Individual Differences in the Correspondence Between Psychological and Physiological Stress Indicators

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Abstract

Stress threatens physical and mental health. Reactions to acute stress comprise multiple levels, including negative thoughts, bodily symptoms and behaviors. Individuals differ in their reaction to acute stress, and importantly, also in the extent to which these levels align, with a closer correspondence between psychological and physiological stress indicators being beneficial for mental health and well-being. This preregistered study investigates such individual differences systematically by inducing psychological (social-evaluative) and physiological (cold water) stress with the Maastricht Acute Stress Test (MAST) in 149 healthy adults. Participants indicated the extent to which they perceived to be stressed and four physiological stress indicators (blood pressure, heart rate, salivary cortisol, alpha-amylase) were measured. Finally, multiple personality traits were assessed as potential moderators, including the Big Five, trait anxiety, and general cognitive ability. In line with previous research, psychological and physiological stress indicators were only weakly correlated. However, considering individual differences in personality, revealed especially conscientiousness and openness as potential moderators of the correspondence between psychological and physiological stress indicators. We propose individual differences in interoceptive abilities as another critical moderator, which deserves further investigation, and discuss how future research on individual differences in psycho-physiological correspondence can contribute to further our understanding of mental and physical diseases.

Keywords: Individual differences, stress, psycho-physiological correspondence, personality, trait anxiety, cognitive ability.

Impact Statement

Stress has substantially increased over the last decade. A missing correspondence between self-reported stress and physiological stress measures has been related to mental illnesses and lower well-being. In revealing that specific personality traits might act as potential moderators of the psycho-physiological correspondence, our research complements recent prospects towards personalized medicine and offers starting points for the development of individualized psychological interventions and prevention programs.

1. Introduction

Stress has been identified as a major health challenge by the American Psychological Association (APA, 2022) and is sometimes even marked as "the health epidemic of the 21st century". Indeed, numerous studies demonstrate associations between *chronic* stress, i.e., the feeling of being overwhelmed by recurring external or internal demands over a longer period of time without being able to recover, and the onset of physical diseases like hypertension and heart attacks (Cohen et al., 2012; Spruill, 2010, Walther & Wirtz, 2023). With regard to the development of mental disorders like depression, anxiety, posttraumatic stress disorder, and chronic pain, stress was identified as a critical vulnerability factor as well (Mills et al., 2019; O'Connor et al., 2021; Schneidermann et al., 2005). Multiple large-scale studies further show that stress has substantially increased over the last decades due to working conditions like multitasking, time pressure or the need to adapt to quickly changing technologies (Schreibauer et al., 2020), making a more comprehensive understanding of the physiological and psychological mechanisms of stress mandatory.

In psychological research, three approaches to investigate stress have emerged (e.g., Walinga, 2010): First, stress as an input relates to research on psychological and physical stressors like time pressure, uncontrollability, critical life events, noise or pain. Second, stress as a transaction denotes the interplay between person and situation, while the question whether this interplay is perceived as stressful depends on appraisal processes (Lazarus & Folkman, 1984). Third, stress as an output refers to the stress reaction that manifests on the subjective-psychological, the physiological and the behavioral level. Psychologically, stress is characterized by heightened levels of perceived tension and anxiety, influencing cognitive processes such as rumination and intrusive thoughts (Cohen et al., 2016). Physiological markers of stress encompass alterations in heart rate, blood pressure, skin conductance level and cortisol release (Allen et al., 2014; Marsland et al., 2017). Finally, disruptions in sleep patterns, changes

in eating habits as well as alcohol and drug consumption exemplify the behavioral manifestations of stress (Geiker et al., 2018; Sinha, 2018).

1.1. The "stressful personality"

Personality traits have been established as both protective and vulnerability factors with regard to stress. In a recent meta-analysis addressing the relationship between the Big Five personality traits and three different conceptualizations of stress (psychological stress response, physiological stress response, type of stressor exposure; Luo et al., 2023), neuroticism was strongly positively, whereas extraversion, agreeableness and conscientiousness were negatively associated with measures of psychological stress perception, i.e., affective states and cognitions. Openness showed a small negative, but no significant across-study relationship with these psychological measures when controlling for small study bias. By contrast, no significant associations were observed between the Big Five and physiological stress measures such as heart rate variability or skin conductance. A potential explanation for this latter finding can be derived from the observation that sometimes only some individuals react with heightened physiological reactivity to the exposure to acute psychological stress, while others demonstrate blunted physiological reactions (Caroll et al., 2017). As long as personality traits are not substantially and consistently linked to both reaction types, potential associations between traits and physiological measures may level out and might thus not be identifiable in the form of a significant correlation across individuals. With respect to the type of stressor exposure (dependent vs. independent on individuals' personality characteristics), only neuroticism, agreeableness and conscientiousness showed significant across-study associations. This third conceptualization of stress is also a critical part of the well-established Exposure-Reactivity model of stress and coping (Bolger & Zuckerman, 1995, Kammeyer-Mueller et al., 2009), which proposes a mediating role for stressor exposure within the relation between personality and the stress reaction, such that, e.g., neuroticism may increase stressor exposure and this heightened exposure in turn leads to a higher stress reaction. On the other hand, the Exposure-

Reactivity model also conceptualizes personality traits as possible moderators between stressor exposure and the stress reaction such that e.g., persons high in neuroticism tend to experience stressors more severely than persons low in neuroticism. Another critical factor to consider when studying the link between personality traits and stress reactions may be individual variation in the correspondence between different response systems of stress (psychological vs. physiological). However, this variation has not been addressed in the context of the Exposure-Reactivity model, so far.

1.2. The missing psycho-physiological correspondence

Although correspondence between different response systems of stress and emotions would be highly functional from an evolutionary perspective in terms of facilitating adequate reactions to external demands (Levenson et al., 1994), empirical studies only show weak associations between psychological, physiological and behavioral stress indicators with substantial variations across different studies. This heterogeneity may be explained by the use of different stress induction and stress placebo procedures, varying measures of stress responses, as well as by the use of two different ways to determine the psycho-physiological correspondence: on a within-subject level, i.e., correspondence of psychological and physiological measures over different trials or across time within individuals, such that trials with higher stress intensity lead to higher psychological and physiological responses (e.g., Mauss et al., 2005) versus on a between-subject level, i.e., correspondence of psychological and physiological measures between different persons, so that persons scoring high on psychological measures tend to score also high on physiological measures (e.g., Sze et al., 2010). With regard to the latter approach, a review of 49 studies using the Trier Social Stress Test (TSST), a well-established method to induce psychological stress, revealed that significant correlations between psychological and physiological stress markers were observed in 25% of the studies only. The authors hypothesized that personality traits might be a critical factor moderating the psycho-physiological correspondence but did not test this hypothesis further

(Campbell & Ehlert, 2012). Similarly, low between-subject psycho-physiological correspondence was also observed in a recent study that used experience sampling and mobile smartwatch sensing to assess psychological and physiological indicators of stress in the everyday life of the participants (Talic et al., 2023). However, these every-day life stress situations were mainly of psychological nature as well. Whether similarly low correspondence between psychological and physiological stress responses exists after facing situations combining psychological and physiological stressors has not yet been investigated.

1.3. Potential moderators of psycho-physiological correspondence

Only few studies considered the possible moderating effects of different (personality) variables on the psycho-physiological correspondence: The pioneer study of Dietzen et al. (2008) examined potential links between psycho-physiological correspondence and attachment style, while higher body awareness has been shown to be positively related with psycho-physiological correspondence in another study (Sze et al., 2010). Addressing clinical relevance, Sommerfeldt et al. (2019) observed that persons with higher correspondence between self-reported stress and heart rate reported higher psychological well-being, fewer depressive symptoms, and lower levels of trait anxiety, denial coping and proinflammatory biomarkers, altogether supporting the significance of psycho-physiological correspondence for both physical and mental health. Finally, the most recent investigation by Talic et al. (2023) found higher conscientiousness and neuroticism to be related to higher psycho-physiological correspondence, while extraversion was associated with lower correspondence. Different associations of personality traits with interoceptive abilities (i.e., abilities of perceiving internal bodily signals) were proposed as potential explanation for these findings. Specifically, interoceptive abilities were assumed to be positively associated with conscientiousness (Ferentzi et al., 2017) and neuroticism (Pearson & Pfeifer, 2022), while extraversion demonstrates typically no association with interoception, most likely due to the inclination of extraversion towards the outside world (Pearson & Pfeifer, 2022). Notably, both studies linking

psycho-physiological correspondence to personality factors considered only one single measure as indicator of the physiological stress response. While Sommerfeldt et al. (2019) focused on heart rate because it is, unlike most other physiological stress indicators, accessible to conscious awareness, Talic et al. (2023) evaluated a transformed stress value based on heart rate variability. However, the use of a single measure as indicator of the physiological stress system might be problematic, as not all persons react in the same way and there are varying amounts of non-responders in respect to different measures (Ellis et al., 2006; Korte et al., 2005). Based on this observation, the use of multiple different physiological indices within the same sample was proposed as a promising means to investigate the link between psycho-physiological correspondence and personality in more detail (Sommerfeldt et al., 2019), but this has – to the best of our knowledge – not yet been realized.

1.4. Aim of the current study

The aim of the current study is to provide a comprehensive investigation of the correspondence between psychological and physiological stress responses in reaction to acute stress. We address the current research gaps (a) by applying a stress induction procedure that comprises exposure to psychological and physiological stressors and causes a marked stress reaction, (b) by assessing a broad range of physiological indicators, and (c) by considering multiple personality traits as potential moderators. Specifically, we used the Maastricht Acute Stress Test (MAST, Smeets et al., 2012) to induce psychological and physiological stress in 149 healthy adults, randomly assigned to a stress group and a placebo group. Subjectively perceived stress (the psychological stress response) was assessed with self-ratings, while heart rate, blood pressure, alpha-amylase and cortisol were obtained as indicators of the physiological stress response. The latter allows for conclusions about the reactivity of both the sympatho-adrenal medullary (SAM) stress axis and the hypothalamic-pituitary-adrenal axis (HPA). The Big Five personality factors (extraversion, neuroticism, agreeableness, conscientiousness, openness) as well as trait anxiety were tested as potential moderators, while the role of general

intelligence as a more cognitive but also health-related dimension of personality (Deary, 2012, 2021; DeYoung, 2011), was explored without a concrete hypothesis.

