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Stress protection by your group – increasing group size reduces physiological stress contagion

Theresa Dorn [a,b], Alisa Auer [a,b], Lisa-Marie Walther [a,b], Christine Sauter [a], Elisabeth Bandle [a], Petra H. Wirtz [a,b,*]

[a] Biological Work and Health Psychology, University of Konstanz, Konstanz, Germany
[b] Centre for the Advanced Study of Collective Behaviour, University of Konstanz, Konstanz, Germany

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ABSTRACT

Background & objectives: Stress contagion refers to the spread of stress from one person to another. We previously established a standardized, controlled experimental paradigm to study stress contagion in humans. While stress contagion effects have been characterized on a physiological level, potential modulating factors are beginning to be understood. Using our paradigm, we tested for the first time whether the number of observers, i.e. observer group size, modulates physiological stress contagion in stress observers.
Methods: Our experimental condition comprised three groups of stress observers varying in group sizes of two ("Group 1", n = 30), three ("Group 2", n = 31), or more observers ("Group 3", n = 31), with each group observing one stressed participant. The data assessment comprised up to 5 healthy young male participants, with one participant randomly assigned to undergo an adapted version of the Trier Social Stress Test ("TSST participants", n = 57) and the remaining participants observing him disguised as panel member(s) ("stress observers", n = 92) in addition to one panel confederate. We repeatedly assessed salivary cortisol, salivary alpha-amylase, and heart rate.
Results: The TSST induced significant increases in all physiological parameters under study (p´s < =.025) without reactivity differences between TSST participants of the three experimental groups (p ś > =.23). When comparing the physiological reactivity to direct stress observation, the stress-observer-groups significantly differed in terms of cortisol (p = .029) with overall higher reactivity in smaller observer groups. Further analyses confirmed a linear effect in terms of higher reactivity with lower observer group size (p = .046). There were no group-by-time interactions in salivary alpha-amylase and heart rate reactivity.
Discussion: Our results suggest that when directly observing stress in other individuals, observer group size has a differential effect on physiological stress contagion systems. While we found evidence for modulating effects on hypothalamic-pituitary-adrenal axis reactivity in terms of higher cortisol stress contagion reactivity with lower observer group size, observer group size did not relate to the sympathetic–adrenal–medullary axis. Potential implications remain to be elucidated.

1. Introduction

In today's increasingly urbanized and socially interconnected world, individuals are often embedded in group settings where stress is not only experienced at the individual level, but also at the interindividual level transmitted between group members (Engert et al., 2019; Srivastava, 2009; White and Buchanan, 2016). The latter phenomenon is referred to as stress contagion and describes the spread of stress from one person to another (Engert et al., 2019; Marheinecke et al., 2025; Nitschke and Bartz, 2023; White and Buchanan, 2016). Stress contagion is considered to comprise both, *resonant* and *vicarious* stress (Engert et al., 2019, 2014). While stress resonance describes a proportional increase in the physiological activation of passive observers and a directly stressed target, vicarious stress evolves independently of a target's actual stress state and is supposed to result from a projection of the observer's own perspective onto the stressful situation. Research on stress contagion increased during the past one and a half decades (see Marheinecke et al., 2025 for a Review). While the findings provide clear evidence for

* Correspondence to: Biological Work and Health Psychology, University of Konstanz, Universitaetsstrasse 10, Konstanz 78464, Germany.
E-mail address: petra.wirtz@uni-konstanz.de (P.H. Wirtz).

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physiological stress contagion in humans, the studies also revealed considerable interindividual variance in the stress contagion responses (Auer et al., 2024; Engert et al., 2014; Erkens et al., 2019; Gallistl et al., 2025; Heilmann et al., 2024; Schury et al., 2020). This variance points to potential modulating factors that influence to what extent and how stress is transmitted.

So far, the role of potential modulating factors on physiological stress contagion is beginning to be understood, although samples, methods, and results have varied greatly across studies in adult participants (see Marheinecke et al., 2025 for a Review). There is evidence for a modulating role of empathy, including trait cognitive empathy (Blasberg et al., 2023; Buchanan et al., 2012; Engert et al., 2014), trait affective empathy (Buchanan et al., 2012; Dimitroff et al., 2017), and state empathy (Engert et al., 2014), although not unequivocally (Blons et al., 2021; Park et al., 2021; Schury et al., 2020). Further, attachment (Gallistl et al., 2025), and the emotional, social, and physical aspects of familiarity and closeness (Blons et al., 2021; Engert et al., 2014; Park et al., 2021; Phan et al., 2019; Schury et al., 2020) have a promoting effect on stress contagion. In addition, the modality in which stress is observed also appears to play a major role. With regard to cortisol, highest responder rates (i.e. $\geq$ 1.5 nmol/l cortisol (Miller et al., 2013)) were observed for direct stress observation (42 % (Auer et al., 2024)), followed by non-direct observation via one-way mirror (22–30 % (Engert et al., 2014; Heilmann et al., 2024)), and via video (16–24 % (Engert et al., 2014; Erkens et al., 2019; Schury et al., 2020)).

