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The effects of acute work stress and appraisal on psychobiological stress responses in a group office environment

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ABSTRACT

Background: The high prevalence of office stress and its detrimental health consequences are of concern to individuals, employers and society at large. Laboratory studies investigating office stress have mostly relied on data from participants that were tested individually on abstract tasks. In this study, we examined the effect of psychosocial office stress and work interruptions on the psychobiological stress response in a realistic but controlled group office environment. We also explored the role of cognitive stress appraisal as an underlying mechanism mediating the relationship between work stressors and the stress response.

Methods and Materials: Ninety participants (44 female; mean age 23.11 ± 3.80) were randomly assigned to either a control condition or one of two experimental conditions in which they were exposed to psychosocial stress with or without prior work interruptions in a realistic multi-participant laboratory setting. To induce psychosocial stress, we adapted the Trier Social Stress Test for Groups to an office environment. Throughout the experiment, we continuously monitored heart rate and heart rate variability. Participants repeatedly reported on their current mood, calmness, wakefulness and perceived stress and gave saliva samples to assess changes in salivary cortisol and salivary alpha-amylase. Additionally, cognitive appraisal of the psychosocial stress test was evaluated.

Results: Our analyses revealed significant group differences for most outcomes during or immediately after the stress test (i.e., mood, calmness, perceived stress, salivary cortisol, heart rate, heart rate variability) and during recovery (i.e., salivary cortisol and heart rate). Interestingly, the condition that experienced work interruptions showed a higher increase of cortisol levels but appraised the stress test as less threatening than individuals that experienced only psychosocial stress. Exploratory mediation analyses revealed a blunted response in subjective measures of stress, which was partially explained by the differences in threat appraisal.

Discussion: The results showed that experimentally induced work stress led to significant responses of subjective measures of stress, the hypothalamic-pituitary-adrenal axis and the autonomic nervous system. However, there appears to be a discrepancy between the psychological and biological responses to preceding work interruptions. Appraising psychosocial stress as less threatening but still as challenging could be an adaptive way of coping and reflect a state of engagement and eustress.

1. Introduction

Stress at the workplace continues to be a major concern for employees and employers. Work stress is known to affect the well-being of employees, is associated with high economic costs and imposes a burden on the public health system (Health and Safety Executive, 2019; Galliker et al., 2018). Indeed, experiencing repeated or prolonged occurrences of acute stress (i.e., chronic stress) can lead to health diseases and disorders

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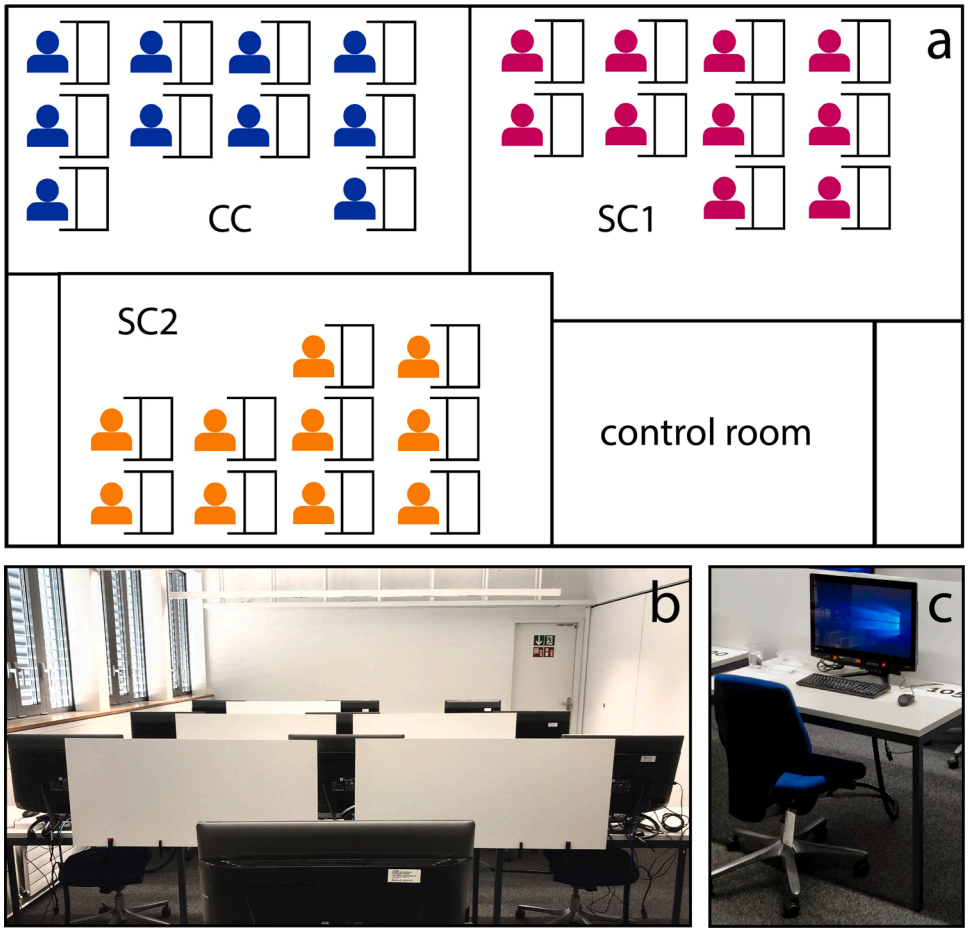

