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Is your stress my stress? A standardized randomized-controlled paradigm to study physiological stress contagion based on direct stress observation

Short title: A standardized stress contagion paradigm

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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 non-stress observing control group 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 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 ("non-stress observers", *n*=30). We repeatedly assessed heart rate, salivary alpha-amylase, salivary cortisol, as well as salivary aldosterone.

Results: Stress observers exhibited greater physiological reactivity to stress observation as compared to non-stress observers to placebo-stress observation in heart rate, salivary alpha-amylase, and cortisol (p's≤.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.

Key words: stress, stress contagion, Trier Social Stress Test, physiological reactivity

1. INTRODUCTION

While the individual experience of and reactivity to direct stress exposure has been extensively studied over decades, comparably little is known about 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, 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 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 review see: (Engert et al., 2019; White and Buchanan, 2016). Hitherto, the physiological contagion of stress was examined between parents (mostly 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 non-stressful control condition controlling for secondary effects of the stress observation setting (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 that includes a non-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 own stress exposure (Blons et al., 2021). In other studies, the observation was realized with observers placed behind the targets 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 and Lengel, 1986). Differing from 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 hypothalamus-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 that is closely

interrelated with SAM and HPA axis (Gideon et al., 2020; Gideon et al., 2022b; Kubzansky and 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 and Davies, 2005; DiBona, 2001). Furthermore, the HPA axis hormone adrenocorticotropic hormone (ACTH) stimulates the release of aldosterone by the zona glomerulosa of the adrenal cortex (Bollag, 2014; Connell and Davies, 2005). Given this interaction between SAM axis, HPA axis, and 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 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 and Sonnentag, 2006; Lovallo, 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 non-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 HR and sAA, the HPA axis parameter cortisol, as well as the RAAS parameter aldosterone before, during, and after (non-)stress (observation). We hypothesized higher physiological reactivity to direct stress observation compared to non-stress observation. Moreover, we

expected stress contagion reactivity of the different stress axes to resemble first-hand stress reactivity, but of minor extent.

2. METHODS

2.1 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 the following self-reported characteristics: age over 30 years, female sex, smoking, BMI > 30 kg/m^2, 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 (Germany).

The study was carried out in accordance with the Declaration of Helsinki principles and was formally approved by the Ethics Committee of the University of Constance, Germany. All participants provided written informed consent prior to participation and received financial compensation (10€/hour).

2.2 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 2 to 4 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 Trier Social Stress Test (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 detailed description of experimental procedure in the stress condition, see *2.3 Stress contagion paradigm – Stress observation condition*). In the placebo-stress observation condition, participants ("non-stress observers") observed a confederate undergoing a version of the Placebo TSST; (Het et al., 2009)) adapted for observation (Observation Placebo TSST, obsPlacTSST, for detailed

description of 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 start of 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 rhythmic of stress hormones (Gideon et al., 2022a; 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.

2.3 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 and Kemeny, 2004). The TSST

procedure includes an audio- and video-taped mock job interview (5 min) followed by a mental arithmetic (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 setting and task, we developed an observation control condition based on the PlacTSST (Het et al., 2009). Instead of observing the obsTSST procedure, non-stress observers observed a male confederate reading out an emotionally neutral story (5 min) followed by an easy mental arithmetic task, namely adding up 5 (5 min) (reading confederate). Comparable to the experimental condition, the non-stress observers were disguised as panel members wearing white coats, and received similar instructions as 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 5 several times in front of an audience to prevent them from being stressed by the non-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 non-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 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 physiological reactions of the stress observers to result explicitly from stress observation, i.e., stress contagion, and not from observation setting or task.

2.4 Physiological assessment

2.4.1 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 40R, Heraeus, Thermo Fisher Scientific, Langenselbold, Germany), aliquoted, and frozen at – 80°C until analysis. Saliva samples were taken at 8 sampling timepoints in all participants (TSST participants, stress observers, and non-stress observers): 10 min before 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 5 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 5 saliva sampling time points in participants (-10, +1, +10, +20, + 120 min) and at 4 sampling time point in reading confederates (-10, +1, +10, +20). For determination of sAA, we used an enzymatic colorimetric assay (alpha Amylase Saliva Assay, RE80111, IBL International GmbH, Hamburg, Germany) following 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 non-stress observer participant are missing because of technical problems.

