017$) and decreased at + 45 min and + 120 min post obsTSST. sAA levels significantly increased from baseline to all post obsTSST measurement time points in stress observers (p 's $\leq .024$) except at + 120 min. With respect to HR, we observed significant increases in HR levels from baseline at + 1 min ($p < .001$) as well as decreases at + 120 min post obsTSST.

Discussion

Here, we investigated for the first time a paradigm to study physiological reactivity to direct stress observation as compared to direct observation of a comparable but placebo-stress control condition to control for secondary effects of the stress observation setting and task. In our standardized, controlled paradigm, participants in the experimental condition directly observed another participant undergoing an adapted version of the TSST. Unlike previous stress contagion studies with direct stress observation, stress observers had no additional own potentially first-hand stress-inducing tasks. In the control condition, participants observed a confederate undergoing an adapted version of the PlacTSST. Moreover, we examined the full reactivity kinetics of the stress contagion responses with respect to the major endocrine stress axes, including the RAAS, for the first time in this context. We repeatedly assessed HR and sAA (SAM axis), salivary cortisol (HPA axis), as well as salivary aldosterone (RAAS) before, during, and after (placebo-)stress (observation) in TSST participants, stress observers, reading confederates, and placebo-stress observers.

As expected, and in line with previous stress studies (e.g., Gideon, Sauter, Pruessner, et al., 2022; Het et al., 2009; Kothgassner et al., 2021), the obsTSST induced higher reactivity with respect to all assessed physiological parameters (sAA, HR, salivary cortisol, and salivary aldosterone) compared to the obsPlacTSST, indicating successful implementation of the stress and placebo-stress experimental manipulation.

Regarding the main findings of our study, we first found that stress observers exhibited higher salivary cortisol, sAA, and HR reactivity to the direct observation of participants undergoing the obsTSST as compared to placebo-stress observers to the observation of the obsPlacTSST of small to medium effect sizes. Notably, this is the first physiological stress contagion study that includes a placebo-stress observation control condition, controlling for secondary effects of the observation setting and task. Consequently, our findings indicate that the observed higher physiological responses in salivary cortisol, sAA, and HR observed in

reaction to stress observation can specifically be attributed to direct stress observation (Green et al., 2014; Shapiro et al., 1978). There were no differences in salivary aldosterone reactivity to stress observation compared to placebo-stress observation. Therefore, in contrast to the SAM axis and the HPA axis, the RAAS does not seem to be sensitive to stress contagion effects. Moreover, this is the first physiological stress contagion study that allowed for direct face-to-face stress observation without additional potentially first-hand stress-inducing tasks such as active stress induction in others as TSST committee member (Buchanan et al., 2012) or preparing for own stress exposure (Blons et al., 2021). Our results underpin the stress contagion responses observed in reaction to non-direct stress observation via video or one-way mirror (Dimitroff et al., 2017; Engert et al., 2014; Engert et al., 2018; Erkens et al., 2019; Park et al., 2021; Schury et al., 2020). Interestingly, responder rates of stress observers, i.e., the percentage of individuals showing a physiologically significant increase in cortisol (≥ 1.5 nmol/l; Miller et al., 2013), in our study with direct stress observation and no additional potentially first-hand stress-inducing tasks, was 41.67%. Non-direct observation via one-way mirror yielded a responder rate of 30% (Engert et al., 2014) and via video of 16–24% (Engert et al., 2014; Erkens et al., 2019; Schury et al., 2020). Unfortunately, cortisol responder rates are not reported in the two hitherto published studies with direct stress observation and additional potentially first-hand stress-inducing tasks (Blons et al., 2021; Buchanan et al., 2012). The higher responder rates with direct stress observation from our study as compared to non-direct stress observation may suggest that the direct access to interactive sensory information within the observed situation is of major importance for the extent of stress contagion (Daft & Lengel, 1986).

Our second main finding relates to the full reactivity kinetics of the observed physiological stress contagion responses. Our observed increases in cortisol and sAA to direct stress observation align with previous studies that investigated physiological stress contagion in terms of significant physiological increases following direct (Buchanan et al., 2012) and indirect stress observation (Engert et al., 2014; Erkens et al., 2019; Schury et al., 2020) in adult

individuals. In contrast to previous studies, we did not limit our analyses to aggregated single measures such as increases from baseline to peak in cortisol and sAA (Buchanan et al., 2012; Engert et al., 2014; Erkens et al., 2019; Schury et al., 2020) or mean HR reactivity (Engert et al., 2014), but investigated the full kinetics of the stress contagion responses. With respect to *cortisol* and *sAA*, the reactivity of stress observers mirrored the reactivity of the TSST participants but less pronounced and in terms of cortisol with earlier peak levels. *HR* was the only parameter also assessed during the observation task. First-hand HR stress reactivity of TSST participants was strongest during the obsTSST, while in stress observers, HR did not change during stress observation but markedly increased after cessation of the obsTSST observation. This absence of HR increases during stress observation may point to an initial freezing response with respect to the SAM axis that may relate to the ambivalence of the situation regarding the origin of the danger, own risk, and the need for potential actions. This may be interpreted in that an ambivalent situation has to be terminated first before a stress contagion response can take place. Notably, the absence of HR increases during the stress observation observed in our study is contradictory to the only other stress contagion study that examined HR during stress observation (Engert et al., 2014) where HR increases were observed already during stress observation. However, in that study, participants observed stress induction by the TSST via one-way mirror or video and not in the same room. Thus, own threat and the need and/or possibility for action, either in terms of escape or defense or in terms of support for the stressed individuals, was precluded, and stress contagion occurred immediately, potentially explaining the divergent HR results.

With regard to mechanisms underlying physiological stress contagion, empathic processes proposed in the context of emotional contagion could also apply to physiological stress contagion (Engert et al., 2019; White & Buchanan, 2016). In explanatory models for empathy, such as the Perception-Action Model for Empathy (PAM; Preston & De Waal, 2002) or the Neurocognitive Model of Emotional Contagion (NMEC; Prochazkova & Kret, 2017), it

is assumed that individuals have shared neural representations for the perception and the generation of actions (Preston & De Waal, 2002). Direct (conscious) perception of an emotional state in another individual, as proposed to be reflected in nonverbal motor movements and physiological activity, automatically activates the corresponding representation in the observing person (Prochazkova & Kret, 2017). Both models posit that this shared neural activation subsequently induces the activation of the respective autonomic and somatic response patterns mimicking the observed emotional state on motor and autonomic levels. In terms of stress contagion, this mimicry would be reflected by the physiological stress contagion response. The models further propose that the physiological mimicry response eventually induces a similar emotional state as observed in the other individual (Preston, 2007; Prochazkova & Kret, 2017). Notably, to allow for automatic activation of the corresponding representation, a certain degree of attention to the state of the other individual is required (Preston, 2007). Also, emotional states can only be mirrored to the degree that a representation of the corresponding state is available (Preston, 2007).

What are potential implications of physiological stress contagion? The physiological activation resulting from stress observation has been proposed to reflect a form of non-verbal communication (Engert et al., 2019). It may point to imminent danger as the source of stress may also pose a threat for oneself or could be interpreted as a silent request for help. In line with this, moderate physiological activation enhances, on the one hand, on the cognitive level attention and alertness towards the surrounding environment and, on the other hand, supports behavioral responses such as assistance for the distressed individual or escaping the threat by providing energy (Calabrese, 2008; Mendl, 1999). Moreover, an empathic stress contagion response could prepare for dealing with possible similar stressors in the future, even if the situation does not have immediate personal relevance (Park et al., 2021). Future studies are needed to investigate whether the possibly frequent (e.g., in environments with chronically stressed others; in the work or family context) but compared to first-hand stress lower

physiological reactivity in response to stress contagion is capable of inducing maladaptive consequences for health as observed with first-hand stress exposure (Chrousos, 2009; McEwen, 1998). In addition, the effects of stress contagion on cognitive and behavioral levels should be further elucidated to obtain a comprehensive understanding of the impact of stress contagion. As not all individuals react physiologically to observing stress in others, further potential modulating factors apart from empathy (Blons et al., 2021; Buchanan et al., 2012; Dimitroff et al., 2017; Engert et al., 2014; Schury et al., 2020), social identity (Blons et al., 2021; Schury et al., 2020), and familiarity (Engert et al., 2014) should be investigated, such as e.g., emotion recognition abilities.

Strengths of our study include the standardized laboratory setting which allowed to control for a variety of confounders. In addition, with our standardized, controlled paradigm, we were for the first time able to investigate stress contagion in a direct, face-to-face observation setting while controlling for secondary effects of the observation setting. Notably, our standardized controlled paradigm was based on well-established paradigms with the stress induction procedure TSST (Kirschbaum et al., 1993) and its control procedure PlacTSST (Het et al., 2009). Remarkably, the observation manipulation, i.e., the irregular composition of the obsTSST panel in terms of participants as passive panel members in addition to the active instructing panel confederate, had not been suspected by any of the stressed participants during the obsTSST as verified by self-report during the debriefing. Moreover, our study design allowed to comprehensively characterize the physiological stress contagion response of stress contagion-responsive human stress axes, including reactivity kinetics. Limitations of our study include the limited generalizability of our findings beyond healthy, medication-free, non-smoking young men. In our proof-of-concept study, we intentionally restricted participation to male participants in order to exclude potential confounding by sex (Kajantie & Phillips, 2006; Liu et al., 2017) and sex composition of the panel (Goodman et al., 2017). Future studies are needed to detect possible sex and gender differences. Moreover, the generalizability of our

laboratory findings to everyday life is unclear despite first evidence for ecological validity for covariation in naturalistic settings in couples (Engert et al., 2018). Notably, in order to minimize potential confounding effects due to interindividual differences in energy availability, TSST participants, stress observers, placebo-stress observers, and reading confederates drank grape juice prior to the start of the experimental manipulation. However, glucose administration by grape juice may have amplified HPA axis reactivity in our male participants (Zänkert et al., 2020) and potentially altered affective reactivity (Kern et al., 2008; Zink et al., 2020). We cannot rule out that the generalizability of our findings has been affected. Furthermore, study groups (e.g., interindividual differences in stress reactivity of TSST participants) may serve as a potential additional source of variance that is not considered in the inferential-statistical procedures of ANOVAs and *t*-tests. While in the present study, this was not a major issue (see Table 2 and Supplemental Material 1), future studies should always consider this potential source of variance and apply alternative analyses when appropriate (see Supplemental Material 1). A statistical shortcoming of our study was the fact that, due to practical reasons, we had two different reading confederates, which is not ideal for the manipulation check (comparisons between TSST participants and reading confederates). Notably, one reading confederate would have been ideal to reduce variance to a minimum. While it is statistically possible to control for interindividual differences of different confederates, the sample size was too small in this case as one confederate worked with nine study groups of placebo-stress observers and the other one with the remaining three. With respect to the study design, a reading confederate should show only very little variation in stress measures across study-groups as he technically reflects a constant across study groups – every existing variation should be solely attributable to small random error. In our study, this variation was still very small despite the fact that a second confederate had to be employed. Nevertheless, future studies should opt for either employing a single confederate in order to avoid any systematic variation or provide each of the several confederates with a sufficient number of study groups so that the systematic interindividual

variability across confederates can be investigated. Moreover, we cannot rule out that the presence of an instructing confederate and/or the assigned role as observer participant may induce mild social evaluative stress in the observer participants. However, to account for these potential confounding effects, among others, the control condition was designed to parallel the experimental condition with the amount of stress of the observed person (i.e., TSST participant or reading confederate) as the only difference between conditions. Thus, while a minor part of the reactivity observed in the stress observation condition may result from mild social evaluative stress (namely to the extent of reactivity observed in the placebo-stress observation control condition), the *difference* in the reactivity between stress observers and placebo-stress observers can most likely be attributed to stress contagion effects. Finally, although our results provide a decent basis for further research on physiological stress contagion, several questions remain open. Future studies are needed to investigate psychological aspects such as feelings of novelty and uncontrollability in all participants in more detail in addition to further potentially modulating factors.

Taken together, our study findings reveal that observing another individual that encounters a stressor leads to activation of the HPA axis and the SAM axis, i.e., physiological stress contagion in the observer. This physiological reactivity is comparable to but less pronounced than physiological reactivity to first-hand stress. The RAAS, on the other hand, does not seem to be sensitive to physiological stress contagion. Future studies are needed to further investigate generalizability to populations other than medication-free, non-smoking, healthy, young men, the role of observation modality and other potential modulating factors as well as implications for health, cognition, and behavior and, thus, everyday life.

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Statement of Ethics: The study was approved by the Ethics Committee of the University of Konstanz and conducted in accordance with the Declaration of Helsinki principles. All participants provided written informed consent.

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Tables

Table 1*Participants Characteristics*

	TSST participants (<i>n</i> = 20)	Stress observer (<i>n</i> = 36)	Placebo-stress observer (<i>n</i> = 30)	<i>p</i>
Age [years]	23.30 ± 0.45 (20–28)	23.14 ± 0.54 (19–30)	23.57 ± 0.46 (20–29)	.82
BMI [kg/m ²]	22.69 ± 0.46 (18.28–26.97)	23.40 ± 0.43 (17.79–29.74)	23.72 ± 0.49 (18.49–29.01)	.36
Baseline sAA [U/ml]	165.20 ± 20.28 (8.24–333.03)	<i>n</i> = 33 159.17 ± 16.21 (33.47–398.05)	<i>n</i> = 29 163.17 ± 20.58 (9.34–527.90)	.98
Baseline salivary cortisol [nmol/l]	4.43 ± .39 (1.53–8.80)	6.17 ± .51 (1.85–15.04)	6.13 ± .61 (2.03–18.55)	.074
Baseline salivary aldosterone [pg/ml]	75.41 ± 6.17 (28.67–136.29)	72.90 ± 4.42 (29.83–133.45)	<i>n</i> = 28 81.11 ± 5.26 (40.11–152.73)	.49
Baseline HR [bpm]	<i>n</i> = 18 74.87 ± 1.96 (63.21–93.22)	<i>n</i> = 29 77.39 ± 2.16 (55.57–101.56)	76.93 ± 1.78 (61.52–102.11)	.70

Note. Values are means ± SE; sAA = salivary alpha-amylase; HR = heart rate assessed with Polar 10; *n* = number of participants.

Table 2

Intraclass-Correlation Coefficients (ICCs) for All Dependent Variables at Every Point in Time with Assessment-Session at the Higher Level and Individual at the Lower Level

	Baseline	Speech task	Mental arithmetic task	+ 1 min	+ 10 min	+ 20 min	+ 30 min	+ 45 min	+ 120 min
Cortisol	.03 (.08)	-	-	0 (.14)	0 (.08)	0 (.12)	0 (.34)	0 (.29)	.44 (0)
sAA	.42 (0)	-	-	.05 (0)	.09 (0)	.09 (0)	-	-	0 (0)
Aldosterone	.40 (.32)	-	-	.25 (.27)	.49 (.24)	.37 (.11)	.44 (.22)	.15 (.09)	.22 (.35)
HR	0 (.02)	0 (.31)	0 (.12)	0 (0)	0 (0)	0 (0)	-	-	.01 (0)
Δ Cortisol	-	-	-	0 (0)	0 (0)	.02 (0)	.08 (0)	0 (0)	0 (0)
Δ sAA	-	-	-	0 (0)	0 (0)	.03 (0)	-	-	0 (.48)
Δ Aldosterone	-	-	-	.20 (0)	.27 (0)	.34 (0)	.13 (0)	.19 (0)	.27 (.11)
Δ HR	-	.24 (.71)	.37 (.21)	.23 (.64)	0 (.26)	0 (.22)	-	-	.23 (.04)

Note. Values reflect the proportion of variance that is attributable to study group. Free values relate to stress observers, values in parentheses relate to placebo-stress observers. Δ reflects a change from baseline. Raw measures for cortisol and aldosterone were transformed using the natural logarithm. Analyses comprise $N = 36$ stress observers for cortisol and aldosterone, $n = 33$ for sAA, and $n = 29$ for HR; $N = 30$ placebo-stress observers for cortisol and HR, $n = 29$ for sAA, and $n = 28$ for aldosterone.

Table 3

Post-Hoc Testing of Differences in Physiological Reactivity between Stress Observers in Reaction to Observation of the obsTSST and Placebo-Stress Observers in Reaction to Observation of the obsPlacTSST as well as between Stress Observers and TSST Participants

		Cortisol		sAA		HR	
		Change in nmol/l from baseline (<i>p</i> -value)	<i>p</i> -value group comparison with stress observers	Change in U/ml from baseline (<i>p</i> -value)	<i>p</i> -value group comparison with stress observers	Change in bpm from baseline (<i>p</i> -value)	<i>p</i> -value group comparison with stress observers
Speech task	Stress observers	-		-		1.10 ± 1.07 (.31)	
	Placebo-stress observers	-	-	-	-	1.10 ± 1.10 (.33)	.99
	TSST participants	-	-	-	-	27.38 ± 4.44 (< .001)	< .001
Mental arithmetic task	Stress observers	-		-		0.49 ± 0.94 (.61)	
	Placebo-stress observers	-	-	-	-	-0.49 ± 0.74 (.52)	.42
	TSST participants	-	-	-	-	26.83 ± 3.92 (< .001)	< .001
+ 1 min	Stress observers	1.61 ± 0.59 (.017)		93.27 ± 14.35 (< .001)		7.13 ± 1.13 (< .001)	

	Placebo-stress observers	-0.74 ± 0.32 (.065)	.002	48.59 ± 10.20 (< .001)	.016	2.22 ± 0.88 (.017)	.001
	TSST participants	5.64 ± 0.93 (< .001)	< .001	192.73 ± 36.70 (< .001)	.005	14.22 ± 2.14 (< .001)	.002
+10 min	Stress observers	1.21 ± 0.67 (.29)		59.09 ± 15.62 (< .001)		1.70 ± 0.83 (.050)	
	Placebo-stress observers	-0.84 ± 0.42 (.053)	.015	12.04 ± 15.25 (.44)	.036	-0.69 ± 1.17 (.56)	.10
	TSST participants	10.49 ± 1.62 (< .001)	< .001	86.78 ± 25.61 (.003)	.33	5.72 ± 1.16 (< .001)	.006
+ 20 min	Stress observers	0.29 ± 0.60 (.91)		26.29 ± 11.07 (.024)		0.28 ± 0.70 (.67)	
	Placebo-stress observers	-1.31 ± 0.53 (.005)	.054	-3.70 ± 11.64 (.75)	.067	-1.01 ± 0.82 (.23)	.23
	TSST participants	9.12 ± 1.69 (< .001)	< .001	68.00 ± 21.92 (.006)	.066	4.75 ± 1.42 (.004)	.003
+ 30 min	Stress observers	-0.54 ± 0.52 (.11)		-		-	
	Placebo-stress observers	-1.54 ± 0.55 (.002)	.52	-	-	-	-
	TSST participants	6.56 ± 1.65 (< .001)	< .001	-	-	-	-
+ 45 min	Stress observers	-1.24 ± 0.45 (.007)		-		-	
	Placebo-stress observers	-1.60 ± 0.56 (.003)	.61	-	-	-	-

	TSST participants	4.14 ± 1.34 (< .001)	< .001	-	-	-	-
	Stress observers	-2.45 ± 0.57 (< .001)		19.30 ± 10.59 (.078)		-6.02 ± 1.17 (< .001)	
+ 120 min	Placebo-stress observers	-1.63 ± 0.69 (.018)	.36	22.73 ± 15.58 (.16)	.85	-6.93 ± 1.15 (< .001)	.58
	TSST participants	-1.24 ± 0.51 (.008)	.17	34.93 ± 24.60 (.17)	.51	-3.88 ± 1.38 (.012)	.25

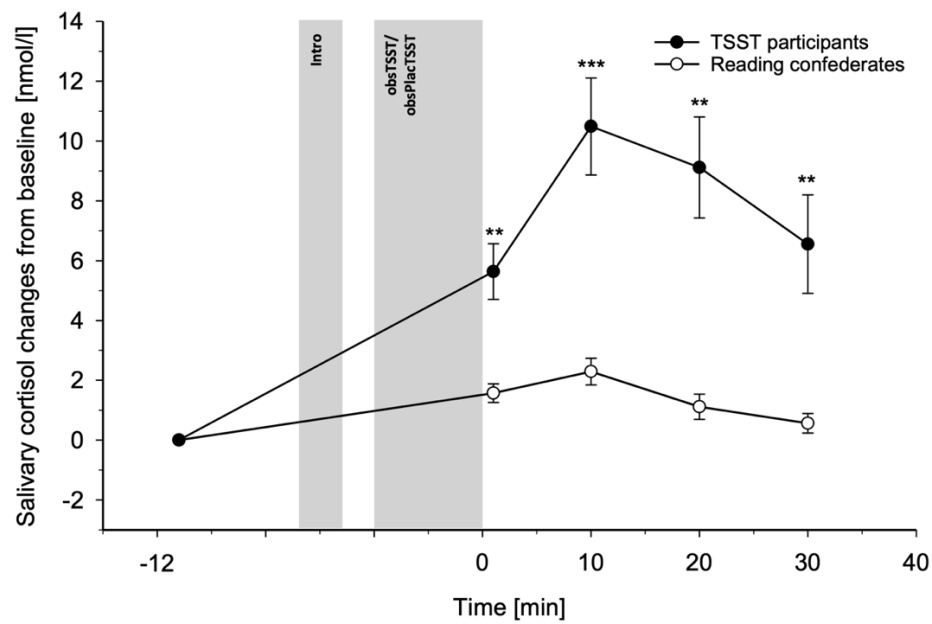
Note. Values are means ± SE if not indicated differently; sAA = salivary alpha-amylase; HR = heart rate assessed with Polar 10; *p*-values change to baseline = main effect of time of repeated measures ANOVAs comprising baseline and respective other individual measurement time point; *p*-values group comparison = interactions group-by-time of repeated measures ANOVAs comprising baseline and respective other individual measurement time point as well as group (TSST participants vs. reading confederate); analyses comprise *N* = 36 stress observers for cortisol, *n* = 33 for sAA, and *n* = 29 for HR; *N* = 30 placebo-stress observers for cortisol and HR, and *n* = 29 for sAA, as well as *N* = 20 TSST participants for cortisol, sAA, and *n* = 18 for HR; significant values are highlighted in bold (*p* < .05).

Figures

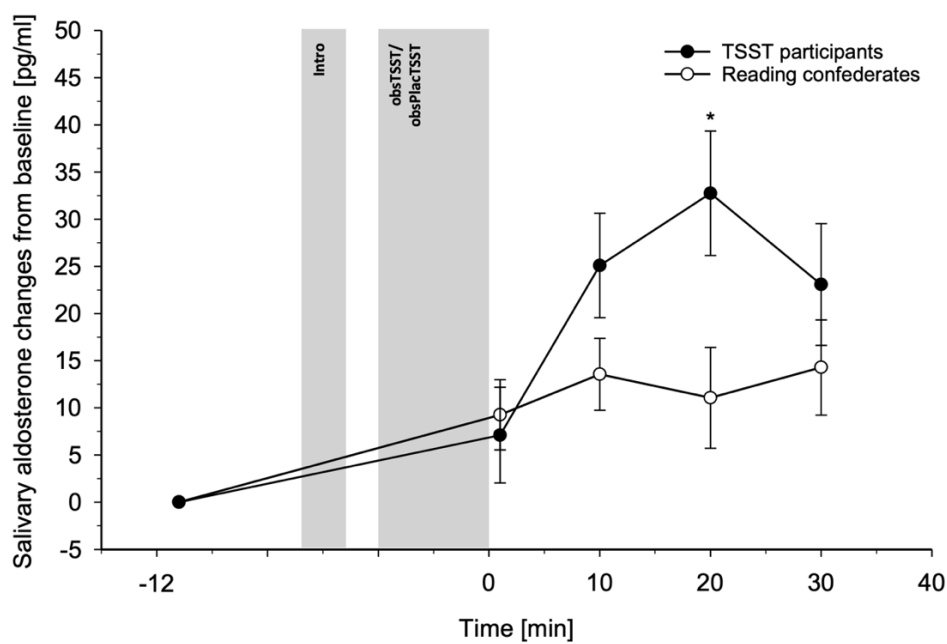
Figure 1

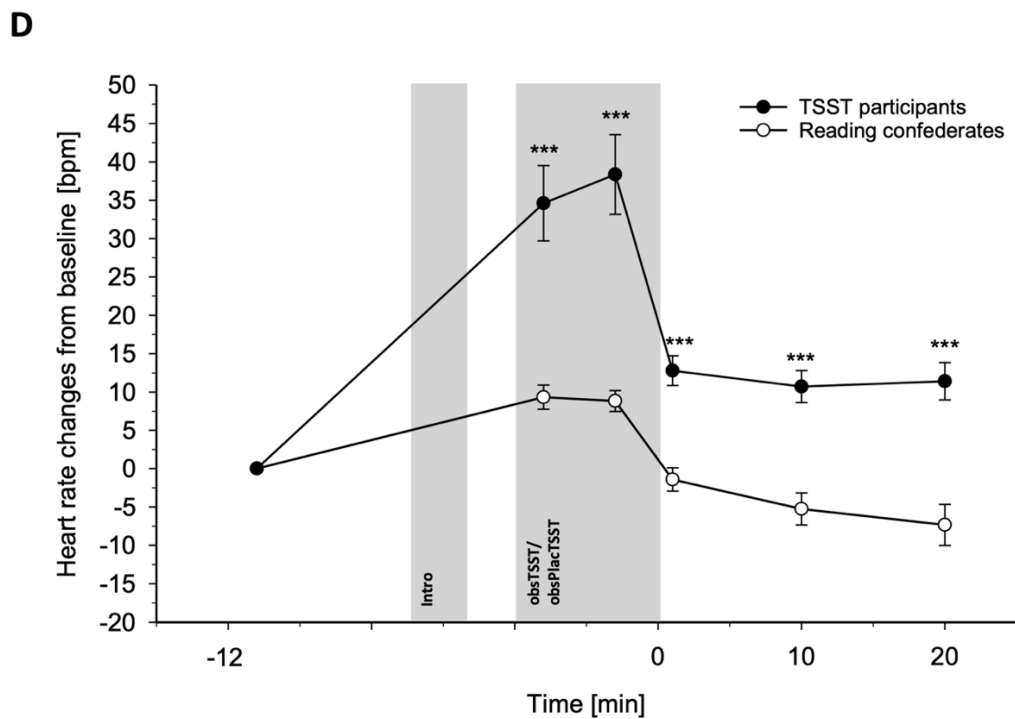
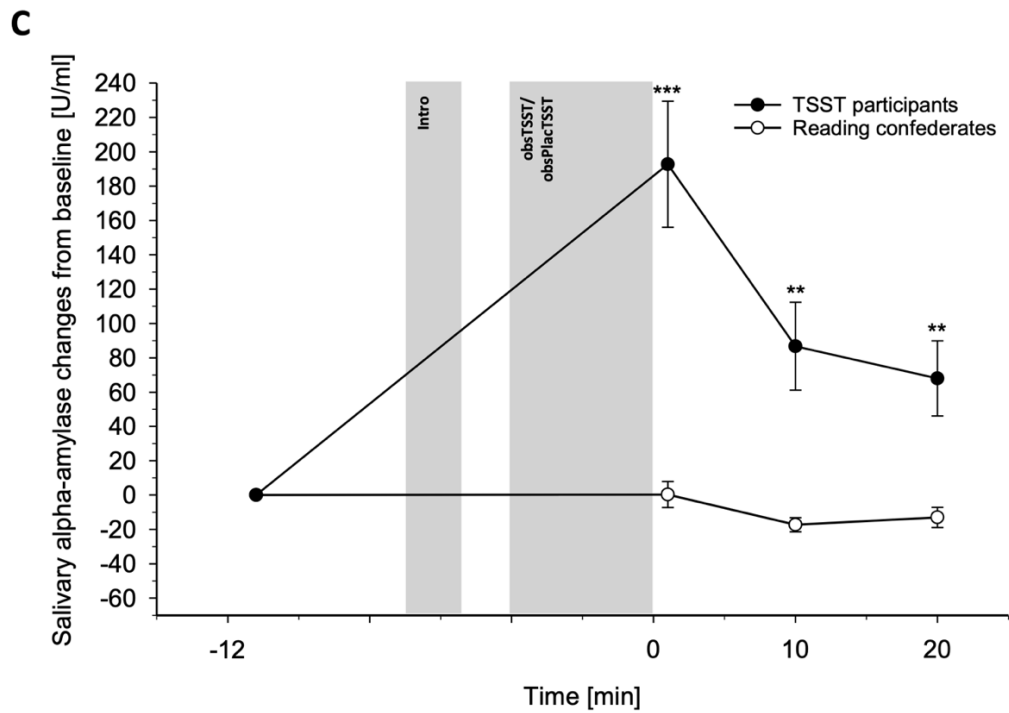
Physiological Reactivity (Mean $\pm$ SE) of TSST Participants to Our Adapted Version of the Trier Social Stress Test (obsTSST) (Black Dots) and of Reading Confederates to Our Adapted Version of the Placebo-TSST (obsPlacTSST) (White Dots)

A



B

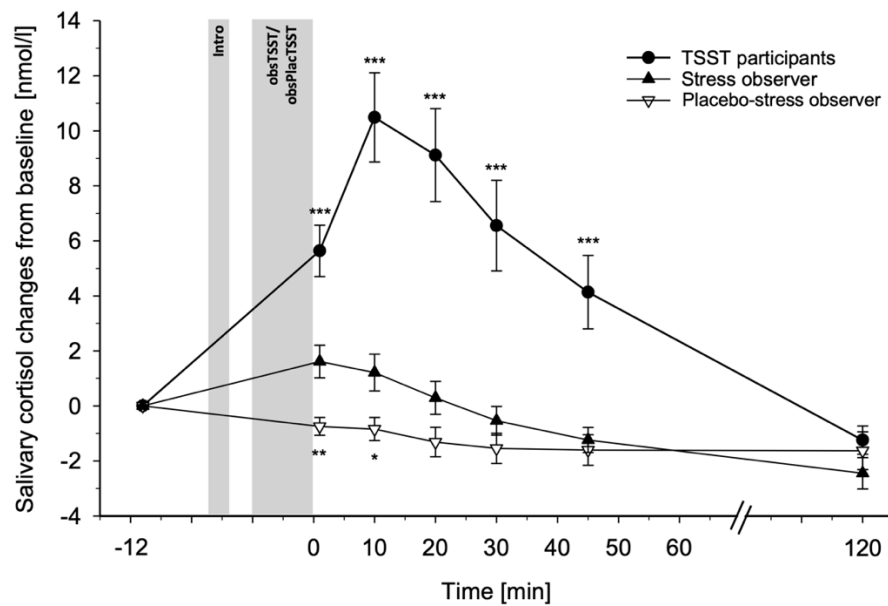
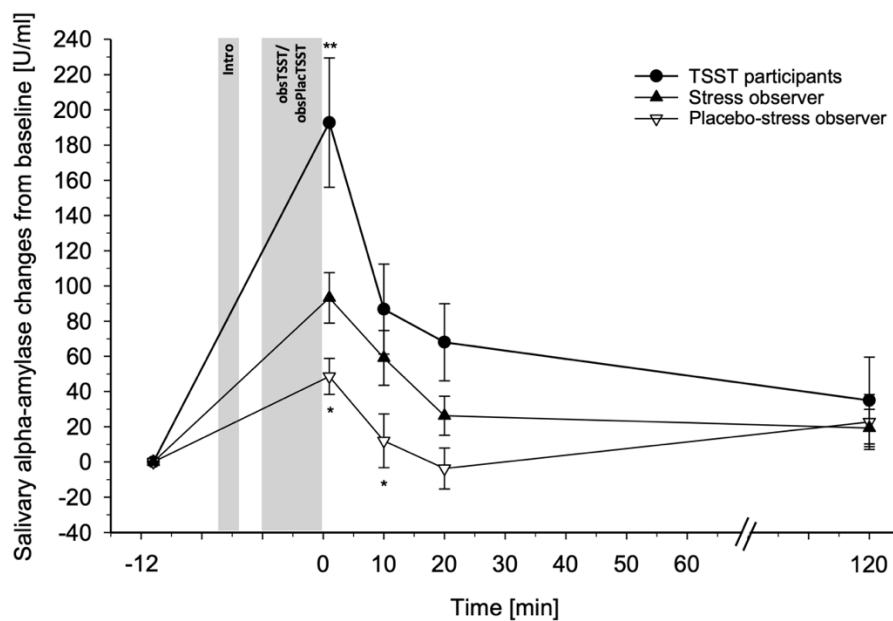


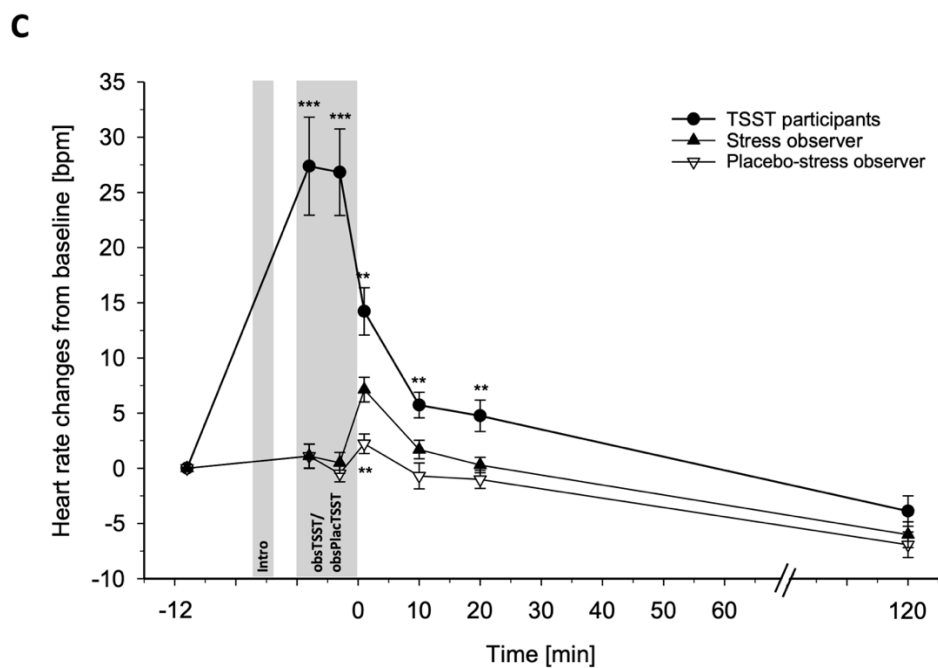


Note. (A) Salivary cortisol. (B) Salivary aldosterone. (C) Salivary alpha-amylase. (D) Heart rate. Asterisks indicate significant group differences between TSST participants and reading confederates (* $p < .05$; ** $p < .01$; *** $p < .001$).