Considering the increasing prevalence of stress in modern society (Gallup Inc., 2025) and the rising population densities, particularly in metropolitan areas (United Nations, 2022), we suggest that the number of observers observing a stressed individual (i.e., *observer group size*) may represent a further relevant aspect of observation modality influencing the amount of physiological stress contagion. So far, a potential modulating effect of observer group size has not yet been investigated. We expect a modulating effect of group size based on the following reasons: First, increasing group size may reduce the intensity of stress contagion via *attentional diffusion*. A factor that likely influences the amount of physiological stress contagion in a group setting is attention within observers. Attention as the ability to focus on specific stimuli or locations in an individual's environment (James, 1890), plays a central role in the emergence of empathic responses. According to the perception-action model (PAM; (Preston and De Waal, 2002)) of empathy, suggested to form a theoretical basis for physiological stress contagion (Engert et al., 2019; White and Buchanan, 2016), the perception of another person's emotional state can automatically activate corresponding emotional and physiological states in the observer, given appropriate attention. In larger observation groups, the attention of individual observers is likely distributed among several social stimuli (Birmingham et al., 2008; Guerin and Innes, 1984), which reduces the attentional focus on the stressed person and thus empathically mediated physiological stress contagion (Engert et al., 2019). Second, increasing group size may reduce the intensity of stress contagion via *diffusion of responsibility*. The latter describes the psychological mechanism by which an individual's sense of responsibility decreases with more people present (Darley and Latané, 1968; Fischer et al., 2011). In social situations, this leads individuals to feel less obliged to act because they assume that others present will act, known as the bystander effect (Darley and Latané, 1968). Indeed, an fMRI study showed that neuronal activity in brain regions associated with the preparation of helping behavior decreases with increasing group size (Hortensius and de Gelder, 2014). Moreover, in line with the idea that perceived social responsibility relates to empathy (Lepron et al., 2015), activity in certain brain regions supposed to be associated with empathic concern were found to be decreased with increasing number of bystanders and thus observers (Decety and Jackson, 2006; Hortensius and de Gelder, 2014). As empathic processes appear to be a key mechanism for stress contagion (Engert et al., 2019) diffusion of responsibility likely buffers the empathic resonance required for physiological stress contagion.

Building on the above-described reasoning, the present study aimed to examine, for the first time, whether observer group size modulates physiological stress contagion in stress observers. Notably, we recently adapted a previous stress contagion paradigm (Buchanan et al., 2012) and established a standardized, controlled experimental paradigm based on direct stress observation to study physiological stress contagion in humans (Auer et al., 2024). Using this paradigm, we focused on those stress axes that we previously identified to be sensitive to physiological stress contagion in observers (Auer et al., 2024) and repeatedly measured heart rate (HR) and salivary alpha-amylase (sAA) (SAM: sympathetic-adrenal-medullary-axis) as well as salivary cortisol (HPA: hypothalamus-pituitary-adrenal-axis) before, during, and after the stress observation task. We hypothesized that increasing observer group size would reduce the intensity of physiological stress contagion in the stress observers. Trait empathy and chronic stress were assessed in all participants to rule out group differences in these potential confounders.

2. Methods

2.1. Study participants

We recruited healthy, medication-free, non-smoking, young men up to 35 years. With respect to potential interference with physiological stress reactivity, we further applied the following inclusion criteria based on self-report: no psychiatric or somatic diseases (including allergies), no regular excessive physical exercise, no illicit drug abuse and no occasional or acute intake of prescribed or non-prescribed medication. 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 single-blind, between-subject design based on Auer et al. (2024). Our experimental condition comprised three groups of stress observers varying in group size, with each group observing one stressed participant. The data assessment comprised 2–5 individuals with one participant randomly assigned to undergo a version of the Trier Social Stress Test (TSST; (Kirschbaum et al., 1993)) adapted for observer stress (Observation TSST, obsTSST; (Auer et al., 2024)) ("TSST participant"). The remaining participant(s) observed the obsTSST disguised as panel member(s) ("stress observers") in addition to one panel confederate who led the obsTSST (for detailed description of experimental procedure, see *2.2.1 Stress contagion paradigm*). The resulting number of observers thus varied between 2 and 5 observers rendering the three experimental groups: stress-observer-group 1 (Group 1: one stress observer and one panel confederate), stress-observer-group 2 (Group 2: two stress observers and one panel confederate) and stress-observer-group 3 (Group 3: three or four stress observers and one panel confederate).

Experimental procedure. Participants abstained from any kind of sports and the consumption of alcohol 24 h prior to study participation. Moreover, they were instructed to avoid caffeinated beverages and flavonoid containing food on the study day. On the study day, participants were invited to individual meeting points at individual times between 11:30 a.m. and 12:00 p.m. to avoid an encounter between participants. 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.

Each experimental procedure started at 1:00 p.m. to rule out potential confounding due to the circadian rhythmic of stress hormones (e.g. Nater et al., 2007; Weitzman et al., 1971). 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, stress observers 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 60 min.