Fig. 1. Floor plan and images of the open office environment. The floor plan on the top (a) illustrates the layout of the laboratory including the control room and the three experimental rooms with abstracted seating arrangements. The image on the bottom left (b) depicts the experimental room of stress condition 1. The image on the bottom right (c) shows the desk, computer and saliva sampling equipment of a single participant. Abbreviations: CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2.

(McEwen, 1998) that include depression (Hammen, 2005), burnout (Iacovides et al., 2003), chronic fatigue syndrome (Coetzee et al., 2019) and cardiovascular diseases (Kivimäki and Kawachi, 2015). In the context of work, acute stress has been defined as a response to an imbalance between the resources available to an individual and the physical, psychological, social and organizational demands (Bakker and Demerouti, 2007). The psychobiological stress response highly depends on the cognitive appraisal of the demands of the job and the resources available to the employee (Lazarus and Folkman, 1984; Kozusznik et al., 2012; Harvey et al., 2010). Besides triggering a psychological response, stress is known to stimulate the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS) in order to activate the organism's ability to successfully cope with encountered stressors (Marques et al., 2010), e.g., heightening vigilance, focusing attention and increasing functioning of the body (Chrousos, 2009). Studies investigating acute stress reactivity in laboratory settings found that challenging tasks lead to increases of perceived arousal and stress as well as a worsening of mood (e.g., Kirschbaum et al., 1999; Boesch et al., 2014). The psychological response is typically accompanied by increased heart rate (HR; von Dawans et al., 2011), reduced heart rate variability (HRV; Taelman et al., 2011), and increased secretion of cortisol and alpha-amylase, which are known indicators of HPA axis and ANS functioning (Petrakova et al., 2015; Kirschbaum et al., 1999).

Understanding the mechanisms and effects of acute work stress is an essential first step in the development of strategies to protect and promote the health of employees. While a considerable amount of research has been devoted to the topic of work stress, important gaps in literature remain regarding the realism of the testing procedure, the type of stressors used in laboratory settings and their effect on the stress response across different systems. This paper makes four contributions towards bridging these gaps.

First, we investigate the extent to which different office stressors affect the psychobiological stress response of healthy subjects in a realistic but controlled work environment. Previous research on office stress has focused on early stress detection using established stress protocols (e.g., Stroop test and arithmetical tasks) rather than the assessment of concomitant psychological and biological stress responses with office-like tasks (cf. Alberdi et al., 2016; Can et al., 2019). To address this lacuna, we created an open floor office environment with three experimental conditions. During the experiment, participants were occupied with basic workload by completing a series of office tasks using a dedicated software. For two of the three experimental conditions, we induced stress by simulating two of the main demands at the workplace: social pressure and work interruptions (Grebner et al., 2010; Health and Safety Executive, 2019). Throughout the experiment, we measured changes in perceived stress, arousal, wakefulness and mood, as well as salivary cortisol, salivary alpha-amylase (sAA), HR and HRV. This allowed us to simultaneously measure responses to work stress across different stress-relevant systems (i.e., subjective measures, indicators of the HPA axis and ANS).