1.5. Hypotheses

H1: Based on previous research on the psycho-physiological correspondence in laboratory (e.g., Campbell & Ehlert, 2012) and real-life settings (Talic et al., 2023), we expect corresponding psychological and physiological stress indicators to show zero or weak positive correlations. Corresponding indicators refer to the matching point in time between psychological and physiological stress indicators which is (a) at the same time point for blood pressure, heart rate, and alpha-amylase capturing the relatively fast sympatho-adrenal medullary (SAM) response, and (b) with a 15-20 minutes time-delay for cortisol depicting the slower hypothalamic-pituitary-adrenal axis (HPA) response. Following the review by Campbell and Ehlert (2012), two different kinds of correspondence scores are considered: (a) the correspondence between the absolute values of psychological and physiological stress, and (b) the correspondence between the absolute value of psychological stress and the change value in physiological stress, while change values refer to the difference between the absolute value and the average between the first two measurements (before stress onset).

H2. We expect the following personality traits to moderate the psycho-physiological correspondence:

H2a. Trait anxiety: Following Sommerfeldt et al. (2019), we expect lower trait anxiety to be related to higher psycho-physiological correspondence.

H2b. Neuroticism: Due to the marked overlap between neuroticism and trait anxiety (e.g., Clark et al., 1994), we initially (see preregistration) expected that lower trait neuroticism is related to higher psycho-physiological correspondence. However, in most recent work of our group (Talic et al., 2023), we observed higher neuroticism to be related to higher psychophysiological correspondence. Thus, and in deviation to our preregistration, the association with neuroticism will be tested rather exploratorily.

H2c. Openness: Based on previous findings that body awareness is positively associated with psycho-physiological correspondence (Sze et al., 2010), and that openness is positively related to body awareness (Ferentzi et al., 2017), we expect higher openness to be related to higher psycho-physiological correspondence.

H2d. Conscientiousness: Based on previous findings that body awareness is positively associated with psycho-physiological correspondence (Sze et al., 2010), and that conscientiousness positively relates to body awareness (Ferentzi et al., 2017), we expect higher conscientiousness to be related to higher psycho-physiological correspondence.

H2e. Extraversion: Based on the findings by Talic et al. (2023) and the rationale that extraversion is associated with an orientation towards the outside world (Pearson & Pfeifer, 2022), we further expect a lower psycho-physiological correspondence for persons with high extraversion scores. Note however, that this hypothesis was not included in our preregistration.

Finally, and as listed in the preregistration under optional analyses, agreeableness and intelligence were also explored as potential moderators. In all analyses, we again differentiate between psycho-physiological correspondence of (a) absolute values and (b) change values.

2. Methods

2.1. Preregistration

Before data analysis, hypotheses, statistical procedures, the sample size, and all variables of interest including their exact operationalization (e.g., which time point was considered) were preregistered in the Open Science Framework: https://osf.io/zd3jf. Note that the present study is part of a larger research project (FOPSID, Fear of Pain, Stress, and Individual Differences in Affect and Cognition) that can be accessed under: https://osf.io/zh3ak/. Note further, that only the first two hypotheses of our preregistration were investigated in the current study (H1, H2), while H3 and H4 will be considered in a following publication.

2.2. Participants and sample size justification

The current study is based on data from $N = 149$ participants that were recruited as part of the FOPSID project at Würzburg University. The sample size was determined by a combination of a priori power calculations and what was feasible during the COVID-19 pandemic. Considering prior research, we expected small to medium effect sizes for the main effects hypothesized in the context of the FOPSID project (e.g., correlative relationship between fear of pain and stress $r = .25$; Timmers et al., 2018). With an alpha level of .05 and a power of .95, this resulted in a sample size of 168 participants (G*Power 3.1; Faul et al., 2007). In order to balance the study groups, a sample size of $N = 180$ was initially planned. However, due to restrictions induced by the COVID-19 pandemic, only complete data from 149 participants could be obtained, thus, slightly diminishing the power to .93. Of these 149 participants, 74 persons (mean age = 24.51 years, $SD = 3.73$) were randomly assigned to the stress group, while 75 persons (mean age = 25.04 years, SD = 4.13) were randomly assigned to the control group. The stress group underwent the Maastricht Acute Stress Test (MAST; Smeets et al., 2012), while the control group participated in a placebo version of the task (Placebo-MAST), where no stress was induced. Note, that we refer to the latter as “mild stress condition” instead of “no stress condition”, as we expect the situation of participating in a psychological experiment not to be completely free of any stressors.

Prior to participation, the following exclusion criteria were assessed via telephone interviews: Age < 18 or > 35; left-handedness; BMI > 30 or < 18; insufficient German language skills (to ensure that instructions are understood); vision and hearing disabilities; Psychology student (beyond the 2nd semester); regular intake of medication affecting the central nervous system; frequent smoking behaviour (> 15 cigarettes per day); drug consumption (including cannabis); history of or acute diagnosis of chronic pain (e.g., headache); episode of severe pain leading to medical treatment within the last six month (e.g., injury); diagnosis of attention-deficit/hyperactivity disorder (ADHD), dyscalculia or dyslexia; acute suffering from any

psychological, neurological, cardiovascular, respiratory or endocrinological diseases; disposition for vertigo or fainting. Note that the majority of these criteria were introduced by the fact that the current study belongs to the larger FOPSID project, which includes the assessment of multiple physiological (e.g., cortisol) and psychological (e.g., attentional control) variables, as well as electroencephalographical recordings (EEG) that can be affected by these factors in an uncontrollable way (e.g., cortisol levels can be influenced by smoking, drugs, and medications; attentional control by ADHD diagnosis; EEG by mixed handedness). Furthermore, participants with any history of chronic pain were excluded to prevent any re-activation and harm to a potentially hypersensitive nociceptive system. Participants were instructed to abstain from coffee two hours prior to measurement, to not drink alcohol for 12h before testing, and to immediately inform the study coordinator if any pain medication was taken on the day of testing (either to postpone the measurement or to exclude the participant when medication was to become regular).

Experimental procedures were performed in accordance with the declaration of Helsinki, approved by the Institutional Review Board (Psychological Institute, University of Würzburg, Germany, GZEK 2020-18) and informed written consent according to the declaration of Helsinki and to the guidelines of the German Psychological Society was obtained from all participants. Participation in the complete FOPSID project was reimbursed with 10€ per hour, except the Virtual Reality (VR) conditioning experiment (not considered in the current study) that was reimbursed with a fixed amount of money (17.50€) to prevent unintentionally reinforcing avoidance behavior (details in Hilger et al., 2023).

2.3. Study procedure

Figure 1 illustrates the general procedure of the FOPSID project including all measurement time points and the assessed variables. The project consisted of three measurement days (with varying time intervals between days), which were passed by all participants in the same order. On day 1, participants were informed about all study details,

informed consent was obtained, all traits were assessed with questionnaires, and intelligence testing took place. On day 2, all participants underwent the Maastricht Acute Stress Test (MAST), a stress induction procedure (or the placebo version of it), before entering an experimental fear conditioning paradigm in VR that was recently developed to investigate the effects of stress on the acquisition and extinction of fear of pain (FoP, Hilger et al., 2023) in accordance with the Fear-Avoidance Model of chronic musculoskeletal pain (Lethem et al., 1983; Vlaeyen & Linton, 2012). For exact timing and more information about the MAST, see Smeets et al. (2012), while the exact procedure and manipulation checks of the VR paradigm are described in detail in Hilger et al. (2023). In brief, the MAST combines physiological and social-evaluative stress exposure by including changing sequences of instructing participants to put their hand in 4°C cold water and counting backwards while receiving negative feedback from the experimenter. After this procedure, the VR fear conditioning paradigm followed for all participants, including a practice phase, a FoP acquisition phase, and a FoP modification or extinction phase. Finally, on day 3, all participants underwent a resting-state EEG recording (data not considered in the present study). While the assessment on day 1 took between 1.5 and 2 hours, the complete experimental procedure on day 2 required 2.5-4 hours (depending on individuals' choices of movements etc.) and the lab visit for EEG measurements lasted between 1,5 and 2 hours. The psychological and physiological state stress measures obtained during the experimental procedure on day 2 together with the trait measures assessed on day 1 present the input for all analyses of the current study.