2.2.1. Stress contagion paradigm

A potential contagion of stress requires a stressed individual encountering other non-stressed individuals. In our experimental procedure, we confronted one randomly selected participant in each data assessment as TSST participant with our version of the well-established psychosocial stress induction procedure TSST (Kirschbaum et al., 1993), adapted for observer stress (obsTSST) that we could previously show to elicit physiological stress responses (Auer et al., 2024). Like the TSST (Kirschbaum et al., 1993), the obsTSST procedure also includes an audio- and video-taped mock job interview (5 min) followed by a mental arithmetic task (5 min) in front of an evaluating panel wearing white coats (Auer et al., 2024). In the original TSST, all panel members are confederates. To allow for direct stress observation, the panel in the obsTSST consisted of up to four 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 like 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, the stress observers were informed that they would not end up in the situation of the TSST participant themselves.

2.3. Physiological assessment

2.3.1. 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 40 R, Heraeus, Thermo Fisher Scientific, Langenselbold, Germany), aliquoted, and frozen at – 80°C until analysis. Saliva samples were taken at seven sampling timepoints in all participants (TSST participants and stress observers): 10 min before start of the experimental procedure as well as + 1, + 10, + 20, + 30, + 45 and + 60 min after the end of the experimental procedure.

Salivary cortisol. Salivary cortisol was measured at all of the seven saliva sampling time points in all participants (-10, +1, +10, +20, +30, +45, +60 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 7.1 % and 3.5 % in our sample. Detection limit was 0.003 µg/dl.

Salivary Alpha-Amylase (sAA). sAA was measured at four saliva sampling time points in all participants (-10, +1, +10, +20 min). 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 9.3 %, and intra-assay CV was 1.6 % in our sample. sAA data of three stress observers (Group 1: n = 1; Group 2: n = 2) and of three TSST participant (Group 2: n = 1; Group 3: n = 2) were incomplete because of insufficient amount of saliva (n = 4) or technical problems (n = 2).

2.3.2. 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 A.S.M.A. application (A.S.M.A. B.V., Amsterdam, The Netherlands). For analyses of HR data in TSST participants and stress observers, means were calculated for seven 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 + 60 min (59–61 min after experimental manipulation). Due to recording problems, HR data of four stress observers (Group 2) and three TSST participants (Group 2: n = 1; Group 3: n = 2) were missing.

2.4. Psychological assessment

2.4.1. Trait empathy

Empathy as a personality trait was assessed using the Saarbrücken Personality Questionnaire (SPF; (Paulus, 2009)), the German version of the Interpersonal Reactivity Index (IRI; (Davis, 1980)). The SPF comprises the affective and cognitive factors of empathy using 16 items distributed across four subscales (perspective taking (PT), empathic concern (EC), personal distress (PD), and the fantasy scale (FS)). The subscales are each measured with four items on a five-point Likert scale (1 = "never" to 5 = "always"), with a maximum score of 20 for each subscale. We calculated the SPF total score according to Paulus (2012) as a measure of empathy. In the present sample, the SPF total score showed a sufficient internal consistency (Cronbach's α=.72, N = 149).

2.4.2. Chronic stress

Chronic stress was assessed using the 12-item short version of the Trier Inventory of Chronic Stress (Screening Scale for Chronic Stress, SSCS; (Schulz et al., 2004)). Participants rate the 12 items on a five-point Likert scale in respect to how often they encountered a certain situation or had a certain experience within the previous three months (0 = "never" to 4 = "very often"). The total score (0–48) was calculated as a measure of chronic stress, with higher scores indicating greater chronic stress. In the present sample, the scale showed a good internal consistency (Cronbach's α =.83, N = 149).

2.5. Statistical analyses

Data were analyzed using SPSS (Version 30.0) statistical software packages for Macintosh (IBM SPSS Statistics, Chicago IL, USA) and are presented as mean ± standard error of the mean (SEM). All tests were two-tailed with the significance level set at $p < .05$ and p-values $< .10$ interpreted as marginally significant. 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). To allow for detection of conservatively expected small effect sizes of $f = .10$ with a power of $(1 - \beta) = .90$ in repeated measures analysis of variance (ANOVA) with 3 groups and 7 repeated measures given the presumed lowest average correlation of $r = .65$ for cortisol, the required sample size is $N = 90$.

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, sAA, and HR, data of all three parameters were transformed using the natural logarithm for use in all statistical analyses. For reasons of clarity, we depict original data or their percentage changes from baseline, respectively, in all Tables and Figures. To protect against violations of sphericity, we applied Huynh-Feld correction where appropriate. Effect size parameter *f* was calculated from partial η^2 (η_p^2) using G*Power for Mcintosh (Version 3.1.9.6; Heinrich Heine University Düsseldorf, Germany) and is 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. To test for differences in participant characteristics, we compared participants of our three observer groups in terms of demographic measures, trait empathy, chronic stress, and baseline levels of physiological measures using univariate analyses of variance (ANOVA). Due to potentially confounding effects of the observed baseline differences in cortisol reactivity in stress observers, we calculated changes from baseline and controlled for cortisol baseline measurement as covariate in all repeated cortisol analyses of stress observers. Moreover, we accounted for effects of age and BMI on cortisol reactivity (Therrien et al., 2010), by performing all repeated cortisol analyses with age and BMI as covariates. To correct for multiple testing in repeated measures analyses using sAA and HR as markers of the SAM-axis, we applied Bonferroni correction for p-values ≤ 0.05.