Salivary cortisol. Salivary cortisol was measured at 7 saliva sampling time points in all participants (-10, +1, +10, +20, +30, +45, +120 min) and at 5 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. Detection limit was 0.003 µg/dl.

Salivary aldosterone. Salivary aldosterone was measured at 7 saliva sampling time points in all participants (-10, +1, +10, +20, +30, +45, +120 min) and at 5 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. Detection limit was 12.07 pg/ml. Due to technical problems, salivary aldosterone data are missing for two non-stress observers and for one reading confederate in one obsPlacTSST.

2.4.2 Heart rate assessment

To obtain information on physiological reactivity *during* 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 experimental manipulation), mock job interview (mins 2-4 of mock job interview), mental arithmetic task (mins 2-4 of mental arithmetic task), saliva sampling time points +1 min (0-2 min after experimental manipulation), +10 min (9-11 min after experimental manipulation), +20 min (19-21 min after experimental manipulation), and +120 min (119-121 min after experimental manipulation). Due to recording problems, HR data of seven stress observers are missing.

In TSST participants and in control condition 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 6 HR measurement timepoints: 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 beginning of the mock job interview, one measurement 2 min after beginning of the mental arithmetic task, as well as three measurements after cessation of the experimental procedure, i.e., + 1, + 10, and + 20 min after cessation of the experimental procedure. HR assessment during the experimental manipulation failed in four TSST participants.

2.5 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 ± 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 Mcintosh (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 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 2 groups and 7 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 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^2_p) using G*Power for Mcintosh (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 by the formula BMI = kg/m^2. For 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 TSST 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 timepoints of HR, sAA, cortisol, and 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 non-stress observation by calculating repeated measures ANOVAs with group (stress observers vs. non-stress observers) as independent variable and repeated measurement of HR, sAA, cortisol, and aldosterone levels as repeated dependent variables. Due to potentially confounding effects of age and BMI on cortisol, sAA, and aldosterone reactivity (Gideon et al., 2022b; 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 timepoint. 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 non-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 conditions were, respectively, 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 timepoint 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.

3. RESULTS

3.1 Participant characteristics

Our final sample comprised 20 TSST participants, 36 stress observers, and 30 non-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 ($2.67 \pm .26$) ($t(30) = .63$, $p = .54$).

3.2 Intraclass-correlation coefficients

Table 2 shows the ICCs for all dependent variables at every measurement timepoint. 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 non-stress observers) which suggests that ANOVAs and *t*-tests should be rather robust. Nevertheless, alternatives for the inferential-statistical procedures are reported in Appendix A. 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.

3.2 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 timepoints and within a specific timepoint (indicated by the standard-error 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 timepoints (p's $\leq .009$) with higher increases in the TSST participants. For salivary aldosterone, higher changes from baseline to separate post-manipulation timepoints in TSST participants compared to reading confederates were only observed +20 min after obsTSST/obsPlacTSST cessation ($p = .035$).

3.3 Observer reactivity: Comparison between stress observers and non-stress observers

As expected, stress observers exhibited greater increases with respect to salivary cortisol, sAA, and HR in reaction to observation of the obsTSST as compared to non-stress observers in reaction to 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 Appendix B for detailed aldosterone data).

We found significant group differences in changes from baseline between stress and non-stress observers at +1 min in all parameters and at +10 min in cortisol and sAA (p's ≤ .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).

3.4 ObsTSST: Comparison between TSST participants and stress observers

To characterize the physiological reactivity to direct stress observation as 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 obsPlacTSST measurement timepoints for cortisol until + 45 min (p's < .001), for sAA at + 1 min (p = .005) as well as for HR at measurement timepoints during and after the obsTSST until + 20 min (p's ≤ .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 an no changes in sAA at + 120 min, TSST participants showed the regular reactivity kinetics with significant increases from baseline at all other measurement timepoints in cortisol, sAA, and HR (p's ≤ .006). In stress observers, cortisol levels significantly increased from baseline to + 1 min post obsTSST (p = .017) and decreased at +45- and +120-min post obsTSST. sAA levels significantly increased from baseline to all post obsTSST measurement timepoints in stress observers (p's ≤ .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 +120 mins post obsTSST.