Figure 2

Physiological Reactivity (Mean $\pm$ SE) of Stress Observers (Black Triangles) and TSST Participants (Black Dots) to Our Adapted Version of the Trier Social Stress Test (obsTSST) and of Placebo-Stress Observers (White Triangles) to Our Adapted Version of the Placebo-TSST (obsPlacTSST) (White Triangles)

A**B**



Note. (A) Salivary cortisol. (B) Salivary alpha-amylase. (C) Heart rate assessed with Polar 10. Asterisks indicate significant group differences between stress observers and the respective other participant group (* $p < .05$; ** $p < .01$; *** $p < .001$).

Supplemental Material

Supplemental Material 1: Alternative Analyses Respecting Study Group as a Potential Source of Variance Using Structural Equation Modeling (SEM)

Statistical Analyses

We used structural equation modeling (SEM) in order to explicitly model variance of the dependent variables on both the study group level (higher level) and the individual level (lower level) for the stress observers and the placebo-stress observers. To this end, we used the wide-data format approach (Curran, 2003, Mehta & Neale, 2005) with the R-package OpenMx (Neale et al., 2016). The data was restructured so that each row of the data frame reflects one study group as the observational unit. Each column of the data frame relates to a specific measurement of one specific dependent variable at a specific measurement time point for a specific person from the study group. Supplemental Material 2, Table S1 gives an example for this data-structure. With this data-structure, it is possible to respect the two-level structure by specifying latent factors with adequate equality constraints and using the full information maximum likelihood (FIML) method (e. g., Voelkle et al., 2012). Supplemental Material 3, Figure S1 depicts an example. The latent factor variance represents the variance of a measure at the study group level and the residual variance represents the variance of a measure at the individual level. All factor loadings are restricted to one, all variances of latent factors are constrained to be equal, all covariances of latent factors are constrained to be equal, all residual variances are constrained to be equal, and all residual covariances are constrained to be equal.

In order to compare stress observers and placebo-stress observers, one model as depicted in Supplemental Material 3, Figure S1 was specified for both, stress observers and placebo-stress observers, respectively. These two analysis groups were compared using multiple-group SEM (mg SEM). In order to compare the analysis group of stress observers with the analysis group of TSST participants, a single group SEM was used as the TSST participants were also part of the study groups (note, however, that the TSST participants, of course, still reflect a different “group” than the group of stress observers in a substantial but not statistical

sense). To this end, the model for the stress observers as depicted in Supplemental Material 3, Figure S1 was expanded to also include the dependent variables for the TSST participants at every point in time (see Supplemental Material 3, Figure S2). The covariances between all TSST dependent variables and all latent study group-factor variables for the stress observers were estimated. For the TSST dependent variables, all variances were constrained to be equal and all covariances were constrained to be equal. All covariances between latent study group factors and TSST dependent variables related to the same point in time were constrained to be equal and all covariances between latent study group factors and TSST dependent variables related to different points in time were constrained to be equal. The exact procedures for the group comparisons are outlined in the following.

Alternatives for the F-Tests of the ANOVAs

For every dependent variable, we computed four different (mg) SEMs. In the first model (“no effects (ne)”), all means were constrained to be equal across points in time *and* groups (i.e., only one general mean for the dependent variable was estimated). In the second model (“main effect group (meg)”), means were still constrained to be equal across points in time but allowed to vary across groups (i.e., two means were estimated, one per group). In the third model (“main effect time (met)”), means were constrained to be equal across groups but were allowed to vary across time (i.e., the number of estimated means equaled the number of points in time for the respective dependent variable). Finally, in the fourth model (“interaction effect (ie)”), means were allowed to vary across measurement time points *and* groups. Likelihood-ratio tests (using χ^2 -statistics) were conducted to compare the models. A significant χ^2 -statistic between the ne model and the meg model indicates a main effect of group and a significant χ^2 -statistic between the ne model and the met model indicates a main effect of time. Significant χ^2 -statistics between the int model and all previous models indicates a significant interaction effect.

For some models, convergence issues occurred. In these cases, the higher level variances were estimated to be much smaller than zero. This is to be expected when higher levels are not an actual source of variance which often was the case here (see the often very low intra-class correlations in Table 2 from the main manuscript). In such cases, convergence problems were resolved by not modeling the higher level.

Alternatives for the Post-Hoc t-Tests

The general mg SEM strategy was the same as for the *F*-tests. However, only one measurement time point was modeled for every group comparison. A restricted model in which the means were constrained to be equal across groups was compared to an unrestricted model in which the two means were allowed to vary across groups using a likelihood-ratio test. A significant χ^2 -statistic suggests a difference in the change from baseline between the groups. Again, variance at the study group-level was sometimes dismissed when convergence problems occurred.

Results

Alternatives for the ANOVAs

Supplemental Material 2, Table S2 displays the results for the alternatives of the ANOVAs. Interaction effects between group and time for the two observer groups were significant for all raw measures except for aldosterone – just as they were with the ANOVAs. Interaction effects were significant for all comparisons between stress observers and TSST participants for all measures that yielded significant stress contagion effects, i.e., cortisol, sAA, and HR.

Alternatives for the Post-Hoc t-Tests

Supplemental Material 2, Table S3 displays the results for the alternatives for the *t*-tests. In general, the same conclusions as for the *t*-tests were yielded. However, there are some exceptions. Concerning the comparison between stress observers and placebo-stress observers,

additional significant differences in changes from baseline were found for cortisol at + 20 min ($p = .015$) and + 45 min ($p = .038$). Concerning the comparison between stress observers and TSST participants, the difference in sAA-change at +10 min was additionally significant ($p = .027$). However, one must of course note that a lot of inferential-statistical tests were calculated in this study and one would expect some false positives, especially when the p -values do not deviate very much from .05. Generally, the more conservative inferential-statistical stance is taken by interpreting the t -tests instead of the χ^2 -tests, as the t -tests yielded less significant results. While it is true that a neglect of a higher level variance component generally underestimates standard errors, modeling higher level variance sources when ICCs are low (see Table 2 from the main manuscript) can result in biases manifesting in the different direction. As higher level variances were often estimated to be negative (but not statistically different from zero) within the SEMs, the likelihood-ratio tests should be interpreted with caution.

Supplemental Material 2: Tables

Table S1

Example for the Wide-Format Data Structure

session	vt1p1	vt1p2	vt1p3	vt2p1	vt2p2	vt2p3	vt3p1	vt3p2	vt3p3
1	150.27	88.02	NA	315.72	108.43	NA	293.39	149.89	NA
2	48.24	109.37	180.72	84.19	150.93	265.46	81.47	87.41	364.03

Note. In this example, two study groups as the observational unit are given. Within the variable names, “v” represents the measured variable (for example, sAA), “t” reflects the measurement time point (ranging from 1 to 3 in this example), and “p” reflects the person within a study group (ranging from 1 to 3 in this example). In the first study group, only two individuals (as observers) were present; in the second study group, there were three observers. Hence, the first row contains missing values (*NA*) in all columns in which p is assigned a 3. These missing values are missing at random and are respected as such using the full information maximum likelihood (FIML) method and adequate equality constraints (see Figure S1). Accordingly, the assignment of a person to a specific index (1, 2, or 3 in this example) is arbitrary and does not influence the results (Mehta & Neale, 2005).

Table S2

Results for the Likelihood-Ratio Tests Serving as Alternatives for the F-Tests

Model comparison	Dependent variable			
	Cortisol	sAA	Aldosterone	HR
ne vs. meg	$\chi^2(1) = 0.45, p = .502$	$\chi^2(1) = 0.47, p = .492$	$\chi^2(1) = 3.19, p = .074$	$\chi^2(1) = 1.87, p = .171$
	$\chi^2(1) = 3.95, p = .047$	$\chi^2(1) = 0.98, p = .322$	-	$\chi^2(1) = 8.31, p = .004$
ne vs. met	$\chi^2(6) = 78.12, p < .001$	$\chi^2(4) = 57.98, p < .001$	$\chi^2(6) = 8.46, p = .206$	$\chi^2(6) = 152.25, p < .001$
	$\chi^2(6) = 105.10, p < .001$	$\chi^2(4) = 62.115, p < .001$	-	$\chi^2(6) = 115.33, p < .001$
ne vs. int	$\chi^2(13) = 102.88, p < .001$	$\chi^2(9) = 72.85, p < .001$	$\chi^2(13) = 18.119, p = .153$	$\chi^2(13) = 171.24, p < .001$
	$\chi^2(13) = 209.38, p < .001$	$\chi^2(9) = 80.451, p < .001$	-	$\chi^2(13) = 236.37, p < .001$
meg vs. int	$\chi^2(12) = 102.43, p < .001$	$\chi^2(8) = 72.38, p < .001$	$\chi^2(12) = 14.93, p = .245$	$\chi^2(12) = 169.36, p < .001$
	$\chi^2(12) = 205.43, p < .001$	$\chi^2(8) = 79.67, p < .001$	-	$\chi^2(12) = 228.06, p < .001$
met vs. int	$\chi^2(7) = 24.76, p < .001$	$\chi^2(5) = 14.86, p = .011$	$\chi^2(7) = 9.65, p = .209$	$\chi^2(7) = 18.98, p = .008$
	$\chi^2(7) = 104.28, p < .001$	$\chi^2(5) = 18.34, p = .003$	-	$\chi^2(7) = 121.04, p < .001$

Note. Values in parentheses reflect the degrees of freedom for the χ^2 -statistic. Dependent variable values for cortisol and aldosterone were transformed using the natural logarithm. The first χ^2 -test in every cell refers to the comparison of stress observers with placebo-stress observers, the second χ^2 -test in every cell refers to the comparison of stress observers with TSST participants.

Table S3

Results for the Likelihood-Ratio Tests Serving as Alternatives for the Post-Hoc t-Tests

Measurement time point	Dependent variable		
	Δ Cortisol	Δ sAA	Δ HR
Speech task	-	-	$\chi^2(1) = 0.05, p = .821$
			$\chi^2(1) = 19.64, p < .001$
Mental arithmetic task	-	-	$\chi^2(1) = 0.76, p = .382$
			$\chi^2(1) = 22.47, p < .001$
+ 1 min	$\chi^2(1) = 12.98, p < .001$	$\chi^2(1) = 6.39, p = .011$	$\chi^2(1) = 7.02, p = .008$
	$\chi^2(1) = 11.89, p < .001$	$\chi^2(1) = 5.06, p = .024$	$\chi^2(1) = 7.23, p = .007$
+ 10 min	$\chi^2(1) = 8.03, p = .005$	$\chi^2(1) = 4.70, p = .030$	$\chi^2(1) = 1.66, p = .197$
	$\chi^2(1) = 21.12, p < .001$	$\chi^2(1) = 1.22, p = .027$	$\chi^2(1) = 9.41, p = .002$
+ 20 min	$\chi^2(1) = 5.93, p = .015$	$\chi^2(1) = 3.47, p = .063$	$\chi^2(1) = 1.11, p = .292$
	$\chi^2(1) = 16.97, p < .001$	$\chi^2(1) = 2.48, p = .115$	$\chi^2(1) = 7.95, p = .005$
+ 30 min	$\chi^2(1) = 2.48, p = .115$	-	-
	$\chi^2(1) = 13.25, p < .001$		
+ 45 min	$\chi^2(1) = 4.29, p = .038$	-	-
	$\chi^2(1) = 12.03, p < .001$		
+ 120 min	$\chi^2(1) = 0.95, p = .329$	$\chi^2(1) = 0.06, p = .808$	$\chi^2(1) = 0.35, p = .554$
	$\chi^2(1) = 2.92, p = .087$	$\chi^2(1) = 0.66, p = .418$	$\chi^2(1) = 1.74, p = .188$

Note. Δ depicts a change from baseline. Values in parentheses reflect the degrees of freedom for the χ^2 -statistic. The first χ^2 -test in every cell refers to the comparison of stress observers with placebo-stress observers, the second χ^2 -test in every cell refers to the comparison of stress observers with TSST participants.

Table S4

Differences in Aldosterone Reactivity between Stress Observers in Reaction to Observation of the obsTSST and Placebo-Stress Observers in Reaction to Observation of the obsPlacTSST

		Aldosterone	
		<i>p</i> -value comparison with stress observers	
interaction effect time x group	Stress observers		
	Placebo-stress observers		.31
	TSST participants		< .001
		Change in nmol/l from baseline (<i>p</i> -value)	<i>p</i> -value group comparison with stress observers
+1 min	Stress observers	2.11 ± 2.93 (.79)	
	Placebo-stress observers	1.68 ± 4.21 (.93)	.93
	TSST participants	7.11 ± 5.07 (.16)	.36
+10 min	Stress observers	−0.18 ± 2.95 (.93)	
	Placebo-stress observers	6.33 ± 3.40 (.085)	.15
	TSST participants	25.10 ± 5.54 (< .001)	< .001
+20 min	Stress observers	0.98 ± 3.30 (.82)	
	Placebo-stress observers	8.54 ± 4.52 (.10)	.17
	TSST participants	32.74 ± 6.60 (< .001)	< .001
+30 min	Stress observers	2.61 ± 2.69 (.54)	
	Placebo-stress observers	5.37 ± 4.68 (.33)	.59
	TSST participants	23.09 ± 6.45 (.003)	.001
+ 45 min	Stress observers	1.96 ± 2.70 (.51)	
	Placebo-stress observers	13.22 ± 4.92 (.022)	.038

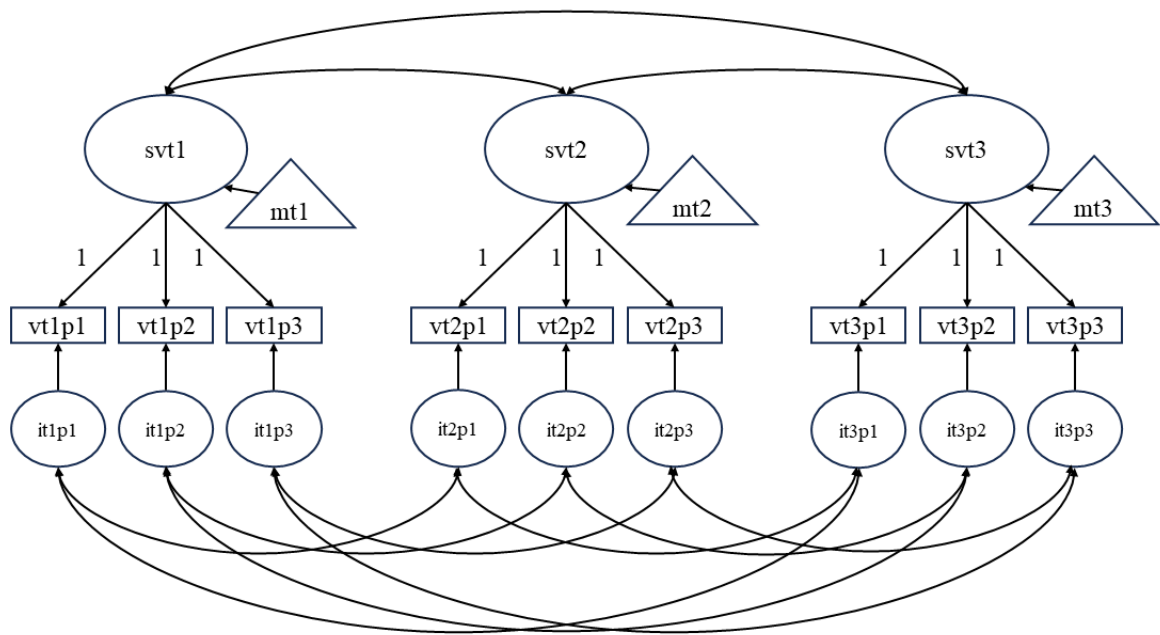
	TSST participants	14.05 ± 6.33 (.057)	.047
	Stress observers	1.52 ± 4.03 (.83)	
+ 120 min	Placebo-stress observers	11.50 ± 5.15 (.054)	.12
	TSST participants	1.70 ± 6.66 (.63)	.98

Note. Values are means ± SE if not indicated differently; *p*-values change to baseline = main effect of time of repeated measures ANOVAs comprising baseline and respective other individual measurement time point; *p*-values group comparison with stress observers = interactions group-by-time of repeated measures ANOVAs comprising baseline and respective other individual measurement timepoint as well as group; analyses comprise *N* = 36 stress observers; *n* = 28 placebo-stress observers, as well as *N* = 20 TSST participants.

Supplemental Material 3: Figures

Figure S1

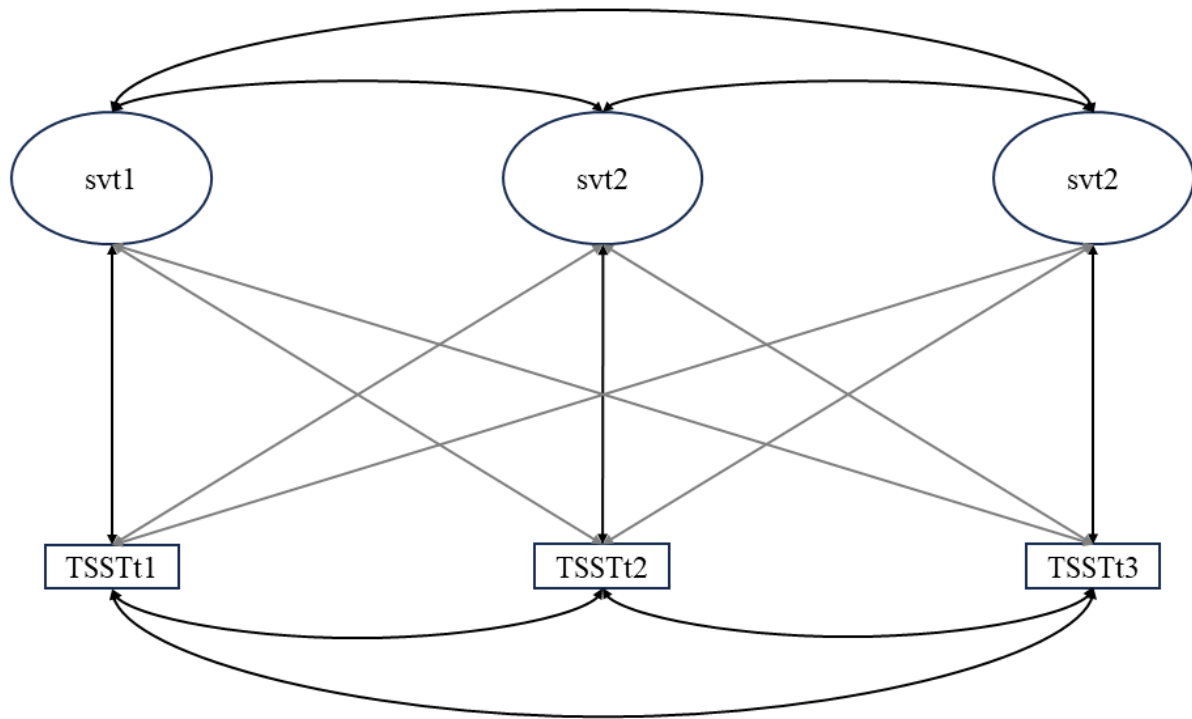
Example for a Structural Equation Model Respecting the Study Group as a Potential Source of Variance (Here, with Three Measurement Time Points)



Note. s = study group-level, v = variable, t = measurement time point, m = mean, p = person, i = individual-level. The variance of the latent factors represents the variance at the study group-level, the residuals represent the variance at the individual level. All latent factor variances are constrained to be equal, all latent factor covariances are constrained to be equal, all residuals are constrained to be equal, and all residual covariances are constrained to be equal.

Figure S2

Example for an Extension of the Structural Equation Model from Figure S1 to Include the Dependent Variables for TSST Participants (Here, with Three Measurement Time Points)



Note. s = study group, v = variable, t = measurement time point. All variances of the TSST variables are constrained to be equal and all covariances between TSST variables are constrained to be equal. All covariances between TSST variables and latent session factors relating to the same measurement time point (straight black lines) are constrained to be equal. All covariances between TSST variables and latent study group factors relating to different measurement time points (straight gray lines) are constrained to be equal. For simplicity, the measurement model for the latent factors as given in Figure S1 is not repeated here and intercepts are not shown.

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APPENDIX B**Research Paper 2****Taking Appreciation to Heart:****Appreciation at Work and Cardiovascular Risk in Male Employees**

This research paper was published in:

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Abstract

Background & Objectives: While perceived appreciation at work has been associated with self-reported health and well-being, studies considering biological health markers are lacking. In this study, we investigated whether appreciation at work would relate to coronary heart disease (CHD) risk as well as the specificity of this proposed association.

Methods: Our study comprised a total of 103 male participants, including apparently healthy, medication-free, non-smoking men in the normotensive to hypertensive range ($n = 70$) as well as medicated hypertensive and CHD patients ($n = 33$). CHD risk was assessed by blood pressure (mean arterial pressure (MAP)), the diabetes marker glycated hemoglobin A1c (HbA1c), blood lipids (total cholesterol (TC)/high-density lipoprotein-cholesterol (HDL-C) ratio), coagulation activity (D-dimer and fibrinogen), and inflammation (interleukin (IL)-6, tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP)). Perceived appreciation at work, as well as potentially confounding psychological factors (social support, self-esteem, and work strain due to lack of appreciation), were measured by self-report questionnaires.

Results: We found higher appreciation at work to relate to lower overall composite CHD risk (p 's $\leq .011$) and, in particular, to lower MAP (p 's $\leq .007$) and lower blood lipids (p 's $\leq .031$) in medication-free participants as well as all participants. This overall association was independent of confounding factors, including related psychological factors (p 's $\leq .049$).

Discussion: Our findings indicate that appreciation at work might be an independent health-promoting resource in terms of CHD risk. Implications include that encouraging appreciation at work may help reduce the development and progression of CHD.

Keywords: appreciation at work, coronary heart disease, blood pressure, HbA1c, blood lipids, coagulation, inflammation, positive cardiovascular health

Introduction

“You did a good job!”, “I need your advice, can you help me?”, “Thank you for your assistance!” Statements like these usually induce a wide range of pleasant feelings as a consequence of the implied appreciation (Basch & Fisher, 2000; Ohly & Schmitt, 2015). In research, appreciation is often captured by similar terms such as respect (Rudolph et al., 2021; van Quaquebeke & Eckloff, 2010), esteem (Siegrist, 1996), acknowledgment (Cleary et al., 2012), or recognition (Wang et al., 2020; Yukl, 2013) that all relate to the same basic idea of valuing someone (Pfister, Jacobshagen, Kälin, & Semmer, 2020). More precisely, appreciation expresses the recognition of a person’s positive qualities by other people (Semmer et al., 2007; Semmer et al., 2019). It, for example, signals the value of a person in social relations in terms of positive qualities such as being likable, competent, or moral, and, thus, supports a positive self-image (Leary & Baumeister, 2000; Semmer et al., 2019).

From a historical perspective, Herzberg et al. (1959) were one of the first to propose the positive effects of recognition or appreciation on motivation and job satisfaction. Given its social relevance (Leary & Baumeister, 2000), appreciation is associated with fundamental human motives, including the motive to get along (Hogan, 1983) and the need to belong (Baumeister & Leary, 1995). Second, in the context of work stress, appreciation or esteem constitutes one of three reward factors in the effort-reward imbalance (ERI) model by Siegrist (1996). According to that model, work stress results from a perceived imbalance between a person’s efforts in relation to rewards at work, with higher rewards, including esteem, counterbalancing the amount of work stress (Siegrist, 1996). Notably, esteem, as assessed by the ERI questionnaire, does not quantify the perceived amount of received appreciation but measures the amount of strain that results from the lack of appreciation at work (Siegrist et al., 2004). Third, appreciation plays a major role in the stress-as-offense-to-self (SOS) theory (Semmer et al., 2007; Semmer et al., 2019), in which threats to the self are considered a major source of stress. Here, appreciation is considered a core resource as it boosts self-esteem and

may buffer stress experiences resulting from threats to the self. Furthermore, in the context of the SOS theory, a pertinent measure of appreciation has been developed (Stocker et al., 2010). Fourth, in addition to self-esteem, appreciation has also been associated with a further stress-reducing resource, namely social support (Cobb, 1976; Lett et al., 2005; Semmer et al., 2008). Functional social support includes the two facets of instrumental support and emotional support (Lett et al., 2005). While instrumental support refers to help concerning the problem at hand, for instance, in terms of tangible help or information, emotional support refers to communicating care, esteem, empathy, and understanding (Lett et al., 2005; Semmer et al., 2008). Thus, in the context of social support, appreciation explicitly indicates emotional support (Lett et al., 2005; Thoits, 1995), but also instrumental support often has an emotional component that transmits appreciation and esteem (Semmer et al., 2008). Nevertheless, there are differences between social support and appreciation. Unlike social support, appreciation can be conveyed in any situation and is often shown in positive situations (e.g., successful cooperation). Not surprisingly, therefore, the effects of appreciation on indicators of well-being have been shown to persist after controlling for social support (Pfister, Jacobshagen, Kälin, et al., 2020; Stocker et al., 2010; Stocker et al., 2019). Fifth, recent leadership concepts that focus on health have acknowledged the relevance of recognition or appreciation by supervisors for employee well-being (Vincent-Höper & Stein, 2019). Taken together, evidence for the relevance of appreciation, in particular in the context of stress and health, comes from several theories and empirical studies that do not explicitly differentiate appreciation from related constructs, including the reward component of the ERI model (i.e., ERI esteem), social support, and self-esteem. These constructs may intertwine with appreciation and, thus, confound the potential effects of appreciation.

Interestingly, despite the outlined decades of linkage between appreciation and the above-described theories and health-relevant concepts, there are comparatively few studies that quantify perceived appreciation at work as a concept of its own right in the context of well-

being and health. With respect to *self-reported well-being*, there is evidence that higher appreciation at work is associated with higher well-being (Gilbert & Kelloway, 2018), serenity (Stocker et al., 2014), enthusiasm and contentment (Pfister, Jacobshagen, Kälin, et al., 2020), job satisfaction (Decker & Van Quaquebeke, 2015; Elfering, Gerhardt, et al., 2017; Pfister, Jacobshagen, Kälin, & Semmer, 2020; Semmer et al., 2005; Stocker et al., 2010; van Quaquebeke & Eckloff, 2010; Wöhrmann et al., 2017), lower feelings of resentment (Pfister, Jacobshagen, Kälin, & Semmer, 2020; Stocker et al., 2010), as well as better psychological functioning, represented by a number of psychological resources including self-esteem (Merino & Privado, 2015). Regarding *self-reported health*, appreciation has been associated with higher levels of subjective health (Wöhrmann et al., 2017), as well as lower levels of depressive symptoms (Pfister, Jacobshagen, Kälin, et al., 2020; Pohrt et al., 2022), anxiety (Pfister, Jacobshagen, Kälin, et al., 2020), emotional exhaustion (Laschinger & Finegan, 2005), and lower-back-pain (Elfering, Häfliger, et al., 2017). In terms of a more *objective assessment of health*, register-based studies show that higher appreciation prospectively predicted a lower risk of sickness absence (Hinkka et al., 2013) and fewer early retirements, both based on disability pension (Sundstrup et al., 2018) as well as self-reported non-disability pension (De Wind et al., 2014). With respect to associations between appreciation and *physiological markers of health*, there is so far only one very recent study that assessed the average situation-specific expectation of appreciation and respect for one's effort and found higher expectations to relate to lower intima-media thickness (IMT) of the carotid artery (Cundiff et al., 2022). Given that the carotid IMT is indicative of the severity of atherosclerosis, the chronic inflammatory process underlying coronary heart disease (CHD) (Libby et al., 2019; Libby & Theroux, 2005; Poredos, 2004; Ross, 1999), the finding of this study point to associations between appreciation and CHD. Notably, this is in line with the concept of positive cardiovascular health (Labarthe et al., 2016), where positive psychological well-being is proposed to relate to cardiovascular health (Boehm & Kubzansky, 2012; Kubzansky et al., 2018; Levine et al., 2021). Indeed, concepts

related to appreciation, in particular higher social support (Barth et al., 2010) and lower ERI (Eddy et al., 2017; Kivimäki & Siegrist, 2016), have been associated with lower CHD risk. However, to the best of our knowledge, data on appreciation and CHD risk are sparse and even absent for a broad measure of appreciation that is not only confined to effort.