To test for successful stress induction by the TSST as well as successful stress contagion response to stress observation under study, we calculated repeated measures AN(C)OVAs across all TSST participants and across all observers, respectively, with cortisol, HR and sAA levels as repeated dependent variables. In addition, to confirm these results, we determined cortisol responder rates. TSST participants and stress observers who showed a minimal cortisol increase of 1.5 nmol/L from baseline to their individual peak were classified as cortisol responders according to Miller et al. (2013). To rule out differences in physiological reactivity of TSST participants of the three observer groups in reaction to the TSST, we further calculated repeated measures AN(C)OVAs with group as independent variable and repeated measurement timepoints of cortisol, sAA or HR as repeated dependent variables.

As our main analyses, we tested for differences in physiological reactivity of the stress-observer-groups in reaction to direct stress observation. We calculated repeated measures AN(C)OVAs with observer group as independent variable and repeated measurement timepoints of cortisol, sAA, or HR levels as repeated dependent variables. Post-hoc testing of significant AN(C)OVA effects between the three observer groups comprised repetition of the analyses for two instead of three groups (i.e. stress-observer-group 1 vs. 2, 1 vs. 3, and 2 vs. 3). As complementary post-hoc analyses, we recalculated repeated measures AN(C)OVAs using observer group size (i.e., 2, 3, 4, or 5 stress observers) as continuous variable to test whether observer group size effects are of linear nature.

3. Results

3.1. Participant characteristics

Our final sample comprised 92 stress observers (Group 1: n = 30, Group 2: n = 31, Group 3: n = 31) and 57 TSST participants (Group 1: n = 30, Group 2: n = 16, Group 3: n = 11). Stress observers in the three experimental groups did not significantly differ in terms of age, BMI, trait empathy, chronic stress, sAA, or HR at baseline (*p*'s $\geq$ .30). However, cortisol at baseline significantly differed between the three experimental groups (F(2, 89) = 6.86, $p = .002$, $\eta_p^2 = .13$, f = .39) with higher levels in the stress-observer-group 2 as compared to the Groups 1and 3 (Comparison of two stress-observer-groups: 1 vs. 2: $p < .001$; 1 vs. 3: $p = .12$; 2 vs. 3: $p = .045$). TSST participants in the three experimental groups did not significantly differ in age, BMI, trait empathy, chronic stress, or physiological measures at baseline (*p*'s $\geq$ .16). Table 1 depicts participants´ characteristics of the stress observers.

Table 1
Participants characteristics.

	Group 1 ($n = 30$)	Group 2 ($n = 31$)	Group 3 ($n = 31$)	*p*
Age [years]	22.33 ± 0.50 (19 – 29)	23.42 ± 0.62 (18 – 30)	23.10 ± 0.48 (19 – 29)	.35
BMI [kg/m^2]	24.48 ± 0.58 (20.31 – 32.31)	23.26 ± 0.51 (18.14 – 29.81)	24.04 ± 0.59 (18.56 – 34.98)	.30
Trait empathy [SPF-score]	40.37 ± 0.96 (25.00 – 51.00)	38.58 ± 1.04 (26.00 – 49.00)	40.10 ± 1.05 (27.00 – 53.00)	.41
Chronic stress [SSCS-score]	20.13 ± 1.19 (6.00 – 33.00)	20.10 ± 1.54 (7.00 – 42.00)	20.52 ± 1.16 (9.00 – 34.00)	.97
Baseline salivary cortisol [nmol/l]	4.19 ± 0.36 (1.71 – 9.52)	6.52 ± 0.48 (1.96 – 12.34)	5.25 ± 0.52 (1.74 – 13.52)	**.002**
Baseline sAA [U/ml]	$n = 29$ 150.24 ± 18.13 (4.58 – 551.93)	$n = 29$ 174.70 ± 19.93 (31.17 – 398.05)	175.25 ± 19.08 (39.94 – 423.15)	.56
Baseline HR [bpm]	74.75 ± 2.33 (55.60 – 109.90)	$n = 27$ 76.82 ± 1.82 (55.50 – 96.00)	76.18 ± 2.04 (54.00 – 95.90)	.69

Footnote. Values are means ± SEM; sAA = salivary alpha-amylase; HR = heart rate; bpm = beats per minute; *n* = number of participants.

3.2. Reactivity of TSST participants in three experimental groups

In TSST participants, the TSST induced significant increases in all physiological parameters under study (main effects of time: cortisol: $F(2.24, 120.75) = 3.64$, $p = .025$, $\eta_p^2 = .06$, f = .25; sAA: F(2.60, 137.69) = 84.25, p = .002, $\eta_p^2 = .61$, f = 1.25; HR: F(2.41, 127.83) = 119.08, $p = .002$, $\eta_p^2 = .69$, f = 1.49; see Fig. 1). In TSST participants, responder rate in terms of cortisol increases of $\geq$ 1.5 nmol/l from baseline (Miller et al., 2013) was 87.72 % (Group 1: 86,67 %; Group 2: 87,50 %; Group 3: 90,91 %). There were no physiological reactivity differences between TSST participants of the three experimental groups (interactions--group-by-time: cortisol: $p = .23$; sAA: $p = .90$; HR: $p = .60$; see Table 2 for descriptives).