4. 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 non-stressful control condition to control for secondary effects of the stress observation setting. In our standardized controlled paradigm, participants in the experimental condition directly observed another participant undergoing an adapted version of the TSST. Differing from 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 (non-)stress (observation) in TSST participants, stress observers, reading confederates, and non-stress observers.

As expected and in line with previous stress studies (e.g., (Gideon et al., 2022b; Het et al., 2009; Kothgassner et al., 2021), the obsTSST induced higher reactivity with respect to all assessed physiological parameters (sAA, HR, salivary cortisol, 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 non-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 non-stress and thus placebo observation control condition and thus controlled 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 the direct stress observation (Green et al., 2014; Shapiro et al., 1978). There were no differences in salivary aldosterone reactivity to stress observation as compared to non-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 notably lower 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 but 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 and 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 are in line 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 heart rate 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/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 and Buchanan, 2016). In explanatory models for empathy such as the Perception-Action Model for Empathy (PAM; (Preston and De Waal, 2002) or the Neurocognitive Model of Emotional Contagion (NMEC; (Prochazkova and Kret, 2017), it is assumed that individuals have shared neural representations for the perception and the generation of actions (Preston and 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 a corresponding representation in the observing person (Prochazkova and Kret, 2017). Both models posit that this shared neural activation subsequently induces the activation of 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 and 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 for 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). In addition, 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 persons 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 and 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, non-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 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 Appendix A), future studies should always consider this potential source of variance and apply alternative analyses when appropriate (see Appendix A). 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 non-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 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 non-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 as 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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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

STATEMENT OF ETHICS

The project/program 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.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are not publicly available but are available from the corresponding author PHW (email address: petra.wirtz@uni-konstanz.de).

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Figure Legends

Figure 1

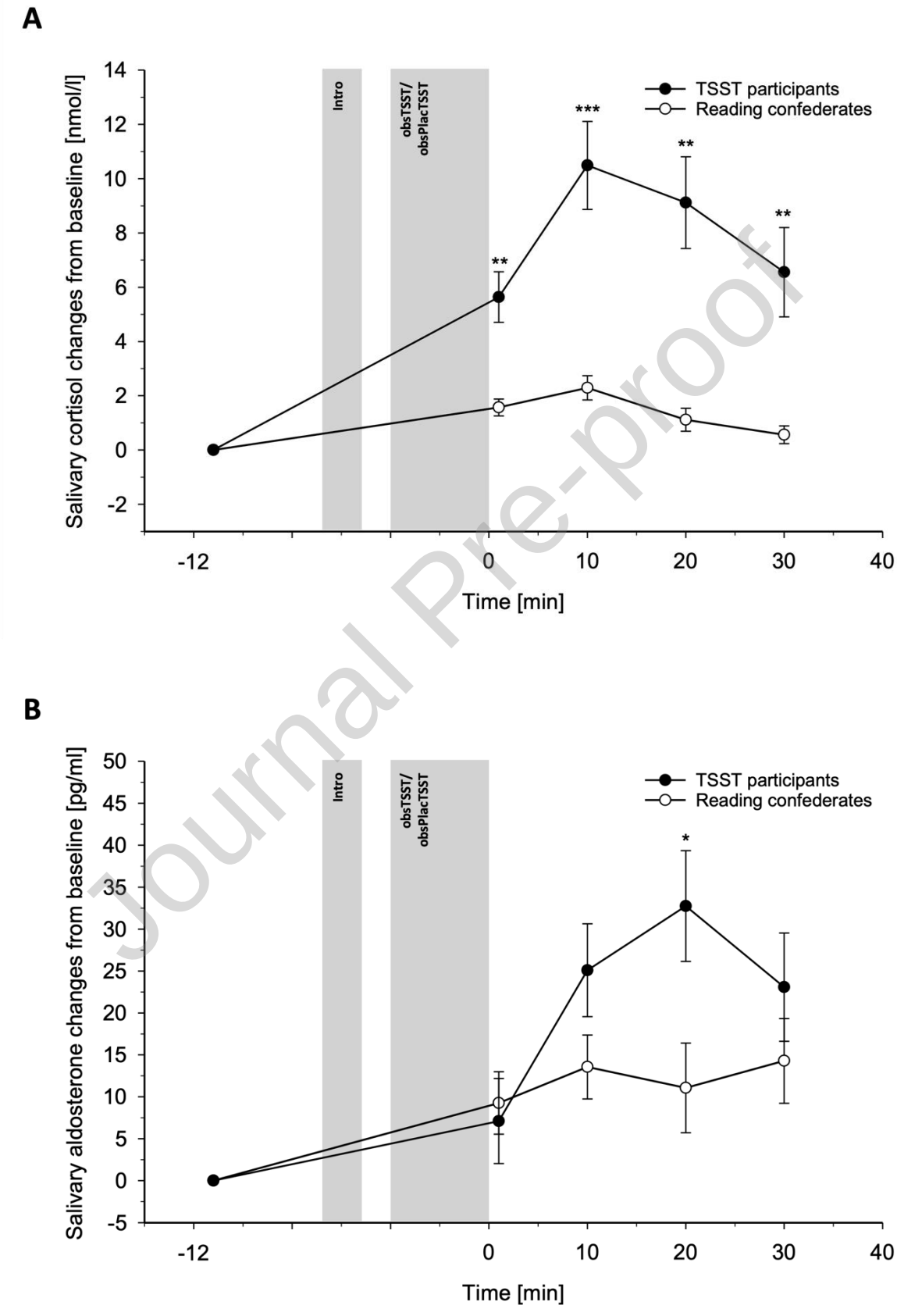
Physiological reactivity (mean ± SE) of TSST participants to our adapted version of the Trier Social Stress Test (obsTSST) (black dots) and of reading confederates to our adapted version of the Placebo-TSST (obsPlacTSST) (white dots). (A) Salivary cortisol. (B) Salivary aldosterone. (C) Salivary alpha-amylase. (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 ± SE) of stress observers (black triangles) and TSST participants (black dots) to our adapted version of the Trier Social Stress Test (obsTSST) and of non-stress observers (white triangles) to our adapted version of the Placebo-TSST (obsPlacTSST) (white triangles). (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 (* $p < .05$;** $p < .01$; *** $p < .001$).