In this study, we set out to investigate whether higher perceived appreciation at work would relate to lower CHD risk as assessed by major biological risk factors under resting conditions in male employees with differential CHD risk. To maximize the score ranges, and, thus, the variability in CHD risk factors, we included apparently healthy participants in the normotensive to hypertensive range, and patients with hypertension or manifest CHD and controlled for relevant confounders. To assess CHD risk in a most comprehensive way, we assessed the major intermediate biological CHD risk factor categories comprising blood lipids, coagulation, and inflammation, in addition to the classical risk factors diabetes and hypertension (e.g., Ridker et al., 2018; Visseren et al., 2021; Wirtz & von Känel, 2017). Apart from diabetes, which was assessed by one marker (glycated hemoglobin A1c, HbA1c, (Sherwani et al., 2016)) all other biological risk factors were assessed by at least two major markers to allow for a representative assessment of the respective risk factor category. In detail, as a linear-single measure of hypertension status, mean arterial pressure (MAP) was calculated from systolic (SPB) and diastolic (DBP) blood pressure (BP) (Fuchs & Whelton, 2020; Lewington et al., 2002; Liu et al., 2021). Moreover, blood lipid assessment included the ratio between total cholesterol (TC) and high-density lipoprotein-cholesterol (HDL-C) (Calling et al., 2021). Coagulation activity was reflected by D-dimer (Danesh et al., 2001; Simes et al., 2018) and fibrinogen (Fibrinogen Studies Collaboration, 2005; Surma & Banach, 2022). Inflammation assessment comprised the acute-phase protein C-reactive protein (CRP) (Ridker et al., 1998; The Emerging Risk Factors Collaboration, 2010) and the cytokines interleukin (IL)-6 and tumor necrosis factor-alpha (TNF- α) (Danesh et al., 2008; Kaptoge et al., 2014; Ridker et al., 2000). In addition, we tested the specificity of the proposed association between

appreciation and CHD risk by additionally controlling for potential psychological confounders, comprising perceived social support (PSS) and the amount of strain induced by a lack of appreciation (ERI esteem) as conceptually related constructs, and self-esteem as a factor that likely interferes with perception of appreciation.

Methods

Study Participants

This study is part of a project assessing psychoneurobiological mechanisms in CHD and essential hypertension (Auer et al., 2022; Degroote et al., 2023; Degroote et al., 2021; Zuccarella-Hackl, von Känel, Thomas, Hauser, et al., 2016; Zuccarella-Hackl, von Känel, Thomas, Kuebler, et al., 2016). Ethical approval was obtained by the Ethics Committee of the State of Bern, Switzerland, and the study was conducted in accordance with the Declaration of Helsinki principles. All participants provided written informed consent and were financially compensated with 20 Swiss Francs.

For the purpose of the current study, we included hypertensive and normotensive men as well as male patients with diagnosed CHD in order to cover the broadest possible range of cardiovascular risk. Besides the exclusion criteria described below, participants had to be employed at the time of the study assessment, working at least 20h per week, and have completed the appreciation at work questionnaire in addition to medical assessment. Due to insufficient medical data, $n = 6$ participants had to be excluded. Furthermore, participants reporting symptoms of infectious disease on the day of the study assessment were excluded ($n = 3$). Our final study sample comprised $N = 103$ participants, with $n = 25$ CHD-patients, $n = 40$ hypertensive participants (32 were medication-free and 8 were medicated at study participation), $n = 32$ normotensive participants, and $n = 6$ with white coat or masked hypertension.

Recruitment and General Inclusion Criteria

The recruitment and assessment of our study participants took place between 2011 and 2016. We invited male CHD patients of the Cardiac Prevention and Rehabilitation Clinic of the Bern University Hospital, who had received their diagnosis at least 6 months earlier, to participate in our study as described previously (Degroote et al., 2023; Zuccarella-Hackl, von Känel, Thomas, Kuebler, et al., 2016). Since all CHD patients require CHD medication, we accepted medication for these patients. Moreover, we recruited apparently healthy (i.e., undiagnosed) medication-free essential hypertensive and normotensive participants with the aid of the Swiss Red Cross of the Canton of Bern as described previously (Degroote et al., 2023; Zuccarella-Hackl, von Känel, Thomas, Hauser, et al., 2016). The final classification of essential hypertension and normotension was based on a two-step assessment procedure comprising home and study BP measurements following our previous methods (e.g., Auer et al., 2022; Degroote et al., 2023) to ensure essential hypertension status validity. Briefly, interested candidates were instructed to measure their BP at home in a seated position after a 15-minute rest using sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofddorp, Netherlands) on three separate days, once in the morning and once in the evening. Based on a maximum of six home BP measurements, participants were preliminarily classified as hypertensive if their average home SBP was ≥ 135 mmHg and/or their average home DBP was ≥ 85 mmHg according to recommendations for home or ambulatory BP measurements (normotensive: SBP < 135 mmHg and DBP < 85 mmHg) (Mancia et al., 2023; Williams et al., 2018). To verify home-measurement-based preliminary classification, trained personnel obtained up to three additional seated baseline BP measurements (see Procedure). For classification regarding study BP measurements, we applied the standard definition of hypertension based on office or clinic BP measurements and classified participants as hypertensive if their average study SBP was ≥ 140 mmHg and/or their average study DBP was ≥ 90 mmHg (normotensive: SBP < 140 mmHg and DBP < 85 mmHg) (Chambers et al., 1999;

Mancia et al., 2023; Williams et al., 2018). Participants with consistent group assignment based on both, home BP and study BP measurements, were classified as hypertensive ($n = 32$) or normotensive ($n = 32$). Participants with deviating classification of home and study BP assessment ($n = 6$) were classified as white coat hypertensive if they displayed normotensive home BP and hypertensive study BP ($n = 3$) and as masked hypertensive if they displayed hypertensive home BP and normotensive study BP ($n = 3$). Notably, we accepted diagnosed hypertensive patients, and, thus, intake of antihypertensive medication, in a small proportion of hypertensive individuals ($n = 8$) to increase sample size, but no other current medication intake (rendering a total of $n = 40$ hypertensive participants).

Procedure

All participants abstained from caffeine and alcohol consumption for 24 hours and consumed a semi-standardized breakfast following written instructions prior to arrival at the lab at 8:00 a.m. In addition to the assessment of height and weight, trained personnel obtained up to three additional seated baseline BP measurements on the dominant arm each after a 15-minute rest by means of sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofddorp, Netherlands). To assess CHD risk factors other than BP, blood samples were collected at 11:30 a.m., i.e., after fasting for 3.5h since arrival.

Psychological Measurements

Appreciation at Work

Appreciation at work was measured with the 10-item Bern Appreciation Scale, which assesses appreciation by supervisors and co-workers (Stocker et al., 2010). Participants were asked to rate on a 7-point response scale (1 = *not at all* to 7 = *very much*) the extent to which statements about different forms of appreciation applied to their work situation, such as compliments, understanding, trust, sympathy, attention, interest, and gratitude (e.g., “My supervisors praise me when I carry out my tasks well” (German: “Mein(e) Vorgesetzte(r) lobt mich, wenn ich meine Aufgaben gut erledige”); “My colleagues show how much they value

my opinion by asking for my advice” (German: “Meine ArbeitskollegInnen fragen mich um Rat und das zeigt mir, dass sie meine Meinung schätzen”). Items were averaged to a total score, with higher scores indicating higher appreciation at work. Psychometric properties of the appreciation total score were found to be adequate with Cronbach’s α ($N = 228$) = .86 (Stocker et al., 2010) and Cronbach’s α ($N = 103$) = .89 in our sample.

Social Support

PSS was assessed by the 8-item subscale of the Berlin Social Support Scale (BSSS) (Schulz & Schwarzer, 2003). On a 6-point response scale (1 = *strongly disagree* to 6 = *strongly agree*), participants were asked to rate their agreement with statements such as “There are people that offer me help when I need it” (German: “Es gibt Menschen, die mir Hilfe anbieten, wenn ich sie brauche”). Items were averaged to compute the PSS score, with higher scores indicating higher PSS. Psychometric properties are adequate with Cronbach’s α ($N = 437$) = .83 for the PSS subscale (Schulz & Schwarzer, 2003) and excellent Cronbach’s α ($N = 103$) = .90 in our sample.

Self-Esteem

We used the 10-item German version (Ferring & Filipp, 1996) of the Rosenberg self-esteem scale (RSES; Rosenberg, 1965) to measure global self-esteem. Participants are asked to rate positive and negative feelings about themselves (e.g., “On the whole, I am satisfied with myself” (German: “Alles in allem bin ich mit mir selbst zufrieden”)) on a 4-point response scale (1 = *strongly disagree* to 4 = *strongly agree*). After recoding where appropriate, items were summarized to a total score, with higher scores indicating higher self-esteem. Psychometric properties have been shown to be adequate with Cronbach’s α ($N=4,988$) = .88 in the reference sample (Roth et al., 2008) and Cronbach’s α ($N = 100$) = .85 in our sample. Notably, $n = 3$ participants (two CHD patients and one normotensive participant) did not fill out the questionnaire.

Strain Induced by Lack of Appreciation (ERI Esteem)

To measure the amount of strain that results from the lack of appreciation at work, we used the 5-item esteem subscale of the German version (Rödel et al., 2004) of the ERI questionnaire (Siegrist et al., 2004). On a 5-point Likert-scale in a two-step rating procedure, participants were asked whether items such as “Considering all my efforts and achievements, I receive the respect and prestige I deserve at work.” (German: “Wenn ich an all die erbrachten Leistungen und Anstrengungen denke, halte ich die erfahrene Anerkennung für angemessen”) would apply or not. If participants indicate a lack of appreciation, they are further asked to rate the resulting extent of distress. Items are summarized to a total score, with higher scores indicating lower strain resulting from lack of appreciation. Psychometric properties have been shown to be adequate (Rödel et al., 2004; Siegrist et al., 2004) with Cronbach’s α ($N = 666$) = .76 for the esteem subscale (Rödel et al., 2004) and Cronbach’s α ($N = 102$) = .73 in our sample. Notably, one normotensive participant did not fill out the questionnaire.

CHD Risk Assessment

We assessed CHD risk by measuring the following biological risk factors: (1) BP, (2) HbA1c, (3) blood lipid profiles in terms of TC/HDL-C ratio, (4) the prothrombotic factors D-dimer and fibrinogen, and (5) the pro-inflammatory measures IL-6, TNF- α , and CRP. Analyses of HbA1c, blood lipids, and prothrombotic factors were performed in the Center for Laboratory Medicine of the Bern University Hospital (Inselgruppe AG, Bern), while analyses of pro-inflammatory measures were performed in the biochemical laboratory of the Biological Work and Health Psychology group at the University of Konstanz.

HbA1c

For assessment of the diabetes marker HbA1c, venous blood was drawn into EDTA-coated Monovettes. Analyses were performed with *in vitro* assays for the quantitative determination of HbA1c IFCC (mmol/mol) in whole blood (Tina-quant®, Roche, Mannheim,

Germany) using Roche/Hitachi Cobas C Systems (Roche, Mannheim, Germany). The inter- and intra-assay coefficients of variation (CVs) were $\leq 1.6\%$ and $\leq 2.0\%$, respectively.

Blood Lipids

TC and HDL-C were measured from heparin-coated Monovettes (Sarstedt Monovette orange). Analyses were performed using *in vitro* assays (enzymatic colorimetric assays, Roche, Mannheim, Germany) for the quantitative determination of blood lipids in human plasma on a Roche/Hitachi Cobas C Analyzer (Roche, Mannheim, Germany). The inter- and intra-assay CVs were $\leq 1.2\%$ and $\leq 2.5\%$ respectively.

Coagulation Activity

To measure coagulation activity in terms of the prothrombotic factors D-dimer and fibrinogen, venous blood was drawn into polypropylene tubes containing 3.8% sodium citrate (Sarstedt, Nümbrecht, Germany). Citrate tubes were immediately centrifuged for 20 min at 4°C at 2000g, and plasma was pipetted into aliquots. D-dimer was analyzed using a particle-enhanced immunoturbidimetric assay for the quantitative determination of D-dimers in human plasma (INNOVANCE® D-Dimer, Siemens Healthcare GmbH, Erlangen, Germany) on a Sysmex CS-5100 (Sysmex Europe, Norderstedt, Germany). Plasma fibrinogen levels were determined by a routine clotting assay applying standard quality procedures following the Clauss method. The intra- and inter-assay CVs were $\leq 7.9\%$.

Pro-inflammatory Measures

For the assessment of inflammation in terms of the pro-inflammatory measures IL-6, TNF- α , and CRP, venous blood was drawn in EDTA-coated Monovettes (Sarstedt, Nümbrecht, Germany) and immediately centrifuged for 10 min at 2000g and 4°C. The obtained plasma was stored at -80°C until analysis. The IL-6 and TNF- α levels were determined with a high sensitivity chemiluminescence sandwich immunoassay (Meso Scale Discovery (MSD), Rockville, USA). CRP was determined using a high-sensitivity enzyme immunoassay (ELISA, IBL Hamburg, Germany). For IL-6, inter- and intra-assay CVs were $\leq 7.3\%$ and $\leq 4.5\%$. For

TNF- α , inter- and intra-assay CVs were $\leq 10.1\%$ and $\leq 3.4\%$. For CRP, inter- and intra-assay CVs were $\leq 6.3\%$ and $\leq 6.9\%$. Notably, CRP could not be analyzed in 14 participants due to problems with blood sampling or processing or an insufficient amount of samples for the analysis.

Statistical Analyses

Data were analyzed using SPSS (Version 29.0) packages for Macintosh (IBM SPSS Statistics, Chicago, IL, USA) and presented as mean $\pm$ standard error of the mean (SEM). All tests were two-tailed, with the significance level set at $p < .05$.

We *a-priori* calculated power analyses using the statistical software G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). To conservatively allow for the detection of small to medium effect sizes of $R^2 = .10$ in linear regression analyses, the required sample size to obtain a power of $(1 - \beta) = .80$ is $N = 74$.

For all participants, we calculated MAP based on up to three study BP measurements by the formula $MAP = (2/3 * \text{mean study DBP}) + (1/3 * \text{mean study SBP})$, as well as body mass index (BMI) by the formula $BMI = \text{kg/m}^2$. To compute an aggregated coagulation index, we averaged z-transformed levels of D-dimer and fibrinogen. For an aggregated inflammatory index, we accordingly averaged z-transformed levels of IL-6, TNF- α , and CRP. Notably, as CRP could not be analyzed in 14 participants, the aggregated inflammatory index for these participants consists of averaged z-transformed levels of IL-6 and TNF- α .

All data were tested for normal distribution using the Kolmogorov-Smirnov test. We used non-normality robust multivariate analyses of covariance (MANCOVAs) (Finch, 2005) and conducted statistical analyses with original data. Effect size parameters partial η^2 (η^2_p ; effect size conventions η^2_p : .01 = small; .06 = medium; .14 = large (Cohen, 1988)) and R^2 changes (ΔR^2 ; effect size conventions R^2 : .02 = small; .13 = medium; .26 = large (Cohen, 1988)) are reported where appropriate.

To test whether appreciation at work is associated with cardiovascular risk, we calculated MANCOVAs. Thereby, appreciation at work was included as a linear independent variable, and MAP, HbA1c, TC/HDL-C ratio, the coagulation index, and the inflammatory index were entered as dependent variables, yielding the latent variable CHD risk. MANCOVAs were conducted without and with controlling for possible confounding effects of medication intake (antihypertensive medication, blood lipid and/or diabetes medication, anticoagulation medication, and other medication intake), age, BMI, and smoking (Cooney et al., 2010; Visseren et al., 2021). Moreover, to completely exclude potential confounding effects of medication intake or smoking, we repeated our analyses with medication-free, non-smoking participants ($n=70$) without and with controlling for age and BMI. Post-hoc testing of significant multivariate effects of appreciation at work comprised hierarchical linear regression analyses with each dependent variable separately regressed on appreciation at work in order to identify the direction of the effect and the variables that mainly account for it.

To test whether appreciation at work would buffer cardiovascular risk independent of important related psychological factors, we repeated the above-described procedure while additionally considering PSS, self-esteem, and/or ERI esteem as covariates.

Results

Group Characteristics

Table 1 provides group characteristics of the 103 male participants, including the 70 medication-free, non-smoking participants. Our study sample comprised employees from various organizations pursuing a wide variety of jobs, such as agriculturists, police officers, computer scientists, consultants, or project managers. Our participants worked on average 43.35 hours/week (SEM = 0.68; range = 20–58). The mean age was 50.15 years (SEM = 0.98; range = 25–71) and the mean BMI was 27.46 kg/m² (SEM = 0.36; range = 19.78–38.90).

Appreciation at Work and Cardiovascular Risk

Over all participants ($N = 103$) MANCOVAs revealed that appreciation at work was significantly related to CHD risk in terms of the dependent variables MAP, HbA1c, TC/HDL-C ratio, the coagulation index, as well as the inflammatory index. Associations were significant both without ($p = .011$, $\eta^2_p = .14$) and with controlling for medication intake, age, BMI, and smoking ($p = .046$, $\eta^2_p = .12$). *Post-hoc* hierarchical linear regression analyses indicated that higher appreciation at work was associated with lower MAP ($\beta = -.27$, $p = .007$, see Figure 1 A; with control variables: $\beta = -.19$, $p = .026$) and with lower blood lipids (TC/HDL-C ratio) ($\beta = -.21$, $p = .031$, see Figure 1 B; although with control variables there was only a trend: $\beta = -.17$, $p = .079$). There were no associations either with HbA1c or the coagulation and inflammatory indices (p 's $\geq .11$). Multivariate effects became stronger when repeating MANCOVAs in medication-free, non-smoking participants ($n = 70$) ($p = .008$, $\eta^2_p = .21$; with control variables age and BMI: $p = .018$, $\eta^2_p = .19$). Again, higher appreciation at work was associated with lower MAP ($\beta = -.35$, $p = .003$; with control variables: $\beta = -.25$, $p = .011$) and lower blood lipids (TC/HDL-C ratio) ($\beta = -.37$, $p = .002$; with control variables: $\beta = -.32$, $p = .005$), but not with HbA1c, or the coagulation and inflammatory indices (p 's $\geq .15$). The results are depicted in more detail in Table 2.

To test for specificity, i.e., whether appreciation-at-work is associated with CHD risk independent of other related psychological constructs, we repeated the above-described analyses and tested whether additional control for PSS, self-esteem, and the amount of strain that results from the lack of appreciation at work (ERI esteem) as covariates either alone or combined would change the obtained results. The results showed that appreciation at work independently related to CHD risk (p 's $\leq .042$, η^2_p 's $\geq .12$; with control variables: p 's $\leq .049$, η^2_p 's $\geq .12$). Neither PSS, self-esteem, nor ERI esteem were associated with CHD risk

independent of appreciation (p 's $\geq .14$; with covariates: p 's $\geq .11$). These results were obtained in analyses including all participants as well as medication-free, non-smoking participants only.

Discussion

In this study, we investigated whether appreciation at work would relate to CHD risk as assessed by major biological risk factors under resting conditions, i.e., MAP, HbA1c, TC/HDL-C ratio, coagulation, as well as inflammation. We further tested for the specificity of the proposed association by additionally controlling for important related psychological factors, i.e., PSS, self-esteem, and the amount of strain induced by a lack of appreciation.

We found that higher appreciation at work was significantly and independently associated with lower overall CHD risk and, in particular, with lower MAP and lower blood lipid levels. These findings corroborate and extend studies that found appreciation at work to be associated with self-reported health and well-being (De Wind et al., 2014; Decker & Van Quaquebeke, 2015; Elfering, Gerhardt, et al., 2017; Elfering, Häfliger, et al., 2017; Gilbert & Kelloway, 2018; Hinkka et al., 2013; Laschinger & Finegan, 2005; Merino & Privado, 2015; Pfister, Jacobshagen, Kälin, & Semmer, 2020; Pfister, Jacobshagen, Kälin, et al., 2020; Pohrt et al., 2022; Semmer et al., 2005; Stocker et al., 2014; Stocker et al., 2010; Sundstrup et al., 2018; van Quaquebeke & Eckloff, 2010; Wöhrmann et al., 2017) to health assessment in terms of biological CHD risk factors. The observed association between perceived appreciation at work and biological CHD risk is in line with the study that found that the appreciation for one's effort to relate to lower IMT (Cundiff et al., 2022). Moreover, the specificity of our results, i.e., that appreciation at work relates to CHD risk independent of PSS, self-esteem, or the amount of strain that results from the lack of appreciation, indicates that the amount of perceived appreciation at work seems to be an independent driving resource with regard to CHD risk.

What *mechanisms* may underlie the observed association between higher appreciation at work and lower CHD risk? First, on the basis of existing theory, we propose that appreciation

may buffer stress effects. Given that work stress is a risk factor for CHD (Brotman et al., 2007; Eddy et al., 2017; Kivimäki & Kawachi, 2015) and appreciation is considered a major resource in the context of work stress according to the ERI model (Siegrist, 1996) and the SOS theory (Semmer et al., 2007; Semmer et al., 2019), we consider appreciation to counteract and, thus, prevent adverse health consequences of work stress. This reasoning is in line with the job-demand resources model (JD-R), with job resources postulated to buffer the negative effects of job demands on strain and health (Bakker & Demerouti, 2017; Demerouti et al., 2001). Indeed, appreciation at work has been shown to buffer the negative effects of illegitimate tasks (Pfister, Jacobshagen, Kälin, et al., 2020), long working hours (Stocker et al., 2010), or work interruptions (Stocker et al., 2019) on indicators of health and well-being. Second, we propose positive effects of appreciation on cardiovascular health, independent of stress. Appreciation induces a wide range of positive feelings (Basch & Fisher, 2000; Ng et al., 2019; Ohly & Schmitt, 2015; Pfister, Jacobshagen, Kälin, et al., 2020; Stocker et al., 2014) and positive psychological well-being, including positive affect, has been shown to relate to lower CHD risk, both in cross-sectional and prospective studies (Boehm & Kubzansky, 2012; Davidson et al., 2010). Indeed, based on the concept of positive cardiovascular health (Labarthe et al., 2016), biological (e.g., lower BP, lower lipids, and lower inflammation), behavioral (e.g., smoking cessation, healthy diet, and physical activity), and psychosocial pathways have been proposed to underlie the association between positive psychological well-being and better cardiovascular health (Kubzansky et al., 2018; Levine et al., 2021; Pressman et al., 2019). Given this, we assume that the observed association between appreciation and better cardiovascular health may similarly be mediated by positive affect pathways. However, this remains to be studied. Taken together, appreciation may not only buffer stress effects with the associated increase in cardiovascular risk but also actively contribute to better cardiovascular health. Given the specificity of our results, future research is needed to further support an evident role of

appreciation in cardiovascular health. Moreover, the potential underlying mechanisms remain to be elucidated.

The *implications* of our findings include encouraging appreciation at work and investigating whether such interventions will ultimately improve cardiovascular health in employees in terms of reducing cardiovascular risk factors and a better prognosis. In general, there are different interaction levels to express appreciation. These interaction levels include the organizational (e.g., policies and programs stating the organization's intention to recognize the work performed by its members), vertical (top-down and bottom-up), horizontal (between team members), external (e.g., in service occupations customers), and social (community's appreciation of the organization or its social value) level (Brun & Dugas, 2008). Moreover, different forms of appreciation include personal appreciation and an appreciation of results, work practice, or job dedication (Brun & Dugas, 2008), but also the assignment of interesting tasks and job design in general (Semmer et al., 2019). For example, in their diary study, Stocker et al. (2014) found that appreciation in small-scale, simple, and economic ways, such as praise and gratitude, predicted positive effects at the end of a workday. However, there are indications that a discrepancy between employees' expectations of appreciation and perceived appreciation at work, especially with regard to supervisors, is not uncommon (Cleary et al., 2012; van Quaquebeke et al., 2009). Reasons for this discrepancy may include underestimation of the positive value and the overestimation of the awkwardness of explicitly expressing appreciation, which may prevent from expressing appreciation (Kumar & Epley, 2018). To allow for an organizational culture that encourages the expression of appreciation, organizations should actively raise awareness of the positive effects of appreciation as well as ways to provide authentic appreciation in order to counteract the potential overestimation of awkwardness. According to Yukl (2013), one should pay attention to (1) recognizing a variety of contributions and achievements, (2) actively searching for contributions to recognize, (3) recognizing improvements in performance, (4) recognizing commendable efforts even if they failed, (5) not

limiting recognition to high-visibility jobs, (6) not limiting recognition to a few best performers, (7) providing specific recognition, (8) providing timely recognition, and (9) using an appropriate form of recognition. Finally, expressing appreciation might not only have a positive impact on employees' health and well-being but also further positive consequences. For instance, on the organizational level, appreciation has been associated with higher work engagement (Babic et al., 2020; Bakker et al., 2007; Wang et al., 2020), lower turnover intentions (Apostel et al., 2018; Ng, 2016), higher intrinsic motivation (Cameron & Pierce, 1994; Deci & Ryan, 2000; Ryan & Deci, 2000), and task performance (Stajkovic & Luthans, 2003). Moreover, positive cross-over effects into the family domain have been found (Babic et al., 2020; Wöhrmann et al., 2017).

The *strengths* of our study include the assessment of CHD risk by major independent biological risk factors under resting conditions. In addition, to maximize the score ranges in CHD risk factors, we included apparently healthy participants in the normotensive to hypertensive range and medicated hypertensive and CHD patients. Moreover, we controlled for a variety of potential confounding variables and tested for specificity of the proposed association. The *limitations* of our study comprise the limited generalizability of our results beyond middle aged, employed men. Furthermore, we recruited blood donors, which, however, should not further limit the generalizability of our findings (Rogers & Glendon, 2003). Notably, given that we included employees from various organizations pursuing a wide variety of jobs, our results are unlikely to be driven by a specific organization or occupation. Nevertheless, potential occupation-specific differences as well as effects of working conditions, including shift work, should be scrutinized in future studies. Moreover, as the meaning and importance of appreciation may diverge in different cultures, further studies in other cultures are needed (Bello et al., 2010). In addition, the medication of our hypertensive and CHD patients could have confounded their CHD risk. However, analyses including all participants as well as medication-free, non-smoking participants yielded very similar results. Similarly, white coat or

masked hypertension may confound CHD risk. However, complementary analyses after excluding white coat or masked hypertensive participants ($n=6$) did not significantly change results (data not shown). In addition, we did not control for physical (in)activity, another important classical CHD risk factor (Li & Siegrist, 2012; Sattelmair et al., 2011). Notably, future studies should assess and consider health behaviors, such as physical activity, given that health behaviors could constitute a potential pathway underlying the association between appreciation at work and CHD risk (Kubzansky et al., 2018; Levine et al., 2021; Pressman et al., 2019; Siegrist & Rödel, 2006). Moreover, our results are cross-sectional and do not allow causal conclusions, and the possible impact of third variables cannot be ruled out. For example, participants who were especially healthy might have triggered more appreciation due to superior performance and positive social behavior.

Taken together, we found higher appreciation at work to significantly and independently relate to lower overall CHD risk, and, in particular, to lower MAP and lower blood lipids. In other words, the current study provides further evidence indicating that appreciation at work is an important resource for health and well-being. Future studies are needed to verify the observed results and to elucidate whether our findings also apply to women and across cultures. Moreover, it should be investigated if different interaction levels, forms, or sources of appreciation may differ in their associations with health and well-being. In addition, future research is needed to determine causality and to elucidate underlying mechanisms.

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Statement of Ethics: The study involving humans was approved by Ethics Committee of the State of Bern, Switzerland (Kantonale Ethikkommission Bern (KEK), reference number KEK-BE: 215/10). The study was conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

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Tables

Table 1*Group Characteristics*

	All participants <i>N</i> = 103	Medication-free, non-smoking participants <i>n</i> = 70
Group assignment	<i>n</i> = 25 CHD patients <i>n</i> = 40 HT <i>n</i> = 32 NT <i>n</i> = 6 wc/m HT	<i>n</i> = 32 HT <i>n</i> = 32 NT <i>n</i> = 6 wc/m HT
Age (years)	50.15 ± 0.98 (25–71)	48.29 ± 1.23 (25–71)
BMI (kg/m ²)	27.46 ± 0.36 (19.78–38.90)	27.27 ± 0.46 (19.78–38.86)
Smoking [% (N)]	3.88 % (<i>n</i> = 4)	-
Working hours	<i>n</i> = 100 43.35 ± 0.68 (20–58)	<i>n</i> = 69 43.40 ± 0.76 (20–55)
Appreciation at work	5.37 ± 0.09 (3.10–6.90)	5.38 ± 0.10 (3.10–6.90)
Perceived social support	5.18 ± 0.06 (3.50–6.00)	5.19 ± 0.08 (3.50–6.00)
Self-esteem	<i>n</i> = 100 34.35 ± 0.43 (20–40)	<i>n</i> = 69 34.32 ± 0.44 (25–40)
Amount of stress induced by lack of appreciation	<i>n</i> = 102 23.29 ± 0.28 (11–25)	<i>N</i> = 69 23.42 ± 0.35 (11–25)
BP (study)		
SBP (mmHg)	138.23 ± 1.54 (109.33–189.67)	138.43 ± 1.92 (109.33–189.67)
DBP (mmHg)	85.03 ± 1.12 (58.33–115.00)	86.00 ± 1.42 (58.33–115.00)
MAP (mmHg)	102.76 ± 1.22 (75.33–139.89)	103.47 ± 1.54 (75.33–139.89)

HbA1c (nmol/mol)	37.08 ± 0.39 (28–50)	36.27 ± 0.45 (28–49)
TC/HDL-C ratio	3.76 ± 0.09 (2.01–6.41)	3.96 ± 0.12 (2.01–6.41)
Coagulation		
Fibrinogen (g/L)	2.58 ± 0.05 (1.47–3.97)	2.55 ± 0.06 (1.47–3.97)
D-Dimer (µg/L)	441.28 ± 26.45 (45–1,481)	458.50 ± 34.59 (45–1,481)
Inflammation		
IL-6 (pg/mL)	0.51 ± 0.03 (0.16–1.52)	0.48 ± 0.03 (0.16–1.52)
TNF-α (pg/mL)	1.98 ± 0.06 (0.70–4.91)	1.95 ± 0.08 (0.70–4.91)
CRP (µg/mL)	<i>n</i> = 89 2.43 ± 0.22 (0.07–9.59)	<i>n</i> = 58 2.66 ± 0.29 (0.11–9.59)

Note. Values are means ± SEM (range) if not indicated differently; BMI = body mass index; BP = blood pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; TC/HDL-C ratio = total cholesterol/high-density lipoprotein-cholesterol ratio; IL-6 = interleukin-6, TNF-α = tumor necrosis factor-α; CRP = C-reactive protein; CHD patients = patients with coronary heart disease; NT = normotensive participants; HT = hypertensive participants; wc/m HT = white coat or masked hypertension; *n* = sample size; deviating sample sizes of a parameter are indicated.