Second, we examine the psychobiology of psychosocial stress in a realistic group office environment. Previous studies were often conducted with single individuals and lacked a social group or socio-evaluative component (e.g., Koldijk et al., 2018). While social interactions are an integral part of office work, they can also be a source of stress if appraised as threatening. To mimic psychosocial stress at the office, we adapted the Trier Social Stress Test for Groups (TSST-G; von Dawans et al., 2011) to fit an office environment. The presence of peers

in the TSST-G adds comparison and social pressure to the inherent socio-evaluative component (Vors et al., 2018).

Third, we investigate the impact of work interruptions on the psychobiological stress response (independent and dependent of psychosocial stress). Studies on work interruptions have primarily investigated job performance and productivity (e.g., Baethge and Rigotti, 2013) rather than stress and well-being (Keller et al., 2020). In office environments, interruptions can arise from various sources including phone calls, chat messages or emails (Akbar et al., 2019). In our study, work interruptions were implemented by giving participants additional tasks via chat messages before and during the anticipation phase of the psychosocial office stress situation.

Fourth, this paper contributes to research by investigating how cognitive appraisal is affected by realistic office stressors and by exploring its role as a potential mediator of the stress reactivity. So far, there are only a few studies on cognitive appraisal of work-related stressors. For example, one study conducted with medical professionals showed that a simulated stressful clinical situation led to increases in threat appraisal, which positively correlated with cortisol levels (Harvey et al., 2010). Another study revealed that work interruptions increased psychological distress, and this relationship was mediated by cognitive appraisal of the interruptions (Ma et al., 2019). Besides exploring the role as a mediating variable, we extend previous research by comparing appraisal of psychosocial office stress with and without prior work interruptions.

Based on the existing literature, we expect psychosocial stress to lead to an activation of psychobiological stress systems, marked by increases in subjective measures of arousal accompanied by a worsening of mood and a rise of salivary cortisol, sAA and HR, and a reduction of HRV. We also expect that prior work interruptions will further heighten this response. Moreover, we expect work interruptions to affect the cognitive appraisal of psychosocial office stress, however, due to the scarcity of literature we investigate this relationship exploratively.

2. Materials and Methods

2.1. Participants

Ninety participants were recruited via the University's online recruitment website. A power analysis using G*Power (Faul et al., 2007) for a repeated measures analysis of variance (ANOVA) revealed that a sample size of 87 participants was sufficient to detect a moderate effect size (f = .3), with a power of 90% and α at .05. All participants were between the ages of 18-40, German speaking and had at least a high-school degree. Exclusion criteria for females included an irregular menstrual cycle, any hormonal contraceptives, pregnancy and breastfeeding, which are known to affect ANS and HPA axis activity (Mezzacappa et al., 2005; Meinlschmidt et al., 2010; Kirschbaum et al., 1999). We also excluded participants who reported acute or chronic mental or somatic illnesses, taking regular medication or any medication to treat acute illnesses during the past two months (e.g., antihistamine, antidepressant, antipsychotic, antihypertensive), substance abuse, or excessive consumption of alcohol, tobacco or psychotropic substances in the past three months. Participants were asked to refrain from vigorous physical exercise, alcohol consumption and chewing gum (24 hours prior to the experiment), from caffeine and food (two hours prior to the experiment) and from brushing their teeth (one hour prior to the experiment).

Participants gave written informed consent and were compensated for participation with 75 Swiss francs. The study was approved by the ETH Zurich's ethics commission (EK 2019-N-34) and was conducted in accordance with the declaration of Helsinki.

2.2. Environment

The ETH Zurich's Decision Science Laboratory was modified to mimic a real-world office environment. We created three separate office spaces (one room per condition) with multiple rows of desks equipped with computers (see Fig. 1). Based on the total amount of participants required by our power analysis, the size of the rooms and the local regulations for office spaces, we placed ten participants per session in each room. Participants were guided through the experiment by a custom-built software (code available at http://u.ethz.ch/xv9hf) that displayed either instruction screens, questionnaires or the office tasks (see Fig. A.1 in the appendix). The experiment sequence was specifically programmed for each condition and synchronized between all participants.