Figures

Figure 1



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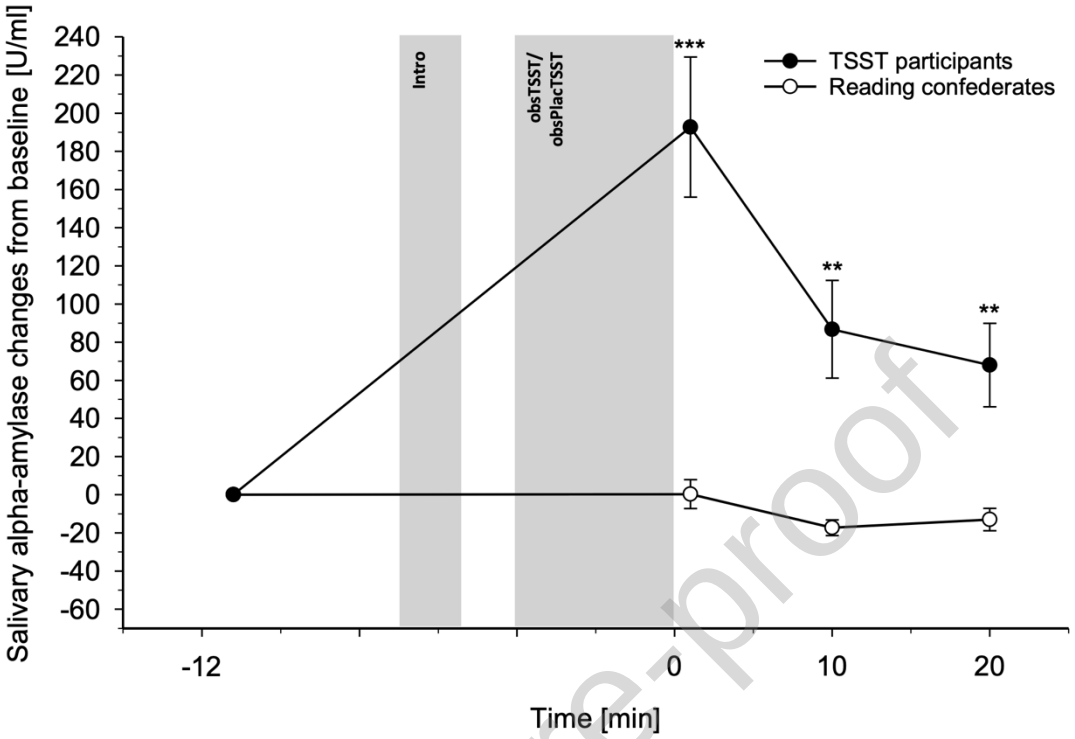