Table 2

Results of MANCOVAs (with Appreciation at Work Predicting the Latent Variable CHD Risk with MAP, HbA1c, TC/HDL-C Ratio, the Coagulation Index as well as the Inflammatory Index as Dependent Variables) in All Participants as well as in Medication-Free, Non-Smoking Participants, and Post-Hoc Linear Regression Analyses with Each Dependent Variable Separately Regressed on Appreciation at Work

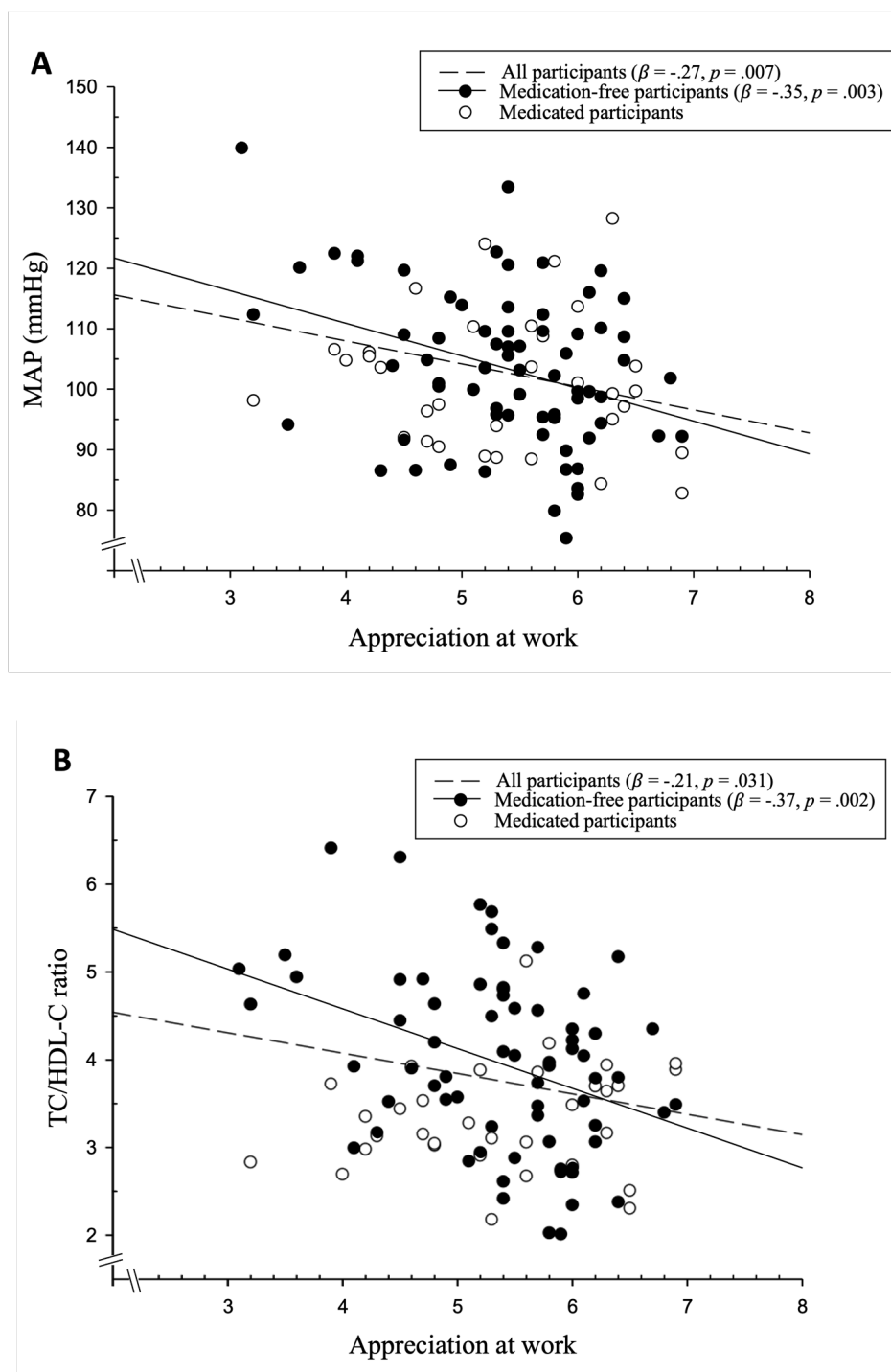
MANCOVAs	All participants ($N = 103$) without covariates (with covariates of medication intake, age, BMI, and smoking)					medication-free, non-smoking participants ($n = 70$) without covariates (with covariates of age and BMI)				
	$[df_{Num}, df_{Den}]$	F	p	η^2_p	Wilk's Λ	$[df_{Num}, df_{Den}]$	F	p	η^2_p	Wilk's Λ
Appreciation at work	[5, 97] (5, 90)	3.16 (2.36)	.011 (.046)	.14 (.12)	.86 (.88)	[5, 64] (5, 62)	3.49 (2.98)	.008 (.018)	.21 (.19)	.79 (.81)
Post-hoc linear regression analyses		β	p	$(\Delta)R^2$			β	p	$(\Delta)R^2$	
MAP		-.27 (-.19)	.007 (.026)	.07 (.04)			-.35 (-.25)	.003 (.011)	.12 (.06)	
HbA1c		-.14 (-.12)	.15 (.24)				.05 (.06)	.70 (.66)		
TC/HDL-C ratio		-.21 (-.17)	.031 (.079)	.05 (.03)			-.37 (-.32)	.002 (.005)	.14 (.10)	
Coagulation index		.15 (.16)	.14 (.11)				.17 (.18)	.15 (.15)		
Inflammation index		-.10 (-.03)	.34 (.72)				-.04 (.01)	.77 (.95)		

Note. MAP = mean arterial pressure; TC/HDL-C ratio = total cholesterol/high-density lipoprotein-cholesterol ratio; coagulation index = averaged z-transformed levels of D-dimer and fibrinogen; inflammation index = averaged z-transformed levels of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and C-reactive protein (CRP), n = sample size; df_{Num} = degrees of freedom numerator; df_{Den} = degrees of freedom denominator; significant values are highlighted in bold ($p < .05$).

Figures

Figure 1

Higher Appreciation at Work Significantly Related (A) Lower Mean Arterial Pressure (MAP) ($\beta = -.27, p = .007, R^2 = .07$) and (B) Lower Blood Lipids (Total Cholesterol (TC)/High-Density Lipoprotein-Cholesterol (HDL-C) Ratio) ($\beta = -.21, p = .031, R^2 = .05$)



APPENDIX C**Research Paper 3****Do Hypertensive Men Spy with an Angry Little Eye?****Anger Recognition in Men with Essential Hypertension – Cross-Sectional and
Prospective Findings**

This research paper was published in:

Auer, A., von Känel, R., Lang, I., Thomas, L., Zuccarella-Hackl, C., Degroote, C., Gideon, A., Wiest, R., & Wirtz, P. H. (2022). Do hypertensive men spy with an angry little eye? Anger recognition in men with essential hypertension – Cross-sectional and prospective findings. *Annals of Behavioral Medicine*, 56(9), 875–889.
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Abstract

Background & Objectives: Higher trait anger has inconsistently been associated with hypertension and hypertension development, but social context in terms of recognition of other persons' anger has been neglected in this context. Here, we investigated anger recognition of facial affect and trait anger in essential hypertensive and normotensive men in addition to prospective associations with blood pressure (BP) increases.

Methods: Baseline assessment comprised a total of 145 participants including 57 essential hypertensive and 65 normotensive men who were otherwise healthy and medication-free. Seventy-two eligible participants additionally completed follow-up assessment 3.1 (± 0.08 SEM) years later to analyze BP changes over time. We assessed emotion recognition of facial affect with a paradigm displaying mixed facial affect of two morphed basic emotions including anger, fear, sadness, and happiness. Trait anger was assessed with the Spielberger trait anger scale.

Results: Cross-sectionally, we found that with increasing BP, hypertensive men overrated anger displayed in facial expressions of mixed emotions as compared to normotensive men (p 's $\leq .019$) while there were no differences in trait anger ($p = .16$). Prospectively, the interaction between mean anger recognition and trait anger independently predicted BP increases from baseline to follow-up (p 's $\leq .043$), in that overrating displayed anger predicted future BP increases only if trait anger was high.

Discussion: Our findings indicate an anger recognition bias in men with essential hypertension and that overrating displayed anger in combination with higher trait anger seems to predict future BP increases. This might be of clinical relevance for the development and progression of hypertension and cardiovascular disease.

Keywords: hypertension, blood pressure, trait anger, anger recognition (bias), mixed emotions

Introduction

Arterial hypertension is a major risk factor for cardiovascular disease. About 95% of patients with arterial hypertension are diagnosed with “essential” hypertension, meaning there is no clear medical explanation for their condition (Carretero & Oparil, 2000). This stimulated research to investigate the potential role of psychosocial factors in essential hypertension. In particular, *anger* as an emotion-related psychosocial factor has been associated with hypertension and hypertension development (for reviews see Diamond, 1982; Rutledge & Hogan, 2002; Schum et al., 2003). Anger is a multidimensional construct and in earlier research, the terms anger, hostility, and aggression were often used interchangeably without clear conceptual distinction (Martin et al., 2000). In this context, an early review indicated associations between anger and hostility and the development of hypertension (Diamond, 1982). Later meta-analyses differentiated between anger experience and anger expression and reported small and highly variable or modest *cross-sectional* relationships between anger and blood pressure (BP) (Schum et al., 2003; Suls et al., 1995) with hostility inconsistently assigned to anger experience (Schum et al., 2003; Suls et al., 1995) and/or expression (Suls et al., 1995). Prospectively, there was support for associations between anger expression and the development of hypertension (Rutledge & Hogan, 2002). Currently, a three-factor model considering trait anger to have distinct affective, behavioral, and cognitive dimensions has been proposed to represent the best conceptual structure (Martin et al., 2000). The affective dimension refers to feelings of anger, the behavioral dimension to aggression and anger expression, and the cognitive dimension to hostility (Buss & Perry, 1992; Martin et al., 2000).

An established scale with proven reliability and validity to assess “trait anger” is the Spielberger trait anger scale (Spielberger, 1988). According to Spielberger (1988), trait anger represents the disposition to frequently and intensively experience anger over a range of situations. Based on this definition, we refer in the following to the affective dimension of trait anger when using the term trait anger. With respect to associations with hypertension we argue

that feelings of anger are accompanied by heightened sympathetic activation (Cacioppo et al., 2000; Kreibig, 2010), and that frequent anger feelings and thus increased sympathetic activation may accumulate over time. According to the allostatic load model (McEwen, 1998), this may result in chronic BP elevation. Some *cross-sectional* studies assessing trait anger accordingly found higher trait anger with increasing resting BP or hypertension (Hosseini et al., 2011; May et al., 2014; Mushtaq & Najam, 2014), whereas others did not (Deffenbacher et al., 1996; Friedman et al., 2001; Larkin & Zayfert, 2004; Mueller et al., 2001). The vast majority of cross-sectional studies reported mixed results where associations between trait anger and BP or hypertension were not supported unequivocally with findings varying between the studies (Beatty & Matthews, 2009; Bishop et al., 2005; Casagrande et al., 2020; Clark, 2006; Durel et al., 1989; Johnson, Schork, & Spielberger, 1987; Johnson, Spielberger, et al., 1987; Laude et al., 1997; van der Ploeg et al., 1985). The above-mentioned meta-analyses summarized different anger scales to experience of anger either with (Schum et al., 2003) or without (Suls et al., 1995) inclusion of the Spielberger trait anger scale (Spielberger, 1988) and found positive associations between anger experience and diastolic BP (DBP) (Suls et al., 1995) and/or systolic BP (SBP) (Schum et al., 2003; Suls et al., 1995) BP. With respect to *prospective* assessment, trait anger according to Spielberger (1988) has not yet been investigated in manifest hypertension. However, few longitudinal studies enrolling normotensive or prehypertensive individuals assessed trait anger in the context of hypertension development. One epidemiological study in a cohort of middle-aged prehypertensive men and women followed for up to 8 years found that about 67% of participants with high baseline trait anger scores developed hypertension (Player et al., 2007). Two other studies in middle-aged normotensive women found increases in trait anger over time but not baseline trait anger to be predictive of BP increases (Markovitz et al., 1991) and hypertension incidence (Räikkönen et al., 2001). In sum, these cross-sectional and prospective findings remain inconclusive and the role of trait anger in hypertension warrants further elucidation.

The inconsistency of the above-summarized findings may relate to the assessment of trait anger by means of a self-report measure that does not capture anger within interpersonal or social context (Jorgensen et al., 1996). The personality in context theory states that personality does not reflect an individual in isolation but rather an individual within interpersonal or social context (Mischel & Shoda, 1995; Zayas et al., 2002). Based on this theory, trait anger may require situational features that in particular elicit feelings of anger and thus the affective dimension of trait anger, and corresponding interrelations. Moreover, the perception-action model for empathy (PAM) posits that in social contexts a person's own emotions strongly interact with the perception and recognition of emotions of others (Preston, 2007). In the sense of emotional contagion individuals come to feel a similar emotional state as the one perceived (Preston, 2007). To recognize a person's emotional state, facial expressions have been proposed to be of particular importance (Ekman, 1982). In line with the personality in context theory and the PAM, social context in terms of facial affect recognition may modulate the associations between personality traits, such as trait anger, and hypertension (development). More specifically, differences in the recognition of anger displayed in another person's face may interact with trait anger (in particular with its affective dimension), and thus modulate situational feelings of anger. This interaction may contribute to hypertension (development) in accordance with the allostatic load model (McEwen, 1998).

So far, evidence from cross-sectional studies points to associations between higher resting BP or hypertension and impaired emotion recognition. A very early study suggests that hypertensive individuals tend to recognize hostility in notably non-standardized and non-validated pictures of facial affect more often than non-hypertensive patients (Thaler et al., 1957). Later studies using standardized, reliable and valid pictures of facial affect reported impaired emotion recognition in borderline hypertensive participants as compared to normotensive controls (Rossi et al., 1986) and in bariatric surgery candidates with a history of hypertension (Manderino et al., 2015). Moreover, within the normotensive up to hypertensive

range higher resting BP (McCubbin et al., 2011; Shukla et al., 2019), elevated DBP (McCubbin et al., 2014), and elevated SBP (McCubbin et al., 2018) were found to relate to reduced accuracy in recognition of facial affect. The hitherto only study in manifest hypertension similarly revealed reduced facial emotion recognition accuracy in prehypertensive and hypertensive participants as compared to normotensive participants (Shukla et al., 2018). Interestingly, notwithstanding the potential role of anger in hypertension, all of these studies investigated different emotions displayed separately and then averaged the proportion of correctly recognized emotions to total recognized emotions, that is. emotion recognition accuracy. So far, two studies considered recognition of separate displayed emotions in borderline hypertensive participants (Rossi et al., 1986) or hypertensive heart disease patients (Yoris et al., 2020) and controls. The recognition of the emotion anger did not differ between groups despite an impaired overall emotion recognition in patients with hypertensive heart disease and borderline hypertensive participants compared to controls (Rossi et al., 1986; Yoris et al., 2020). Meanwhile, sophisticated methods have been developed to assess recognition of facial affect more specifically by displaying mixed facial affect comprising blends of two morphed basic emotions (Kaiser et al., 2019). This methodology allows to identify a recognition bias towards one emotion (e.g., anger) over others. To the best of our knowledge, neither recognition of mixed facial affect nor prospective associations regarding recognition of facial affect have been studied in the context of hypertension. Also, trait anger combined with anger recognition of facial affect has not yet been investigated in essential hypertension either cross-sectionally or prospectively.

We, therefore, set out to specifically investigate anger recognition and trait anger in otherwise healthy essential hypertensive and normotensive men and to determine prospective associations with BP changes. We assessed emotion recognition accuracy (i.e., underrating or overrating displayed emotion) of electronically morphed pictures of facial affect displaying two blended basic emotions (anger, fear, sadness, happiness). Cross-sectionally, we hypothesized

that hypertensive men would differ from normotensive controls in emotion recognition accuracy of anger displayed in pictures of mixed facial affect. Moreover, we expected prospective associations between anger recognition, trait anger, and BP increases over an average 3-year follow-up period. In particular, given the personality in context theory (Mischel & Shoda, 1995; Zayas et al., 2002) and the PAM (Preston, 2007), we expected prospective associations between the interaction of anger recognition with trait anger and future BP increases.

Methods

Study Participants

This study is part of a series of studies assessing psychoneurobiological mechanisms in essential hypertension and coronary heart disease (Zuccarella-Hackl, von Känel, Thomas, Hauser, et al., 2016; Zuccarella-Hackl, von Känel, Thomas, Kuebler, et al., 2016). The project was approved by the Ethics Committee of the State of Bern, Switzerland and conducted in accordance with the Declaration of Helsinki principles. All participants provided written informed consent for both assessments and were financially compensated for each assessment with 20 Swiss Francs.

For the *baseline cross-sectional assessment* of the present study, we recruited apparently healthy participants with BP in the normotensive to hypertensive range. The final cross-sectional sample comprised 145 participants with 65 normotensive participants, 57 essential hypertensive participants, and 23 participants without clear classification (16 white coat hypertensive participants, 7 masked hypertensive participants) (see below).

Due to gender differences regarding cardiovascular risk and facial affect recognition (Hall & Matsumoto, 2004), only men were included in the study. Recruitment was carried out with the aid of the Swiss Red Cross of the Canton of Bern. In detail, members of our study team accompanied the mobile blood-donation unit of the Swiss Red Cross that routinely records BP before blood donation. Male blood donors with elevated or normal BP expressing interest in

participating in the study were given study information asking for the following inclusion criteria: age 18–80 years; nonsmoker (≤ 3 cigarettes per day; to exclude confounding influences of smoking on BP (Omvik, 1996)); no current intake of medication. In addition, eligible hypertensive participants provided blood samples for routine assessment of serum creatinine, calcium, sodium, and potassium to test for potential cases with secondary hypertension. No eligible hypertensive participant showed critical values indicative of secondary hypertension post-hoc and all participants were included. Notably, calcium, sodium, and potassium could not be analyzed in eight hypertensive participants, in two of them, creatinine could not be analyzed either. To rule out diabetes, we measured HbA1c in all participants. HbA1c could not be assessed in two hypertensive, five normotensive, and three participants with white coat hypertension because of technical problems. Apart from hypertension-related criteria, normotensive controls had to meet the same inclusion and exclusion criteria as hypertensive participants verified by telephone interview using an extensive health questionnaire (Wirtz et al., 2008; Zuccarella-Hackl, von Känel, Thomas, Hauser, et al., 2016). Subjects who reported any chronic or acute clinical psychiatric disorders, current use of medication, heart disease, elevated cholesterol, elevated blood sugar and diabetes, liver and renal diseases, chronic obstructive pulmonary disease, rheumatic diseases, HIV, cancer, current infectious diseases, regular strenuous physical exercise, smoking, alcohol and illicit drug abuse were not eligible for study participation.

All 145 participants of the baseline assessment were invited for the *follow-up assessment* scheduled between 2 and 5 years later with a final sample of 72 participants completing both assessments (*mean (standard error of the mean (SEM))* = 3.06 (0.08)). Apart from study exclusion due to start of regular BP medication intake ($n=11$), reasons for drop-out comprised: severe illness ($n = 7$), no interest ($n = 10$), not reachable by phone ($n = 11$), lack of time ($n = 17$), or no reason given ($n = 17$). Notably, we initially intended to schedule for a 2-

year follow-up period that had to be prolonged due to organizational reasons. Drop-out rates did not differ between study groups ($\chi^2(2) = 1.35, p = .51$).

Assessment of Essential Hypertension

Classification of essential hypertension and normotension was based on a two-step assessment procedure to allow for detection of white coat and masked hypertension and thus, to ensure essential hypertension status validity. (1) *Home BP measurement*. Following written instructions, each interested candidate was instructed to measure BP on 3 separate days at home using sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofddorp, Netherlands). Home BP measurements were to be obtained in a seated position after a 15-minute rest twice per day (once in the morning and once in the evening). Based on a maximum of six home BP measurements, participants were preliminary classified as hypertensive if the average home SBP was ≥ 135 mmHg and/or the average home DBP was ≥ 85 mmHg according to recommendations for home or ambulatory BP measurements (Williams et al., 2018). Correspondingly, participants were preliminary classified as normotensive if average home SBP was < 135 mmHg and average home DBP was < 85 mmHg. (2) *Study BP measurement*. To verify the home-measurement-based preliminary classification of each participant as hypertensive or normotensive, trained personnel obtained up to three additional seated baseline BP measurements each after a 15-minute rest (three measurements: $n = 126$; two measurements: $n = 19$). BP was again assessed by means of sphygmomanometry (Omron M6; Omron Healthcare Europe B.V., Hoofddorp, Netherlands). For classification regarding study BP measurements, we applied the regular definition of hypertension based on office or clinic BP measurements and classified participants as hypertensive if their average study SBP was ≥ 140 mmHg and/or their average study DBP was ≥ 90 mmHg (Chambers et al., 1999; Williams et al., 2018). Participants were classified as normotensive if their average study SBP was < 140 mmHg and their average study DBP was < 90 mmHg.

Based on this two-step procedure, we recruited a total of 145 participants including 54 hypertensive and 65 normotensive participants with consistent group assignment based on both, home BP and study BP measurements. Three participants who were hypertensive based on self-report did not measure their BP at home due to technical problems and were classified according to study BP measurements only, rendering 57 hypertensive participants in total. Twenty-three participants deviated between home and study BP assessment with 16 participants displaying normotensive home BP and hypertensive study BP (white coat hypertension) and 7 participants displaying hypertensive home BP and normotensive study BP (masked hypertension).

Experimental Procedure

All participants abstained from caffeine and alcohol consumption for 24 hours and consumed a semi-standardized breakfast following written instructions prior to arrival at the lab at 8:00 a.m. Besides the assessment of height and weight as well as blood sampling for secondary hypertension screening, three seated *study baseline BP measurements* were performed each after a 15-minute rest. These study baseline BP measurements were used (1) for the final classification of essential hypertension or normotension as described above and (2) to calculate mean arterial pressure (MAP) (see below). The mixed emotion paradigm to assess emotion recognition (see below) started at 9:45 a.m. The experimental procedure was repeated at the follow-up assessment.

Mixed Emotion Paradigm

To assess emotion recognition, we used the mixed emotion forced-choice paradigm by Kaiser et al. (2019). Facial pictures of two male and two female actors were chosen from the “NimStim set of facial expression” (Tottenham et al., 2009) that we morphed from 30% to 70% of one emotional expression to another in 10% steps. This procedure resulted in a series of five different affect intensity pictures with blends of two morphed basic emotions, for example: 30%-anger/70%-sadness, 40%-anger/60%-sadness, 50%-anger/50%-sadness, 60%-anger/40%-sadness, and 70%-anger/30%-sadness (see Supplemental Material 1, Figure S1).

Affect intensity pictures comprised six blended emotion-combinations: anger to sadness, anger to fear, anger to happiness, fear to sadness, fear to happiness, and happiness to sadness. All pictures were projected separately on a screen in a randomized order. In a forced-choice decision task, participants were asked to choose as quickly as possible which of the two possible emotions was displayed by the presented facial expression. A corresponding button press stopped the presentation, and another face was displayed.

Pictures were randomly presented within two blocks for each blended emotion-combination (e.g. anger-fear). Each block contained 20 pictures of all four faces in all five affect intensities in a randomized order. In total, 12 blocks of the six blended emotion combinations were presented rendering a total of 240 pictures (four faces x five affect intensities x two blocks x six blended emotion-combinations). We averaged the percentage of anger recognition for each pairing of anger intensity (30/70%, 40/60%, 50/50%, 60/40%, 70/30%) with anger-other emotion combination (anger-fear, anger-happiness, anger-sadness), based on eight button presses (four faces x two blocks). We calculated *anger recognition accuracy* as the difference between an individual's percentage of anger recognition and the actually displayed anger intensity (30%-70%, see Figure 1B and 1C displaying emotion recognition accuracy of five emotion intensities averaged over the different emotion combinations). The closer to zero the accuracy score is, the more accurate the facial expression had been recognized, with positive scores indicating overrating and negative scores indicating underrating of the respective emotion. We accordingly averaged the percentages of recognition of fear, happiness, and sadness and calculated emotion recognition accuracy of non-anger emotions.

Trait Anger

We used the *trait anger* subscale of the German version of the State Trait Anger Expression Inventory (STAXI) to assess the affective dimension of anger (Schwenkmezger et al., 1992; Spielberger, 1988). The trait anger scale asks people to rate themselves with ten items such as "I am quick tempered" (German: „Ich werde schnell ärgerlich“) on a four-point Likert

scale (1 = *never* to 4 = *almost always*). We summarized items to compute the total trait anger score with higher scores indicating a greater level of trait anger. The trait anger scale has previously shown excellent psychometric quality (Eckhardt et al., 2004). In our study, $n = 17$ participants did not fill out the questionnaire and trait anger scores were available for $n = 128$ participants with acceptable internal consistency in our sample (Cronbach's $\alpha = .77$).

Statistical Analyses

Data were analyzed using SPSS (Version 26.0) statistical software packages for Macintosh (IBM SPSS Statistics, Chicago, IL, USA) and presented as mean $\pm$ standard error of the mean (SEM). All tests were two-tailed with the significance level set at $p < .05$.

We a-priori calculated power-analyses using the statistical software G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). Expecting effects of small to medium effect size (Shukla et al., 2018) of $f = .15$ with a power of .90 and an average correlation of the repeated measures of $r = .11$ in a conservatively calculated 2 (groups) x 5 (affect intensities) repeated measures analysis of variance (ANOVA) for one emotion combination, the required sample size is $N=138$ in cross-sectional analyses. For prospective analyses, the required sample size to detect medium effects of $R^2 = .13$ in linear regression analyses is $N = 73$.

For all participants, we calculated MAP at baseline and follow-up assessment based on the up to three study BP measurements by the formula $\text{MAP} = (2/3 * \text{mean study DBP}) + (1/3 * \text{mean study SBP})$ as well as body mass index (BMI) by the formula $\text{BMI} = \text{kg/m}^2$. As MAP had greater predictive power for cardiovascular mortality in the general population as compared to SBP and DBP separately (Lewington et al., 2002), we used MAP as combined BP measure in our linear analyses. Analyses considering trait anger were conducted threefold: first, with missings excluded (trait anger missings in cross-sectional analyses: $n = 17$ (nine normotensive participants, six hypertensive participants, two participants with white coat or masked hypertension); in prospective analyses: $n = 3$ (two normotensive participants, one

hypertensive participant)) as depicted in tables and figures, second, by replacing missings with group means of trait-anger scores, and third, by replacing missings using multiple imputation as implemented in SPSS (Version 26.0).

All data were tested for normal distribution and homogeneity of variance using Kolmogorov-Smirnov and Levene's tests prior to statistical analyses. Statistical analyses were conducted with original untransformed data because (1) our sample size is larger than $n = 30$ (Field, 2018), (2) we used AN(C)OVAs, that are in general relatively robust against violations of normality (Berkovits et al., 2000; Glass et al., 1972; Harwell, 2003; Harwell et al., 1992; Schmider et al., 2010), and (3) data transformation did not improve distribution of skewed emotion recognition accuracies. Furthermore, effect size parameters f were calculated from partial η^2 (η_p^2) using G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). Effect size parameters f (effect size conventions f : .10 = small; .25 = medium; .40 = large (Cohen, 1988)) and R^2 changes (ΔR^2 ; effect size conventions R^2 : .02 = small; .13 = medium; .26 = large (Cohen, 1988)) are reported where appropriate.

To compare group characteristics at baseline and test for differences between follow-up and drop-out participants, we used independent t -tests and χ^2 -tests where appropriate.

Emotion Recognition in Essential Hypertension

To test whether hypertensive participants differ from normotensive participants in anger recognition accuracy, we applied the following procedure: first, we calculated a repeated measures ANOVA with group as independent variable and two factors representing anger recognition accuracy as dependent variable. The first factor consists of the three displayed contrasting emotion combinations (*anger-fear*, *anger-happiness*, *anger-sadness*), and the second factor comprises the five displayed affect intensities of anger (30/70%, 40/60%, 50/50%, 60/40%, 70/30%). Given age effects in emotion recognition (Ruffman et al., 2008) and computer-based tasks (Band & Kok, 2000; Hubel et al., 2013), we conducted all analyses without and with controlling for age as covariate. Second, we tested for linear associations

between anger recognition accuracy and MAP as a continuous measure of hypertension by recalculating repeated measures AN(C)OVAs but using MAP as the independent variable instead of group, again without and with covarying for age. Third, to test whether trait anger relates to the hypothesized associations between hypertension/MAP and anger recognition accuracy, either directly, or as a moderating factor, we repeated the above-described two-step procedure entering trait anger without and with the interaction trait anger-by-group (or trait anger-by-MAP).

We performed the above-described two-step procedure accordingly for *fear*, *happiness*, and *sadness* recognition accuracy as dependent variables.

Prediction of MAP Change in Prospective Follow-Up Assessment

To test for potential *direct associations*, we calculated hierarchical linear regressions with MAP change (MAP follow-up minus baseline assessment) as dependent variable and *mean* anger recognition accuracy (i.e. anger recognition accuracy averaged over all affect intensities and anger other-emotion combinations) and/or trait anger as independent variable(s). To test for *moderation effects*, we additionally entered the corresponding interaction terms between *mean* anger recognition accuracy and trait anger as independent variables. All prospective analyses were conducted twice: first, including participants with consistent group assignment only ($n = 62$), and second, including all participants ($n = 72$) as depicted in the prospective analyses figure with missing trait anger scores excluded. Whereas participants with antihypertensive medication intake at follow-up were a-priori excluded ($n = 11$), we controlled for blood lipids and diabetes medication intake ($n = 6$) as well as any other medication intake ($n = 7$) that started since baseline. Each analysis was performed without and with controlling for baseline age, baseline MAP (Berntson et al., 1994), BMI changes (BMI follow-up minus baseline assessment), and medication-intake as potential confounders as well as days between baseline and follow-up assessment. The latter covariate was needed to at least statistically control for the follow-up differences ranging from 2 up to 5 years.

Notably, all interaction terms were calculated after prior z-transformation of the respective composing variables, and moderation testing comprised z-transformed independent variables.

Results

Group Characteristics

Table 1 depicts group characteristics of the 145 participants studied at baseline and group characteristics of the 72 participants studied during the follow-up assessment. As expected, hypertensive participants displayed higher average SBP, DBP, MAP, and BMI compared to normotensive participants (p 's $< .001$) as well as comparable age ($p = .54$) at baseline. Furthermore, hypertensive and normotensive participants did not significantly differ in their trait anger scores ($p = .16$) or any other group characteristics (p 's $\geq .42$). There were no significant group differences between the follow-up and the drop-out participants in any baseline characteristic (see Table 2).

Cross-Sectional Analyses: Emotion Recognition in Essential Hypertension

Anger Recognition

Over all affect intensities and contrasting anger emotion combinations, hypertensive participants significantly differed from normotensive participants in anger recognition accuracy ($F(1, 120) = 7.37, p = .008, \eta^2_p = .058, f = .25$; with age as covariate: $F(1, 119) = 7.40, p = .008, \eta^2_p = .059, f = .25$; see Figures 1A and 1B). More precisely, hypertensive participants displayed lower accuracy in recognizing anger in terms of comparably overrating displayed anger. These group differences remained when additionally controlling for trait anger without and with missing trait anger scores replaced either with group means or multiple imputation (p 's $\leq .011$) with trait anger not being related to anger recognition (p 's $\geq .20$). Moreover, trait anger did not moderate the observed group differences in anger recognition (p 's $\geq .22$).

Complementary analyses using MAP as linear independent variable instead of group confirmed the above findings: over all affect intensities and contrasting anger emotion

combinations higher MAP significantly related to anger recognition accuracy in that displayed anger is overrated with increasing MAP ($F(1, 120) = 5.62, p = .019, \eta^2_p = .045, f = .22$; with age as covariate: $F(1, 119) = 5.70, p = .019, \eta^2_p = .046, f = .22$; see Figure 2). Again, these associations were independent of trait anger without and with missing trait anger scores replaced either with group means or multiple imputation (p 's $\leq .028$) while trait anger did not relate to anger recognition (p 's $\geq .17$). Also, trait anger did not moderate the association between MAP and anger recognition (p 's $\geq .17$).

Non-Anger Emotion Recognition

We further tested whether hypertensive participants would also differ from normotensive controls in non-anger emotion recognition (i.e. fear, happiness, or sadness), again by comparing groups and complementary association testing with MAP. There were no significant findings except for a borderline significant finding regarding sadness recognition accuracy in relation to MAP, notably in the opposite direction as compared to anger recognition and not independent of age (without age as covariate $p = .087$; with age as covariate $p = .12$). Figures 1A and 1C depict non-anger emotion recognition accuracy in hypertensive and normotensive participants, and Figure 2 depicts corresponding relations with MAP.