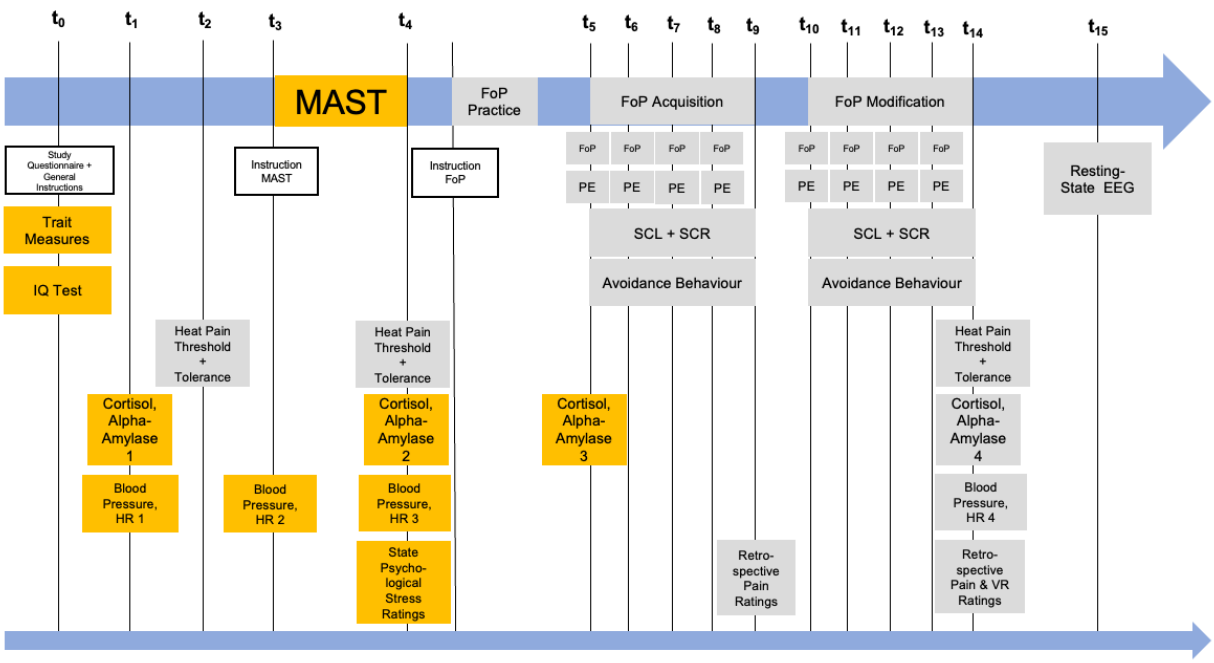

Figure 1. Flowchart of study procedure and assessed variables. Variables analyzed and presented in the current study are highlighted by yellow boxes, while variables not considered here are depicted in grey. Day 1 (t_0): Study information, personality trait assessments, and intelligence testing. Day 2 (t_1-t_{14}): Stress induction procedure and experimental fear conditioning paradigm in VR. Cortisol and alpha-amylase were assessed four times: In the beginning of the whole experiment (baseline, t_1), directly after the Maastricht Acute Stress Test (MAST, t_4), before the acquisition phase of the VR paradigm (exactly 10 min after finishing the MAST, t_5), and at the end of the experiment (t_{14}). Blood pressure and heart rate were also obtained four times: At the beginning (baseline, t_1), directly before the MAST (t_3), directly after the MAST (t_4), and at the end of the experiment (t_{14}). Subjective ratings of stress were assessed directly after finishing the MAST (t_4). Day 3 (t_{15}): Electroencephalographical (EEG) resting-state recordings (data not included in the present study). FoP, fear of pain; HR, heart rate; PE, pain expectancy; SCL, skin conductance level; SCR, skin conductance response.

2.4. Outcome variables

2.4.1. Psychological state stress

Subjectively perceived stress and negative affect were assessed directly after the MAST (reflecting induced stress) or the Placebo-MAST (reflecting mild stress levels without explicit experimental manipulation) (t_4), respectively. As recommended in Timmer et al. (2018) for the

MAST, three general items on stress, pain, and unpleasantness were used together with 10 item from the negative affect subscale of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988; German version by Breyer & Bluemke, 2016; Krohne et al., 1996). The three general items were "How stressful was this procedure right now?" (for stress), "How painful was this procedure right now?" (for pain), and "How unpleasant was this procedure right now?" (for unpleasantness). These items were responded on visual analogue scales, ranging from 0 (*not at all*) to 100 (*extremely*). For the PANAS items, participants were asked to indicate to which extent they felt the different emotions in the current moment, ranging from 1 (*not at all*) to 5 (*very strong*), for example "distressed", "upset", or "irritable". All 13 items combined showed high reliability in our sample ($\omega = .86$ and $\omega = .85$ for the MAST and Placebo-MAST groups, respectively), such that we derived a single state psychological stress score (for details, see the section *Statistical Analyses*).

2.4.2. Physiological State Stress

The state stress response of the sympatho-adrenal-medullary (SAM) system was assessed via systolic and diastolic blood pressure, heart rate, and salivary alpha-amylase, while the response of the hypothalamus-pituitary-adrenal-axis (HPA) was examined via salivary cortisol. For most comprehensive insights and as outlined in our preregistration in detail, all variables were operationalized in two ways: a) By absolute scores obtained when the stress response is assumed to be maximal and b) by change scores calculated as difference between this score and a baseline measurement.

Heart rate. Heart rate (HR) was assessed in beats per minute using the Sanitas SBM 21 (Breuer GmbH, Ulm, Germany) oscillometric blood pressure monitor with arm cuff. Heart rate values obtained at the end of the stress induction procedure, i.e., at the same time point as psychological stress ratings (t_4), were considered as absolute scores (HR_{abs}), while the difference between this score and a baseline score (average across both time points before the stress induction procedure: t_1, t_3) served as change score (HR_{change}).

Blood pressure. Systolic and diastolic blood pressure (BP) in mmHg were also measured using the Sanitas SBM 21 (Breuer GmbH, Ulm, Germany) oscillometric BP monitor. Similarly as for heart rate, diastolic and systolic blood pressure obtained at the end of the stress induction procedure (t_4) served as absolute scores (BP_{abs}), while change scores (BP_{change}) were calculated as differences between these scores and the baseline (again averaged across both time points before the stress induction procedure: t_1, t_3).

Alpha-amylase. The same definitions and timing of absolute (AA_{abs}) and change scores ($AA_{change} = t_4 - (t_1+t_3)/2$) refer also to salivary alpha-amylase.

Cortisol. Due to the slower hypothalamic-pituitary-adrenal axis (HPA) response to stress, cortisol values assessed exactly 10 minutes after finishing the stress induction (t_5) served as absolute scores. Change scores were calculated as the difference between this score and the baseline at t_1.

Concentrations of alpha-amylase and cortisol were assessed by salivary samples and analyzed in an external laboratory at Dresden University. It was ensured that all saliva samples kept frozen at -20°C until analysis. Concentrations of alpha-amylase in saliva were measured by an enzyme kinetic method: Saliva was processed on a Genesis RSP8/150 liquid handling system (Tecan, Crailsheim, Germany). First, saliva was diluted 1:625 with double-distilled water by the liquid handling system. Twenty microliters of diluted saliva and standard were then transferred into standard transparent 96-well microplates (Roth, Karlsruhe, Germany). Standard was prepared from ‘‘Calibrator f.a.s.’’ solution (Roche Diagnostics, Mannheim, Germany) with concentrations of 326, 163, 81.5, 40.75, 20.38, 10.19, and 5.01 U/l alpha-amylase, respectively, and bidest water as zero standard. Afterwards, 80 ml of substrate reagent (*a*-amylase EPS Sys; Roche Diagnostics, Mannheim, Germany) was pipetted into each well by using a multichannel pipette. The microplate containing sample and substrate was then warmed to 37°C by incubation into a waterbath for 90 s. Immediately afterwards, a first interference measurement was obtained at a wavelength of 405 nm using a standard ELISA reader (Anthos

Labtech HT2, Anthos, Krefeld, Germany). The plate was then incubated for another 5 min at 37°C in the waterbath, before a second measurement at 405 nm was taken. Increases in absorbance were calculated for unknowns and standards. Increases of absorbance of diluted samples were transformed to alpha-amylase concentrations using a linear regression calculated for each microplate (Graphpad Prism 4.0c for MacOSX, Graphpad Software, San Diego, CA).

For the analyses of cortisol concentrations, salivettes were first centrifuged at 3,000 rpm for 5 min after thawing, which resulted in a clear supernatant of low viscosity. The exact concentrations were then derived by using commercially available chemiluminescence immunoassay with high sensitivity (IBL International, Hamburg, Germany). All intra- and interassay coefficients for alpha-amylase and cortisol were below 9% indicating good measurement quality. For more details about the analytical procedure, see Rohleder et al. (2006).

2.5 Moderator variables

2.5.1. Trait anxiety

Trait anxiety was assessed together with all other trait variables prior to the experimental manipulation on a separate day (t_0) using the trait anxiety subscale ($\alpha = .88$) from the German version of the State-Trait-Depression-Inventory (STADI; Laux et al., 2013). The trait part of the STADI includes 20 items consisting of statements about the person, e.g., “I enjoy life”. Participants were asked to indicate on a 4-point scale how often each statement holds true from 1 = “not at all” to 4 = “a lot”.

2.5.2. Personality traits

The Big Five personality traits (openness, conscientiousness, extraversion, agreeableness, neuroticism) were also assessed at t_0 using the German version (Borkenau & Ostendorf, 1993) of the NEO-FFI (Costa & McCrae, 1992). The questionnaire consisted of 60 items, measuring each trait with 12 items. These items were statements about how the participants perceive themselves, e.g., “I am not easily disturbed”, and were rated on 5-point

Likert scales ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). Scale reliabilities were reported to range between α = .72 and α = .87 (Borkenau & Ostendorf, 1993).

2.5.3. Intelligence

Finally, also general intelligence was obtained at t_0 with the German version of Raven's Advanced Matrices (APM; Bulheller & Häcker, 1998; Raven & Court, 1998). The APM is a non-verbal intelligence assessment intended to capture general intelligence relatively independent of culture, knowledge and previous education. Note that the advanced rather than the standard version was used to best differentiate also in the higher ability range as can be expected for a primarily student sample. For all further analyses, the total number of correct responses was used (APM sum score). Reliability was reported to be α = .85 (Bulheller & Häcker, 1998).

2.6 Statistical analyses

All analyses were performed using the software R (R Core Team, 2021). Specifically, linear and moderated regressions were implemented with the lm() function and 95% confidence intervals were obtained with the confint() function. Prior to analyses, outliers (values deviating more than three standard deviations from the sample mean) were replaced with values two standard deviations from the sample mean (for an analogous procedure, see e.g., Timmers et al., 2018). Due to largely differing value ranges across psychological and physiological variables, all scores were tested for normal distribution using the Shapiro-Wilk test as well as graphical inspections. As almost all distributions were non-normal, Spearman's ρ was used as correlation coefficient. Further, all scores were *z*-standardized before entering the multiple regression models, while Table 1 illustrates the unstandardized scores.

To obtain a composite score for state psychological stress, the ten negative affect items of the PANAS and the three single items assessing stress, pain, and unpleasantness (see *Measures* for details) were combined into a single composite score of psychological stress (PS) after running an exploratory factor analysis using the fa() function. To obtain a single score for

blood pressure, we calculated the mean arterial pressure based on systolic and diastolic blood pressure using the formula provided by Meany et al. (2000). In a post-hoc analysis, which was not pre-registered, we obtained a composite score for state physiological stress analogously to the composite score for state psychological stress, i.e., by obtaining the factor score of an exploratory factor analysis based on the four physiological variables. All analyses were rerun with that latent physiological stress factor as variable of interest to gain insight into a potential superiority of a composite physiological score as compared to separate physiological stress indicators.