D

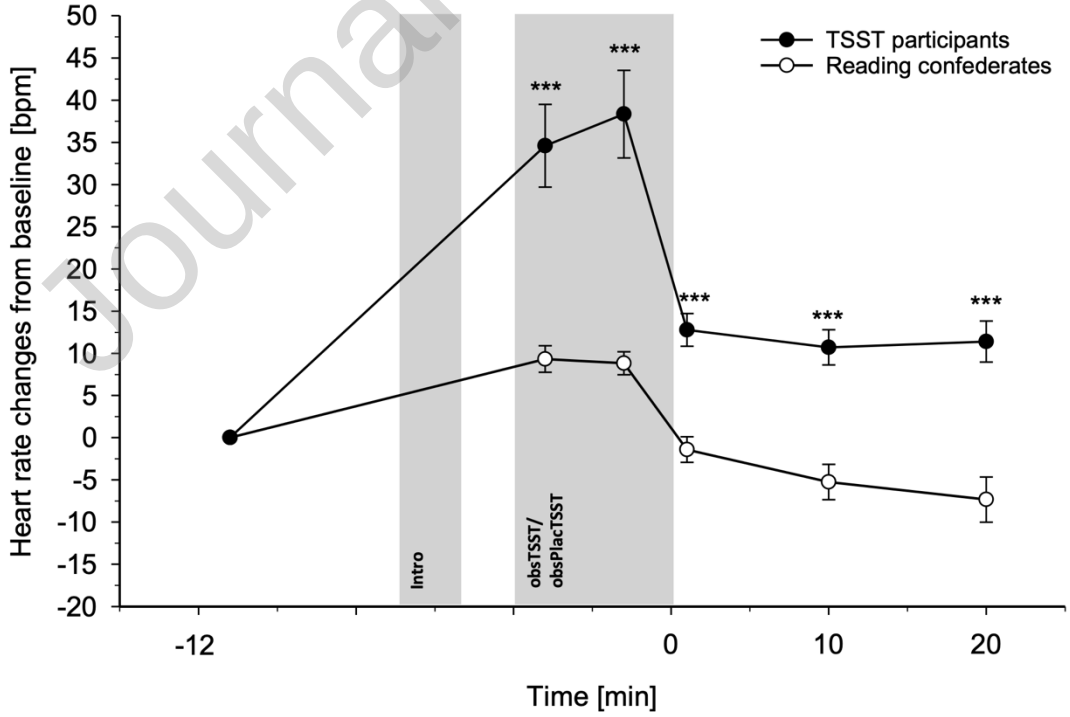