2.3. Procedure

Data was collected during three sessions of 30 participants each conducted on consecutive afternoons. In the recruitment process, participants were informed that they would perform a series of challenging office tasks while their psychobiological responses were measured. During the orientation phase, we explained the basic workload tasks and

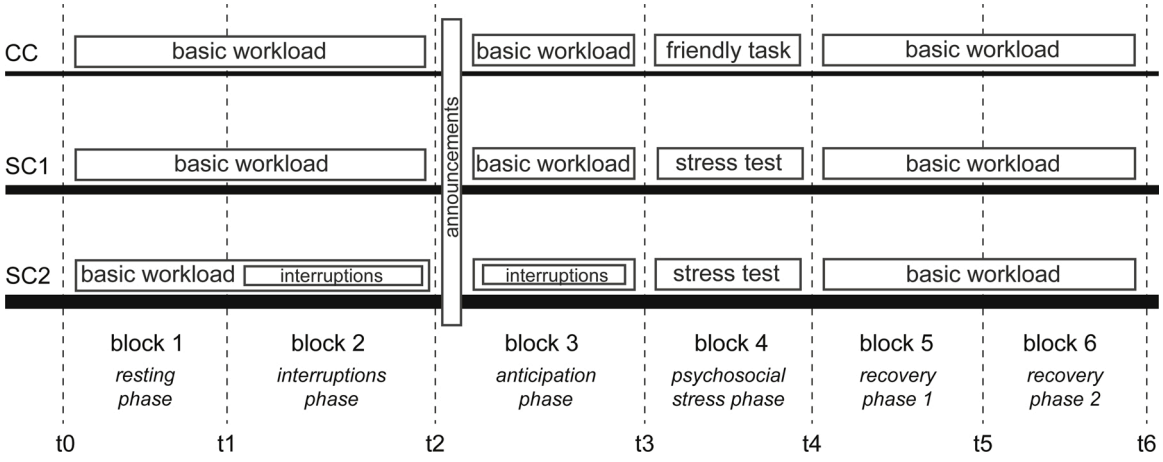

Fig. 2. Study protocol. Saliva samples and repeated questionnaire data were obtained at t1 to t6. Block 1 lasted 15 minutes, block 2 and 3 lasted 20 minutes, while blocks 4, 5 and 6 lasted 15 minutes. Cardiac features were obtained from 5-minute segments and averaged within blocks. Abbreviations: CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2; t0 = −15 minutes; t1 = +0 minutes; t2 = +20 minutes; t3 = +40 minutes; t4 = +55 minutes; t5 = +70 minutes; t6 = +85 minutes.

the communication properties of the chat window. Importantly, participants were not yet informed about the different experimental conditions and upcoming stress-eliciting tasks (i.e., interruptions and psychosocial stress). Participants were also informed through an instructional video accompanied by oral instructions of the experimenters on how to correctly give saliva samples. Following the attachment of the ambulant heart monitoring device, participants were guided to the laboratory and randomly separated into three conditions: Control condition (CC), stress condition 1 (SC1) and stress condition 2 (SC2). From this point on, the participants remained seated at their assigned office desks. Prior to the experiment, participants were asked to complete a set of questionnaires to assess their sociodemographic and psychological baseline characteristics. The actual experiment consisted of six blocks (see Fig. 2) and lasted 85 minutes. At the end of each block, participants completed psychological state questionnaires and provided saliva samples. Saliva samples were clearly labeled and organized on each desk. The experiment software prompted the participants at each time point to commence with the saliva collection. Research assistants monitored the correct handling of the samples. Cardiac activity was continuously recorded throughout the experiment. In all blocks, except block 4, participants in all conditions were asked to perform basic workload tasks using the experiment software. In block 4, both stress conditions were subjected to a situation designed to induce psychosocial stress while the CC underwent a friendly version of the situation. The psychosocial stress test was introduced 20 minutes earlier (in the beginning of block 3) in order to induce anticipatory stress. In blocks 2 and 3, SC2 was subjected to additional stress through work interruptions.

2.3.1. Stress protocol

Basic workload – All participants were asked to behave as employees of a fictitious insurance company and complete their tasks to the best of their ability. They received three different types of tasks via their email inbox. Specifically, participants had to digitize handwritten scans, compute sales numbers for company employees and schedule appointments between clients and employees.