The MAST and Placebo-MAST groups were analyzed separately. Respective models are presented in the results section. Statistical significance was assumed at $p < .05$. Furthermore, effect sizes and their 95% confidence intervals were computed. To account for multiple comparisons, all *p*-values were corrected with the Benjamini-Hochberg correction ($\alpha = .05$) within the same model across the MAST and Placebo-MAST conditions, respectively (Benjamini & Hochberg, 1995). Since the Holm-Bonferroni-Method has been evaluated as too conservative, especially when it comes to exploratory analyses, we considered the Benjamini-Hochberg correction as the most appropriate method (instead of the Holm-Bonferroni, which was initially specified in our preregistration, Holm, 1979). For most comprehensive insights and to enhance clarity, both corrected and uncorrected *p*-values are reported and the exact model specifications are described in the beginning of the respective results sections.

2.7 Data and code availability

All analysis code can be accessed at the Open Science Framework under: https://osf.io/zh3ak/files/osfstorage. The data can be obtained from the authors by request.

3. Results

3.1. Descriptive statistics and manipulation checks

Descriptive statistics and reliabilities for each variable and each group (MAST vs. Placebo-MAST) are listed in Table 1. As indicated by McDonald's ω coefficients, internal consistencies ranged between $\omega = .75$ and $\omega = .92$ and were remarkably similar across the MAST and the Placebo-MAST group.

To examine whether the stress induction procedure was successful, i.e., whether the MAST group demonstrated a marked stress response, while the Placebo-MAST group did not, one-sided Welch's *t*-tests were conducted for psychological and physiological state stress measures. In respect to all other variables, for which no group differences were assumed (e.g., personality traits), two-sided Welch's *t*-tests were performed. As depicted in Table 1, all psychological and physiological state stress measures differed significantly between the two groups in the expected direction (higher stress in the MAST group) with mostly moderate to large effect sizes (Cohen, 1992; uncorrected for multiple comparisons). The only exception were heart rate values, that did not differ significantly between both groups. In sum, these manipulation checks suggest that the stress induction procedure was successful and induced a marked stress response in the MAST group as compared to the Placebo-MAST group. This stress response became visible on psychological and physiological levels.

Spearman correlations among all study variables for the MAST and the Placebo-MAST group, respectively, are listed in Table 2 (for exact *p*-values see Supplementary Table S1). As expected, psychological and physiological stress indicators (i.e., state psychological stress ratings, absolute and change values of heart rate, blood pressure, alpha-amylase, and cortisol) were not significantly correlated with each other. Neuroticism was the only Big Five personality trait which significantly correlated with state psychological stress in both experimental groups ($\rho = .26$, $p = .028$ and $\rho = .34$, $p = .003$ in the MAST and the Placebo-MAST group,

respectively; uncorrected for multiple comparisons), while trait anxiety was only significantly correlated with state psychological stress in the Placebo-MAST group ($\rho = .27$, $p = .020$) and all other personality traits demonstrated no significant association.

The physiological stress indicators were all significantly positively inter-correlated with one exception (i.e., change in cortisol was negatively related to heart rate in the Placebo-MAST group, $\rho = -.25$, $p = .030$).

3.2. Psycho-physiological correspondence (H1)

Our first hypothesis proposes psychological and physiological stress indicators to show zero or only weak positive correlations. To test this assumption, we firstly conducted linear regression analyses with the latent state psychological stress factor as outcome variable, and one physiological variable and age, respectively, as predictor variables. These preliminary models using only one physiological predictor variable (i.e., Models 1-8) are presented in Table S2 of the OSM. Out of all physiological variables, only cortisol demonstrated a statistically significant relationship with psychological stress, while this applies only for the MAST group and only when considering uncorrected *p*-values (Model 4; $\beta = .21$, $p = .024$). Accordingly, Model 4 (MAST) showed the highest R^2 of .11 which was statistically significant with $F(2,71) = 4.38$; $p = .016$. In contrast, all other models were far away from being significant with a mean R^2 of .04. We then, secondly, ran two comprehensive models for each group, comprising all physiological (absolute or change, respectively) variables and age as predictors, and psychological stress as outcome (see Table 3). Absolute and change values of cortisol were the only significant predictors of psychological stress besides the covariate age, but again only in the MAST group and only when considering uncorrected *p*-values (Model 9; $\beta = .24$, $p = .016$, $R^2 = .14$; Model 10; $\beta = .21$, $p = .041$, $R^2 = .11$).

In sum, we found no significant correspondence between psychological and physiological stress indicators irrespective of whether stress was induced or not and irrespective of the physiological stress indicator considered. This supports our first hypothesis.

3.3. Moderators of the psycho-physiological correspondence (H2)

Our second hypothesis proposes that personality traits may moderate the psycho-physiological correspondence. To evaluate this presumption statistically, we conducted moderated regression analyses including all absolute or change indicators of physiological stress, respectively (i.e., the four variables heart rate, blood pressure, alpha-amylase, and cortisol), the covariate age, the respective trait of interest (i.e., anxiety, openness, conscientiousness, neuroticism, extraversion, agreeableness, and intelligence) as well as the interaction terms between the respective trait of interest and all physiological variables (Models 11-24). Respective results are listed in Table 4 and Supplementary Table S3. Specifically, while main effects of personality traits and the exact moderation terms are listed in Table 4, main effects of the four physiological variables and of the covariate age are displayed in Table S3. Since the interactions and not the main effects were of interest to answer the moderation hypotheses, main effects are not discussed in detail in the following text. Note further, that as almost no moderation effect reached a p-value < .05 when correcting for the number of comparisons, effects whose uncorrected p-value reached statistical significance are also reported in the text.

At first, we expected lower trait anxiety to be related to higher psycho-physiological correspondence (H2a). This was true for the correspondence between psychological stress and blood pressure in the MAST group (Model 11; $\beta = -.24$, $p = .014$) as well as for the correspondence between psychological stress and alpha-amylase in the Placebo-MAST group (Model 11; $\beta = -.16$, $p = .014$). However, in the MAST group, we also identified a moderator effect of trait anxiety in the opposite direction as for alpha-amylase (Model 11; $\beta = .17$, $p = .038$). Overall, the R^2 of this Modell was .26 and the model was statistically significant with $F(10,62) = 2.16$; $p = .033$. No significant moderator effects for trait anxiety were observed in respect to the other physiological stress indicators (all $p > .05$; Table 3).

Second, we explored the relation between neuroticism and psycho-physiological correspondence as previous evidence was contradictory (H2b). Model 17 indicated a positive relation in the Placebo-MAST group only, i.e., higher neuroticism was associated with higher correspondence between psychological stress and absolute heart rate (Model 17; $\beta = .15$, $p = .031$).

Third, we expected higher openness to be related to higher psycho-physiological correspondence (H2c). In line with this hypothesis, higher openness was positively associated with higher correspondence between psychological stress and absolute alpha-amylase (Model 13; $\beta = .37$, $p = .002$, $p_{adj} = .011$) as well as to higher correspondence between psychological stress and the change in alpha-amylase (Model 14; $\beta = .34$, $p = .009$). Both such moderation effects apply to the MAST group only, meaning after stress was induced. Contrary to our hypothesis, higher openness was associated with lower correspondence between psychological stress and absolute cortisol in the MAST group (Model 13; $\beta = -.26$, $p = .009$), while no significant moderation effects were observed for the Placebo-MAST group and in respect to the other physiological indicators (all $p > .05$; Table 3). Overall, Model 13 (MAST) reached statistical significance with $R^2 = .31$ ($F(10,62) = 2.74$; $p = .007$).

Fourth, we proposed higher conscientiousness to be related to higher psycho-physiological correspondence (H2d). Supporting this hypothesis, conscientiousness was associated with higher correspondence between psychological stress and absolute blood pressure (Model 15; $\beta = .23$, $p = .021$) as well as to a higher correspondence between psychological stress and the change in cortisol (Model 16; $\beta = .29$, $p = .003$, $p_{adj} = .017$). Again, both were true for the MAST group only, i.e., after stress was induced. Contrary to our expectations, conscientiousness was associated with lower correspondence between psychological stress and the change in alpha-amylase in the MAST group (Model 16; $\beta = -.32$, $p = .002$, $p_{adj} = .015$). Overall Model 16 (MAST) reached a R^2 of .31 which was statistically significant with $F(10,62) = 2.8$ ($p = .006$).

Finally, we explored extraversion, agreeableness and intelligence as potential moderators of the psycho-physiological correspondence. For extraversion and intelligence, not any moderation effect reached statistical significance (see Models 19, 20, 23, and 24 in Table 4). For agreeableness, only one association was statistically significant: Higher agreeableness was related to a higher correspondence between psychological stress and absolute heart rate in the MAST group (Model 21; $\beta = .29$, $p = .011$).

3.4. Post-hoc analyses: A composite score of physiological stress

To investigate whether a composite score of physiological stress might yield additional insights into psycho-physiological correspondence, we repeated all analyses using aggregated absolute and change physiological stress scores, respectively. Note that this analysis was considered post-hoc, i.e., it was not pre-registered. Descriptive statistics are listed in Table S4, and correlations to all other study variables are displayed in Table S5. Notably and in clear contrast to the psychological stress variables, the physiological stress variables showed unsatisfying reliability coefficients ($\omega = .12$ to $\omega = .45$). This suggests low internal consistency of the four indicators heart rate, blood pressure, alpha-amylase and cortisol irrespective of whether stress was induced or not. Moderation effects between traits of interest and the psycho-physiological correspondence considering this composite score of physiological stress are presented in Table S6 (Models 25 to 40). Across all models, no single moderation effect reached statistical significance.

4. **Discussion**

Although a correspondence between psychological and physiological stress indicators might be advantageous from an evolutionary perspective (Levenson et al., 1994) and was proposed as relevant for physical and mental health (Sommerfeldt et al. (2019), previous research suggests rather low or non-existent correspondence between psychological and physiological indicators of acute stress (Campbell & Ehlert, 2012, Russell, 2003). The current

study provides a comprehensive test of this correspondence. It closes the gaps of previous research (a) by applying a stress induction procedure that comprises exposure to rather high psychological *and* physiological stressors as well as a control condition in which no stress was induced and (b) by considering four physiological indicators of stress (blood pressure, heart rate, alpha-amylase, cortisol) as well as multiple personality traits (trait anxiety, the Big Five, intelligence) as potential moderator variables. This experimental design allowed determining whether the correspondence and the hypothesized moderator effects will emerge under considerably high stress and under mild stress as well as with respect to multiple stress indicators. Overall, no significant correspondence between physiological and psychological stress variables was observed, however, conscientiousness and openness emerged as significant moderators requiring further investigation.