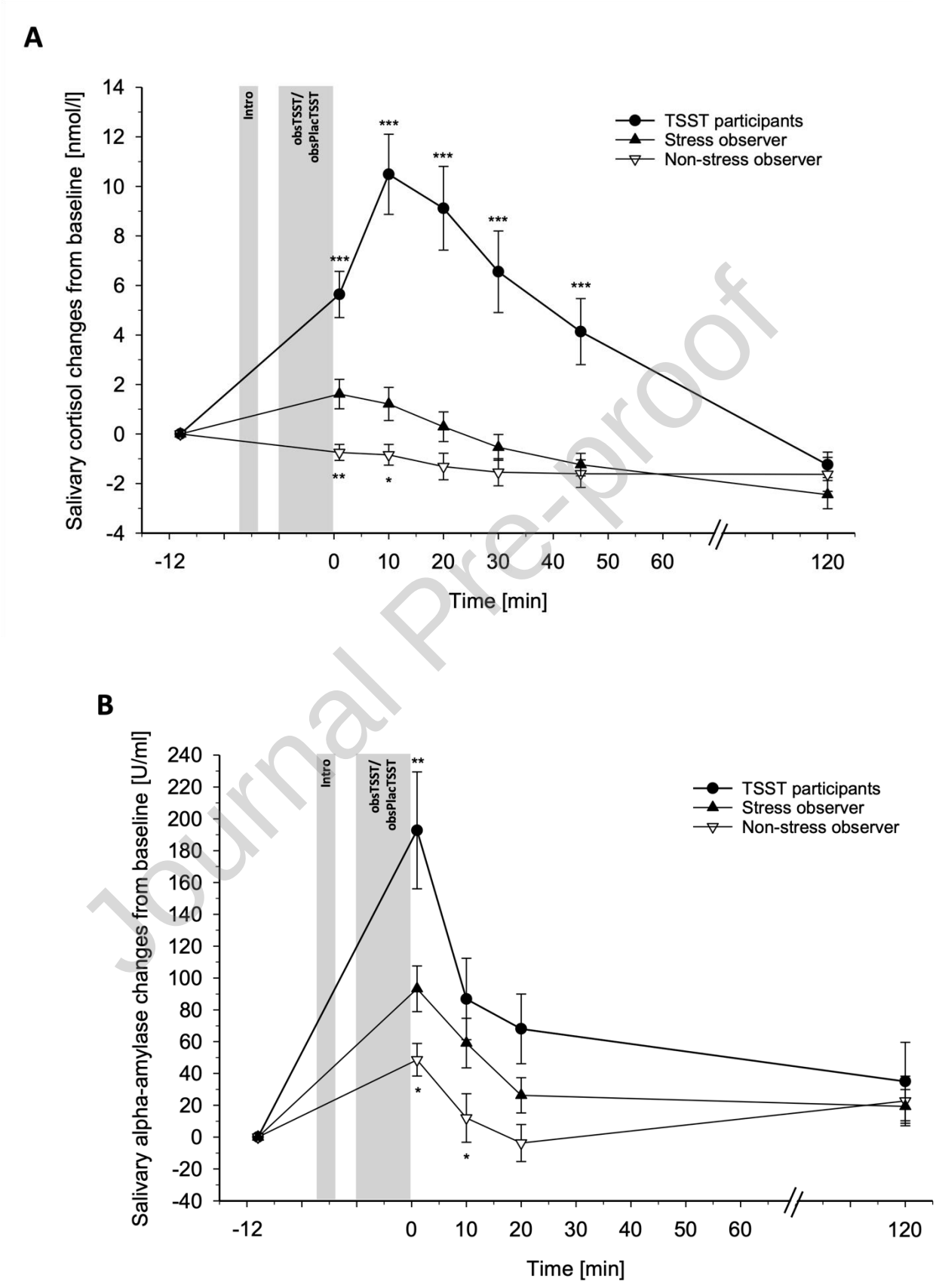
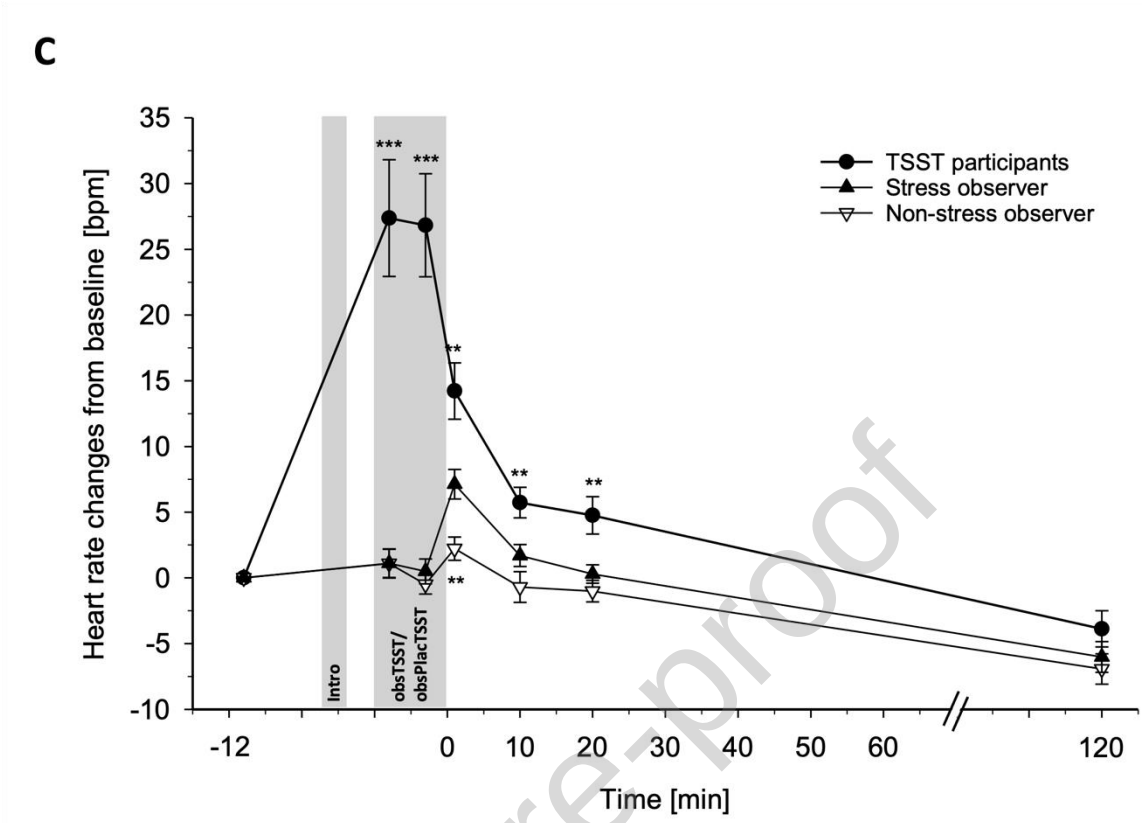