Prospective analyses: Prediction of MAP Change by Anger Recognition, Trait anger, and their Interaction

Direct Effects

We calculated hierarchical linear regression analyses with MAP change as dependent variable and mean anger recognition accuracy and/or trait anger without and with missing trait anger scores replaced (with group means/ multiple imputation) as independent variables. There were no direct associations with MAP change either when considering participants with consistent baseline group assignment only (mean anger recognition accuracy: p 's $\geq .76$; trait anger: p 's $\geq .55$) or when considering all follow-up participants including white coat and masked hypertension (mean anger recognition accuracy: p 's $\geq .74$; trait anger: p 's $\geq .75$). Controlling

for the a-priori defined set of confounders (i.e. baseline age, baseline MAP, BMI change, days between baseline and follow-up assessment, and medication-intake) did not significantly change these results (mean anger recognition accuracy: p 's $\geq .46$; trait anger: p 's $\geq .18$). Notably, of these confounders, higher baseline MAP (β 's $\leq -.25$, p 's $\leq .039$, ΔR^2 's $\geq .05$) related to decreases or lower increases in MAP from baseline to follow-up. Higher BMI increases (β 's $\geq .44$, p 's $< .001$, ΔR^2 's $\geq .19$) related to higher MAP increases from baseline to follow-up.

Moderation Effects

Moderation testing revealed that the interaction between mean anger recognition accuracy and trait anger without missings replaced significantly predicted MAP change both, in participants with consistent baseline group assignment ($\beta = .38$, $p = .006$, $\Delta R^2 = .13$) and when considering all follow-up participants including white coat and masked hypertension ($\beta = .33$, $p = .009$, $\Delta R^2 = .10$). These associations remained when replacing missing trait anger scores with group means or multiple imputation (p 's $\leq .009$). Notably, controlling for confounders did not change the significance of the observed moderation effects (p 's $\leq .043$). Again, higher BMI increases (β 's $\geq .46$, p 's $< .001$, ΔR^2 's $\geq .18$) related to higher MAP increases over time and higher baseline MAP (marginal significantly) related to decreases or lower increases in MAP over time (β 's $\leq -.21$, p 's $\leq .083$, ΔR^2 's $\geq .03$).

Figure 3 depicts moderation by trait anger: overrating of displayed anger predicted higher MAP increases from baseline to follow-up only if trait anger was high but not if trait anger was low.

Discussion

Here, we investigated cross-sectionally whether essential hypertensive men differ from normotensive men in anger recognition and trait anger. We additionally examined prospective associations of anger recognition and trait anger with BP changes over an average 3-year follow-up period.

Cross-sectionally, we found that hypertensive men overrated displayed anger as compared to normotensive men. In other words, hypertensive men tend to recognize anger more often when evaluating ambiguous facial expressions comprising anger plus another emotion. These results were confirmed in complementary analyses using MAP as continuous hypertension measure, suggesting a linear relationship with increasing BP. We argue that the observed recognition bias towards anger in hypertension is in line with previous studies reporting impaired emotion recognition in the context of hypertension (Manderino et al., 2015; McCubbin et al., 2014; McCubbin et al., 2011; McCubbin et al., 2018; Rossi et al., 1986; Shukla et al., 2018; Shukla et al., 2019; Yoris et al., 2020) due to the following: while we used pictures displaying mixed facial affect allowing to identify a recognition bias towards one emotion over others, these studies presented facial expressions displaying prototypical single basic emotions. Emotion recognition accuracy was then averaged over several emotions by calculating the proportion of correctly recognized emotions (“hits”) to total recognized emotions (“hits+errors”). Notably, observers tend to perceive other emotions in addition to displayed prototypical basic emotion in facial expressions at least to a certain degree (Ekman et al., 1987). Given this, our findings suggest that hypertensive men correctly identify displayed anger but may overrate anger in facial expressions of non-anger emotions leading to increased error rates in total, that is, over all emotions. As a consequence, emotion recognition accuracy as proportion of hits to hits+errors would be impaired as previously observed in the context of hypertension over all emotions (Manderino et al., 2015; McCubbin et al., 2014; McCubbin et al., 2011; McCubbin et al., 2018; Rossi et al., 1986; Shukla et al., 2018; Shukla et al., 2019; Yoris et al., 2020), but not when considering anger separately (Rossi et al., 1986; Yoris et al., 2020). Notably, we could not observe relevant group differences in non-anger emotion recognition or relations to MAP.

Furthermore, we did not find group differences between hypertensive and normotensive men in trait anger and trait anger was neither associated with anger recognition accuracy nor

did it moderate the associations between anger recognition and group or MAP. In line with previous studies that did not find higher trait anger with increasing resting BP or hypertension (Deffenbacher et al., 1996; Friedman et al., 2001; Larkin & Zayfert, 2004; Mueller et al., 2001), our findings further question an independent role for trait anger in essential hypertension.

Prospectively, our analyses did not support expected direct associations between anger recognition or trait anger and BP increases over an average 3-year follow-up period. However, the interaction between mean anger recognition accuracy and trait anger predicted MAP changes from baseline to follow-up as expected. With regard to *clinical implications* these results suggest that in men, high trait anger together with a recognition bias towards anger contribute to further BP increases and thus progression of hypertension and related cardiovascular risk. This finding is in line with the personality in context theory that personality does not reflect an individual in isolation but rather an individual within interpersonal or social context (Mischel & Shoda, 1995; Zayas et al., 2002). Our results indicate that trait anger seems to require situational features such as perceiving anger in facial expressions of interaction partners to elicit anger, or feelings of anger, respectively. Moreover, it is conceivable that trait anger predisposes to select anger-related situations and to modulate social situations (Zayas et al., 2002). The observed interaction can be interpreted based on the allostatic load model (McEwen, 1998): Given that feelings of anger are characterized by heightened sympathetic activation (Cacioppo et al., 2000; Kreibig, 2010), frequent anger feelings and thus increased sympathetic activation may accumulate over time, and result in chronic BP elevation. Notably, these implications should be treated with caution, as our participants were hypertensive and normotensive men. It remains to be elucidated whether our findings and thus the resulting implications also apply to women with hypertension.

We can only speculate about *mechanisms* underlying the observed anger recognition bias in hypertension in our study participants. On the one hand, the observed anger recognition bias could predispose to BP increases and thus hypertension development. According to the

PAM (Preston, 2007), in the sense of anger contagion, perceiving anger in interaction partners likely results in own feelings of anger with concomitant increased sympathetic activation (Kreibig, 2010) that may accumulate over time (McEwen, 1998). At the same time, impaired emotion recognition that others observed in hypertension (Manderino et al., 2015; McCubbin et al., 2014; McCubbin et al., 2011; McCubbin et al., 2018; Rossi et al., 1986; Shukla et al., 2018; Shukla et al., 2019; Yoris et al., 2020) may lead to inadequate social behavior (van Kleef, 2009) resulting in reduced quality of social relationships with lower social support, social distancing and isolation. As a consequence, stress-buffering is reduced (Christenfeld & Gerin, 2000; Lepore, 1998), giving rise to more stressful experiences and further BP increases. On the other hand, an anger recognition bias could be a consequence of increased BP or hypertension. In order to respond quickly to potential environmental threats in social contexts, angry faces are in general detected more quickly and accurately than happy or other non-threatening faces, referred to as anger superiority effect (Horstmann & Bauland, 2006; Pinkham et al., 2010). Due to their BP hyperactivity, individuals with hypertension might be on higher alert and thus more sensitive to perceive threatening stimuli. This may result in an overestimation of threatful stimuli such as angry faces. It is also conceivable that both proposed mechanisms interact to increase BP. Additional mechanisms may comprise integrated neurophysiological pathways that control both, resting BP and affect (McCubbin et al., 2014; McCubbin et al., 2018; Porges, 2003; Thayer & Lane, 2000).

As outlined before, an anger recognition bias, especially if combined with trait anger, may contribute to the development and progression of essential hypertension and related cardiovascular risk. *Therapeutic implications* include to not only address anger conventionally with regard to individual's responses to anger (Glancy & Saini, 2005) but to additionally target anger recognition by training the perception of the social environment, especially emotion recognition of facial affect. For example, experimental modification of biases in emotion recognition by promoting perception of happiness over anger in ambiguous facial expressions

has been shown to decrease state anger (Penton-Voak et al., 2013). Further positive effects of emotion recognition training may comprise improved social behavior (van Kleef, 2009) that in turn can increase social support with its beneficial effects on cardiovascular health (Christenfeld & Gerin, 2000; Lepore, 1998). Again, generalization of our findings to populations other than men with and without hypertension is unclear.

Strengths of our study include the prospective design, which allowed participant monitoring over an average follow-up period of 3 years. Second, we had a rigid hypertension assessment procedure ensuring hypertension status validity by including assessment of both, home and study BP combined with exclusion of the diagnosis of secondary hypertension. This procedure also allows to detect white coat and masked hypertension. Third, in real-life social contexts facial expressions of emotions are proposed to appear not as straightforward as prototypical facial expressions of basic emotions, but rather ambiguous (Aviezer et al., 2017; Hassin et al., 2013). Additionally, depending on the situation, natural emotional expressions are more likely subtle or mixed. Therefore, our mixed emotion paradigm may be more ecologically valid than prototypical emotion recognition assessment. Fourth, combined investigation of both, trait anger and anger recognition of mixed facial affect, may shed light on how these factors interact in the development of essential hypertension, revealing more specific potential mechanisms. Last, we controlled for a variety of potential confounders.

Limitations of our study are that the generalizability of our results is limited to normotensive and hypertensive but otherwise healthy and medication-free men. Also, we recruited blood donors, which however should not further limit the generalizability of our findings (Rogers & Glendon, 2003). Second, ecological validity of our paradigm is unclear. Our displayed mixed facial expressions were morphed static pictures and do not necessarily reflect natural facial expressions. Also, our displayed facial expressions were isolated from body motion, posture, and prosody limiting the amount of perceivable information (de Gelder et al., 2015). Third, modern conceptualizations consider trait anger to not only have an affective dimension as

measured by the Spielberger trait anger scale (Spielberger, 1988) but also behavioral and cognitive dimensions with potentially different impacts on cardiovascular health (Martin et al., 2000; Suls, 2013). Finally, participant number of our prospective assessment was substantially reduced. Whereas start of antihypertensive medication intake was a major exclusion reason inherent in our study design, we feel that the reduced and less flexible offers for follow-up assessment time-points due to organizational reasons may have contributed to a higher drop-out rate. Notably, drop-out and follow-up participants did not substantially differ in their characteristics (see Table 2).

Taken together, our findings provide for the first-time evidence for an anger recognition bias in essential hypertension in terms of overrating anger displayed in facial expressions of mixed emotions. Noteworthy, in our study normotensive and hypertensive men did not differ in trait anger. The proposed clinical significance of our findings is supported by our prospective data with anger recognition interacting with trait anger in the prediction of BP increases over an average period of 3 years. Future studies are needed to verify the observed results obtained in hypertensive men and whether they extend to women with hypertension since gender differences in facial affect recognition have been described (Hall & Matsumoto, 2004). In addition, the potential role of anger recognition and trait anger in hypertension development and ecological validity of the mixed emotions paradigm warrant further investigation.

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Tables

Table 1

Baseline and Follow-Up Group Characteristics of Medication-Free Hypertensive Participants, Normotensive Participants, and Participants with White Coat or Masked Hypertension

	Baseline group characteristics (N=145)			Follow-up group characteristics (N=72)					
	NT n=65	HT n=57	wc/m HT n=23	p-value			NT n=40	HT n=22	wc/m HT n=10
				NT vs. HT	NT vs. wc/m HT	HT vs. wc/m HT			
Age (yrs)	49.82 ± 1.50 (25–78)	51.14 ± 1.53 (26–74)	53.43 ± 3.08 (21–73)	.54	.25	.46	51.90 ± 1.91 (31–81)	56.14 ± 2.72 (31–77)	53.30 ± 4.88 (33–73)
BMI (kg/m ²)	25.27 ± 0.30 (19.78–30.85)	28.71 ± 0.55 (20.35–38.86)	26.96 ± 0.73 (21.55–35.26)	< .001	.040	.077	24.89 ± 0.38 (20.07–28.59)	29.14 ± 1.00 (23.40–40.44)	28.10 ± 1.13 (23.72–33.44)
BP (home)		n = 54							
SBP (mmHg)	121.87 ± 0.82 (105.17–133.83)	145.26 ± 1.04 (132.17–162.33)	130.37 ± 2.16 (115.67–148.33)	< .001	< .001	< .001			
DBP (mmHg)	71.72 ± 0.71 (60.00–82.33)	87.00 ± 0.89 (75.67–103.00)	77.27 ± 1.14 (68.33–84.83)	< .001	< .001	< .001			
BP (study)									
SBP (mmHg)	126.57 ± 1.05 (108.67–139.67)	153.60 ± 1.48 (129.33–189.67)	141.60 ± 2.67 (109.50–177.00)	< .001	< .001	< .001	128.80 ± 1.85 (102.33–161.67)	151.98 ± 4.15 (102.67–181.67)	147.10 ± 4.27 (123.67–167.00)
DBP (mmHg)	77.59 ± 0.87 (58.33–88.67)	95.47 ± 1.14 (73.67–115.00)	88.15 ± 1.26 (75.00–96.67)	< .001	< .001	< .001	78.33 ± 1.22 (55.67–92.67)	91.06 ± 2.73 (65.67–112.00)	87.83 ± 1.59 (81.00–93.67)
MAP (mmHg)	93.92 ± 0.86 (75.33–104.56)	114.85 ± 1.17 (95.78–139.89)	105.97 ± 1.54 (86.50–117.44)	< .001	< .001	< .001	95.15 ± 1.34 (71.22–111.78)	111.37 ± 3.00 (79.56–135.22)	107.59 ± 2.05 (95.22–115.78)
MAP change (mmHg)							1.35 ± 1.06 (-13.8–11.44)	-2.97 ± 2.39 (-40.00–14.22)	0.89 ± 1.83 (-7.00–8.72)
Trait anger	n = 56 16.63 ± 0.45 (10–29)	n = 51 17.69 ± 0.60 (11–29)	n = 21 18.14 ± 0.71 (12–24)	.16	.079	.66			

High school degree (Swiss "Matura" or "Fachhochschulreife") [% (N)]	<i>n</i> = 57 49.12 (28)	<i>n</i> = 53 41.51 (22)	39.13 (9)	.42	.42	.85	<i>n</i> = 38 57.90 (22)	45.45 (10)	50.00 (5)
Working status							<i>n</i> = 39		
Working [% (N)]	86.15 (56)	84.21 (48)	65.22 (15)	.76	.029	.060	84.62 (33)	68.18 (15)	60.00 (6)
Retired [% (N)]	13.85 (9)	14.04 (8)	34.78 (8)	.98	.029	.036	15.39 (6)	27.27 (6)	40.00 (4)
Days (baseline till follow-up)							1091.68 ± 40.65 (651–1923)	1102.45 ± 48.90 (875–1598)	1242.30 ± 75.70 (952–1678)

Note. Values are *means* ± *SEM* (range) if not indicated differently; BMI = body mass index; BP = blood pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; MAP change = follow-up MAP minus baseline MAP; NT = normotensive participants; HT = hypertensive participants; wc/m HT = white coat or masked hypertension; *n* = sample size; deviating sample sizes of a parameter are indicated; significant values are highlighted in bold ($p < .05$).

Table 2

Comparison of Baseline Group Characteristics between Eligible Participants Completing Follow-Up Assessment ($n = 72$) and Participants Who Dropped Out ($n=73$)

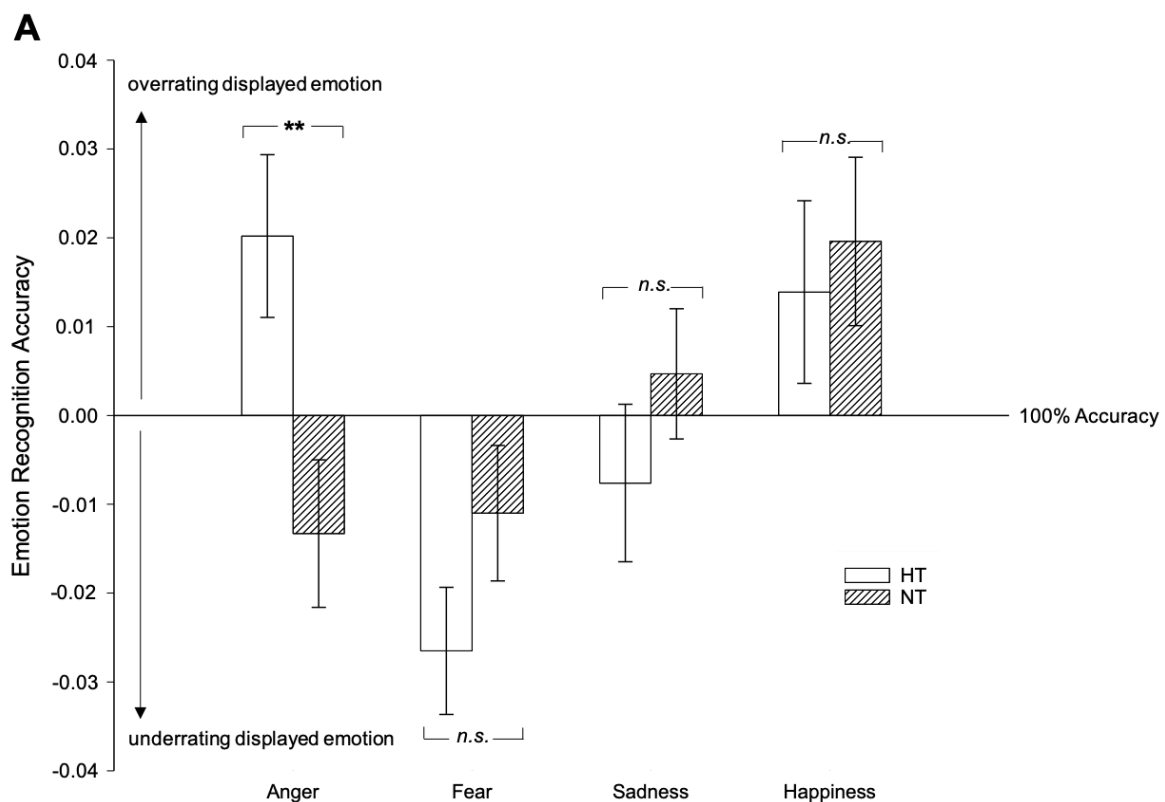
	Follow-up sample ($n=72$)			Drop-out participants ($n=73$)			Follow-up vs. Drop-out		
	NT $n=40$	HT $n=22$	wc/m HT $n=10$	NT $n=25$	HT $n=35$	wc/m HT $n=13$	<i>p</i> -value		
							NT	HT	wc/m HT
Age (yrs)	49.50 ± 1.92 (28–78)	53.73 ± 2.74 (28–74)	50.40 ± 4.85 (29–70)	50.32 ± 2.45 (25–78)	49.51 ± 1.78 (26–74)	56.77 ± 4.01 (21–73)	.79	.18	.40
BMI (kg/m ²)	24.89 ± 0.40 (19.78–28.87)	29.21 ± 1.00 (20.35–38.86)	27.53 ± 1.04 (23.25–32.99)	25.88 ± 0.45 (21.93–30.85)	28.40 ± 0.63 (22.67–37.74)	26.53 ± 1.03 (21.55–35.26)	.11	.47	.51
BP (study)									
SBP (mmHg)	126.48 ± 1.31 (109.33–139.67)	154.06 ± 2.71 (135.67–188.00)	142.62 ± 5.38 (109.50–177.00)	126.73 ± 1.80 (108.67–138.00)	153.30 ± 1.74 (129.33–189.67)	140.81 ± 2.50 (121.33–157.00)	.91	.81	.75
DBP (mmHg)	77.47 ± 1.13 (58.33–87.67)	94.47 ± 2.31 (73.67–113.67)	88.73 ± 1.80 (75.00–94.00)	77.79 ± 1.37 (62.67–88.67)	96.10 ± 1.18 (86.00–115.00)	87.71 ± 1.81 (78.67–96.67)	.86	.49	.70
MAP (mmHg)	93.80 ± 1.11 (75.33–104.56)	114.33 ± 2.30 (95.78–137.11)	106.69 ± 2.68 (86.50–117.44)	94.10 ± 1.40 (79.83–104.56)	115.17 ± 1.26 (104.89–139.89)	105.41 ± 1.87 (93.11–115.89)	.87	.73	.69
Trait anger	$n = 38$ 16.79 ± 0.60 (10–29)	$n = 21$ 18.10 ± 1.05 (12–29)	$n = 10$ 18.50 ± 0.90 (15–22)	$n = 18$ 16.28 ± 0.61 (12–22)	$n = 30$ 17.40 ± 0.73 (11–28)	$n = 11$ 17.82 ± 1.13 (12–24)	.60	.58	.65

Note. Values are means ± SEM (range); BMI = body mass index; BP = blood pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; NT = normotensive participants; HT = hypertensive participants; wc/m HT = white coat or masked hypertension; n = sample size; deviating sample sizes of a parameter are indicated

Figures

Figure 1

*Cross Sectional Analyses: (A) Emotion Recognition Accuracy of Anger, Fear, Sadness, and Happiness Averaged Over All Affect Intensities and the Different Emotion Combinations in Hypertensive (HT) Compared to Normotensive (NT) Participants $\pm$ Standard Error of the Mean (SEM). Over All Affect Intensities and Contrasting Emotion Combinations, HT Significantly Differed from NT in Anger Recognition Accuracy ($p = .008$), But Not in Emotion Recognition Accuracy of Other Emotions. Asterisks Indicate Significant Group Differences between HT and NT (** $p < .01$; n.s. = Not Significant). (B) Anger Recognition Accuracy of the Five Displayed Anger Intensities Averaged Over Three Anger-Other Emotion Combinations in HT Compared to NT $\pm$ SEM. (C) Non-Anger Emotion Recognition Accuracy of the Five Displayed Non-Anger Emotion Intensities Averaged Over the Different Emotion Combinations (i.e., Average Accuracy of Displayed Fear, Sadness, and Happiness per Intensity) in HT Compared to NT $\pm$ SEM*



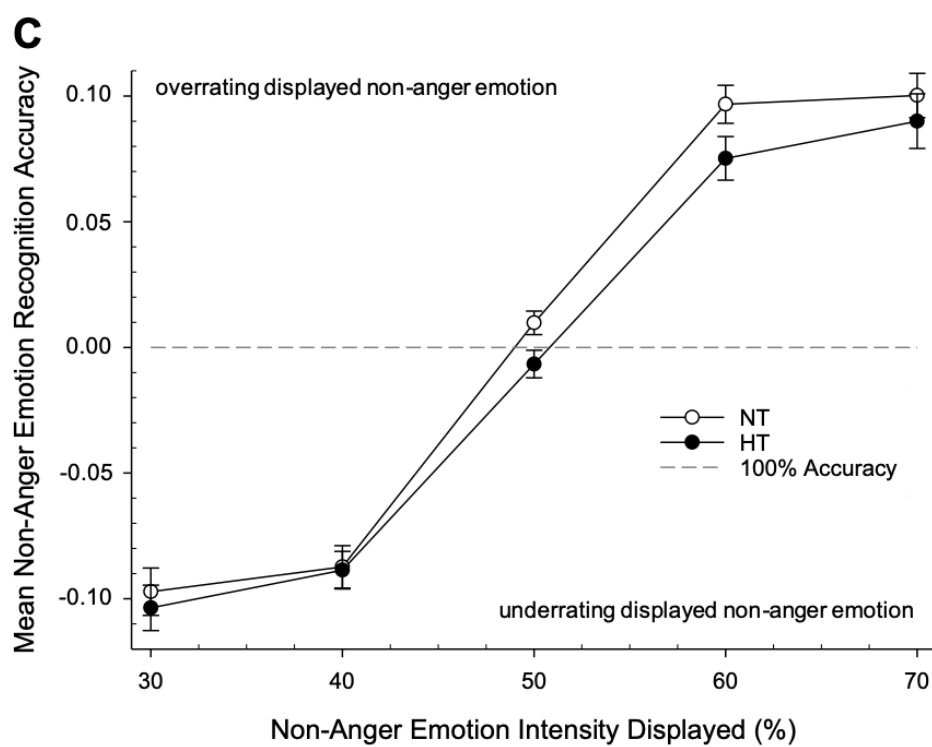
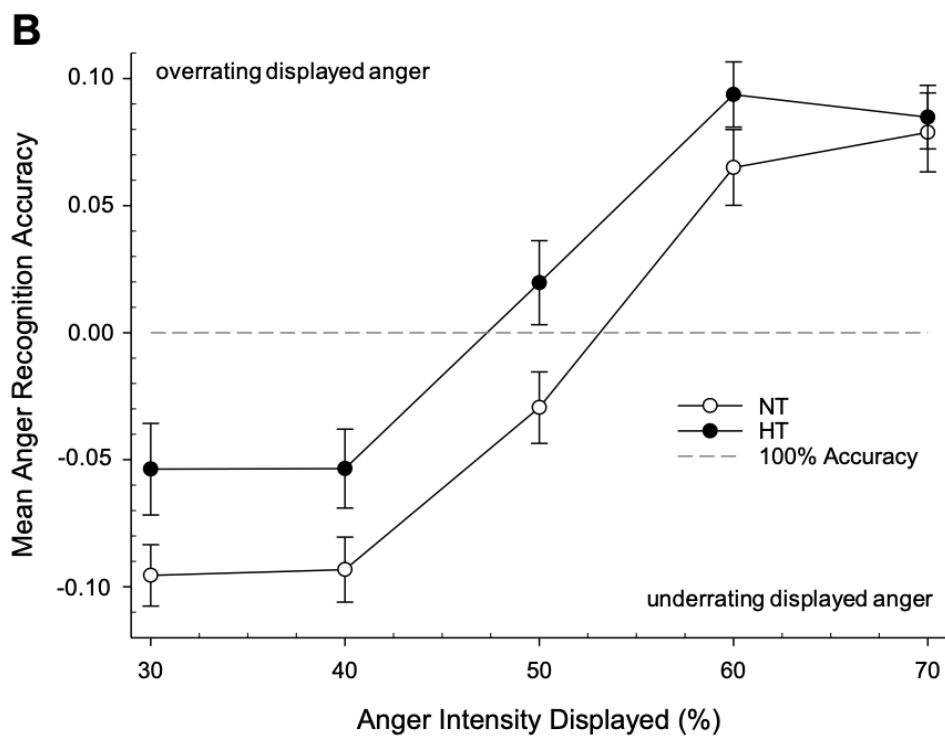
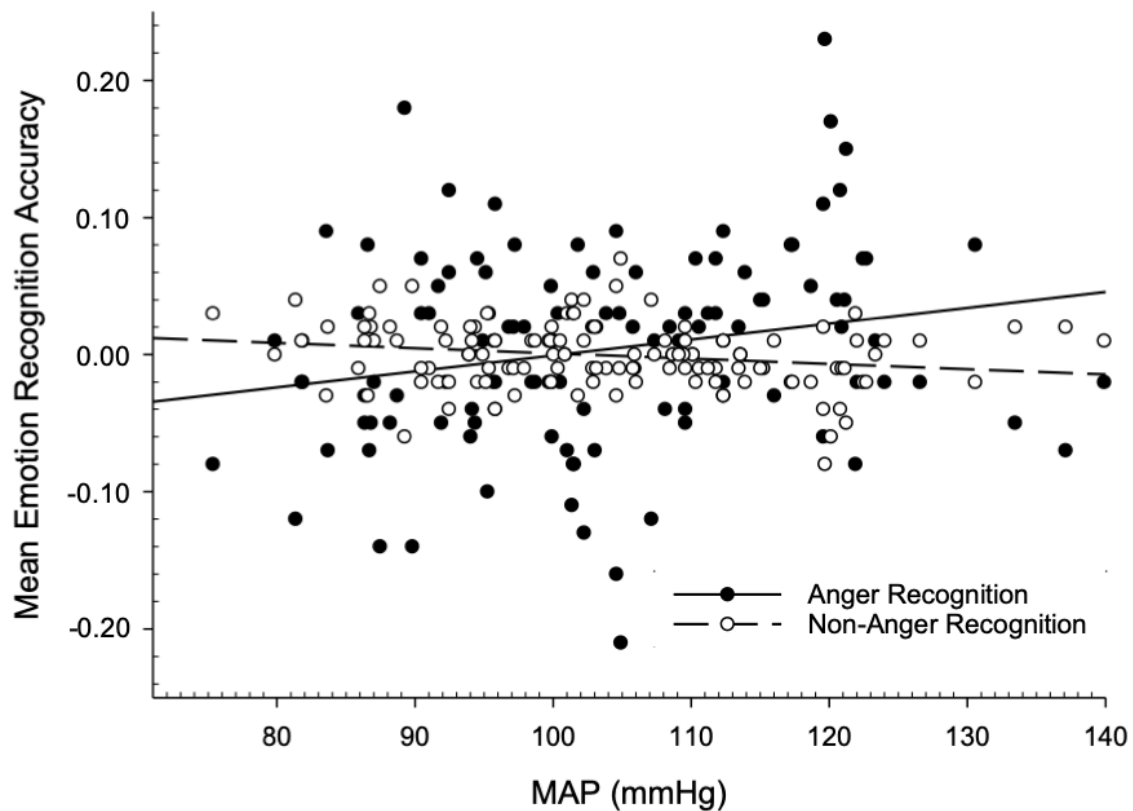


Figure 2

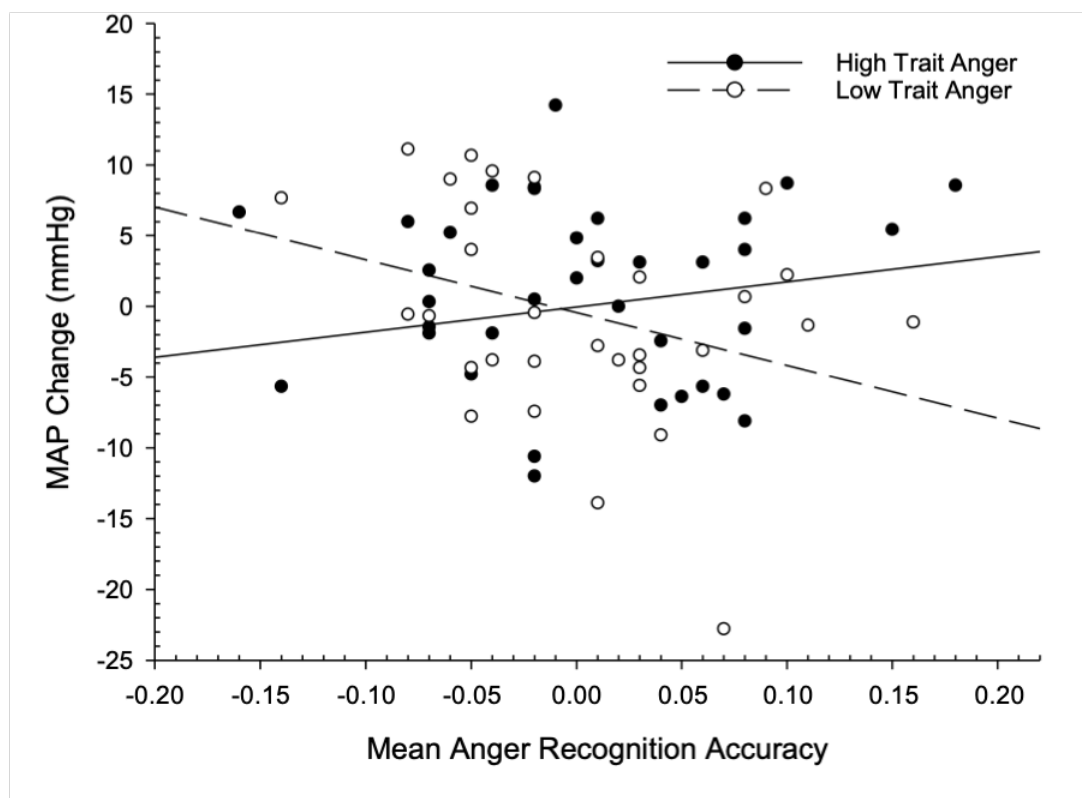
Cross Sectional Analyses: Linear Associations between Mean Arterial Pressure (MAP) and Anger Recognition Accuracy Averaged Over All Affect Intensities and Anger-Other Emotion Combinations (Solid Line and Black Dots) and between MAP and Non-Anger Recognition Accuracy (i.e., Accuracy of Displayed Fear, Sadness, and Happiness) Averaged Over All Affect Intensities and the Different Emotion Combinations (Dotted Line and White Dots)



Note. Over all affect intensities and contrasting emotion combinations, MAP significantly related to anger recognition accuracy ($p=.019$), but not to emotion recognition accuracy of other emotions.