Psychosocial stress – Participants in the SC1 and SC2 were subjected to an adapted version of the TSST-G to induce socio-evaluative threat and uncontrollability (von Dawans et al., 2011). Two actors (male and female) interacted directly with the participants on two occasions. The actors were first introduced as human resources personnel by a line manager via the chat window in block 3. Shortly after, the actors entered the room and announced that they would conduct interviews in 20 minutes to find the most suitable candidate for a promotion. Participants were then told to continue working on their office tasks while mentally preparing for the upcoming interview. The actors returned at the beginning of block 4 and the male actor began questioning all participants in a random order while the female actor took notes. The actors behaved in a reserved manner and let participants speak freely for one minute each. In the CC, participants also interacted with an actor portraying human resources personnel. However, they were treated in a friendly manner and asked to read aloud a short work-related dialogue (i.e., a sales interview for the fictitious insurance company) in unison. Participants and the HR employee alternately read out the lines of the dialogue. Thus, similarly to the control condition in the original TSST-G (von Dawans et al., 2011), participants in the CC performed a task that is comparable to the task of SC1 and SC2 but without any socio-evaluative threat and uncontrollability.

Work interruptions – During blocks 2 and 3, participants in SC2 received continual chat messages from their line manager. These messages asked participants to summarize certain aspects of the work they had conducted and immediately share the retrieved information via chat.

2.4. Psychological measures

Subjective stress load was assessed with the Multidimensional Mood State Questionnaire (MDMQ; Steyer et al., 1997) and the Self-Assessment Manikin (SAM; Bradley and Lang, 1994) at the end of each block. The MDMQ includes the scales $MDMQ_{good\ mood}$ ("good mood–bad mood"), $MDMQ_{calmness}$ ("calmness–nervousness") and $MDMQ_{wakefulness}$ ("wakefulness–sleepiness") with 4 items each. Note that lower scores on the MDMQ subscales indicate bad mood, nervousness or sleepiness. The SAM has 9 gradations and is used to measure valence, arousal and dominance with 1 item per scale. Participants also answered a single item pertaining to their perceived level of stress (i.e., "How stressed are you in this moment?").

Table 1
Baseline characteristics.

	CC		SC1		SC2		*p*
Age [years]	24.07	(3.35)	21.77	(3.46)	23.50	(4.26)	.02 [a]
BMI [kg/m^2]	22.00	(2.19)	21.07	(2.81)	22.21	(2.97)	.15
Chronic stress [TICS SSCS]	6.47	(5.19)	6.63	(4.00)	6.60	(4.99)	.99
Vital exhaustion [MQ]	13.80	(4.02)	13.60	(3.22)	13.63	(4.03)	.89
Depression [BSI_{DEP}]	0.26	(0.27)	0.37	(0.52)	0.40	(0.60)	.86
$MDMQ_{good\ mood}$ at t1	16.97	(2.19)	17.27	(2.10)	16.70	(2.97)	.81
$MDMQ_{calmness}$ at t1	15.80	(3.06)	15.60	(2.86)	15.40	(2.80)	.91
$MDMQ_{wakefulness}$ at t1	15.33	(2.89)	15.03	(2.70)	15.70	(2.82)	.44
$SAM_{valence}$ at t1	3.07	(1.23)	3.13	(1.25)	3.13	(1.20)	.98
$SAM_{arousal}$ at t1	7.63	(1.43)	7.03	(1.47)	7.07	(1.82)	.07
$SAM_{dominance}$ at t1	4.73	(1.51)	5.30	(1.18)	5.23	(1.45)	.25
Perceived stress at t1	1.73	(0.87)	1.87	(0.86)	1.73	(0.83)	.56
Salivary cortisol [nmol/l] at t1	3.70	(1.93)	4.58	(2.94)	3.46	(1.76)	.33
sAA [U/ml] at t1	164.36	(139.92)	149.70	(122.71)	173.90	(118.19)	.71
HR [bpm] during block 1	70.54	(7.54)	74.37	(11.92)	72.49	(10.88)	.39
RMSSD [ms] during block 1	53.57	(32.81)	46.49	(30.68)	46.47	(26.01)	.59
HF [ms^2] during block 1	439.77	(378.89)	504.80	(754.88)	464.78	(573.54)	.90
LF [ms^2] during block 1	922.65	(495.81)	755.37	(521.57)	1020.85	(938.47)	.31
LF/HF during block 1	3.12	(1.95)	3.46	(2.98)	4.23	(2.89)	.35