3.3. Reactivity of stress observers in three experimental groups

Across all three stress-observer-groups, the observation of the TSST induced (marginally) significant increases in sAA, HR, and cortisol reactivity (main effects of time: sAA: $F(2.73, 240.41) = 46.09$, $p = .002$, $\eta_p^2 = .34$, f = .72; HR: F(4.57, 397.40) = 27.11, $p = .002$, $\eta_p^2 = .24$, f = .56; cortisol: without covariates: F(3.37, 215.38) = 22.99, $p < .001$, $\eta_p^2 = .20$, f = .50; with covariates: F(3.19, 280.95) = 2.42, $p = .063$, $\eta_p^2 = .027$, f = .17) (see Fig. 2). Across all stress observers, responder rate was 30.43 %. Responder rates in the different observer groups were 40 % in Group 1, 25.81 % in Group 2, and 25.81 % in Group 3.

When comparing their physiological reactivity to direct stress observation, the stress-observer-groups significantly differed in terms of cortisol (interactions group-by-time: cortisol: $F(6.62, 284.58) = 2.32$, $p = .029$, $\eta_p^2 = .05$, $f = .23$) with overall higher reactivity in smaller stress-observer-groups (see Fig. 3). Post-hoc testing of the significant group-by-time interaction revealed significantly higher cortisol reactivity in Group 1 as compared to Group 3 ($F(2.94, 164.57) = 2.94$, $p = .036$, $\eta_p^2 = .05$, $f = .23$) and marginally significant higher cortisol reactivity as compared to Group 2 ($F(3.42, 191.76) = 2.29$, $p = .072$, $\eta_p^2 = .04$, $f = .20$). Moreover, Group 2 showed marginally significant higher cortisol reactivity as compared to Group 3 ($F(3.46, 197.43) = 2.17$, $p = .083$, $\eta_p^2 = .04$, $f = .20$). Complementary analyses using observer group size as continuous variable further confirmed a linear effect with higher reactivity the lower observer group size ($F(3.28, 285.47) = 2.63$, $p = .046$, $\eta_p^2 = .03$, $f = .18$). There were no group-by-time interactions in sAA and HR reactivity, either in terms of group comparisons (sAA: $F(5.58, 240.00) = .61$, $p = .71$, $\eta_p^2 = .01$, $f = .10$; HR: $F(9.50, 403.64)$

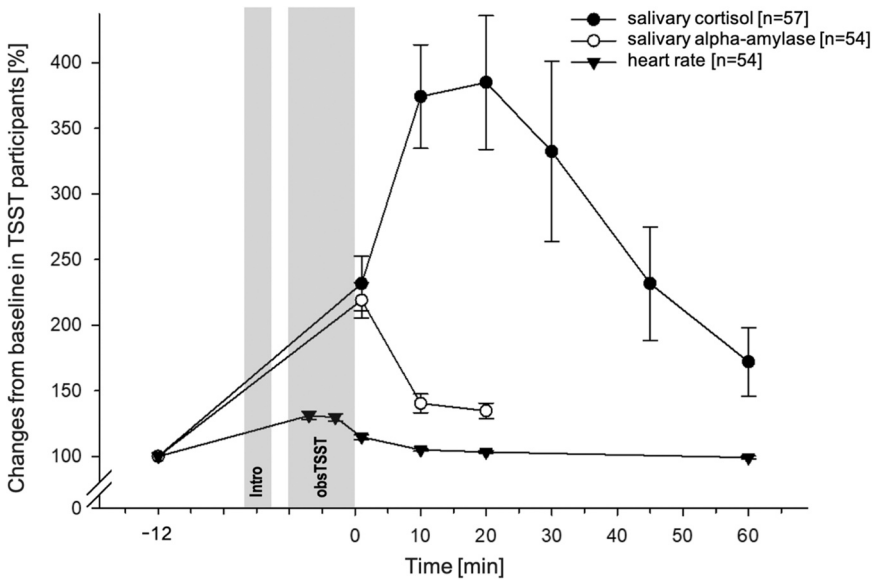

Fig. 1. Physiological stress reactivity of TSST participants. Comparison of the physiological reactivity of TSST participants in reaction to the observer TSST (obsTSST) in salivary cortisol (black dots), salivary alpha-amylase (white dots), and heart rate (black triangles).