4.1. Study design allows comparing responses to high versus mild acute stress

With the exception of heart rate, all psychological and physiological stress measures differed significantly between the two groups in the expected direction (higher stress in the MAST group), which strongly suggests that the stress manipulation was successful. Although heart rate differed also in the expected direction between both groups, this difference did not reach statistical significance. A potential reason for this could be that compared to common references for resting-state heart rate (Ostchega et al., 2011) and heart rate following stress induction (Arza et al., 2008; Jaime et al., 2022), the heart rate values of our Placebo group seemed unexpectedly high, most likely due to the per-se at least as mildly stressful perceived laboratory situation. Why this is only reflected in heart rate values and not in the other stress indicators, could be explained by the possibility that heart rate may more directly mirror a general anticipatory arousal response, that is independent from specific psychosocial stress responses (e.g., Engert et al., 2017; Petrakova et al., 2015), or by the possibility that stress-induced heart reactions reach their maximum faster as compared to other indicators so that no further increase is possible after passing a certain value (e.g., Engert et al. 2017; Nater et al.

2006; Petrakova et al. 2015). Nevertheless, it is noteworthy to highlight that all other indicators demonstrated a significant stress response with mostly moderate to large effect sizes (Cohen, 1992) – an important prerequisite to validly study the psycho-physiological correspondence of stress markers that has not been explicitly considered by previous studies (Sommerfeldt et al., 2019).

4.2. No psycho-physiological correspondence at the group-average level (H1)

In accordance with previous research and in line with our first hypothesis, we did not find any significant correspondence between psychological stress and the four physiological stress indicators after controlling for multiple testing. With regard to unadjusted *p*-values, cortisol was the sole physiological stress indicator demonstrating a significant correlation with psychological stress - in the MAST group only. This is at least partially in accordance with the meta-analysis of Campbell and Ehlert (2012) who observed significant correlations between cortisol and psychological stress indicators in 25% of the studies involving the TSST. At least in respect to our study, one may speculate that this cortisol-specificity of the correspondence may result from a closer timing of both assessments, as psychological stress was measured directly after the MAST (10 min after the acute stress induction started) and the cortisol response requires about 10 minutes until reaching its maximum (Smeets et al., 2012). Further, cortisol release is modulated by the activity of the HPA axis, which contrasts with the other obtained physiological stress indicators reflecting SAM activity. However, as the observed association did not pass the corrected significance threshold, and these potential explanations were considered post-hoc, further research including larger samples is essentially required to elucidate the potential cortisol-specificity (or HPA axis-specificity) of the observed effect.

Further, it is noteworthy that the uncorrected cortisol-specific effect refers only to models testing for psycho-physiological correspondence in the MAST group, while the models considering the Placebo-MAST group performed descriptively worse and did not show any significant effect. This suggests the induction of a marked stress response on a group level as

an important prerequisite for validly studying psycho-physiological correspondence. On the one hand, physiological stress symptoms below a certain threshold might be difficult to notice (e.g., a heart rate increase), and thus can hardly affect self-reported (psychological) stress ratings. On the other hand, psycho-physiological correspondence is evolutionary most functional under high stress, as physiological and psychological stress reactions running in parallel increase the probability to survive and take respective actions (see also Engert et al. 2017 for a similar reasoning).

4.3. Psycho-physiological correspondence is moderated by personality traits (H2)

In our second hypothesis, we proposed that individual differences in basic personality traits are linked to the question of whether psychological and physiological stress indicators align, i.e., whether psycho-physiological correspondence can be observed. To this aim, the Big Five personality factors, trait anxiety and general intelligence were tested as potential moderators for the correspondence between psychological stress and four indicators of physiological stress (absolute and change scores) in the MAST and the Placebo-MAST group (i.e., a total of 7 x 4 x 2 x 2 = 112 tests).

Before controlling for multiple testing, eleven significant interactions were observed out of which six were in the expected direction and three passed the corrected *p*-threshold (two in the expected direction): In line with our hypotheses conscientiousness and openness showed four significant interaction effects indicating higher conscientiousness/openness to be related to higher psycho-physiological correspondence. Two out of these effects stayed significant even after controlling for multiple testing. These two personality traits, however, also showed two interaction effects in the opposite direction out of which one survived the correction for multiple testing. A further contra-hypotheses interaction effect was identified for neuroticism (higher correspondence in persons scoring higher in neuroticism) while two expected effects (higher correspondence in persons scoring higher trait anxiety) and one effect in the opposite direction emerged for trait anxiety as well as one unexpected effect for agreeableness (higher

correspondence in persons with lower agreeableness scores). Of note, all effects regarding neuroticism, trait anxiety and agreeableness did not pass the significance threshold when correcting for multiple testing. Finally, no effects were observed for extraversion and general intelligence.

Regarding the four investigated physiological stress indicators blood pressure, heart rate, alpha-amylase and cortisol, the latter two endocrinological markers were involved in the three interaction effects that stayed significant after controlling for multiple testing. Specifically, the correspondence between psychological stress ratings and the absolute value of alpha-amylase was moderated by openness (in the hypothesized direction: higher correspondence – higher openness), while the correspondence between psychological stress and the alpha-amylase change score was moderated by conscientiousness (in the unexpected direction: higher correspondence – lower conscientiousness). The third significant moderation effect was again in line with our hypotheses and also observed for conscientiousness but with respect to cortisol change scores (higher correspondence – higher conscientiousness). Although Taliç et al. (2023) addressed (rather mild) everyday life stressors, the latter effect corroborates their observation that conscientiousness moderated the correspondence between psychological stress and heart rate variability. The higher body awareness of persons scoring higher on conscientiousness (Ferentzi et al., 2017) might provide a potential explanation and would align with the well-documented ability of conscientiousness to predict physical and mental well-being (Ozer & Benet-Martinez, 2006; Soto, 2019) as well as with first reports suggesting that psycho-physiological correspondence is positively associated with important well-being indicators (Sommerfeldt, et al., 2019). However, we did not expect to observe an opposite effect with respect to alpha-amylase change scores. Potentially, this could be explained by the fact that cortisol changes reflect an increase in HPA activity (Tsigos & Chrousos, 2002) and alpha-amylase indicates reactivity of the SAM axis (Rohleder et al., 2004), while both stress axes differ in their timing and, most importantly, potentially also in which direction higher

conscientiousness influences their responsivity. Specifically, it can be speculated that higher conscientiousness facilitates the alignment of psychological stress ratings and HPA reactivity, while it may reduce the alignment between psychological stress ratings and SAM responses for which alpha-amylase might be a more direct marker compared to heart rate and blood pressure for which no effects were observed. However, this very vague interpretation was proposed post-hoc and has to be tested in future studies comprehensively.

Finally, the significant interaction effect of openness, indicating higher correspondence between psychological stress and absolute alpha-amylase in persons scoring higher on openness was again in the expected direction. We hypothesized this direction before based on former research demonstrating higher openness to be related to higher body awareness. However, if our above-outlined interpretation of the contradictory effect of alpha-amylase change scores in respect to conscientiousness holds true, this finding would reveal an important distinction between the consideration of change versus absolute scores of alpha-amylase that has to be taken into account in future research.

A final note refers to the fact that nine out of the 11 uncorrected significant effects were observed in the MAST group only, i.e., after exposure to marked psychological (social-evaluative) and physiological (cold water) stress. This applies also to all three interaction effects that survived the correction for multiple testing. Thus, it seems that only under a certain amount of stress, personality factors come into play (conscientiousness and openness) and align psychological and physiological stress responses. Personality theories which do *not* conceptualize traits as fully decontextualized constructs provide potential interpretations for this observation. For example, the Cybernetic Big Five Theory (CB5T; DeYoung, 2015) assumes that traits are conditional on the presence of specific classes of stimuli. Similarly, Trait Activation Theory (TAT; Tett et al., 2021) posits that individual differences in personality traits are only expressed in thoughts, feelings and especially behaviors when triggered by trait-relevant situational cues, e.g., it is in social situations when it becomes evident who is sociable.

When we consider psycho-physiological correspondence as a trait-like phenomenon, high stress might present the class of stimuli or trait-relevant situation for individual differences in the correspondence (inclusively their moderation by further personality factors) to become visible. Although this has to be further investigated in future studies, we speculate that personality-related individual differences in interoception in general, and in particular in the ability to notice and become aware of bodily stress symptoms might play a critical role.

Limitations and Future Directions

Overall, the results of our study are mixed: While H1 was confirmed, the evidence for H2 differed with respect to the physiological indicators, the personality traits proposed as moderators and the direction of the interaction effects. Thus, and from a purely calculative perspective only 2 out of 80 hypotheses regarding the potential moderating effects of personality traits on the psycho-physiological correspondence were supported after correcting for multiple testing and 9 out of 80 hypotheses were confirmed when considering uncorrected *p*-values. Thus, most of our hypotheses were actually *not* supported. At the same time, it would be premature to conclude that personality traits do not moderate the psychophysiological correspondence at all. Rather our study showcases that potential moderating effects of personality traits may depend on (a) the respective trait, (b) the measures for psychological and physiological stress, (c) the intensity, duration and type of the stress and (d) the calculation of the psycho-physiological correspondence (within or between subjects). Therefore, we recommend future studies to evaluate these factors (as well as their combinations) systematically to allow for more specific hypotheses in future research. For example, more specific personality traits or trait-like skills such as body awareness or interceptive abilities that are more directly related to the perception of physiological stress symptoms should be included. In addition, the notion of psycho-physiological correspondence implicitly assumes an association between all kinds of psychological stress and all kinds of physiological stress markers. However, this assumption is much too broad and undifferentiated both in terms of the

psychological stress measure and in terms of the physiological stress markers. One and the same score on a psychological stress scale could stem from or be associated with different emotions (e.g., anxiety, anger, disgust) and it seems therefore reasonable or even mandatory for future studies to operationalize the construct of psycho-physiological correspondence in a much more fine-grained way - both on the psychological and on the physiological side. This would result in several correspondence types between specific self-reported stress-related emotions and specific physiological stress indicators, each to be considered separately and to be tested for potential moderation effects of personality traits. Since our study was the first one to investigate the psycho-physiological convergence in a stress and in a placebo condition and because we found at least some preliminary evidence that the correspondence was more pronounced under stress, it also seems worthwhile to investigate such different types of psycho-physiological correspondences in different more or less stressful contexts over shorter and longer time periods. Another and more profound issue is related to the question whether the assumption of high evolutionary functionality of psycho-physiological correspondence (Levenson et al., 1994) is valid under all circumstances. For example, rescue workers in both civil and military contexts may experience high levels of physiological stress but may have to suppress the respective signals on the psychological level in order to complete a necessary and life-saving emergency medical procedure.