Baseline characteristics (mean and standard deviation) and *p*-values of F-tests of group differences. $N = 90$, $N = 88$ for cardiac measures. Abbreviations: CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2; BMI = Body mass index; kg/m^2 = kilogram per meter squared; TICS SSCS = Screening Scale of the Trier Inventory for the Assessment of Chronic Stress; MQ = Maastricht Questionnaire; BSI_{DEP} = Depression subscale of the Brief Symptom Inventory; MDMQ = Multidimensional Mood Questionnaire; SAM = Self-Assessment Manikin; nmol/l = Nanomole per liter; sAA = Salivary alpha-amylase; U/ml = Units per milliliter; HR = Heart rate; bpm = Beats per minute; RMSSD = Root mean square of successive differences; ms = Milliseconds; HF = Power in the high frequency range (0.15-0.4 Hz) of the power spectral density of the normal-to-normal heartbeat time series; ms^2 = Milliseconds squared; LF = Power in the low frequency range (0.04-0.15 Hz) of the power spectral density of the normal-to-normal heartbeat time series; LF/HF = LF to HF ratio.

[a] $p < .05$.

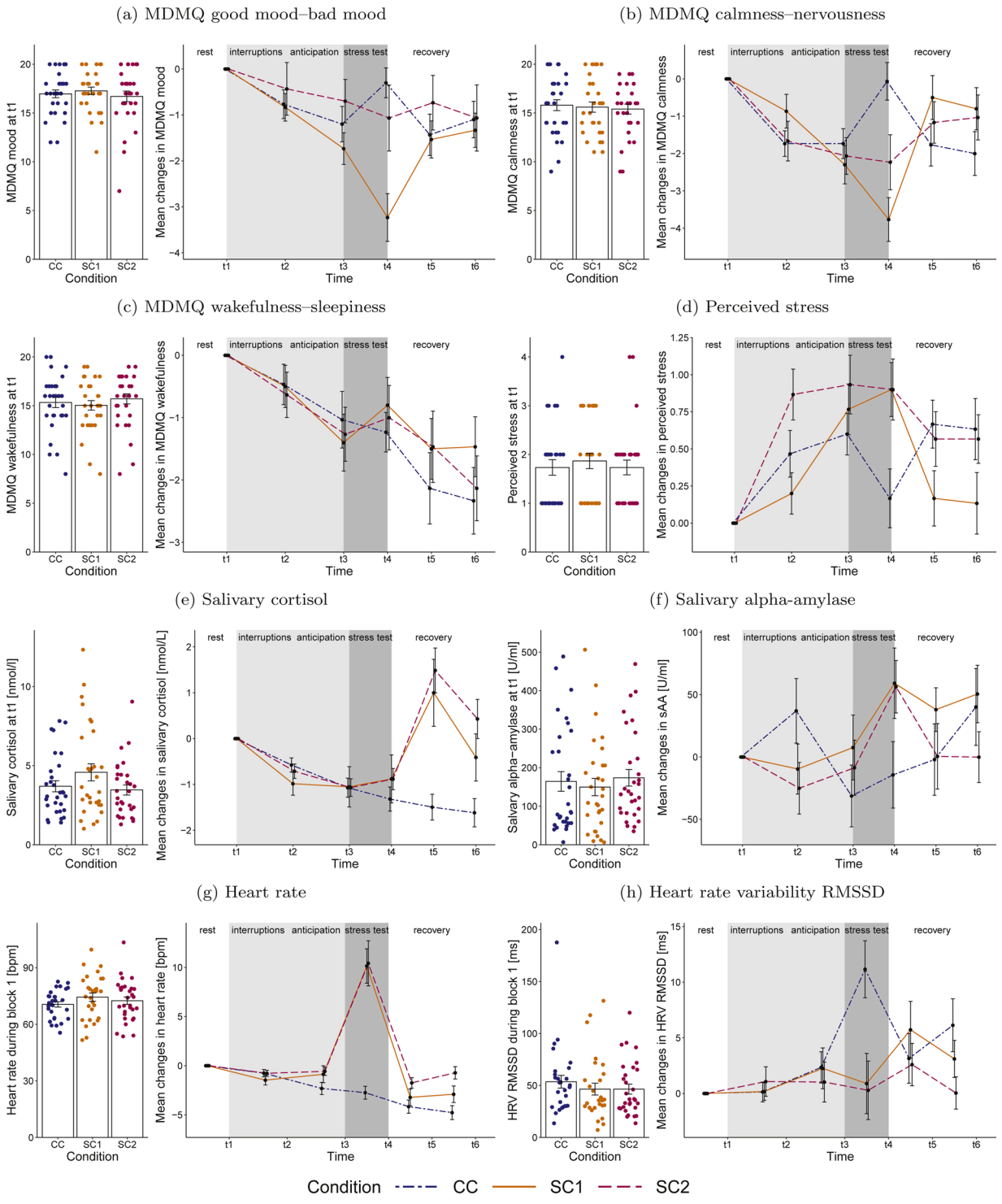

Fig. 3. Bar charts of baseline measurements during the resting phase and plots of the respective repeated measures for MDMQ subscales (a–c), perceived stress item (d), salivary cortisol (e), salivary alpha-amylase (f), heart rate (g) and heart rate variability RMSSD (h). Data points of the baseline measurements are jittered horizontally and data points of the repeated measurements are baseline-corrected for better visualization. Abbreviations: CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2; MDMQ = Multidimensional Mood Questionnaire; nmol/l = Nanomole per liter; sAA = Salivary alpha-amylase; U/ml = Units per milliliter; bpm = Beats per minute; HRV = Heart rate variability; RMSSD = Root mean square of successive differences; ms = Milliseconds.

Cognitive stress appraisal was collected with subscales *threat* and *challenge* of the Primary Appraisal Secondary Appraisal questionnaire (PASA; Gaab et al., 2005) with 16 items to evaluate anticipatory stress. Participants were asked to complete the PASA at the end of block 3 immediately before the psychosocial stress test.