Table 2
Descriptives for stress response across the TSST participants.

	time	Group 1 (n = 30)	Group 2 (n = 16)	Group 3 (n = 11)
Cortisol	1	4.64 ± 0.36	5.29 ± 0.92	3.82 ± 0.58
	2	8.31 ± 0.78	11.32 ± 1.81	8.78 ± 1.47
	3	13.69 ± 1.22	15.98 ± 2.35	11.92 ± 2.24
	4	13.55 ± 1.37	13.88 ± 1.92	12.47 ± 2.11
	5	10.79 ± 1.27	10.46 ± 1.39	10.27 ± 1.41
	6	7.83 ± 0.99	8.00 ± 1.02	7.15 ± 0.86
	7	5.96 ± 0.62 (n = 30)	6.47 ± 0.90 (n = 15)	5.76 ± 0.72 (n = 9)
sAA	1	170.96 ± 15.22	146.59 ± 25.84	120.47 ± 23.87
	2	346.60 ± 36.32	309.77 ± 60.21	238.68 ± 39.82
	3	234.29 ± 26.00	226.09 ± 48.60	150.11 ± 33.11
	4	232.22 ± 24.11 (n = 30)	211.45 ± 46.26 (n = 15)	145.06 ± 32.98 (n = 9)
HR	1	77.29 ± 2.16	78.39 ± 2.72	73.93 ± 2.61
	TSST_1	98.46 ± 3.26	101.90 ± 4.75	103.50 ± 6.71
	TSST_2	98.14 ± 3.26	101.75 ± 4.90	99.51 ± 6.05
	2	86.35 ± 2.56	92.04 ± 3.45	87.36 ± 5.30
	3	78.89 ± 1.89	83.78 ± 2.59	80.33 ± 2.68
	4	78.01 ± 1.82	82.53 ± 2.25	77.47 ± 3.15
	5	75.17 ± 1.76	78.42 ± 2.22	73.97 ± 2.81

Footnote. Values are means ± SEM; sAA = salivary alpha-amylase; HR = heart rate

= .95, $p = .48$, $\eta_p^2 = .02$, $f = .14$), or when using observer group size as continuous variable (sAA: $F(2.76, 239.96) = 1.50$, $p = .22$, $\eta_p^2 = .02$, $f = .14$; HR: $F(4.69, 403.18) = 1.21$, $p = .31$, $\eta_p^2 = .01$, $f = .10$).

4. Discussion

Here, we investigated for the first time whether the number of observers, i.e. observer group size, modulates physiological stress contagion in stress observers. Using our standardized experimental paradigm (Auer et al., 2024), we varied observer group size in groups of two, three, and four to five observers. We focused on stress axes sensitive to physiological stress contagion (Auer et al., 2024) and repeatedly measured HR and sAA (SAM axis), as well as salivary cortisol (HPA axis), before, during, and after the stress observation task in stress observers.

Overall, successful stress induction and stress contagion was verified with responder rates of almost 90 % in TSST participants and more than 30 % in our observer participants corroborating previous research (Auer et al., 2024; Blasberg et al., 2023; Engert et al., 2014; Erkens et al., 2019; Heilmann et al., 2024; Schury et al., 2020). The TSST participants in the three subject groups did not differ in their physiological reactivity to the obsTSST. Our main finding was that increasing observer group size reduced the intensity of physiological stress contagion in terms of salivary cortisol and thus the HPA axis in our stress observers. In more detail, when comparing the three stress-observer-groups, the smallest group with two observers (Group 1) showed the highest cortisol reactivity and the largest group with four or five observers (Group 3) the lowest. However, there were no group differences in sAA and HR contagion reactivity suggesting that in contrast to the HPA axis, the SAM axis does not seem to be sensitive to the modulating effect of observer group size on stress contagion.

What *mechanisms* may underly these findings? Auer et al. (2024) validated the paradigm used in our study in order to control for secondary effects of the task by a placebo-TSST control group and found that observation in the control condition did not lead to cortisol increases in the placebo-TSST observers. Based on these findings, we mainly attribute our observation-induced cortisol increase differences between the three observer groups to stress contagion effects but cannot completely rule out potential influences of first-hand stress. Furthermore, while the three observer groups differed in their cortisol stress contagion responses, with lowest responder rates in the stress observers of the two larger groups compared to the smallest group, the TSST participants in the respective groups did not statistically differ in their cortisol stress reactivity and even showed slightly higher responder rates with increasing observer group size. Considering this discrepancy between the cortisol reactivities of TSST participants and observers, our findings point to vicarious rather than resonant stress. Notably, there were no differences in trait empathy between the observer groups that may have influenced the observed reactivity differences.

Moreover, based on studies investigating first-hand stress, the SAM axis is more responsible for the rapid, non-specific alarm response to immediate threats (Goldstein and Kopin, 2008). As the focus here is on the rapid provision of energy for the fight-or-flight response, there is no time for extensive cognitive processing of the stressful situation. In contrast, the HPA axis reacts slower and more sensitively to aspects reflecting *cognitive processing* including psychosocial evaluation, cognitive appraisals, and interpersonal contextual features (Dickerson and Kemeny, 2004; Lazarus, 1984). Given that we found evidence for