Finally, the suggested avenues for future research should be complemented by intensive longitudinal studies focused on single participants (i.e., case studies). Such idiographic investigations allow detecting potential idiosyncratic correspondence patterns: For some individuals there may be a correspondence with regard to certain psycho-psychological pairings (e.g., psychological stress and cortisol) but not for other pairings (e.g., psychological stress and heart rate) and there may be individuals for whom there is no correspondence at all. Resulting idiosyncratic correspondence types may then be aggregated to clusters across individuals showing similar patterns and can be investigated further, for instance, with regard to relations

with personality traits. To conclude, we recommend future studies to (a) consider a broad range of both psychological and physiological stress indicators, (b) to investigate specific types of psycho-physiological correspondence resulting from certain pairings of indicators, (c) to include concepts reflecting interoceptive abilities and body awareness, (d) to systematically vary the intensity and duration of stressors, and (e) to combine large-sample nomothetic approaches with longitudinal idiographic research.

Implications and Conclusion

This study is the first to comprehensively investigate the potential moderating role of basic personality traits on the correspondence between psychological stress and multiple physiological stress markers. In extension to previous research, we used an experimental paradigm that included both the induction of psychological and physiological stress (the MAST) as well as a control condition in which no stress was explicitly induced (the Placebo-MAST). Although most of the hypothesized moderations failed to reach statistical significance, and some interaction effects turned out to contradict our hypotheses, our study presents promising starting points for future research and reveals that the specific situational circumstances as well as participants' personality traits need to be taken into account when studying the psycho-physiological correspondence of stress indicators. For future research, we propose that (a) the consideration of more specific personality traits or trait-like skills with regard to different corresponding pairings of stress-specific emotions and specific physiological indicators, (b) the systematic variation of stress intensity and laboratory vs. real life contexts, and (c) the combination of nomothetic and idiographic analyses as promising avenues to disentangle the "mystery of the missing psychophysiological correspondence".

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Conflict of Interest Disclosure

The authors declare no conflicts of interest.

Ethics Approval Statement

All experimental procedures were in accordance with the declaration of Helsinki and approved by the local ethics committee (Psychological Institute, University of Würzburg, Germany, GZEK 2020-18). Informed written consent according to the declaration of Helsinki and to the guidelines of the German Psychological Society was obtained from all participants.

CRediT authorship contribution statement

Kirsten Hilger: Conceptualization, Methodology, Formal Analysis, Project Administration, Writing – Original Draft, Supervision, Funding acquisition. **Irma Talic:** Conceptualization, Formal Analyses, Writing – Original Draft. **Karl-Heinz Renner:** Conceptualization, Writing – Original Draft, Supervision, Funding.

Supplementary Material

Supplementary Material associated with this article can be found in a separate document.

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Table 1

Descriptive statistics and difference tests for MAST and Placebo-MAST groups

	MAST				Placebo-MAST				Welch's *t*-test			
	M	*SD*	Min, Max	ω	*M*	*SD*	Min, Max	ω	*t*	df	*p*	*d*
Age	24.51	3.73	18, 35	--	25.04	4.13	18, 34	--	0.82	145.82	.416	0.13
Psychological variables												
State psychological stress^{m}	0.61	0.83	-0.97, 2.86	--	-0.60	0.57	-1.19, 1.88	--	-10.29**	129.04	<.001	1.69
Trait-Anxiety	2.13	0.30	1.30, 2.80	.91	2.12	0.28	1.65, 2.94	.92	-0.13	145.57	.899	0.02
Neuroticism	1.44	0.63	0.25, 3.17	.87	1.55	0.64	0.08, 2.92	.88	1.06	147	.292	0.17
Openness	2.73	0.47	1.33, 3.75	.75	2.74	0.50	1.50, 3.83	.78	0.12	146.68	.908	0.02
Conscientiousness	2.56	0.71	0.83, 4.00	.91	2.59	0.63	1.08, 3.83	.90	0.26	144.55	.796	0.04
Extraversion	2.33	0.58	1.08, 3.75	.86	2.46	0.61	0.92, 3.58	.88	1.29	146.75	.201	0.21
Agreeableness	2.56	0.56	1.10, 3.67	.81	2.65	0.55	1.36, 4.00	.80	1.03	146.86	.304	0.17
Intelligence	27.54	4.66	14.00, 35.00	--	26.29	4.27	17.00, 36.00	--	-1.70	145.48	.091	0.28
Physiological variables												
HR_{change}^{m}	-5.65	10.18	-41.36, 17.00	--	0.70	14.00	-50.00, 64.50	--	3.17	135.19	.999	0.52
HR_{abs}^{m}	76.00	14.35	46.00, 120.95	--	80.68	18.05	51.00, 123.00	--	1.75	140.64	.959	0.29
BP_{change}^{m}	4.21	6.71	-10.27, 19.69	--	-1.96	7.16	-23.84, 20.50	--	-5.42**	146.63	<.001	0.89
BP_{abs}^{m}	106.97	9.25	81.83, 124.95	--	100.47	8.82	74.65, 120.89	--	-4.39**	146.47	<.001	0.72
AA_{change}^{m}	31.80	50.60	-56.80, 157.73	--	-3.19	43.45	-128.11, 134.28	--	-4.48**	140.78	<.001	0.74
AA_{abs}^{m}	128.26	107.77	7.15, 472.16	--	92.55	94.65	4.42, 453.85	--	-2.13*	141.64	.018	0.35
$Cortisol_{change}^{m}$	5.64	6.43	-9.79, 23.67	--	-1.44	3.08	-10.94, 4.73	--	-8.55**	104.49	<.001	1.40
$Cortisol_{abs}^{m}$	10.88	6.41	1.27, 25.49	--	4.20	2.62	0.48, 13.76	--	-8.32**	96.56	<.001	1.37

Note. ^{m} = psychological and physiological variables that capture the stress reaction following the experimental manipulation in the MAST group and are thus assumed to differ between groups (i.e., manipulation check). Statistical significance of group differences was assessed with Welch's *t*-tests. For variables assumed to differ between groups, Welch's *t*-tests were conducted one-sided, while all other variables were tested two-sided. Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score), that is, a positive value indicates an increase in stress following experimental manipulation, while a negative value indicates a decrease in stress. Note that while this table presents values in their original measurement scale, all variables were z-standardized before entering statistical regression models, testing our main hypotheses. Sample size (*N*) of the MAST group = 74; *N* of the Placebo-MAST group = 75. * = significant at $p < .05$, ** = significant at $p < .001$ (uncorrected for multiple comparisons). HR = heart rate; BP = blood pressure; AA = alpha amylase.

Table 2

Correlations between all study variables

Variable	1	2	3	4	5	6	7	8	9
1. State psychological stress	---								
2. Openness	.00 /.07	---							
3. Conscientiousness	.07 / -.11	-.16 / .08	---						
4. Extraversion	-.05 / -.12	.22 / .21	.21 / .18	---					
5. Agreeableness	-.15 / .02	.15 / .05	-.18 / .17	.33* / .36*	---				
6. Neuroticism	.26* / .34*	-.10 / .01	-.42** / -.29*	-.27*/ .40**	-.14 / -.34*	---			
7. Trait-Anxiety	.15 / .27*	.05 / .16	-.15 / -.22	.20 / .01	-.20 / -.13	.16 / .40**	---		
8. Intelligence	-.08 / .15	.14 / -.09	-.03 / -.06	-.09 / .17	.15 / .22	-.21 / -.19	-.20 / -.12	---	
9. HR_{change}	.05 / -.01	-.11 / .04	.02 / -.18	-.07 / -.04	-.21 / .09	.02 / .10	-.05 / -.05	.10 / -.02	---
10. HR_{abs}	.10 / .13	.08 / .08	-.13 / -.30*	-.02 / -.07	.06 / -.04	.30* / .08	.14 /.19	-.17 / .20	.03 / .26*
11. BP_{change}	-.13 / .02	.00 / .05	-.02 / .11	-.06 / -.02	-.08 / -.01	.01 / -.22	-.32* / -.08	.20 / .07	.28* / .07
12. BP_{abs}	.08 / -.09	.16 / .03	.11 / -.11	.07 /-.13	-.22 / .01	.01 / -.08	-.07 / -.02	-.04 / -.03	.21 / .19
13. AA_{change}	.03 / -.05	-.08 / -.04	.07 / .01	.00 / .16	.10 / .01	-.18 / -.19	-.15 / .10	.12 / .11	-.01 / -.31*
14. AA_{abs}	-.04 / -.07	.04 / .04	.12 / .10	-.05 / .27*	.06 / .17	-.21 / -.11	-.24* / .03	.12 / .16	-.02 / .02
15. $Cortisol_{change}$	.13 / -.13	.02 / .02	.09 / .35*	-.04 / -.09	.08 / -.19	-.08 / -.02	-.21 / -.15	.07 / -.26*	.07 / -.25*
16. $Cortisol_{abs}$	.17 / .19	.12 / .10	.04 / -.09	-.02 / .08	.13 / .01	-.03 / .06	-.27* / -.17	.06 / -.09	.08 / .14

Note. The table lists Spearman's rank-order correlation coefficients ρ between all study variables. Coefficients before the slash (/) represent correlations in the MAST group ($N = 74$), while coefficients after the slash indicate correlations in the Placebo-MAST group ($N = 75$). Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score). * = significant at $p < .05$, ** = significant at $p < .001$ (uncorrected for multiple comparisons; for exact p-values see Supplementary Table S1). HR = heart rate; BP = blood pressure; AA = alpha amylase.