Psychological baseline assessment was done prior to block 1. In addition to the subjective stress load measures described above, we also assessed participants' levels of chronic stress, vital exhaustion and

Table 2
Mediation analysis: Indirect effect of stress condition on the psychobiological peak reactivity and recovery through threat appraisal.

	Direct effect			Indirect effect			
	b	*p*	*BCa CI*	*b*	*p*	*BCa CI*	*υ*
$MDMQ_{good\ mood}$							
peak reactivity	1.45	.10	[-0.24, 3.60]	0.71	.03	[0.09, 1.72][a]	0.01
recovery	−1.47	.03	[-2.83, -0.19][a]	−0.43	.06	[-1.28, -0.03][a]	0.01
$MDMQ_{calmness}$							
peak reactivity	0.93	.31	[-1.09, 2.98]	0.60	.06	[0.04, 1.58][a]	0.01
recovery	−1.16	.23	[-3.31, 0.70]	−0.60	.05	[-1.72, -0.06][a]	0.01
$MDMQ_{wakefulness}$							
peak reactivity	−0.82	.27	[-1.93, 0.77]	0.62	.03	[0.09, 1.47][a]	0.01
recovery	0.13	.84	[-1.37, 0.91]	−0.34	.06	[-0.84, -0.02][a]	0.01
Perceived stress							
peak reactivity	0.22	.47	[-0.41, 0.69]	−0.22	.03	[-0.57, -0.03][a]	0.01
recovery	0.20	.47	[-0.30, 0.78]	0.24	.02	[0.04, 0.60][a]	0.01
Salivary cortisol							
peak reactivity	9.11	.79	[-41.60, 59.00]	−0.34	.96	[-15.70, 20.60]	0.00
ecovery	14.69	.23	[-8.53, 37.60]	−4.73	.15	[-13.61, 1.35]	0.00
sAA							
peak reactivity	−599.75	.60	[-2923.04, 1912.04]	−13.50	.97	[-775.63, 525.73]	0.00
recovery	−1380.10	.08	[-2852.08, 187.30]	416.86	.10	[-13.49, 1152.60]	0.01
HR							
peak reactivity	0.82	.78	[-4.48, 6.46]	−0.55	.48	[-3.57, 0.86]	0.00
recovery	1.17	.66	[-4.53, 5.92]	0.75	.27	[-0.34, 3.62]	0.00
RMSSD							
peak reactivity	−2.29	.62	[-10.60, 5.48]	1.68	.23	[-0.54, 5.87]	0.00
recovery	−0.95	.83	[-8.13, 5.57]	−1.51	.16	[-4.72, 0.26]	0.00

Results of non-parametric bootstrap mediation analysis run with 1000 simulations (N = 60 for psychological and biochemical measures; N = 59 for cardiac measures). Abbreviations: *b* = Unstandardized regression coefficient; *p* = Probability value; *υ* = Effect size upsilon; *BCa CI* = 95% bias-corrected and accelerated bootstrap confidence interval; MDMQ = Multidimensional Mood Questionnaire; sAA = Salivary alpha-amylase; HR = Heart rate; RMSSD = Root mean square of successive differences.