Figure 2





TABLES

Table 1. Participants characteristics

	TSST participants (*n* = 20)	**Stress observer** (*n* = 36)	**Non-stress observer** (*n* = 30)	***p***
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)	*n* = 33 159.17 ± 16.21 (33.47 – 398.05)	*n* = 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)	*n* = 28 81.11 ± 5.26 (40.11 – 152.73)	.49
Baseline HR [bpm]	*n* = 18 74.87 ± 1.96 (63.21 – 93.22)	*n* = 29 77.39 ± 2.16 (55.57 – 101.56)	76.93 ± 1.78 (61.52 – 102.11)	.70

Footnote. 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)

Footnote. Values reflect the proportion of variance that is attributable to study group. Free values relate to stress-observers, values in parentheses relate to non-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 non-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 non-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 (*p*-value)	*p*-value group comparison with stress observers	Change in U/ml from baseline (*p*-value)	*p*-value group comparison with stress observers	Change in bpm from baseline (*p*-value)	*p*-value group comparison with stress observers
Speech task	Stress observers	-		-		1.10 ± 1.07 (.31)	
	Non-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)	
	Non-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)**	
	Non-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)	
	Non-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)	
	Non-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)		-		-	

	Non-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)**		-		-	
	Non-stress observers	-1.60 ± 0.56 **(.003)**	.61	-	-	-	-
	TSST participants	4.14 ± 1.34 **(< .001)**	**< .001**	-	-	-	-
+ 120 min	Stress observers	-2.45 ± 0.57 **(<.001)**		19.30 ± 10.59 (.078)		-6.02 ± 1.17 **(<.001)**	
	Non-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

Footnote. 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 timepoint; *p*-values group comparison = interactions group-by-time of repeated measures ANOVAs comprising baseline and respective other individual measurement timepoint 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 non-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 ($p<.05$).

DECLARATION OF INTEREST

None.

HIGHLIGHTS

- Introduction of a standardized controlled experimental stress contagion paradigm
- Stress observation induces a physiological stress contagion response
- Stress contagion reactivity resembles first-hand stress reactivity but less pronounced
- HPA and SAM axis are stress contagion responsive systems as opposed to the RAAS