Table 2 (continued)

Correlations between all study variables

Variable	10	11	12	13	14	15	16
10. HR_{abs}	---						
11. BP_{change}	-.10 / -.05	---					
12. BP_{abs}	.02 / .03	.43**/.49**	---				
13. AA_{change}	-.07 / .13	.09 / .00	.18 / .00	---			
14. AA_{abs}	.01 / .26*	.17 / -.11	.29* /.08	.55**/.26*	---		
15. $Cortisol_{change}$	-.20 / -.25*	.25* / -.03	.20 / -.21	.30* / .08	.48**/ -.05	---	
16. $Cortisol_{abs}$	-.14 / .16	.21 / .07	.29* / -.03	.23* / -.14	.41**/ -.09	.78**/ .12	---

Note. The table lists Spearman's rank-order correlation coefficients ρ between all study variables. Coefficients before the slash (/) represent correlations in the MAST group ($N = 74$), while coefficients after the slash indicate correlations in the Placebo-MAST group ($N = 75$). Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score). * = significant at $p < .05$, ** = significant at $p < .001$ (uncorrected for multiple comparisons; for exact *p*-values see Supplementary Table S1). HR = heart rate; BP = blood pressure; AA = alpha amylase.

Table 3

Multiple regression models on psycho-physiological correspondence (H1)

	State psychological stress (MAST)					State psychological stress (placebo-MAST)				
	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)
Model 9					.14 (.067)					.08 (.357)
HR_{abs}	.15 (0.11)	.169	.339	[-.07, .37]		.10 (0.06)	.102	.246	[-.02, .23]	
BP_{abs}	.00 (0.10)	.963	.963	[-.21, .20]		.03 (0.07)	.649	.779	[-.11, 18]	
AA_{abs}	-.11 (0.10)	.273	.413	[-.30, .09]		-.08 (0.08)	.275	.413	[-.23, .07]	
$Cortisol_{abs}$	.24 (0.10)	.016*	.065	[.05, .44]		.05 (0.16)	.780	.851	[-.28, .37]	
Age	-.22 (0.10)	.039*	.117	[-.43, -.01]		-.06 (0.07)	.393	.524	[-.20, .08]	
Model 10					.11 (.149)					.06 (.556)
HR_{change}	.05 (0.12)	.702	.766	[-.20, .29]		-.01 (0.06)	.882	.882	[-.13, .11]	
BP_{change}	-.13 (0.11)	.271	.464	[-.35, .10]		.03 (0.07)	.693	.766	[-.12, .17]	
AA_{change}	-.09 (0.10)	.364	.515	[-.29, .11]		-.07 (0.08)	.386	.515	[-.23, .09]	
$Cortisol_{change}$	.21 (0.10)	.041*	.122	[.01, .41]		-.16 (0.14)	.257	.464	[-.44, .12]	
Age	-.24 (0.11)	.032*	.122	[-.45, -.02]		-.08 (0.07)	.228	.464	[-.21, .05]	

Note. Absolute scores (indicated by the subscript abs) represent stress scores after experimental stress induction (MAST) or time-corresponding values in the control group, respectively (Placebo-MAST). Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score). * = significant at $p < .05$, ** = significant at $p < .001$. HR = heart rate; BP = blood pressure; AA = alpha amylase; β = standardized regression coefficient; *SE* = standard error; p_{adj} = Benjamini-Hochberg corrected *p*-values.

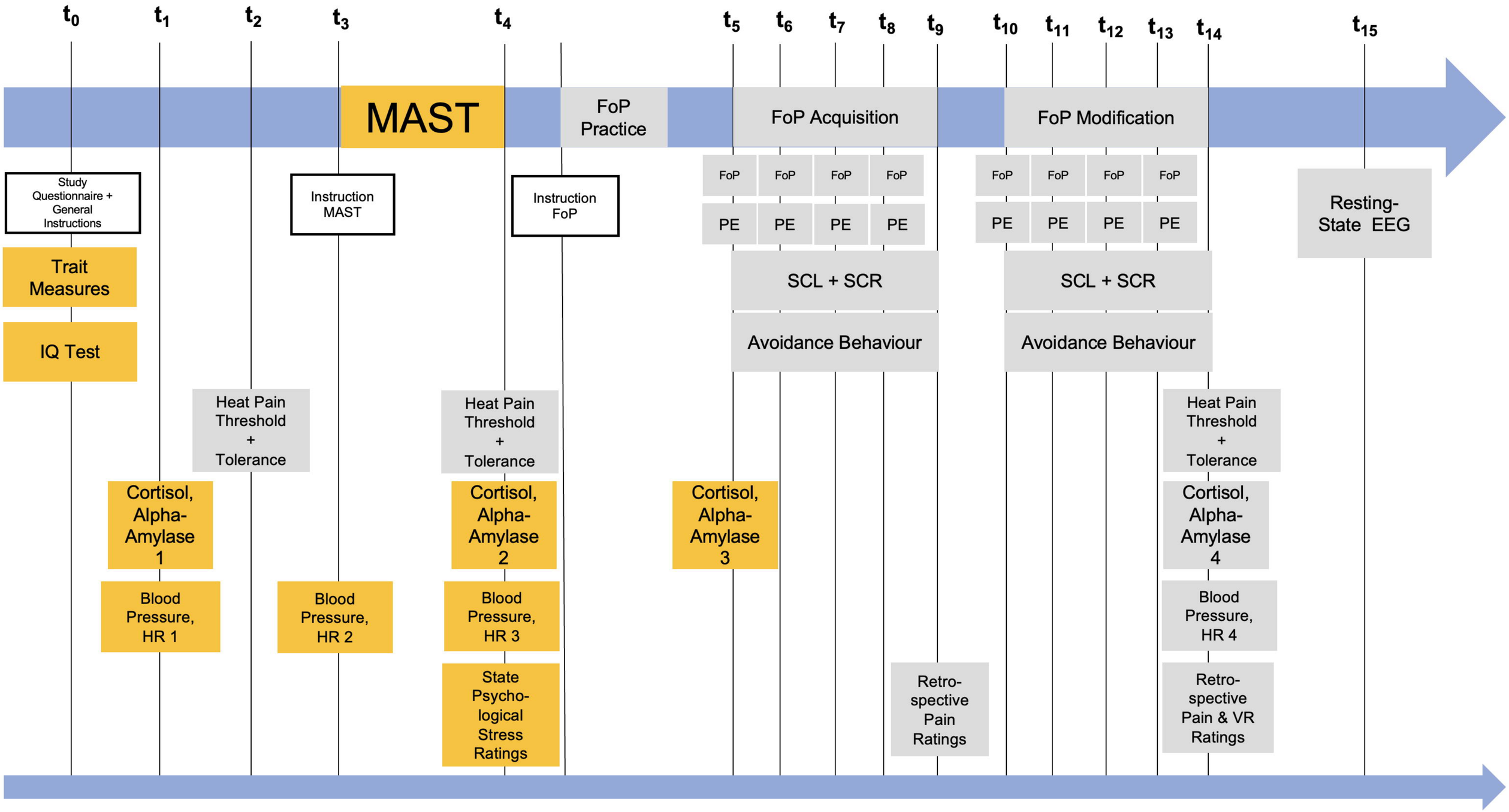
Table 4

Multiple regression models testing moderators of psycho-physiological correspondence (H2)

	State psychological stress (MAST)					State psychological stress (placebo-MAST)				
	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)
Model 11					.26* (.033)					.22 (.086)
Anx	.20 (0.12)	.111	.272	[-.05, .44]		.13 (0.14)	.380	.464	[-.16, .42]	
Anx*HR_{abs}	-.08 (0.13)	.263	.386	[-.34, .18]		.08 (0.07)	.130	.278	[-.06, .22]	
Anx*BP_{abs}	-.24 (0.11)	.014*	.061	[-.46, -.03]		.10 (0.08)	.085	.235	[-.05, .25]	
Anx*AA_{abs}	.17 (0.09)	.038*	.140	[-.02, .36]		-.16 (0.07)	.014*	.061	[-.31, -.02]	
Anx*$Cortisol_{abs}$	-.11 (0.10)	.139	.278	[-.31, .09]		-.15 (0.19)	.220	.354	[-.53, .24]	
Model 12					.13 (.548)					.20 (.133)
Anx	.03 (0.14)	.821	.903	[-.25, .31]		.39 (0.13)	.003*	.021*	[.14, .64]	
Anx*HR_{change}	-.05 (0.15)	.368	.495	[-.34, .24]		.06 (0.07)	.175	.427	[-.07, .20]	
Anx*BP_{change}	.00 (0.14)	.500	.611	[-.27, .27]		.13 (0.08)	.068	.214	[-.04, .30]	
Anx*AA_{change}	.07 (0.11)	.280	.449	[-.16, .29]		-.07 (0.07)	.167	.427	[-.21, .07]	
Anx*$Cortisol_{change}$	-.08 (0.09)	.203	.446	[-.26, .11]		.27 (0.16)	.050	.184	[-.05, .60]	
Model 13					.31* (.007)					.10 (.721)
O	.03 (0.11)	.753	.828	[-.18, .25]		-.01 (0.13)	.928	.928	[-.26, .24]	
O*HR_{abs}	-.02 (0.10)	.411	.573	[-.22, .18]		.05 (0.07)	.213	.468	[-.08, .19]	
O*BP_{abs}	-.01 (0.13)	.481	.623	[-.27, .25]		.07 (0.08)	.194	.468	[-.10, .24]	
O*AA_{abs}	.37 (0.12)	.002*	.011*	[.13, .61]		-.11 (0.15)	.241	.471	[-.41, .19]	
O*$Cortisol_{abs}$	-.26 (0.11)	.009*	.051	[-.48, -.05]		-.08 (0.16)	.310	.487	[-.39, .23]	
Model 14					.22 (.089)					.08 (.850)
O	.03 (0.12)	.773	.810	[-.20, .26]		.09 (0.11)	.428	.524	[-.14, .31]	
O*HR_{change}	.15 (0.17)	.187	.479	[-.18, .48]		-.04 (0.10)	.327	.479	[-.24, .15]	
O*BP_{change}	-.22 (0.13)	.051	.224	[-.48, .04]		-.03 (0.07)	.321	.479	[-.17, .11]	
O*AA_{change}	.34 (0.14)	.009*	.067	[.06, .62]		.05 (0.09)	.281	.479	[-.12, .23]	
O*$Cortisol_{change}$	-.05 (0.10)	.308	.479	[-.26, .16]		.03 (0.12)	.411	.524	[-.21, .26]	
Model 15					.23 (.078)					.13 (.513)
C	.04 (0.11)	.746	.782	[-.18, .25]		-.15 (0.17)	.376	.567	[-.49, .19]	
C*HR_{abs}	.03 (0.08)	.347	.567	[-.14, .20]		.13 (0.09)	.069	.302	[-.04, .30]	
C*BP_{abs}	.23 (0.11)	.021*	.150	[.01, .45]		-.13 (0.10)	.096	.310	[-.32, .07]	
C*AA_{abs}	-.03 (0.12)	.413	.567	[-.27, .21]		-.03 (0.09)	.362	.567	[-.21, .14]	
C*$Cortisol_{abs}$	.03 (0.11)	.387	.567	[-.18, .24]		-.25 (0.22)	.135	.328	[-.69, .20]	
Model 16					.31* (.006)					.09 (.799)
C	.02 (0.11)	.858	.858	[-.20, .24]		.12 (0.13)	.366	.537	[-.15, .39]	
C*HR_{change}	.03 (0.09)	.383	.537	[-.15, .21]		.07 (0.08)	.187	.411	[-.09, .24]	
C*BP_{change}	.20 (0.12)	.050	.157	[-.04, .43]		-.03 (0.12)	.415	.537	[-.26, .21]	
C*AA_{change}	-.32 (0.11)	.002*	.015*	[-.53, -.10]		.03 (0.10)	.368	.537	[-.17, .24]	
C*$Cortisol_{change}$	.29 (0.10)	.003*	.017*	[.08, .49]		.19 (0.19)	.167	.408	[-.20, .57]	
Model 17					.18 (.227)					.20 (.156)
N	.12 (0.12)	.312	.651	[-.12, .36]		.03 (0.13)	.818	.867	[-.24, .30]	
N*HR_{abs}	.06 (0.12)	.622	.805	[-.17, .29]		.15 (0.07)	.031*	.171	[.01, .29]	
N*BP_{abs}	-.10 (0.11)	.393	.686	[-.33, .13]		.04 (0.09)	.659	.805	[-.14, .21]	
N*AA_{abs}	-.03 (0.13)	.827	.867	[-.28, .23]		-.04 (0.10)	.723	.837	[-.24, .17]	
N*$Cortisol_{abs}$	.10 (0.12)	.411	.686	[-.14, .33]		-.13 (0.17)	.437	.686	[-.46, .20]	