Figure 3

Prospective Analyses: Moderation by Trait Anger: Mean Anger Recognition Accuracy Predicting Mean Arterial Pressure (MAP) Change Depending on Trait Anger. The Interaction between Mean Anger Recognition Accuracy and Trait Anger Significantly Predicted MAP Change ($p=.009$): Overrating of Displayed Anger Predicted Higher MAP Increases from Baseline to Follow-Up if Trait Anger Was High (Solid Line and Black Dots), but Not if Trait Anger was Low (Dotted Line and White Dots)

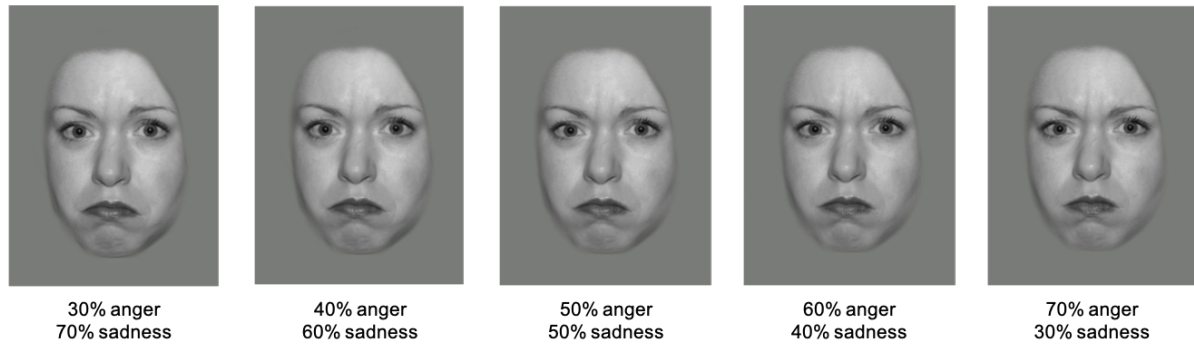


Note. For illustrative purposes (but not for modeling and testing), we categorized trait anger levels based on median split and depict levels below median as “low trait anger” and levels above median as “high trait anger”.

Supplemental Material

Supplemental Material 1: Figures**Figure S1**

Affect Intensity Pictures with Blends of Anger and Sadness Developed by Domes (Kaiser et al., 2019) Based on Faces of the NimStim Set of Facial Expressions (Tottenham et al., 2009)



References Supplemental Material

- Tottenham, N., Tanaka, J. W., Leon, A. C., McCarry, T., Nurse, M., Hare, T. A., Marcus, D. J., Westerlund, A., Casey, B. J., & Nelson, C. (2009). The NimStim set of facial expressions: Judgments from untrained research participants. *Psychiatry Research*, 168(3), 242-249. <https://doi.org/10.1016/j.psychres.2008.05.006>
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APPENDIX D**Research Paper 4****Beneficial Effects of a Cognitive-Behavioral Occupational Stress Management Group
Training: The Mediating Role of Changing Cognitions**

This research paper was published in:

Wirtz, P. H., Auer, A., Semmer, N. K., Ehlert, U., & Nussbeck, F. W. (2024). Beneficial effects of a cognitive-behavioral occupational stress management group training: The mediating role of changing cognitions. *Frontiers in Psychology*, 14, 1232172.
<https://doi.org/10.3389/fpsyg.2023.1232172>

Abstract

Background & Objectives: While the effectiveness of cognitive-behavioral stress management trainings (SMT) is well documented, the underlying mechanisms, especially in an occupational context, are not fully understood. We tested whether SMT-induced improvements in stress management skills, particularly in the mastery of changing cognitions, may explain beneficial SMT effects.

Methods: Our non-randomized controlled trial comprised 108 employees of a German health insurance company with 65 of them participating in a cognitive-behavioral SMT and 43 participating in an alternative control training (AT). As outcome variables we repeatedly assessed stress-related (functional stress management skills, relaxation, stress reactivity, exhaustion), work-related (job dissatisfaction), and specific-context-related (social support, trait anger) measures at baseline, two weeks, and three months after the trainings. Functional stress management skills and, in particular, a subscale assessing perceived mastery of changing cognitions (“cognitive-strategies-and-problem-solving”) were tested as mediators of change.

Results: Repeated measures (M)AN(C)OVAs and complementary multigroup latent difference models confirmed improvements in all outcomes in the SMT-group compared to the AT-group ($p \leq .015$). Multivariate mediation path analyses revealed that, regarding mechanisms of change, the subscale cognitive-strategies-and-problem-solving was identified as the most important mediator for all outcomes (95%CIs for expected increases in SMT- vs. AT-group=[lower limits(LLs) $\geq .004$]; 95%CIs for expected decreases in SMT- vs. AT-group=[upper limits(ULs) $\leq -.078$]) except for job dissatisfaction.

Discussion: Our findings confirm that employees can effectively learn to master stress reduction techniques and consequently lower the resulting burden. Moreover, beneficial SMT effects seem to result from improvements in functional stress management skills, particularly in the ability to change cognitions. This points to the importance of training cognitive techniques.

Keywords: cognitive-behavioral stress management training, cognitive restructuring, perceived mastery of functional stress management skills, exhaustion, job dissatisfaction, trait anger, group training

Introduction

Stress is a major risk factor for health impairments that causes substantial financial burden and that affects individuals, organizations, and societies (e.g., Brotman et al., 2007; Hassard et al., 2014; Hassard et al., 2017). Adverse health consequences of chronic stress exposure, in particular, in the context of work, include not only exhaustion but also increased risk, both for somatic diseases such as cardiovascular disease, and for mental diseases (Appels, 1999; Aronsson et al., 2017; Kivimäki & Steptoe, 2018; Stansfeld & Candy, 2006).

Stress management interventions aim to reduce stress and the resulting burden. Evidence from meta-analyses confirms the high effectiveness of person-focused stress management trainings (SMTs) with cognitive-behavioral elements (Kröll et al., 2017; Richardson & Rothstein, 2008; Saunders et al., 1996; van der Klink et al., 2001). The core element of cognitive-behavioral SMTs are cognitive techniques, including cognitive restructuring (Beck, 1967; Ellis, 1962) and resulting self-instructions (Meichenbaum, 1985, 2017), but also systematic problem solving (e.g., D’Zurilla & Nezu, 1982). Cognitive techniques are often combined with relaxation techniques such as progressive muscle relaxation (PMR) (Jacobsen, 1929), as in the well-established Stress Inoculation Training (SIT) by Meichenbaum (1985). Moreover, in later cognitive-behavioral SMT manuals, these techniques were trained in specific contexts such as anger, work-life balance, or social context (e.g., Kaluza, 2018a, 2018b; Siegrist & Silberhorn, 1998; Wiegard et al., 2000; Williams & Williams, 2010). Other SMTs use cognitive techniques more or less explicitly when working on changes in perceptions and cognitions that consequently result in increased relaxation or calmness (Bond, 2004; Bond & Hayes, 2002; Kabat-Zinn, 1982, 1990, 1994). Our occupational cognitive-behavioral SMT included the basic stress reduction techniques of cognitive restructuring, self-instructions, systematic problem-solving, and relaxation techniques based on the SIT by Meichenbaum (1985). In line with other occupational stress reduction programs, these techniques were applied to and trained in stress-relevant specific contexts, including anger

management and assertiveness (Siegrist & Silberhorn, 1998; Wiegard et al., 2000), work-life balance (Kaluza, 1996; Reschke & Schröder, 2000; Wagner-Link, 2001), identification and activation of resources (Siegrist & Silberhorn, 1998), social support (Reschke & Schröder, 2000; Wagner-Link, 2001), and perfectionism (Siegrist & Silberhorn, 1998; Wiegard et al., 2000) (see Figure 1).

The effectiveness of SMTs has been examined regarding a broad range of psychological (e.g., stress, coping, general mental health, work satisfaction, social support), physiological (e.g., blood pressure, stress hormones), and organizational outcome measures (e.g., absenteeism, job performance) (Kröll et al., 2017; Richardson & Rothstein, 2008; Riley et al., 2017; van der Klink et al., 2001). Overall, meta-analyses revealed medium to large effect sizes for beneficial effects of cognitive-behavioral SMTs, in particular, for psychological measures (Kröll et al., 2017; Richardson & Rothstein, 2008; Saunders et al., 1996; van der Klink et al., 2001). In our study, we assessed psychological measures that we expected to be improved by our SMT based on its contents (i.e., basic stress reduction techniques and their training in stress-relevant specific contexts; see Figure 1). More precisely, the assessed psychological outcome measures comprise *stress-related measures*, i.e., perceived mastery of functional stress management skills, including subscales, relaxation after work, stress reactivity (in particular, to work overload), vital exhaustion (VE), and the *work-related measure* job dissatisfaction. In addition, we included *specific-context-related measures*, i.e., perceived social support (PSS) and trait anger. Notably, we expected improvements in stress- and work-related measures to result from successful training of the basic stress reduction techniques and the overall training, while we expected the specific-context-related measures to improve, in particular, from successful training of the basic stress reduction techniques in the respective specific contexts.

While the effectiveness of cognitive-behavioral SMTs is well documented, the underlying psychological mechanisms are not fully understood. The transactional model of stress by Lazarus and Folkman (1984) can help to understand the psychological mechanisms

underlying effective, successful cognitive-behavioral SMTs. According to the transactional model of stress, a stress reaction results from the perceived imbalance between situational demands and personal resources based on cognitive appraisal processes, with primary appraisal addressing harm, threat, and challenge of a situation and secondary appraisal addressing the person's coping options (Lazarus & Folkman, 1984). Accordingly, changing the cognitive appraisal of a stressful situation by means of cognitive techniques should change the resulting stress experience. Indeed, based on quantitative assessment, such as the frequency of using a certain coping strategy, there is emerging evidence that coping in terms of *changing cognitions* may play a role in mediating the beneficial SMT effects on stress and mental health outcomes (Bond & Bunce, 2000; Brinkborg et al., 2011; Flaxman & Bond, 2010; Gaab et al., 2003; Hammerfald et al., 2006; Keogh et al., 2006; Leung et al., 2010; Lloyd et al., 2013). However, according to the framework of moderators, mediators, and mechanisms of change of occupational SMTs proposed by Bunce (1997), changes in terms of *mastery* and, thus, quality of coping are relevant to induce beneficial SMT outcomes.

A standardized questionnaire for the assessment of *perceived mastery of functional stress management skills* (Wirtz et al., 2013) was validated previously based on the Measure-of-Current-Status (MOCS) by Carver (2005), both including a subscale that assesses perceived mastery of changing cognitions using cognitive strategies such as problem solving. To date, mediation effects of perceived mastery of changing cognitions on beneficial psychological outcomes have only been investigated in one study, notably in the context of cancer and in an individual therapeutic setting. In that study Marsland et al. (2020) reported mediating effects via perceived mastery of changing cognitions in addition to mediation effects via MOCS-total-score on intervention-induced improvements in perceived stress and anxiety in mothers of children newly diagnosed with cancer. Notably, the cognitive-behavioral therapeutic intervention was specifically tailored for cancer caregivers with a special focus on coping with cancer. This finding is in line with a study in cancer patients that found SMT-induced

improvements in quality of life and in benefit-finding from cancer to be mediated by changes in the MOCS-total-score (Penedo et al., 2004; Penedo et al., 2006). So far, several questions regarding generalization of these findings remain unclear. First, it needs to be elucidated whether mediating effects of functional stress management skills and changing cognitions can be generalized to a broader range of outcome measures, including further stress-related outcome measures, but also work-related and more specific outcome measures, such as anger and social support. Second, it remains unclear whether the findings of a specifically tailored therapeutic intervention can be extended to occupational SMTs, i.e., SMTs targeting stress management on an occupational and non-cancer specific basis. This would be of particular importance in the context of health prevention, given the above-outlined adverse health effects of occupational stress. Third, it is unclear whether findings, in particular, with regard to changing cognition of the individual therapeutic setting, can be extended to a group-based SMT. Given that group-based trainings would allow a substantial increase in efficiency by helping more people within the same time with the given resources, it would be of particular importance to show that a group-based trainings are capable of changing cognitions that in turn mediate effectiveness of the trainings. Based on this reasoning, we aimed at investigating perceived mastery of functional stress management skills and its subscales not only as a stress-related outcome variable but also by considering significant intervention-induced improvements in the respective scales also as mediators of beneficial SMT effects.

Taken together, in the current study, we investigated the effects and underlying psychological mechanisms of an occupational cognitive-behavioral SMT compared to alternative control trainings (ATs) over a period of 3 months. Figure 1 provides a structured overview of contents and outcome measures of our SMT including specific hypotheses regarding SMT effects on outcome measures. We hypothesized that the SMT, on the one hand, would increase the stress-related measures of perceived mastery of functional stress management skills and relaxation after work as well as the specific-context-related measure of

PSS compared to AT. On the other hand, we hypothesized that the SMT would reduce the stress-related measures of VE and stress reactivity (in particular, to work overload), the work-related measure of job dissatisfaction, and the specific-context-related measure of trait anger compared to AT (see Figure 1). Moreover, with respect to underlying mechanisms, we expected that intervention-induced increases in perceived mastery of functional stress management skills in general and in the subscales assessing specific stress management skills would mediate the hypothesized positive effects of the intervention. Given the importance of cognitions in the context of stress (Lazarus & Folkman, 1984), we specifically hypothesized that increases in the stress management skill subscale “perceived mastery of changing cognitions” would mediate improvements in all outcome variables.

Methods

Study Participants and Procedure

We conducted a field study in employees (aged 18–65 years) of a German health insurance company located all over Germany. Parts of this study were used for psychometric validation of the Inventory for Assessment of Stress Management Skills (Wirtz et al., 2013) and for a doctoral thesis (Stein, 2007) (for more information see Supplemental Material 1). The study was carried out in accordance with the Declaration of Helsinki principles and was formally approved by the company’s board of management and staff council. All participants provided written informed consent.

A non-randomized controlled trial with an active control group was conducted in Germany. As personnel development procedures on a voluntary basis, the company offered its employees participation in different group trainings, including our cognitive-behavioral SMT. Our control group underwent one of two ATs, either on the structure and organization of the company and its corporate guidelines (“Basic Training”) or on working strategies with respect to communication and cooperation (“Working efficiently”). None of the ATs were explicitly related to stress or stress management. Outcome measures were assessed at three points in time

(T) in all training groups: at baseline, i.e., immediately before beginning of the respective training (“baseline”, i.e., T1), at two weeks (“post”, i.e., T2), and at three months (“follow-up”, i.e., T3) after the end of the training. For the experimental *SMT-group*, attendees of 7 SMT groups were asked to participate in the study. All 65 attendees volunteered for the study and participated in the baseline assessment ($N = 65$). A total of 56 participants finished both parts of the SMT. Reasons for not attending the second part of the SMT and thus completing the training were disease or high workload ($n = 8$), and one participant denied participating. Post assessment data were provided by 50 participants and follow-up assessment data were provided by 36 participants. For the *AT-group*, participants of the personnel development trainings “Basic training” and “Working efficiently” taking place during a time-frame close to the SMTs were asked for participation. A total of 43 participants volunteered to participate in the AT-group at baseline, with 36 persons providing post assessment and 30 persons completing follow-up assessment. Participants’ allocation to the study groups and participants’ flow through the study are depicted in Figure 2.

Intervention

Our occupational SMT addresses basic stress reduction techniques and their application and training in specific (mostly work-related) contexts using cognitive-behavioral techniques. We specifically tailored a group-oriented four-day training that consisted of two blocks. Each block was conducted over two consecutive workdays with a four-week time interval between the first and the second block to allow for individual training and “homework”. Group sizes varied between 7 and 12 participants, with two trainers per group. As already outlined in the introduction, the SMT addressed the basic stress reduction techniques of cognitive restructuring, self-instructions, systematic problem-solving, and relaxation techniques, based on the Stress Inoculation Training by Meichenbaum (1985). In line with other occupational stress reduction programs, these techniques were applied to and trained in stress-relevant specific contexts including anger management and assertiveness (Siegrist & Silberhorn, 1998;

Wiegard et al., 2000), work-life balance (Kaluza, 1996; Reschke & Schröder, 2000; Wagner-Link, 2001), identification and activation of resources (Siegrist & Silberhorn, 1998), social support (Reschke & Schröder, 2000; Wagner-Link, 2001), and perfectionism (Siegrist & Silberhorn, 1998; Wiegard et al., 2000) (see Figure 1). Each technique or topic, respectively, was introduced by brief theoretical inputs to allow for a maximum of supervised stress management practice during the training based on participants' stress problems.

Measures

Given potential confounding effects of baseline work stress, anxiety, and depression on presumed changes in outcome variables over time, we assessed these variables at baseline (T1) to test for group differences in addition to sociodemographic data. As outcome variables we repeatedly assessed the stress-related measures of perceived mastery of functional stress management skills (total-score and subscales), relaxation after work, stress reactivity (total score and subscale reactivity-to-work-overload), and VE, the work-related measure of job dissatisfaction, and the specific-context-related measures of PSS and trait anger at baseline (T1), as well as two weeks (T2) and three months (T3) after the end of the trainings. Notably, perceived mastery of functional stress management skills (total-score and subscales) were first tested as outcome measures when analyzing changes over time in the study groups. Second, significant training-induced improvements in these scales were further tested as mediators of change in the mediation models.

Baseline-Only Assessment: Work Stress, Anxiety, and Depression

Effort-reward imbalance (ERI) assesses stressful experiences at work with a questionnaire consisting of two scales measuring perceived effort (scale effort: 5 items) and experienced or anticipated rewards (scale reward: 11 items) (Rödel et al., 2004; Siegrist, 1996; Siegrist et al., 2004). The ratio of effort and reward was calculated according to established recommendations (Siegrist et al., 2004), yielding a quantitative estimate of the effort-reward imbalance, with higher values indicating a higher degree of stressful experience at work.

Cronbach's α ($N = 102$) in our sample was .74 for "effort" and .80 for "reward". Anxiety was assessed by the 20-item trait version of the state-trait-anxiety-inventory (STAI) (Laux et al., 1981), with higher scores indicating higher anxiety. The extent of depression was assessed with 15 items by the short version of the "Allgemeine Depressionsskala" (ADS-K) (Hautzinger & Bailer, 1993), with higher values indicating higher depressive symptomatology. The ADS-K is the German version of the "Center for Epidemiological Studies Depression Scale" (CES-D) (Radloff, 1977). In our sample Cronbach's α was .87 for anxiety ($N = 93$) and .86 for depression ($N = 94$).

Stress-Related Measures

Mastery of Functional Stress Management Skills. We used the 14-item Inventory for Assessment of Stress Management Skills (German "Inventar zur Erfassung von Stressbewältigungsfertigkeiten", ISBF) to assess perceived mastery of functional stress management skills (Wirtz et al., 2013). We computed the total-score and the five subscales "cognitive-strategies-and-problem-solving" (corresponding to the MOCS-scale "coping confidence"; Carver, 2006), "adequate-anger-expression-and-assertiveness", "identification-and-use-of-social-resources", "relaxation-abilities", and "perception-of-bodily-tension". Notably, the items of the subscale "cognitive-strategies-and-problem-solving" assess perceived mastery of changing cognitions using cognitive strategies, including strategies to solve occurring problems. Participants are asked to rate on a 5-point response scale how well they can perform on each of the items (e.g., "I can easily stop and re-examine my thoughts to gain a new perspective" (German: "Ich kann meine Gedanken leicht stoppen und überprüfen, um zu neuen Perspektiven zu gelangen"); 1 = *I cannot do this at all* to 5 = *I can do this extremely well*). Higher scores indicate better mastery of functional stress management skills. Reliability (Cronbach's α ($N = 332$) = .83) and validity have been found to be adequate (Wirtz et al., 2013) with comparable Cronbach's α ($N = 108$) = .82 in our sample at baseline.

Relaxation after Work. Relaxation after work was assessed by the 4-item subscale of the Recovery Experience Questionnaire that measures unwinding and recuperation from work during leisure time (Sonnentag & Fritz, 2007). Relaxation, as assessed by this questionnaire, refers to a process often associated with leisure activities characterized by a state of low activation and increased positive affect. Participants rated all items on a 5-point response scale (e.g., “I do relaxing things” (German: “Am Feierabend unternehme ich Dinge, bei denen ich entspannen kann”); 1 = *I do not agree* to 5 = *I fully agree*). Items are summed up to a total-score with higher scores, with higher scores indicating higher relaxation after work. Psychometric properties were found to be adequate with Cronbach’s α ($N = 930$) = .85 for the relaxation subscale (Sonntag & Fritz, 2007) and comparable with Cronbach’s α ($N = 108$) = .88 in our sample at baseline.

Stress Reactivity. Subjective stress reactivity was measured using the Stress-Reactivity-Scale (SRS) (Schulz et al., 2005). The SRS comprises 29 items forming six scales that assess intensity of different aspects of stress reactions to different types of stress situations. The scales are summarized to a total-score assessing general stress reactivity, with higher scores indicating higher stress reactivity. Each item describes a potentially stressful situation (e.g., “When I have little time for a job to be done...” (German: “Wenn ich für meine Arbeit wenig Zeit habe...”)) and asks participants to choose their typical response out of three potential responses depending on the potentially stressful situation (e.g., “*I usually stay calm*”, “*I usually feel uneasy*”, “*I usually get quite agitated*” (German: “bleibe ich meist ruhig”, “werde ich meist unruhig”, “werde ich meist ziemlich hektisch”)). In this study, we used the SRS total score and the subscale “reactivity-to-work-overload”. Reliability (Cronbach’s α ($N = 975$) = .91) and validity have been found to be adequate (Schulz et al., 2005) with comparable Cronbach’s α ($N = 108$) = .90 in our sample at baseline.

Vital Exhaustion. We assessed VE using the German version of the short form of the Maastricht Exhaustion Questionnaire (Kopp et al., 1998; Kudielka et al., 2004; Wirtz et al.,

2003). Nine items that are summarized to a total-score ask about the presence or absence of undue tiredness, trouble falling asleep, repeated waking up at night, general malaise, listlessness, irritability, loss of energy, demoralization, and waking up exhausted (e.g., “Do you often feel tired?” (German: „Fühlen Sie sich oft müde?“); 2 = *yes*, 1 = *I don’t know*, 0 = *no*). Higher scores indicate higher VE. Psychometric properties of the short form of the Maastricht Exhaustion Questionnaire have been shown to be adequate with Cronbach’s α ($N = 822$) = .84 (Kudielka et al., 2004). In our sample, Cronbach’s α ($N = 105$) was .84 at baseline.

Work-Related Measures

Job Dissatisfaction. As a measure of job dissatisfaction, we measured a resigned attitude towards one’s job, i.e., an attitude of resigned and resentful acceptance of unpleasant conditions at work, with four items (Baillod & Semmer, 1994; Grebner et al., 2005). Participants were asked to rate on a 7-point response scale how often they have thoughts such as “My job is not ideal, but it could be worse.” (German: „Meine Arbeit ist zwar nicht gerade ideal, aber schließlich könnte sie noch schlimmer sein; 1 = *never* to 7 = *always*). Items are summarized to a total-score with higher scores indicating higher resigned attitude towards one’s job. The internal consistency has been found to be between Cronbach’s $\alpha = .68$ (Baillod & Semmer, 1994) and Cronbach’s $\alpha = .72$ (Grebner et al., 2005) with Cronbach’s α ($N = 108$) = .65 in our sample at baseline.

Specific-Context-Related Measures

Perceived Social Support. PSS was assessed by the 8-item subscale of the Berlin Social Support Scale (BSSS) (Schulz & Schwarzer, 2003). Participants were asked to rate their agreement with statements such as “There are people that offer me help when I need it” (German: “Es gibt Menschen, die mir Hilfe anbieten, wenn ich sie brauche”) on a 4-point response scale (1 = *disagree* to 4 = *agree*). Items were averaged to compute the PSS score, with higher scores indicating higher PSS. Psychometric properties were found to be adequate with

Cronbach's α ($N = 437$) = .83 for the PSS subscale (Schulz & Schwarzer, 2003) and comparable Cronbach's α ($N = 108$) = .91 in our sample at baseline.

Trait Anger. We used the 10-item trait anger subscale of the German version of the State Trait Anger Expression Inventory (STAXI) to assess participant's disposition to experience anger (Schwenkmezger et al., 1992). Participants were asked to rate themselves with items such as "I am quick tempered" (German: "Ich werde schnell ärgerlich") on a four-point response scale (1 = *never* to 4 = *almost always*). Items were summarized to a total-trait-anger-score with higher scores, indicating a greater level of trait anger. Psychometric properties were found to be adequate with Cronbach's α ($N = 990$) = .71 (Schwenkmezger et al., 1992) and good Cronbach's α ($N = 107$) = .84 in our sample at baseline.

Statistical Analyses

We conducted our analyses with SPSS (Version 28.0; IBM SPSS Statistics, Chicago, IL, USA) and Mplus (Version 8.6; Muthén & Muthén, Los Angeles, CA, USA) for Macintosh. Data are presented as mean $\pm$ standard error of the mean (SEM). Unless indicated differently, tests were two-tailed with the significance level set at a p -value of $< .05$. We a-priori calculated power-analyses using the statistical software G*Power for Macintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany). Based on previous research (Hammerfeld et al., 2006; Kröll et al., 2017; Limm et al., 2011; Richardson & Rothstein, 2008; van der Klink et al., 2001), we conservatively expected (1) small to medium sized group differences between SMT- and AT-groups (i.e., $f = .15$ in a 2 (group) $\times$ 3 (T) repeated measures ANOVA) and (2) presumed the lowest average correlation of repeated measures to be $r = .40$. To obtain a power of $(1 - \beta) = .80$ analyses have to be run on $N=88$ participants. All data were tested for normal distribution and homogeneity of variance using Kolmogorov-Smirnov and Levene's tests. We applied Huynh-Feld correction where appropriate. As not all assumptions were completely fulfilled and due to the number of missing data at follow-up assessments (see Figure 2 and Table 1), we conducted complementary robust analyses with Mplus. We used the robust full-

information maximum-likelihood (FIML) estimator implemented in Mplus to estimate our models. In case of missing data, FIML performs well if the data are at least missing at random (MAR). Little's MCAR test ($\chi^2(288) = 241.85, p = .98$; 15 missing data patterns in the data) revealed that we may consider the data to be missing (completely) at random. If applicable, we report various model fit indices, including Chi-Square Goodness-of-Fit test with a nonsignificant chi-square value indicating a good model fit, Root Mean Square Error of Approximation (RMSEA) with values below 0.05 indicating a good model fit, Comparative Fit Index (CFI) with values above 0.95 indicating a good model fit, and Standardized Root Mean Square Residual (SRMR) with values below 0.05 indicating a good model fit. Effect size parameters partial η^2 (η^2_p ; effect size conventions η^2_p : .01 = small; .06 = medium; .14 = large) and d (effect size conventions $|d|$: .20 = small; .50 = medium; .80 = large) are reported where appropriate.

Changes Over Time in the Study Groups

To test whether the SMT induced improvements in stress-related (perceived mastery of functional stress management skills (ISBF-total-score and its subscales), relaxation after work, VE, stress reactivity (SRS-total-score and the subscale reactivity-to-work-overload)), work-related (job dissatisfaction), and specific-context-related (trait anger, PSS) outcome measures compared to the AT, we first conducted *repeated measures MANOVAs* in SPSS with group as independent variable and main (i.e., without subscales) outcome measures (T1, T2, and T3) as dependent variables. Outcome variables with expected increases over time (i.e., ISBF-total-score, relaxation after work and PSS; see Figure 1) were recoded for MANOVAs that a decrease always represents an improvement. Because of missing data, we ran two repeated measures MANOVAs. In the first MANOVA, we excluded all participants with missing values (listwise deletion), while we used “last observation carried forward” (LOCF) in the second MANOVA. *Post-hoc* testing of significant three-way (i.e., outcome variables-by-time-by-group) interaction effects comprised repeated measures ANOVAs for all outcome variables, including subscales.

Given the baseline difference in gender between the groups but not in age, baseline work stress, anxiety, and depression (see Table 1), we conducted all analyses without and with controlling for gender. Second, we estimated *multigroup latent difference models* (LD models; McArdle, 1988) in MPlus as complementary analyses, allowing us to perform analyses including all participants. More precisely, to model the actual change of our participants in response to SMT or ATs, we estimated baseline-change-models. In these models, increases and decreases at post (T2) or follow-up (T3) as compared to baseline (T1) are modelled as differences between latent factor scores separately for the SMT- and the AT-group (see Figure 3 for a simplified graphical representation). Thus, the LD model allows us to examine baseline scores and mean differential changes over time, i.e., from pre-to-post and from pre-to-follow-up. These baseline scores and, most importantly, mean differential changes over time are tested based on two-tailed *p*-values of *z*-tests. Effect sizes *d* for these effects are calculated by dividing unstandardized change scores by the standard deviation of a variable at T1. *Post-hoc*, we used one-tailed, independent *t*-tests to compare means at baseline and mean changes over time (“pre-to-post” and “pre-to-follow-up”) between SMT- and AT-group. Thus, these models provide results similar to those of repeated measures (M)ANOVAs. However, important advantages are that the LD models are generally more robust, missings can be estimated using FIML, and they allow modeling of all kind of changes, including nonlinear changes. Notably, we specified two LD models: First, in the main LD model, we tested the differential trajectories for the seven main outcome variables. Second, in an additional LD model, we accordingly tested the differential trajectories for the five ISBF subscales and the SRS subscale reactivity-to-work-overload.

Mediation Analyses

To test whether significant intervention-induced changes in perceived mastery of functional stress management skills would mediate significant intervention-induced changes in the other outcome variables, we ran multivariate mediation analyses with bootstrapping in Mplus. More specifically, we specified path models to examine whether the outcome variables

at T3 can be predicted by SMT participation and whether this effect is mediated by changes in functional stress management skills (T2) while controlling for baseline scores. Thus, our mediation models allow us to consider the temporal precedence of variables influencing each other and thus to test for true mediation. In our main mediation model, we used the ISBF-total-score as mediator of the SMT effects on all other main outcomes (see Figure 4). In the additional mediation model, we used the SRS subscale reactivity-to-work-overload as the only dependent variable as it is statistically not possible to analyze SRS-total-score and its subscale reactivity-to-work-overload simultaneously as outcome variables in one path model. In *post-hoc* mediation models, we accordingly tested whether significant changes in ISBF subscales instead of the total-score would mediate training effects on other outcome variables. Notably, sample size varies slightly between mediation models because of missing data treatment in Mplus with exclusion of participants with missing baseline values or complete lack of post-training data (see Table 1) in such models. In all path models, statistical significance was evaluated based on one-sided 95% bootstrap confidence intervals (CI).