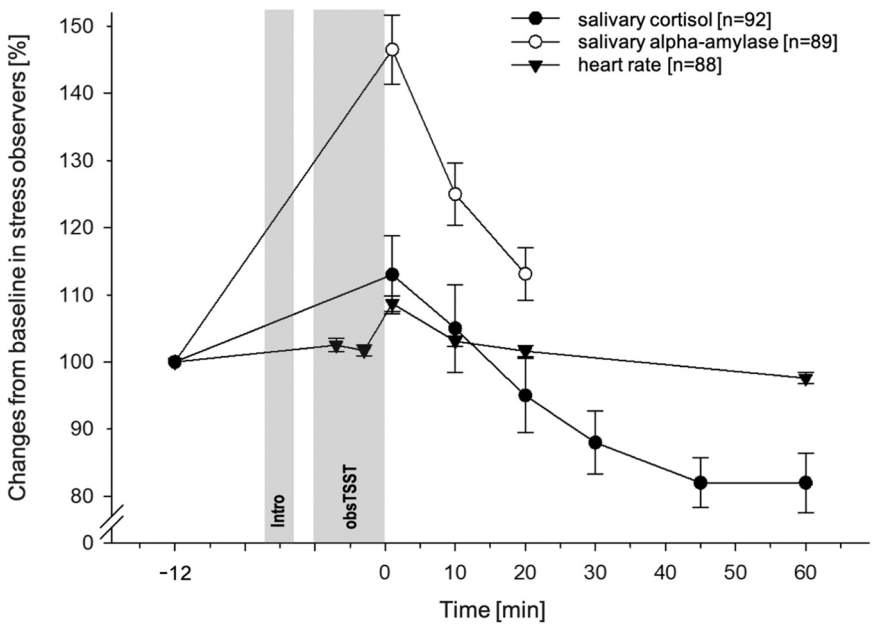

Fig. 2. Physiological stress reactivity of stress observers. Comparison of the physiological reactivity of stress observers in reaction to the observer TSST (obsTSST) in salivary cortisol (black dots), salivary alpha-amylase (white dots), and heart rate (black triangles).

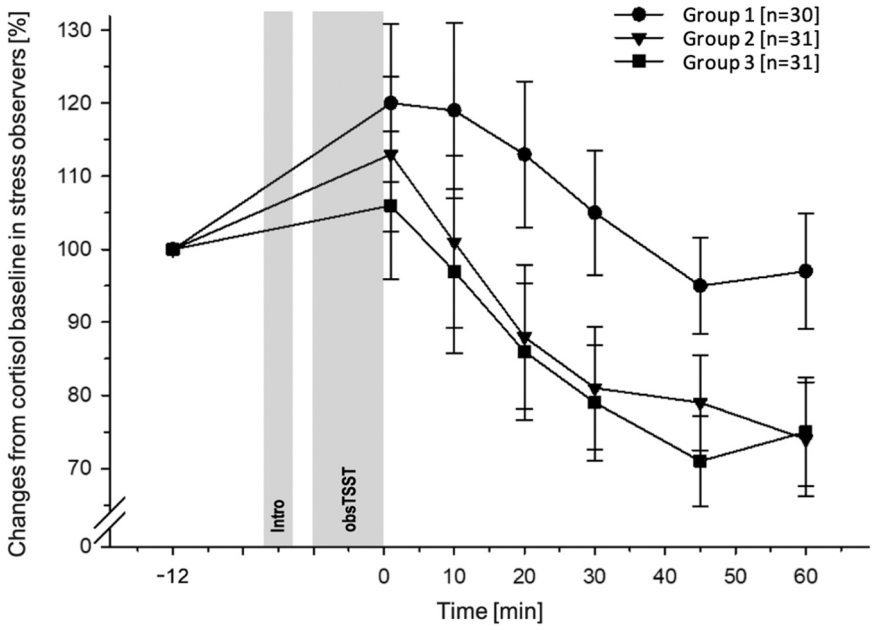

Fig. 3. Salivary cortisol reactivity of stress observers with varying group size. Comparison of the cortisol reactivity of stress observers in reaction to observer TSST (obsTSST) in the three experimental groups: Group 1 (two observers, black dots), Group 2 (three observers, black triangles) and Group 3 (four and five observers, black squares).

observer group size effects on HPA axis but not SAM axis reactivity, we speculate that different group sizes may comparably induce alterations in cognitive processing underlying the differential cortisol contagion responses in the three observer groups.

More precisely, we speculatively propose *changes in attention* and *diffusion of responsibility* as potential mechanisms in cortisol stress contagion group size effects. Attention as a cognitive process that enables individuals to selectively prioritize and process sensory information (James, 1890) plays a central role in social perception and, thus, in the emergence of empathic responses (Preston and De Waal, 2002). In the context of stress contagion, a focused allocation of attention towards the stressed person might be essential for the accurate perception of emotional cues that underly empathic processes and consequently contagion effects in observers (Engert et al., 2019; Preston and De Waal, 2002; White and Buchanan, 2016). In group settings, the attentional resources of individual observers are divided among multiple social stimuli with increasing number of people present (Birmingham et al., 2008; Guerin and Innes, 1984). Thus, with increasing group size, less attention may remain for the stressed person and empathically mediated cortisol stress contagion would be reduced (Engert et al., 2019). Moreover, diffusion of responsibility as a cognitive process based on the mental evaluation of social contexts describes the psychological mechanism by which an individual's sense of responsibility decreases with more people present (Darley and Latané, 1968). In social situations, as

induced by our direct observation task in a group setting, this leads individuals to feel less obliged to act because of the assumption that others present will act (bystander effect; (Darley and Latané, 1968)). Therefore, we assume that the more observers present, the lower the individual sense of responsibility might be, which consequently could lead to reduced emotional significance, less empathic resonance, and thus lower cortisol stress contagion responses in the observers.