	State psychological stress (MAST)					State psychological stress (placebo-MAST)				
	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)	β (*SE*)	*p*	p_{adj}	95% CI	R^2 (*p*)
Model 18					.17 (.246)					.14 (.430)
N	.26 (0.13)	.046*	.337	[.00, .52]		.20 (0.13)	.114	.551	[-.05, .45]	
N*HR_{change}	.07 (0.09)	.478	.741	[-.12, .25]		.08 (0.10)	.410	.709	[-.12, .29]	
N*BP_{change}	-.14 (0.13)	.289	.707	[-.40, .12]		-.05 (0.08)	.539	.741	[-.20, .11]	
N*AA_{change}	.10 (0.12)	.419	.709	[-.14, .34]		.03 (0.09)	.757	.833	[-.16, .21]	
N*$Cortisol_{change}$	-.08 (0.12)	.520	.741	[-.33, .17]		.10 (0.20)	.612	.771	[-.30, .51]	
Model 19					.25* (.037)					.13 (.521)
E	-.01 (0.10)	.887	.892	[-.21, .19]		.06 (0.14)	.662	.890	[-.21, .33]	
E*HR_{abs}	.21 (0.12)	.082	.224	[-.03, .46]		-.08 (0.07)	.274	.547	[-.23, .07]	
E*BP_{abs}	.04 (0.10)	.718	.890	[-.17, .25]		.03 (0.07)	.687	.890	[-.11, .17]	
E*AA_{abs}	.13 (0.09)	.149	.364	[-.05, .31]		-.02 (0.08)	.805	.890	[-.19, .15]	
E*$Cortisol_{abs}$	-.23 (0.13)	.081	.224	[-.49, .03]		.21 (0.17)	.227	.500	[-.14, .56]	
Model 20					.14 (.420)					.11 (.641)
E	-.06 (0.11)	.633	.918	[-.28, .17]		-.19 (0.12)	.113	.498	[-.43, 05]	
E*HR_{change}	-.06 (0.18)	.714	.918	[-.42, .29]		-.06 (0.06)	.329	.759	[-.18, .06]	
E*BP_{change}	-.11 (0.14)	.436	.799	[-.40, .17]		-.02 (0.08)	.807	.918	[-.17, .13]	
E*AA_{change}	-.03 (0.10)	.755	.918	[-.23, .17]		-.05 (0.09)	.551	.918	[-.23, .12]	
E*$Cortisol_{change}$	-.02 (0.12)	.878	.918	[-.26, .22]		-.11 (0.12)	.367	.759	[-.35, .13]	
Model 21					.29* (.011)					.15 (.401)
A	-.22 (0.10)	.037*	.130	[-.43, -.01]		.20 (0.13)	.124	.302	[-.06, .46]	
A*HR_{abs}	.29 (0.11)	.011*	.081	[.07, .51]		-.12 (0.06)	.065	.179	[-.24, .01]	
A*BP_{abs}	.14 (0.11)	.230	.380	[-.09, .37]		.09 (0.08)	.242	.380	[-.06, .24]	
A*AA_{abs}	.14 (0.12)	.230	.380	[-.09, .37]		.06 (0.10)	.550	.672	[-.14, .26]	
A*$Cortisol_{abs}$	-.05 (0.11)	.672	.778	[-.26, .17]		.18 (0.17)	.287	.421	[-.15, .51]	
Model 22					.21 (.125)					.07 (.891)
A	-.08 (0.12)	.508	.840	[-.33, .16]		.06 (0.13)	.672	.840	[-.21, .33]	
A*HR_{change}	.25 (0.14)	.079	.348	[-.03, .54]		-.08 (0.08)	.314	.645	[-.24, .08]	
A*BP_{change}	-.19 (0.13)	.155	.567	[-.46, .07]		.03 (0.07)	.640	.840	[-.11, .17]	
A*AA_{change}	.06 (0.11)	.592	.840	[-.15, .27]		.01 (0.08)	.861	.947	[-.15, .18]	
A*$Cortisol_{change}$	.07 (0.11)	.535	.840	[-.15, .28]		.01 (0.16)	.973	.973	[-.31, .32]	
Model 23					.19 (.165)					.12 (.554)
Int	-.06 (0.11)	.618	.800	[-.28, .17]		.12 (0.13)	.368	.713	[-.14, .38]	
Int*HR_{abs}	-.10 (0.13)	.455	.714	[-.36, .16]		-.07 (0.08)	.409	.713	[-.23, .10]	
Int*BP_{abs}	.04 (0.12)	.752	.808	[-.20, .28]		.07 (0.09)	.421	.713	[-.10, .24]	
Int*AA_{abs}	-.02 (0.10)	.882	.882	[-.22, .19]		-.03 (0.09)	.757	.808	[-.20, .14]	
Int*$Cortisol_{abs}$	.14 (0.09)	.107	.394	[-.03, .32]		.11 (0.18)	.550	.800	[-.25, .46]	
Model 24					.17 (.288)					.11 (.657)
Int	-.03 (0.12)	.782	.882	[-.27, .21]		.00 (0.12)	.980	.980	[-.24, .25]	
Int*HR_{change}	.11 (0.14)	.431	.675	[-.17, .39]		.06 (0.08)	.460	.675	[-.09, .21]	
Int*BP_{change}	-.07 (0.14)	.632	.818	[-.36, .22]		.14 (0.09)	.136	.580	[-.05, .33]	
Int*AA_{change}	.14 (0.11)	.226	.580	[-.09, .36]		-.08 (0.08)	.317	.580	[-.24, .08]	
Int*$Cortisol_{change}$	.12 (0.14)	.411	.675	[-.17, .40]		-.05 (0.19)	.802	.882	[-.42, .32]	

Note. Absolute scores (indicated by the subscript abs) represent stress scores after experimental stress induction (MAST) or time-corresponding values in the control group, respectively (Placebo-MAST). Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score). Moderation effects for anxiety, openness and conscientiousness (Models 11-16) were directed such that one-sided *p*-values are reported. Moderation effects for neuroticism, extraversion, agreeableness and intelligence (Models 17 – 24) were undirected such that two-sided *p*-values are reported. Each model additionally contained the physiological variables (absolute or change values of HR, BP, AA, and cortisol, respectively) and age as predictor variables. For the sake of brevity, these main effects are not displayed here, but shown in Supplementary Table S3. Change scores (indicated by subscript) were calculated such that the baseline score was subtracted from the score following stress induction or Placebo, respectively (i.e., score after experimental manipulation – baseline score), that is, a positive value indicates an increase in stress following experimental manipulation, while a negative value indicates a decrease in stress. * = significant at $p < .05$, ** = significant at $p < .001$. HR = heart rate; BP = blood pressure; AA = alpha amylase; Anx = anxiety; O = openness; C = conscientiousness; N = neuroticism; E = extraversion; A = agreeableness; Int = intelligence; β = standardized regression coefficient; *SE* = standard error; p_{adj} = Benjamini-Hochberg corrected *p*-values.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MAST
FoP Practice
FoP Acquisition
FoP Modification
Study Questionnaire + General Instructions
Trait Measures
IQ Test
Instruction MAST
Instruction FoP
FoP
PE
SCL + SCR
Avoidance Behaviour
Resting-State EEG
Heat Pain Threshold + Tolerance
Cortisol, Alpha-Amylase 1
Cortisol, Alpha-Amylase 2
Cortisol, Alpha-Amylase 3
Cortisol, Alpha-Amylase 4
Blood Pressure, HR 1
Blood Pressure, HR 2
Blood Pressure, HR 3
Blood Pressure, HR 4
State Psycho-logical Stress Ratings
Retro-spective Pain Ratings
Retro-spective Pain & VR Ratings