Results

Group Characteristics

The total study sample consisted of 108 participants, with 65 participants in the SMT-group and 43 participants in the AT-group. The characteristics of the study groups at T1, T2, and T3 are detailed in Table 1. At T1, the SMT- and the AT-group did not significantly differ in age, ISBF-total-score, PSS, and job dissatisfaction (p 's $\geq .25$). Moreover, there were no significant baseline group differences in work stress, anxiety, and depression between study groups (p 's $\geq .20$). However, the SMT-group had lower scores in relaxation after work, higher scores in VE, SRS-total-score, and trait anger, as well as a higher proportion of women as compared to the AT-group (p 's $\leq .040$).

Changes Over Time in the Study Groups

Repeated measures MANOVAs with group (SMT- vs. AT-group) as independent variable and the seven main outcome measures as repeated (T1, T2, and T3) dependent variables with listwise deletion revealed a statistically significant three-way interaction between outcome variables, time, and group ($F(5.08, 304.56) = 17.08, p < .001, \eta^2_p = .22$; with gender as covariate: $F(5.18, 300.47) = 13.47, p < .001, \eta^2_p = .19$). Corresponding repeated measures MANOVAs with LOCF revealed similar results (three-way interaction: $F(4.56, 464.56) = 19.15, p < .001, \eta^2_p = .16$; with gender as covariate: $F(4.64, 463.54) = 16.61, p < .001, \eta^2_p = .14$). The significant three-way interactions indicate differential trajectories of the main outcome variables depending on group, which we further explored *post-hoc*, again with missings excluded as well as LOCF, each without and with controlling for gender. Repeated measures ANOVAs for each main outcome variable separately revealed significant interactions of time-by-group with beneficial changes (i.e., increases in ISBF-total-score, relaxation after work, and PSS, and decreases in VE, SRS-total-score, job dissatisfaction, and trait anger) in the SMT-group compared to the AT-group for each of the seven outcome variables (p 's $\leq .010$). Further subscale *post-hoc* testing revealed significant interactions of time-by-group for the ISBF subscales cognitive-strategies-and-problem-solving (p 's $\leq .002$), identification-and-use-of-social-resources (p 's $\leq .037$), relaxation-abilities (p 's $\leq .001$), and for the SRS subscale reactivity-to-work-overload (p 's $< .001$), all with improvements in the SMT-group as compared to the AT-group. The ISBF subscale adequate-anger-expression-and-assertiveness was significant with LOCF, both without and with controlling for gender and with listwise deletion when controlling for gender (p 's $\leq .045$). No significant interaction was found for the ISBF subscale perception-of-bodily-tension (p 's $\geq .11$). Detailed results of *post-hoc* repeated measures ANOVAs are provided in Supplemental Material 4, Table S1.

Complementary LD models further confirmed the obtained results. As results of the repeated measures (M)ANOVAs were independent of gender, this variable was not considered

in LD models to reduce complexity of the models. Notably, running alternative LD models on residuals controlled for gender did not change results (data not shown). Table 2 provides detailed results. With regard to the *main LD model*, we found the expected increases from “pre-to-post” and “pre-to-follow-up” for ISBF-total-score and relaxation after work (p 's $\leq .001$) and the expected decreases from “pre-to-post” and “pre-to-follow-up” for VE, SRS-total-score, job dissatisfaction, and trait anger (p 's $\leq .048$) in the SMT-group. For PSS, where both groups displayed high values at baseline, the increase from “pre-to-post” did not reach statistical significance ($p = .063$), whereas the increase from “pre-to-follow-up” assessment did ($p = .001$). Overall, effect sizes of change within the SMT-group are small (d 's $\leq .20$), except for $d(T2-T1 \text{ ISBF-total-score}) = 1.09$, $d(T3-T1 \text{ ISBF-total-score}) = 1.40$, and $d(T3-T1 \text{ PSS}) = 0.56$. For the AT-group we found either non-significant changes or statistically significant but non-beneficial changes over time, indicating either preservation of status quo or deterioration. *Post-hoc* calculated group comparisons of change scores between SMT- and AT-group revealed that all comparisons became significant with beneficial changes in the SMT-group. With regard to the *additional LD model* with the six subscales of interest, we found the expected decreases in the SMT-group from “pre-to-post” and “pre-to-follow-up” assessment for the SRS subscale reactivity-to-work-overload (p 's $< .001$) and the expected increases from “pre-to-post” and/or “pre-to-follow-up” assessment for all ISBF subscales (p 's $\leq .004$) except for the subscale perception-of-bodily-tension, which did not significantly change over time (p 's $\geq .17$). In the AT-group, we again observed statistically non-significant changes or significant non-beneficial changes over time. *Post-hoc* calculated group comparisons of change scores revealed significant group differences in change scores from “pre-to-post” and/or “pre-to-follow-up” in all subscales of interest, again with beneficial effects in the SMT group as compared to the control group.

Mediation Analyses

For reasons of clarity, we present mediation results in a condensed way. For complete results of mediation analyses with ISBF-total-score as mediator, see Table 3. For mediation analyses with ISBF subscales as mediators see Supplemental Material 4, Table S2 (model fit information) and Table S3 (model results). Statistical significance was evaluated based on one-sided 95% bootstrap CIs and only the relevant CI (lower (LL) or upper (UL)) limits for statistical significance are presented.

With regard to the *ISBF-total-score as mediator*, the main mediation model (see Figure 4; $\chi^2(36) = 39.22, p = .33$; RMSEA = .03 (90%CI = [.000; .087]); CFI = .99; SRMR = .08) and the additional mediation model ($\chi^2(1) = 0.09, p = .76$; RMSEA = .00 (90%CI = [.000; .192]); CFI = 1.00; SRMR = .01) fit the data well. In the main mediation model, we found total effects of group, i.e., training, on all outcome variables (95%CIs for expected increases in SMT- vs. AT-group = [LLs $\geq .176$]; 95%CIs for expected decreases in SMT- vs. AT-group = [ULs $\leq -.154$]). These total effects decompose into a mediated, i.e., indirect effect (reflecting the specified training effects via ISBF-total-score T2) and a direct effect of group, i.e., training effect on the outcome variable (reflecting unspecific training effects independent of ISBF-total-score changes). We found indirect effects supporting the expected mediation model via ISBF-total-score for relaxation after work (95%CI = [LL = 0.033]), VE (95%CI = [UL = -0.116]), SRS-total-score (95%CI = [UL = -0.085]), and trait anger (95%CI = [UL = -0.045]). Furthermore, we observed direct effects for relaxation after work (95%CI = [LL = 0.081]), SRS-total-score (95%CI = [UL = -0.116]), and job dissatisfaction (95%CI = [UL = -0.097]), but not for PSS, VE, and trait anger. The significant direct effect of group, i.e., training, on ISBF-total-score T2 (95%CI = [LL = 0.320]) reflects training-induced increases in functional stress management skills. In the additional mediation model with the SRS subscale reactivity-to-work-overload as outcome variable, we also found a statistically significant total effect (95%CI = [UL = -0.220]) with a statistically significant indirect (95%CI = [UL = -0.067]), but

no direct effect. The direct effect of group, i.e., training, on ISBF-total-score T2 (95%CI = [LL = 0.310]) was again significant.

With respect to mediation analyses with the *ISBF subscales as mediators*, the subscale cognitive-strategies-and-problem-solving proved to be the most important mediator since we found indirect effects via this subscale for all outcomes (main model and additional model: 95%CIs for expected increases in SMT- vs. AT-group = [LLs $\geq$.004]; 95%CIs for expected decreases in SMT- vs. AT-group = [ULs $\leq$ -.078]), except job dissatisfaction. In addition, the subscale identification-and-use-of-social-resources had indirect effects on VE and the SRS subscale reactivity-to-work-overload (95%CIs = [ULs $\leq$ -.002]). For the ISBF subscales adequate-anger-expression-and-assertiveness and relaxation-abilities, there were no or fewer indirect effects. Mediation analyses with the ISBF subscales as mediators are described in more detail in Supplemental Material 2. Taken together, over all mediation analyses, we found indirect effects via ISBF-total-score and/or subscales for all outcome variables except for job dissatisfaction, supporting our hypothesis that SMT effects are mediated via functional stress management skills and, in particular, via cognitive-strategies-and-problem-solving, i.e., perceived mastery of changing cognition.

Discussion

In this study we investigated the effects of an occupational cognitive-behavioral SMT compared to ATs on different outcome measures and examined mediating effects of perceived mastery of functional stress management skills, in particular, of changing cognitions, over a period of 3 months.

We first found the expected beneficial *effects of the SMT* on stress-related, work-related, and specific-context-related measures. More specifically, regarding stress-related measures, we observed improvements in terms of increases in perceived mastery of functional stress management skills in total (ISBF-total-score), including improvements in the subscales cognitive-strategies-and-problem-solving, adequate-anger-expression-and-assertiveness,

identification-and-use-of-social-resources, and relaxation-abilities. Moreover, we found improvements in terms of increases in relaxation after work and decreases in stress reactivity (in particular, to work overload) and exhaustion. We also found decreases in the work-related measure of job dissatisfaction. With respect to specific-context-related measures, social support increased and anger decreased. Overall, our findings are in line with the results of previous meta-analyses showing beneficial effects of cognitive-behavioral SMTs especially with regard to psychological measures (Kröll et al., 2017; Richardson & Rothstein, 2008; Saunders et al., 1996; van der Klink et al., 2001). In more detail, beneficial effects of cognitive-behavioral SMTs have previously been shown for comparable stress-related (e.g., recovery experiences and thus relaxation (Siu et al., 2014), stress reactivity (Limm et al., 2011), exhaustion (Brinkborg et al., 2011; Higgins, 1986; Kushnir & Malkinson, 1993; Lloyd et al., 2013; Norvell et al., 1987; Ojala et al., 2019; Riley et al., 2017; van Rhenen et al., 2005)), work-related (e.g., job (dis)satisfaction (Bunce & West, 1996; Cecil & Forman, 1990; Forman, 1981; Maddi et al., 1998; Nickel et al., 2007)), and specific-context-related (e.g., anger (Keyes & Dean, 1988; Nickel et al., 2007), social support (Freedy & Hobfoll, 1994; Maddi et al., 1998)) measures. Notably, these beneficial effects have not been observed unequivocally (Bond & Bunce, 2000; de Jong & Emmelkamp, 2000; Freedy & Hobfoll, 1994; Grønningæter et al., 1992; Munz et al., 2001; Siu et al., 2014; Willert et al., 2009). With respect to cognitive-behavioral SMT effects on stress management skills, our results are in line with findings of non-clinical studies assessing frequency of use of coping (Alkhawaldeh et al., 2020; Bekker et al., 2001; de Jong & Emmelkamp, 2000; Gardiner et al., 2004; Long, 1988; Willert et al., 2009; Żołnierczyk-Zreda, 2002) and with findings of clinical studies (Antoni et al., 2006; Gudenkauf et al., 2015; Jensen et al., 2013; Marsland et al., 2020; Penedo et al., 2004; Penedo et al., 2006) and one non-clinical study (Riley et al., 2017) assessing perceived mastery of stress management skills. Notably, these studies found improvements in coping or stress management skills in at least one scale.

Overall, most studies report improvements in some outcome measures and to the best of our knowledge, only few studies report a similar broad range of beneficial SMT effects.

In the present SMT, participants were trained in cognitive restructuring, self-instructions, systematic problem solving, and relaxation as *basic stress reduction techniques*. As outlined in the introduction and depicted in Figure 1, we consider the reported increases in the ISBF subscales cognitive-strategies-and-problem-solving and relaxation-abilities as well as in the relaxation after work scale as a direct result of successful training. Moreover, as we explicitly trained the basic techniques in the contexts of anger and social support, we further interpret the observed improvements in the ISBF subscales adequate-anger-expression-and-assertiveness and identification-and-use-of-social-resources as well as in PSS and trait anger to result from successful technique acquisition and mastery in the respective contexts. In contrast, as there were no specific parts of the training addressing exhaustion, stress reactivity, or job dissatisfaction, the observed beneficial training effects in these outcomes more likely result from the total training (see Figure 1). There were no SMT-induced improvements in the ISBF subscale perception-of-bodily-tension, a skill that notably was not explicitly trained.

Second, regarding the *mechanisms underlying the observed SMT effects* on outcome variables other than ISBF scales, our mediation analyses confirmed mediating effects of perceived mastery of functional stress management skills in general and specifically of the subscale cognitive-strategies-and-problem-solving; these results apply to all outcome variables except job dissatisfaction. This suggests that the effectiveness of our occupational cognitive-behavioral SMT can specifically be attributed to improvements in perceived mastery of functional stress management skills. In this regard, our results provide empirical evidence in support of the framework of moderators, mediators, and mechanisms of change of occupational SMTs proposed by Bunce (1997), where changes in terms of mastery of coping are relevant to induce beneficial SMT outcomes. In more detail, the ISBF-total-score emerged as a mediator of change for almost all outcomes (except for job dissatisfaction and social support). This is in

line with the finding that *active training* of coping skills is of principal importance for beneficial SMT effects (West et al., 1984) and, for the first time, extends results from clinical studies in the context of cancer (Marsland et al., 2020; Penedo et al., 2004; Penedo et al., 2006) to a non-clinical occupational context. Moreover, as expected, based on the transactional model of stress (Lazarus & Folkman, 1984), we found the ISBF subscale cognitive-strategies-and-problem-solving to mediate training effects on almost all outcome variables, with job dissatisfaction being the only exception. This suggests that mastery of changing cognitions seems to be the most important stress management skill corroborating findings on this subscale in mothers of children newly diagnosed with cancer (Marsland et al., 2020). With regard to the non-clinical context, this finding further supports and extends the findings that beneficial changes in dysfunctional cognition, stress appraisal, or psychological flexibility mediate or at least relate to improvements after SMT (Bond & Bunce, 2000; Brinkborg et al., 2011; Flaxman & Bond, 2010; Gaab et al., 2003; Hammerfald et al., 2006; Keogh et al., 2006; Leung et al., 2010; Lloyd et al., 2013).

In addition, we observed the subscale identification-and-use-of-social-resources to mediate training effects on exhaustion and the SRS subscale reactivity-to-work-overload. This finding can be interpreted in the context of the social support-reactivity hypothesis where social support is supposed to buffer stress effects with resulting beneficial effects on health (Christenfeld & Gerin, 2000; Lepore, 1998). Mediation results for the ISBF subscales adequate-anger-expression-and-assertiveness and relaxation-abilities are discussed in Supplemental Material 3. Interestingly, the observed improvements in job dissatisfaction were not mediated by SMT-induced improvements in functional stress management skills. This suggests that other mechanisms have to account for the improvement in job dissatisfaction. We can only speculate that general training effects may play a role. However, it is also conceivable that the offer to undergo a SMT without financial costs during worktime at full payment is perceived as appreciation and concern by a responsible employer that cares for employees. At the same time,

the finding of non-mediation by stress management skills in general and cognitive strategies, in particular, also suggests that there are limits that even optimal individual coping cannot overcome. It needs to be considered that a stressful environment is capable of inducing stress in probably everyone, if the level of stress induction is high enough, particularly, if the level of control or reward is low (e.g., Karasek, 1979; Siegrist, 1996). Therefore, the responsibility for health and well-being should not be left only at the individual employee level. Instead, it should be considered an explicit responsibility of employers to establish a workplace compatible for health and well-being of employees. Overall, achieving the best effects for a healthy workplace requires responsibility not only at the level of the individual, but also of the work group, leader, and organization (Nielsen & Christensen, 2021). Notably, cognitive-behavioral SMT are promising for contributing to a healthy workplace at the individual level. However, they are unlikely to compensate substantial workplace deficits that require activity at further levels.

Clinical implications of our study include that cognitive-behavioral SMTs seem to be an effective way to teach employees techniques to reduce stress and resulting the burden that are primarily mediated by improvements in perceived mastery of changing cognitions. Therefore, SMTs should particularly pay attention to sufficiently teach and actively train and practice techniques such as cognitive restructuring, self-instructions, and systematic problem-solving. Further implications are that the group-setting of medium group size (7-12 participants) and a combination of teaching basic stress reduction techniques and training them in specific stress-related contexts seem well-suited for successful training effects. Given that other cognitive-behavioral SMTs have already been shown to have long-lasting beneficial effects over several years (Herr et al., 2018; Li et al., 2017), it is conceivable that our results are of similar persistence.

Strengths of our study include the comparison of our SMT-group with an AT-group to account for unspecific placebo effects of interventions in general. Second, the SMT was carried out according to a protocol based on well-established stress reduction programs (Kaluza, 1996;

Meichenbaum, 2017; Reschke & Schröder, 2000; Siegrist & Silberhorn, 1998; Wagner-Link, 2001; Wiegard et al., 2000). Third, our study design with post and follow-up assessment allowed for considering the temporal precedence of variables influencing each other and thus to test for a true mediation. Finally, we used different and, in particular, state-of-the-art statistical methods to deal with missings, including listwise deletion as well as LOCF and FIML, following the intention to treat approach, that, notably, all provided comparable results. *Limitations* of our study include the relatively low sample size, in particular, regarding the return rate of the follow-up assessment questionnaires (see Figure 2) that we, however, compensated for statistically. Second, we could not use a randomized group allocation as the company offered participation in the different group trainings as personnel development procedure on a voluntary basis. Third, the SMT-group had less favorable baseline levels in some outcome measures (relaxation after work, VE, stress reactivity, and trait anger) at baseline than the AT-group, suggesting that the SMT-group was more stressed at study entry, which may have influenced the potential for improvements. We interpret these baseline differences to result from the voluntary, non-randomized group assignment. In addition, the SMT-group comprised a higher proportion of women as compared to the AT-group, which however did not affect our results. Given this, further studies should include a randomized group assignment to overcome these limitations of our field study.

Taken together, our findings provide further evidence that occupational cognitive-behavioral SMTs are effective in reducing stress experience and the resulting burden as indicated by the beneficial effects of our SMT on our outcome measures. Moreover, beneficial effects of cognitive-behavioral SMTs seem to be mediated by SMT-induced improvements in perceived mastery of functional stress management skills, in particular, of changing cognitions. Future studies are needed to confirm these results in larger samples with randomized group allocation and to determine whether results can be generalized to other occupational sectors than health insurance and to different working conditions. Moreover, it should be examined

how such interventions on an individual level could be combined with interventions addressing demands and resources at a group, leader, and organizational level to achieve best effects (Nielsen & Christensen, 2021).

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Tables

Table 1

Group Characteristics of the Stress Management Training (SMT)-Group and the Alternative Training (AT)-Group at Timepoint (T) 1, T2, and T3

	Group characteristics T1 (N = 108)			Group characteristics T2 (N = 86)		Group characteristics T3 (N = 66)	
	SMT-group n = 65	AT-group n = 43	p-value	SMT-group n = 50	AT-group n = 36	SMT-group n = 36	AT-group n = 30
Gender (N)	52 women 13 men n = 59	26 women 16 men n = 42	.040				
Age (years)	32.88 ± 1.09 (20–54)	31.02 ± 1.11 (19–49)	.25				
ISBF-total-score	2.95 ± 0.07 (1.50–4.00)	3.02 ± 0.08 (2.07–4.14)	.53	3.25 ± 0.06 (2.36–4.21)	2.88 ± 0.08 (1.86–3.57)	n = 35 3.45 ± 0.06 (2.57–4.14)	2.87 ± 0.10 (1.71–3.64)
CogProb	2.92 ± 0.09 (1.00–4.60)	3.13 ± 0.09 (1.80–4.00)	.13	3.22 ± 0.06 (2.20–4.20)	2.93 ± 0.10 (2.00–4.00)	n = 35 3.35 ± 0.08 (2.00–4.40)	2.96 ± 0.12 (1.60–4.00)
AngExAs	3.21 ± 0.09 (1.33–5.00)	3.22 ± 0.09 (2.00–4.67)	.96	3.33 ± 0.10 (1.33–5.00)	3.24 ± 0.10 (2.00–4.00)	n = 35 3.51 ± 0.08 (2.67–5.00)	3.12 ± 0.11 (2.00–4.00)
SocRes	3.08 ± 0.13 (1.00–5.00)	3.43 ± 0.15 (2.00–5.00)	.08	3.30 ± 0.15 (1.00–5.00)	3.18 ± 0.15 (2.00–5.00)	n = 35 3.39 ± 0.14 (1.00–5.00)	3.02 ± 0.17 (1.50–5.00)
RelaxAb	2.02 ± 0.11 (1.00–4.50)	2.05 ± 0.12 (1.00–4.00)	.85	2.81 ± 0.12 (1.00–4.50)	2.04 ± 0.13 (1.00–4.00)	n = 35 3.01 ± 0.14 (1.00–5.00)	2.25 ± 0.18 (1.00–4.00)
PBodTens	3.43 ± 0.13 (1.00–5.00)	3.00 ± 0.16 (1.00–5.00)	.036	3.59 ± 0.14 (1.00–5.00)	2.76 ± 0.15 (1.00–4.00)	n = 35 3.39 ± 0.15 (1.00–5.00)	2.72 ± 0.15 (1.00–4.00)
Relaxation after work	12.95 ± 0.45 (4–20)	14.84 ± 0.43 (8–20)	.005	14.30 ± 0.46 (5–20)	13.97 ± 0.48 (7–19)	n = 35 14.89 ± 0.56 (8–20)	13.17 ± 0.53 (7–20)

PSS	3.57 ± 0.07 (1.50–4.00)	3.54 ± 0.07 (2.25–4.00)	.74	3.65 ± 0.07 (1.50–4.00)	3.43 ± 0.09 (2.38–4.00)	<i>n</i> = 35 3.71 ± 0.08 (1.38–4.00)	3.22 ± 0.11 (2.00–4.00)
VE	<i>n</i> = 62 11.00 ± 0.59 (0–18)	7.23 ± 0.66 (0–17)	< .001	7.60 ± 0.62 (0–16)	8.97 ± 0.83 (0–18)	<i>n</i> = 35 7.46 ± 0.90 (0–18)	8.20 ± 0.98 (0–17)
SRS-total-score	65.74 ± 0.98 (51–84)	54.51 ± 1.22 (38–68)	< .001	56.90 ± 1.26 (44–79)	56.61 ± 1.53 (39–78)	52.75 ± 1.18 (42–66)	58.17 ± 1.75 (42–78)
RWO	10.68 ± 0.30 (5–15)	8.70 ± 0.27 (5–11)	< .001	9.10 ± 0.31 (5–14)	8.75 ± 0.36 (5–12)	8.17 ± 0.37 (5–13)	9.30 ± 0.42 (5–13)
Resigned attitude toward one's job	11.43 ± 0.56 (4–21)	11.79 ± 0.69 (4–20)	.68	10.14 ± 0.68 (4–19)	12.25 ± 0.69 (5–21)	<i>n</i> = 35 9.69 ± 0.72 (4–20)	13.07 ± 0.76 (5–21)
Trait anger	<i>n</i> = 64 20.28 ± 0.66 (10–37)	18.19 ± 0.59 (12–31)	.028	<i>n</i> = 49 17.90 ± 0.57 (10–27)	18.86 ± 0.69 (13–30)	<i>n</i> = 34 17.47 ± 0.73 (11–27)	19.30 ± 0.78 (13–29)
ERI ratio	<i>n</i> = 60 0.82 ± 0.04 (0.22–2.12)	<i>n</i> = 42 0.74 ± 0.04 (0.29–1.67)	.20				
STAI	<i>n</i> = 58 39.52 ± 1.10 (24–63)	<i>n</i> = 35 38.43 ± 1.23 (24–54)	.53				
ADS-K	<i>n</i> = 58 9.78 ± 0.91 (0–31)	<i>n</i> = 36 10.92 ± 1.05 (0–26)	.42				

Note. Values are means ± SEM (range) if not indicated differently; ISBF-total-score = Inventory for Assessment of Stress Management Skills total-score; CogProb = ISBF subscale cognitive-strategies-and-problem-solving; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of-social-resources, RelaxAb = ISBF subscale relaxation-abilities; PBodTens = ISBF subscale perception-of-bodily-tension; PSS = perceived social support; VE = vital exhaustion; SRS-total-score = Stress-Reactivity-Scale total-score; RWO = SRS subscale reactivity-to-work-overload; ERI ratio = effort reward imbalance ratio; STAI = state-trait-anxiety-inventory; ADS-K = German version of the “Center for Epidemiological Studies Depression Scale”; T1 = timepoint 1; T2 = timepoint 2; T3 = timepoint 3; SMT-group = stress management training group; AT-group = alternative training group; *n* = sample size; deviating sample sizes of a parameter are indicated. *p*-values = two-tailed *p*-values either of χ^2 -test (gender) or independent t-tests (all other variables); significant values are highlighted in bold (*p* < .05).

Table 2

Unstandardized Baseline Values and Change Scores of the Multigroup Latent Difference Models (LD models) and Post-Hoc Group Comparison Between the Stress Management Training (SMT)-Group and the Alternative Training (AT)-Group

	Baseline (T1)			Change “pre-to-post” (T2–T1)			Change “pre-to-follow-up” (T3–T1)		
	SMT-group	AT-group	group comparison	SMT-group	AT-group	group comparison	SMT-group	AT-group	group comparison
	<i>Mean</i>	<i>Mean</i>	<i>p</i> -value (effect size)	<i>Mean Change</i> <i>p</i> -value (effect size)	<i>Mean Change</i> <i>p</i> -value (effect size)	<i>p</i> -value (effect size)	<i>Mean Change</i> <i>p</i> -value (effect size)	<i>Mean Change</i> <i>p</i> -value (effect size)	<i>p</i> -value (effect size)
ISBF-total-score ^a	2.95	3.02	.26 (0.13)	0.30 <.001 (1.09)	–0.13 .010 (–0.48)	<.001 (–1.14)	0.39 <.001 (1.40)	–0.14 .064 (–0.49)	<.001 (–1.05)
CogProb ^b	2.92	3.13	0.055 (0.32)	0.28 <.001 (0.53)	–0.17 .032 (–0.51)	<.001 (–0.85)	0.40 <.001 (0.75)	–0.13 .14 (–0.40)	<.001 (–0.86)
AngExAs ^b	3.21	3.22	.48 (0.01)	0.12 .28 (0.22)	0.03 .75 (0.08)	.29 (–0.11)	0.37 <.001 (0.71)	–0.09 .47 (–0.22)	.002 (–0.58)
SocRes ^b	3.09	3.43	.038 (0.35)	0.26 .004 (0.24)	–0.15 .27 (–0.16)	.007 (–0.49)	0.18 .13 (0.17)	–0.38 .005 (–0.42)	.001 (–0.65)
RelaxAb ^b	2.02	2.05	.42 (0.04)	0.84 <.001 (1.19)	0.00 .99 (0.00)	<.001 (–1.11)	1.09 <.001 (1.55)	0.23 .12 (0.40)	<.001 (–0.82)
PBodTens ^b	3.43	3.00	.016 (–0.43)	0.17 .17 (0.16)	–0.16 .20 (–0.16)	.031 (–0.37)	0.11 .43 (0.11)	–0.20 .16 (–0.20)	.064 (–0.30)
Relaxation after work ^a	12.95	14.84	.001 (0.60)	1.26 .001 (0.10)	–0.74 .10 (–0.10)	.001 (–0.66)	1.75 <.001 (0.14)	–1.33 .017 (–0.17)	<.001 (–0.87)
PSS ^a	3.57	3.54	.37 (–0.07)	0.08 .063 (0.31)	–0.10 .13 (–0.52)	.012 (–0.45)	0.15 .001 (0.56)	–0.21 .010 (–1.05)	<.001 (–0.76)
VE ^a	10.95	7.23	<.001 (–0.83)	–3.15 <.001 (–0.14)	2.03 .001 (0.11)	<.001 (1.11)	–3.77 <.001 (–0.17)	1.62 .040 (0.09)	<.001 (0.97)
SRS-total-score ^a	65.74	54.51	<.001 (–1.42)	–8.76 <.001 (–0.14)	3.16 .018 (0.05)	<.001 (1.44)	–12.54 <.001 (–0.20)	4.51 .025 (0.07)	<.001 (1.48)
RWO ^b	10.68	8.70	<.001 (–0.97)	–1.46 <.001 (–0.25)	0.14 .66 (0.04)	<.001 (0.79)	–2.48 <.001 (–0.43)	0.44 .32 (0.14)	<.001 (0.94)
Resigned attitude toward one’s job ^a	11.43	11.79	.34 (0.08)	–1.02 .048 (–0.05)	0.73 .23 (0.04)	.015 (0.43)	–1.56 .007 (–0.08)	1.14 .14 (0.06)	.003 (0.55)

Trait anger ^a	20.35	18.19	.008 (-0.48)	-2.34 <.001 (-0.08)	0.98 .20 (0.07)	<.001 (0.67)	-2.35 .001 (-0.08)	1.28 .16 (0.09)	.001 (0.62)

Note. All results of the two multigroup latent difference models (LD models) are depicted in this table ^a = results of main LD model, i.e., with main outcome variables; ^b = results of additional LD model, i.e., with subscales of interest; ISBF-total-score = Inventory for Assessment of Stress Management Skills total-score; CogProb = ISBF subscale cognitive-strategies-and-problem-solving; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of-social-resources, RelaxAb = ISBF subscale relaxation-abilities; PBodTens = ISBF subscale perception-of-bodily-tension; PSS = perceived social support; VE = vital exhaustion; SRS-total-score = Stress-Reactivity-Scale total-score; RWO = SRS subscale reactivity-to-work-overload; ERI ratio = effort reward imbalance ratio; T1 = timepoint 1; T2 = timepoint 2; T3 = timepoint 3; SMT-group = stress management training group; AT-group = alternative training group; *p*-values group comparison = one-tailed *p*-values of post-hoc independent t-tests with effect size *d*; *p*-values mean change = two-tailed *p*-values of *z*-tests with effect sizes calculated by dividing unstandardized change score by the standard deviation of the variable at T1; significant values are highlighted in bold (*p* < .05).