Interestingly, in addition to our main finding, we observed despite our randomized group assignment baseline group differences in salivary cortisol levels between our three observer groups. Post-hoc testing revealed that Group 2 had significantly higher baseline levels as compared to Groups 1 and 3 that however were within the normal physiological range with respect to daytime (Kirschbaum et al., 1999). Groups 1 and 3 in contrast did not differ from each other. Notably, we statistically accounted for cortisol baseline differences in our analyses of stress contagion reactivity differences and thus controlled for potential confounding effects of baseline differences on our main observer group size stress contagion effect. Furthermore, since Groups 1 and 3 did not differ significantly in their cortisol levels at baseline and as we particularly observed cortisol increase differences between observer Group 1 and 3, these latter group differences are unlikely confounded by cortisol baseline differences. Nevertheless, it remains unclear why baseline levels were increased in Group 2 and given that there was no association with increasing observer group size, we consider a random effect most likely, maybe related to a potential anticipatory stress response (Lazarus, 1984; Monat et al., 1972).

What are potential *implications* of our findings? Notably, stress contagion as the physiological activation resulting from stress observation has been proposed to reflect a form of non-verbal communication (Engert et al., 2019) that may point to imminent danger as a potential threat for oneself or that 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. On the other hand, it supports behavioral responses such as assistance for the distressed individual or escaping the threat by providing energy (Calabrese, 2008; Dhabhar, 2009). With increasing group size, individual attention and alertness can be reduced as the monitoring of the environment as well as the need to act is distributed among several group members (Roberts, 1996; Tedeschi et al., 2021). The higher baseline cortisol level with increasing group size might prepare for the energy needed for social expectation, comparison, or evaluation processes that can be anticipated when entering new social situations.

Strengths of our study include the use of a validated standardized experimental paradigm (Auer et al., 2024) designed to investigate stress contagion in a direct, face-to-face observation setting. The use of a highly controlled laboratory setting with randomized group assignment of homogenously selected participants allows to control for a variety of potential confounding factors. Consequently, we interpret the observed group differences in salivary cortisol stress contagion reactivity to specifically result from the modulating effect of observer group size. *Limitations* of our study include the limited generalizability of our findings beyond healthy, medication-free, non-smoking, young men. Future studies are needed to confirm the findings in women and larger sample sizes with greater diversity. Despite our randomized group allocation, we faced baseline differences in salivary cortisol in observer Group 2 that we however statistically controlled in order to account for possible confounding effects. Furthermore, due to our instruction to maintain a neutral facial expression during the observation task, we cannot rule out that empathic processes may have been reduced, that emotion regulation processes have been induced across all observers, or that a certain level of first-hand stress may have occurred in this direct face-to-face observation task. Nevertheless, all of our observer groups had exactly the same instructions while observing comparably stressed TSST participants and we observed group differences despite the exactly same instructions and similar stress reactivity of the TSST participants. Notably, in real world scenarios, we assume that stress contagion is usually combined with a certain amount of first-hand stress due to the direct (even non-verbal) interaction with a stressed individual that requires a certain amount of reactivity of the observer, either verbal or non-verbal, or in terms of behavior. Given this reasoning, our direct face-to-face observation paradigm may provide advantages regarding external validity. Also, we did not assess potential feelings of power, emotion regulation, or state empathy in the observer groups which may have influenced their stress contagion reactivity. Finally, we had differences in the sample sizes of our TSST participant groups as a result of our experimental design that focused on equal sample sizes within observer groups.

Taken together, our study findings reveal that with increasing observer group size, the intensity of physiological stress contagion, or vicarious stress respectively, in terms of salivary cortisol and thus the HPA axis is reduced in our stress observers. The SAM axis (HR and sAA), on the other hand, does not seem to be sensitive to the modulating effect of observer group size on stress contagion. Nevertheless, although our results provide a decent basis for further research on physiological stress contagion and its modulating factors, several questions such as regarding mediating mechanisms remain open. Future studies are needed to confirm our findings in larger and more diverse samples that better allow for linear mixed modelling as an alternative statistical approach and related post-hoc testing of potential time-point differences. In addition, future research should further investigate the generalizability of our results and thus the role of observation modality and other potential modulating factors to populations other than healthy young men, as well as underlying mechanisms and implications for health, cognition, behavior, and everyday life.

CRediT authorship contribution statement

Theresa Dorn: Writing – original draft, Investigation, Data curation, Formal analysis, Visualization. **Alisa Auer**: Investigation, Project administration, Writing – review & editing. **Lisa-Marie Walther**: Investigation, Project administration, Writing – review & editing. **Christine Sauter**: Investigation, Writing – review & editing. **Elisabeth Bandle**: Investigation, Writing – review & editing. **Petra Wirtz**: Conceptualization, Methodology, Funding acquisition, Writing – original draft, Supervision.

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.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

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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