Table 3

Standardized Estimates and Bootstrapped 90% Confidence Intervals (CI) of the Total, Direct, and Mediated (Indirect) Effects of the Mediation Models with ISBF-Total-Score as Mediator

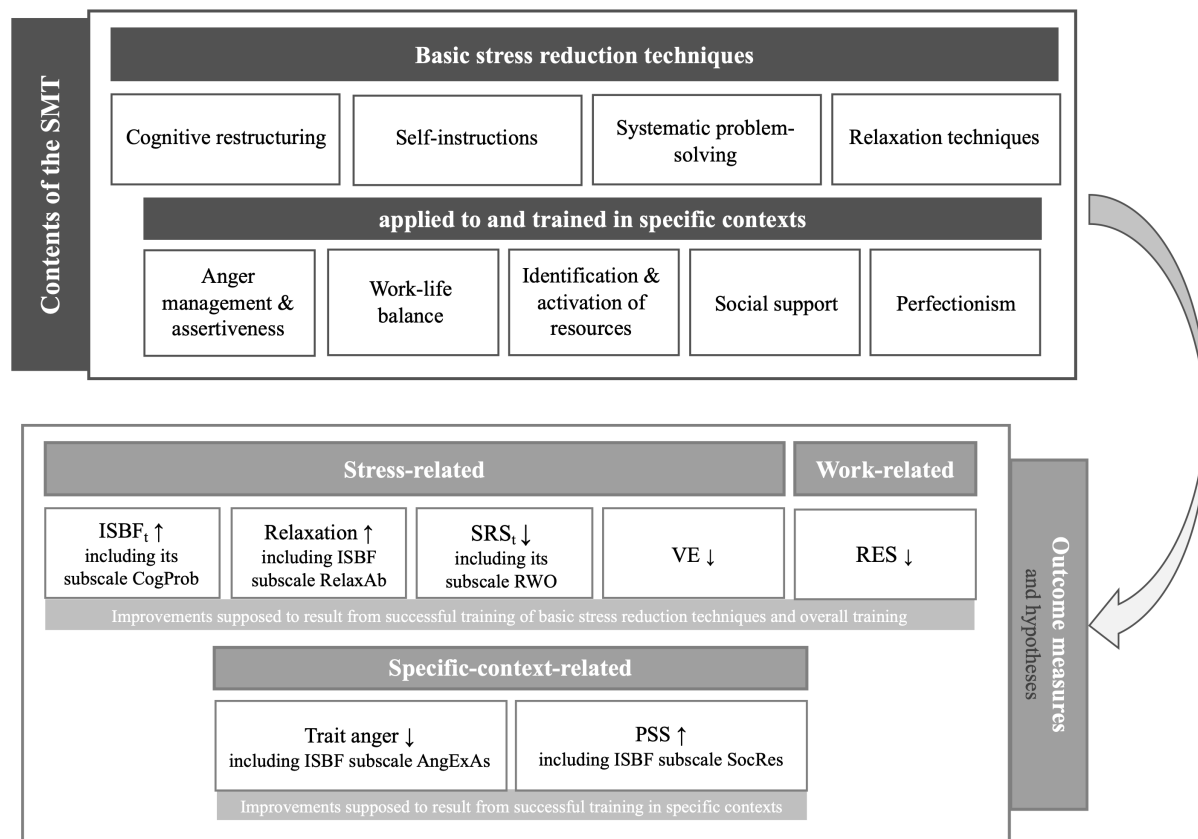
	Total effect			Direct effect of group (SMT- /AT-group)			Mediated indirect effect via ISBF-total-score T2		
	Estimate	Lower Limit	Upper Limit	Estimate	Lower Limit	Upper Limit	Estimate	Lower Limit	Upper Limit
Main mediation model (<i>n</i>=84)									
Relaxation after work T3	0.46	<i>0.310</i>	0.589	0.31	<i>0.081</i>	0.481	0.15	<i>0.033</i>	0.323
PSS T3	0.31	<i>0.176</i>	0.473	0.18	<i>-0.029</i>	0.392	0.13	<i>0.000</i>	0.304
VE T3	-0.39	<i>-0.557</i>	<i>-0.218</i>	-0.16	<i>-0.355</i>	<i>0.070</i>	-0.24	<i>-0.401</i>	<i>-0.116</i>
SRS-total-score T3	-0.60	<i>-0.769</i>	<i>-0.439</i>	-0.36	<i>-0.606</i>	<i>-0.116</i>	-0.24	<i>-0.429</i>	<i>-0.085</i>
Resigned attitude toward one's job T3	-0.41	<i>-0.554</i>	<i>-0.248</i>	-0.30	<i>-0.532</i>	<i>-0.097</i>	-0.10	<i>-0.245</i>	<i>0.064</i>
Trait anger T3	-0.33	<i>-0.497</i>	<i>-0.154</i>	-0.11	<i>-0.363</i>	<i>0.145</i>	-0.22	<i>-0.419</i>	<i>-0.045</i>
ISBF-total-score T2				0.43	<i>0.320</i>	0.530			
Additional mediation model (<i>n</i>=87)									
RWO T3	-0.39	<i>-0.572</i>	<i>-0.220</i>	-0.18	<i>-0.421</i>	<i>0.058</i>	-0.21	<i>-0.390</i>	<i>-0.067</i>
ISBF-total-score T2				0.42	<i>0.310</i>	0.518			

Note. *N* = 10,000 Bootstrapping resamples; *n* = sample size; T2 = timepoint 2; T3 = timepoint 3; PSS = perceived social support; VE = vital exhaustion; SRS-total-score = Stress-Reactivity-Scale total-score; ISBF-total-score = Inventory for Assessment of Stress Management Skills total-score; RWO = SRS subscale reactivity-to-work-overload; SMT-group = stress management training group; AT-group = alternative training group; statistical significance was evaluated based one-sided 95% bootstrap confidence intervals (CI), the relevant CI limits for statistical significance are highlighted in italics; significant estimates based on CIs are highlighted in bold (*p* < .05).

Figures

Figure 1

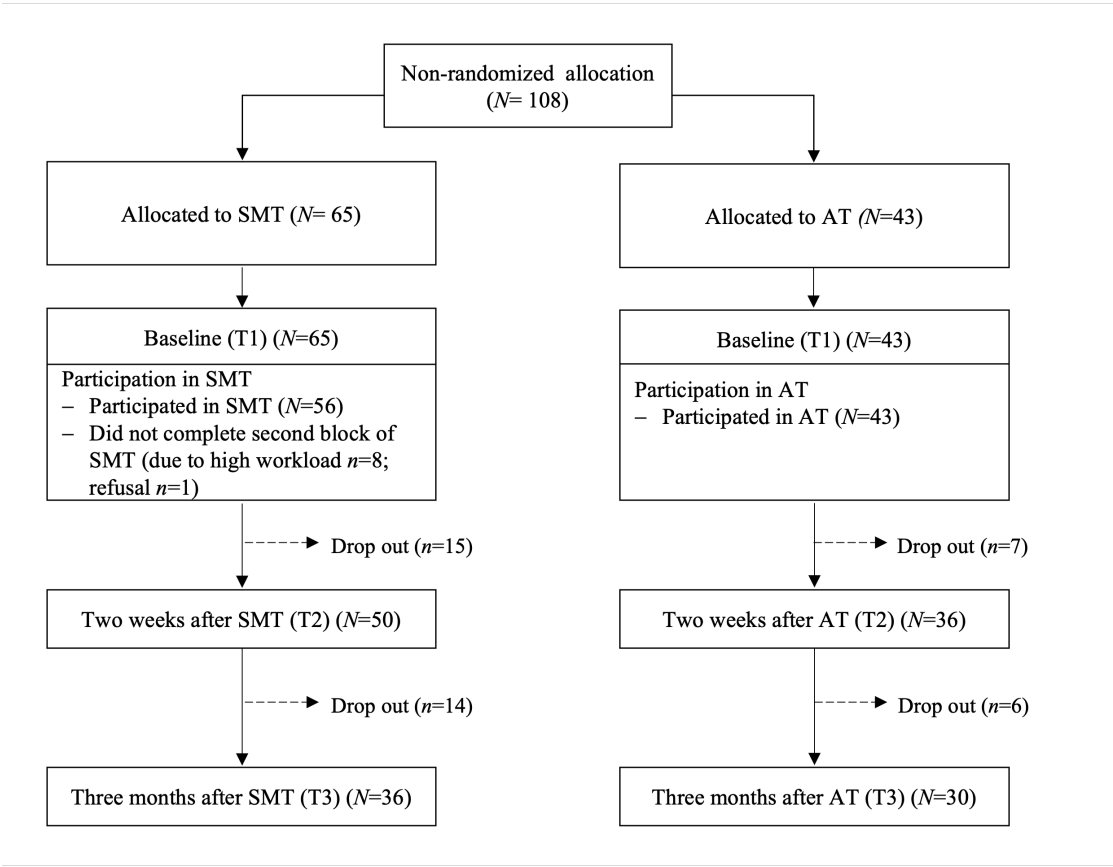
Content and Structure of the Stress Management Training (SMT) Related to Hypothesized Changes in Stress-related, Work-related, and Specific-Context-Related Outcome Measures



Note. ISBF_t=Inventory for Assessment of Stress Management Skills total-score; Relaxation=Relaxation after work; SRS_t = Stress-Reactivity-Scale total-score; VE = vital exhaustion; RES = resigned attitude toward one's job; PSS=perceived social support; CogProb = ISBF subscale cognitive-strategies-and-problem-solving; RelaxAb = ISBF subscale relaxation-abilities; RWO = SRS subscale reactivity-to-work-overload; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of-social-resources; ↑ = hypothesized increases; ↓ = hypothesized decreases.

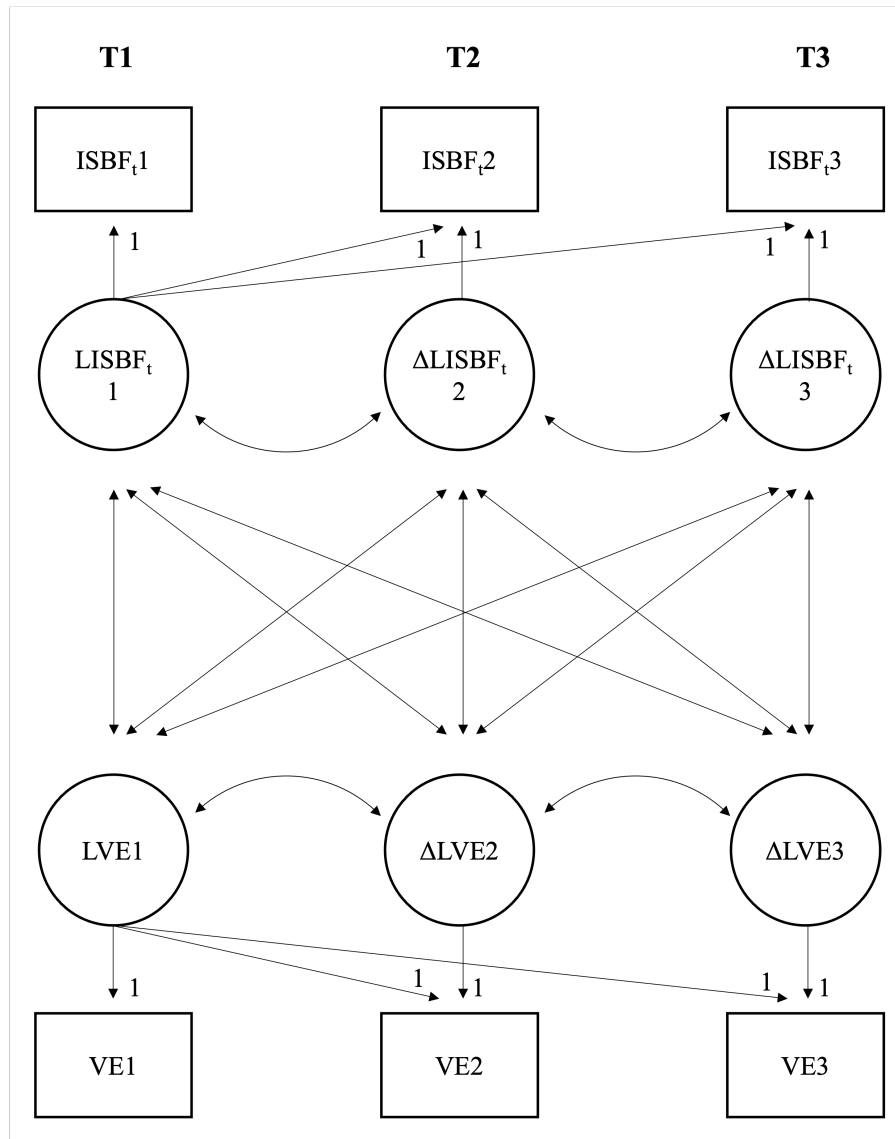
Figure 2

Participants' Allocation to Study Groups and Participant Flow Through the Study



Note. SMT = stress management training; AT = alternative training; N = sample size; T = timepoint.

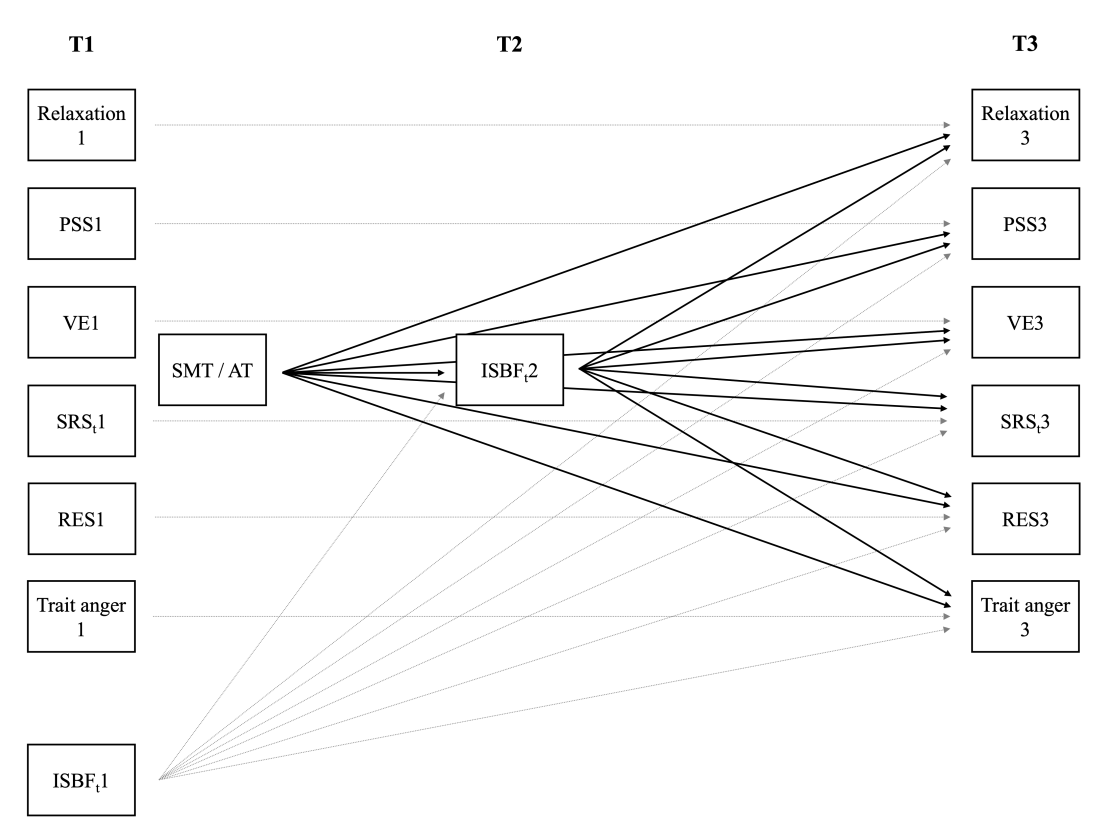
A Simplified Graphical Representation of the Main Multigroup Latent Difference Model (LD Model) Based on Two (Instead of Seven) Outcome Variables, Namely Inventory for Assessment of Stress Management Skills Total-Score (ISBF_i) and Vital Exhaustion (VE)



Note. One baseline-change model is calculated for the stress management training (SMT)-group and simultaneously one for the alternative training (AT)-group (i.e., multigroup). Mean baseline values were calculated for timepoint (T) 1 (LISBF_{T1} and LVE1). Baseline-change scores were calculated for T2 and T3 (Δ LISBF_{T2} and Δ LVE2: T2-T1; Δ LISBF_{T3} and Δ LVE3: T3-T1).

Figure 4

The Main Mediation Model with Inventory for Assessment of Stress Management Skills Total-Score (ISBF_t) at Timepoint (T) 2 as Mediator and the Other Main Outcome Variables at T3 as Dependent Variables (Black Lines) While Controlling for Their Baseline (T1), i.e., Before Beginning of the Stress Management Training (SMT) or the Alternative Training (AT) (Gray Lines)



Note. For reasons of clarity, we did not depict correlations across variables at T1 and at T3, and between T1 and group allocation. Relaxation=Relaxation after work; PSS=perceived social support; VE = vital exhaustion; SRS_t = Stress-Reactivity-Scale total-score; RES = resigned attitude toward one's job.

Supplemental Material

Supplemental Material 1: Previous Publication of Data of the Study

Parts of this study were used for psychometric validation of the ISBF (Wirtz et al., 2013). More precisely, within the employee sample 3 of that publication, we used the baseline assessment of the questionnaires ISBF (total-score and all subscales), SRS total-score, BSSS subscale PSS, and VE of all participants to calculate the factor structure and internal consistency of the ISBF scales as well its convergent validity with the three other scales. Moreover, preliminary data of the study were used for a doctoral thesis assessing SMT effects, including 30 participants of the experimental SMT-group and 20 participants of the AT-group (Stein, 2007). In that thesis, the effects of the SMT as compared to the AT were tested on most outcome variables except for the main questionnaire of interest of the present study, namely the ISBF-total-score and its subscales, and except for the SRS-total-score and its subscale stress-reactivity-to-work-overload.

Supplemental Material 2: Results of Mediation Analyses with the ISBF Subscales as Mediators

Due to the sample size, we had to examine the *ISBF subscales as mediators* in separate path analytic models. For each ISBF subscales, that significantly changed over time, i.e. cognitive-strategies-and-problem-solving, adequate-anger-expression-and-assertiveness, identification-and-use-of-social-resources, and relaxation-abilities, we again specified two models, i.e., a main mediation model and an additional mediation model, resulting in a total of 8 models. All 8 models fit the data (acceptably) well (main models: χ^2 's(36) $\leq$ 46.37, p 's $\geq$.12; RMSEAs $\leq$.06 (90%CIs = [LLs = .000; ULs $\leq$.103]); CFIs $\geq$.97; SRMRs $\leq$.09 and additional models: χ^2 's(1) $\leq$ 2.87, p 's $\geq$.09; RMSEAs $\leq$.15 (90%CIs = [LLs = .000; ULs $\leq$.358]); CFIs $\geq$.94; SRMRs $\leq$.05). In all 8 models, we found total effects of group, i.e., training, on all outcome variables (95%CIs for expected increases in SMT- vs. AT-group = [LLs $\geq$.158]; 95%CIs for expected decreases in SMT- vs. AT-group = [ULs $\leq$ -.120]). The subscale cognitive-strategies-and-problem-solving proved to be the most important mediator since we found indirect effects via this subscale for all outcomes (main model and additional model: 95%CIs for expected increases in SMT- vs. AT-group = [LLs $\geq$.004]; 95%CIs for expected decreases in SMT- vs. AT-group = [ULs $\leq$ -.078]) except job dissatisfaction. There were direct effects of group, i.e., training, for relaxation after work (95%CI = [LL = 0.180]), SRS-total-score (95%CI = [UL = -0.070]), and job dissatisfaction (95%CI = [UL = -0.160]), but not for PSS, VE, trait anger, and SRS subscale reactivity-to-work-overload. We also found a direct effect of group and thus training on the subscale cognitive-strategies-and-problem-solving T2 (95%CIs = [LLs $\geq$.237]). Regarding the further subscales, we found indirect effects via the subscale identification-and-use-of-social-resources for VE and the SRS subscale reactivity-to-work-overload (95%CIs = [ULs $\leq$ -.002]) in the expected direction and via the subscale relaxation-abilities for PSS (95%CI = [LL = -0.213]) contrary to our expectations in a negative direction. There were no indirect effects via the subscale adequate-anger-expression-and-

assertiveness. We, moreover, found direct effects of group, i.e., training, for all outcome variables (95%CIs for expected increases in SMT- vs. AT-group = $[LLs \geq .157]$; 95%CIs for expected decreases in SMT- vs. AT-group = $[ULs \leq -.069]$) except for trait anger in the main mediation model with the subscale relaxation-abilities as mediator. We also found direct effects of the training on T2 of the ISBF subscales identification-and-use-of-social-resources, and relaxation-ability (95%CIs = $[LLs \geq .028]$), but not on T2 of adequate-anger-expression-and-assertiveness.

Supplemental Material 3: Discussion of Mediation Results of the ISBF Subscales

Relaxation-Abilities and Adequate-Anger-Expression-and-Assertiveness

We found an indirect effect via the subscale relaxation-abilities on PSS, but contrary to our expectations in a negative direction. Here, a bias due to a ceiling effect is conceivable as both groups displayed very high PSS values at baseline. Notably, in clinical studies in the context of cancer, i.e., in highly stressed population, there is evidence that perceived relaxation-abilities mediate or at least relate to beneficial SMT effects (Antoni et al., 2006; Jensen et al., 2013; Marsland et al., 2020), although not unequivocally (Gudenkauf et al., 2015). Especially in highly stressed populations it is possible that first remedy through relaxation is necessary in order to train other techniques with a clear head. This would increase importance of perceived relaxation-abilities for beneficial SMT effects in highly stressed participants, but not in occupational participants in general. Interestingly, there were no indirect effects via the subscale adequate-anger-expression-and-assertiveness. Given that adequate-anger-expression-and-assertiveness could also be a result of successfully using other coping strategies such as cognitive-strategies-and-problem-solving, we speculate that this subscale is rather an outcome of successful SMTs than a mediator of change. This consideration is in line with cognitive-behavioral interventions being commonly used to reduce anger (Beck & Fernandez, 1998; Fava et al., 1991; Gerzina & Drummond, 2000; Lee & DiGiuseppe, 2018). However, the study in mothers of children newly diagnosed with cancer found SMT-induced increases in perceived assertiveness to mediate psychological health outcomes after SMT (Marsland et al., 2020).

Supplemental Material 4: Tables

Table S1

Results of Post-Hoc 2 (Group) x 3 (Timepoints) Repeated Measures ANOVAs without and with the Covariate Gender

	Listwise deletion without covariate: $N=62$ ($n=32$ SMT-group; $n=30$ AT-group) (with covariate: $N=61$ ($n=32$ SMT-group; $n=29$ AT-group))				Last observation carried forward (LOCF) without covariate: $N=104$ ($n=61$ SMT-group; $n=43$ AT-group) (with covariate: $N=103$ ($n=61$ SMT-group; $n=42$ AT-group))			
	$[df_{Num}, df_{Den}]$	F	p	η^2_p	$[df_{Num}, df_{Den}]$	F	p	η^2_p
ISBF-total-score	[1.57, 94.15] (1.60, 92.96)	11.63 (7.54)	< .001 (.002)	.16 (.12)	[1.41, 143.54] (1.42, 142.28)	19.33 (16.17)	< .001 (< .001)	.16 (.14)
CogProb	[1.67, 100.16] (1.66, 93.39)	10.51 (7.69)	< .001 (.002)	.15 (.12)	[1.46, 148.98] (1.46, 146.29)	14.76 (12.42)	< .001 (< .001)	.13 (.11)
AngExAs	[1.77, 106.11] (1.80, 104.17)	2.84 (3.34)	.069 (.044)	.05 (.05)	[1.60, 163.57] (1.63, 162.63)	3.79 (3.41)	.033 (.045)	.04 (.03)
SocRes	[1.82, 109.04] (1.85, 107.43)	4.61 (3.50)	.014 (.037)	.07 (.06)	[1.69, 172.59] (1.71, 171.10)	7.07 (6.57)	.002 (.003)	.07 (.06)
RelaxAb	[2, 120] (2, 116)	10.53 (7.43)	< .001 (< .001)	.15 (.11)	[1.75, 178.36] (1.77, 177.25)	12.34 (10.18)	< .001 (< .001)	.11 (.09)
PBodTens	[1.77, 106.25] (1.79, 104.07)	.21 (.06)	.79 (.93)		[1.51, 154.22] (1.52, 152.06)	2.34 (2.19)	.11 (.13)	
Relaxation after work	[1.68, 100.69] (1.67, 96.95)	17.08 (14.63)	< .001 (< .001)	.22 (.20)	[1.50, 152.79] (1.49, 148.61)	16.86 (13.94)	< .001 (< .001)	.14 (.12)
PSS	[2, 120] (2, 116)	5.28 (5.09)	.006 (.008)	.08 (.08)	[1.85, 188.64] (1.87, 186.71)	8.30 (7.97)	< .001 (< .001)	.08 (.07)
VE	[2, 120] (2, 116)	15.13 (12.31)	< .001 (< .001)	.20 (.18)	[1.83, 186.52] (1.85, 185.39)	19.72 (16.03)	< .001 (< .001)	.16 (.14)

SRS-total-score	[1.86, 111.62] (1.89, 109.65)	33.07 (25.82)	< . .001 (< . .001)	.36 (.31)	[1.70, 173.66] (1.72, 172.29)	36.20 (31.54)	< . .001 (< . .001)	.26 (.24)
RWO	[1.74, 104.55] (1.79, 103.96)	15.87 (13.52)	< . .001 (< . .001)	.21 (.19)	[1.67, 169.94] (1.70, 170.22)	14.47 (12.90)	< . .001 (< . .001)	.12 (.11)
Resigned attitude toward one's job	[1.81, 108.37] (1.84, 106.92)	7.98 (5.51)	< . .001 (.007)	.12 (.09)	[1.66, 169.12] (1.68, 167.70)	8.33 (7.07)	< . .001 (.002)	.08 (.07)
Trait anger	[1.76, 105.64] (1.77, 102.68)	6.44 (5.16)	. .003 (.010)	.10 (.08)	[1.63, 166.08] (1.63, 163.29)	9.05 (7.37)	< . .001 (.002)	.08 (.07)

Note. Group-by-time interactions of post-hoc repeated measures ANOVAs without covariate and with covariate gender in parentheses and italics; ISBF-total-score = Inventory for Assessment of Stress Management Skills total-score; CogProb = ISBF subscale cognitive-strategies-and-problem-solving; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of-social-resources, RelaxAb = ISBF subscale relaxation-abilities; PBodTens = ISBF subscale perception-of-bodily-tension; PSS = perceived social support; VE = vital exhaustion; SRS-total-score = Stress-Reactivity-Scale total-score; RWO = SRS subscale reactivity-to-work-overload; n = sample size; SMT-group = stress management training group; AT-group = alternative training group; df_{Num} = degrees of freedom numerator; df_{Den} = degrees of freedom denominator; significant values are highlighted in bold ($p < .05$).

Table S2*Model Fit Information of Mediation Models with ISBF Subscales as Mediator*

	χ^2	RMSEA	CFI	SRMR
Main mediation models (<i>n</i> =84)				
via subscale CogProb T2	$\chi^2(36) = 46.37, p = .12$	.06 (90%CI = [.000; .103])	.97	.08
via subscale AngExAs T2	$\chi^2(36) = 41.49, p = .24$	.04 (90%CI = [.000; .092])	.98	.09
via subscale SocRes T2	$\chi^2(36) = 37.16, p = .42$	.02 (90%CI = [.000; .081])	1.00	.08
via subscale RelaxAb T2	$\chi^2(36) = 45.85, p = .13$	.06 (90%CI = [.000; .102])	.97	.08
Additional mediation models (<i>n</i> =87)				
via subscale CogProb T2	$\chi^2(1) = 0.84, p = .36$	.00 (90%CI = [.000; .274])	1.00	.02
via subscale AngExAs T2	$\chi^2(1) = 2.87, p = .09$	.15 (90%CI = [.000; .358])	.94	.05
via subscale SocRes T2	$\chi^2(1) = 1.33, p = .25$	.06 (90%CI = [.000; .300])	1.00	.03
via subscale RelaxAb T2	$\chi^2(1) = 0.65, p = .42$	.00 (90%CI = [.000; .262])	1.00	.02

Note. Main mediation models = SRS-total-score and other main outcome variables as dependent variables; additional mediation models = SRS subscale reactivity-to-work-overload as only dependent variable; *n* = sample size; T2 = timepoint 2; CogProb = Inventory for Assessment of Stress Management Skills (ISBF) subscale cognitive-strategies-and-problem-solving; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of social-resources, RelaxAb = ISBF subscale relaxation-abilities; RWO = Stress-Reactivity-Scale (SRS) subscale reactivity-to-work-overload; χ^2 = Chi-Square Test of Model Fit; RMSEA = Root Mean Square Error Of Approximation; CFI = Comparative Fit Index; SRMR = Standardized Root Mean Square Residual; CI = confidence interval.

Table S3

Standardized Estimates and Bootstrapped 90% Confidence Intervals (CI) of the Total, Direct, and Mediated (Indirect) Effects of Mediation Models with ISBF Subscales as Mediators

	Total effect			Direct effect of group (SMT- /AT-group)			Mediated indirect effect via subscale CogProb T2		
	Estimate	Lower Limit	Upper Limit	Estimate	Lower Limit	Upper Limit	Estimate	Lower Limit	Upper Limit
Main mediation model (n=84)									
Relaxation after work T3	0.45	<i>0.301</i>	0.586	0.36	<i>0.180</i>	0.508	0.09	<i>0.004</i>	0.205
PSS T3	0.30	<i>0.162</i>	0.458	0.11	<i>-0.015</i>	0.273	0.19	<i>0.107</i>	0.279
VE T3	-0.36	<i>-0.527</i>	<i>-0.175</i>	-0.16	-0.363	<i>0.061</i>	-0.20	-0.324	<i>-0.098</i>
SRS-total-score T3	-0.54	-0.704	<i>-0.380</i>	-0.24	-0.415	<i>-0.070</i>	-0.30	-0.428	<i>-0.186</i>
Resigned attitude toward one’s job T3	-0.40	-0.561	<i>-0.226</i>	-0.36	-0.544	<i>-0.160</i>	-0.04	-0.159	<i>0.063</i>
Trait anger T3	-0.30	-0.477	<i>-0.120</i>	-0.12	-0.310	<i>0.089</i>	-0.19	-0.320	<i>-0.078</i>
CogProb T2				0.37	<i>0.241</i>	0.487			
Additional mediation model (n=87)									
RWO T3	-0.36	-0.540	<i>-0.176</i>	-0.12	-0.324	<i>0.086</i>	-0.24	-0.356	<i>-0.137</i>
CogProb T2				0.36	<i>0.237</i>	0.482			
							Mediated indirect effect via subscale AngExAs T2		
Main mediation model (n=84)									
Relaxation after work T3	0.47	<i>0.327</i>	0.601	0.46	<i>0.305</i>	0.589	0.02	<i>-0.012</i>	0.067

[illegible]

RWO T3	−0.40	−0.579	−0.226	−0.35	−0.527	−0.156	−0.05	−0.153	−0.002
SocRes T2				0.16	0.029	0.290			
							Mediated indirect effect via subscale RelaxAb T2		
Main mediation model (n=84)									
Relaxation after work T3	0.45	0.288	0.591	0.41	0.198	0.598	0.04	−0.059	0.149
PSS T3	0.29	0.158	0.457	0.43	0.273	0.604	−0.14	−0.213	−0.059
VE T3	−0.37	−0.537	−0.188	−0.40	−0.608	−0.197	0.04	−0.068	0.161
SRS-total-score T3	−0.59	−0.744	−0.441	−0.67	−0.864	−0.488	0.08	−0.030	0.200
Resigned attitude toward one’s job T3	−0.39	−0.553	−0.229	−0.41	−0.680	−0.153	0.02	−0.114	0.175
Trait anger T3	−0.30	−0.492	−0.123	−0.26	−0.533	0.018	−0.05	−0.203	0.093
RelaxAb T2				0.44	0.312	0.562			
Additional mediation model (n=87)									
RWO T3	−0.40	−0.583	−0.223	−0.51	−0.715	−0.293	0.11	−0.027	0.216
RelaxAb T2				0.45	0.318	0.568			

Note. $N = 10,000$ Bootstrapping resamples; n = sample size; T2 = timepoint 2; T3 = timepoint 3; PSS = perceived social support; VE = vital exhaustion; SRS-total-score = Stress-Reactivity-Scale total-score; RWO = SRS subscale reactivity-to-work-overload; CogProb = Inventory for Assessment of Stress Management Skills (ISBF) subscale cognitive-strategies-and-problem-solving; AngExAs = ISBF subscale adequate-anger-expression-and-assertiveness; SocRes = ISBF subscale identification-and-use-of-social-resources, RelaxAb = ISBF subscale relaxation-abilities; SMT-group = stress management training group; AT-group = alternative training group; statistical significance was evaluated based on one-sided 95% bootstrap confidence intervals (CI), the relevant CI limits for statistical significance are highlighted in italics; significant estimates based on CIs are highlighted in bold ($p < .05$).

References Supplemental Material

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