[a] Confidence interval contains the value zero and is therefore significant.

depression (as work stress-related symptomatologies) in order to characterize our sample. Chronic stress was measured with the screening scale (SSCS) of the short version of the Trier Inventory for the Assessment of Chronic Stress consisting of 6 items resulting in a total score of 0 to 24 (TICS-2-K; Schulz et al., 2004). Vital exhaustion was assessed using the Maastricht Questionnaire with 9 items (MQ; Appels et al., 1987). Depression was measured with 6 items taken from the Brief Symptom Inventory (BSI; Spitzer et al., 2011).

2.5. Biochemical measures

Saliva samples were collected by asking participants to chew gently on cotton rolls (Salivette, Sarstedt, Sevelen, Switzerland) for two minutes at the end of each block while they completed the psychological state questionnaires. After collection, samples were stored at -20°C as well as analyzed at the biochemical laboratory of the Department of Clinical Psychology and Psychotherapy at the University of Zurich. Saliva samples were thawed and then centrifuged at 3000 rpm for 10 minutes to reach low-viscosity of saliva.

Salivary cortisol, as an index of HPA axis activity (Lovallo and Buchanan, 2017), was assessed using an immunoassay with time-resolved fluorescence detection. The sensitivity of cortisol was 0.004 μg/dl and inter- and intra-assay coefficients of variance (CVs) were < 10%.

Salivary alpha-amylase (sAA) levels, as a biomarker primarily of the SNS (Petrakova et al., 2015), were determined with an enzymatic colorimetric assay (Roche diagnostics GmbH, Mannheim, Germany) according to the guidelines of the International Federation of Clinical Chemistry with a lower detection limit of 3 U/l. One missing value was replaced with the corresponding condition mean at the time point. Inter- and intra-assay CVs were 7.2 and 7.0, respectively.

2.6. Cardiac activity measures

Cardiac activity was monitored continuously with an ambulant electrocardiogram device, the Firstbeat Bodyguard 2 (Firstbeat Technologies Oy; Jyväskylä, Finland), to capture changes in both the SNS and PSNS (Berntson et al., 2017). Due to a technical failure, the recordings of one participant in the CC and one participant in SC1 were excluded from all analyses.

Heart rate and heart rate variability measures were derived from the recorded R-R intervals in accordance with the standards of measurement established by the Task Force on HRV (Malik et al., 1996). Raw recordings were filtered for artifacts and ectopic beats using the R software package "RHRV" and its adaptive threshold-based filtering algorithm described in listing 2.4.1 of Martínez et al. (2017). This algorithm rejects beats that differ too much from the previous and following beats or from a rolling mean with respect to certain relative and adaptive threshold values. The following initial threshold parameters were chosen based on visual inspection of the filtering results and using the notation of listing 2.4.1 in Martínez et al. (2017): `ULAST=15; LONG=500; UMEAN=3*ULAST; MINIMUM=12; MAXIMUM=30; MINBPM=35; MAXBPM=200`. Wrongly detected and ectopic beats were removed while missed beats or other outliers were visually inspected and corrected with linear interpolation (less than 0.7% of the data). After artifact correction, mean heart rate (HR) and HRV measures were determined on 5-minute intervals for all such intervals that fell entirely within a block and did not overlap with the preceding interval or any saliva sample collections. Values were averaged per block (i.e., blocks 1 and 3 to 6: 2× 5 minutes, block 2: 3× 5 minutes). For block 3, only the intervals after the announcement of the psychosocial stress test were considered. To quantify HRV we used the measure RMSSD (i.e., the square root of the mean of the squared differences of successive normal-to-normal (NN) heartbeat intervals), which has been found to be significantly lowered in response to stress (Kim et al., 2018). In addition, we considered the power in the low (LF) and high (HF) frequency ranges (i.e., 0.04-0.15 Hz and 0.15-0.4 Hz, respectively) of the power spectral density of the NN time series, as well as the ratio of LF to HF (LF/HF). The power spectral density was estimated via Fast Fourier Transform from the NN-tachogram interpolated at 4 Hz using cubic splines. HF is correlated

with RMSSD (Kleiger et al., 2005) and has similarly been found to be significantly lowered in response to stress, while LF and LF/HF tend to increase (Giannakakis et al., 2019).

2.7. Statistical analyses

Data analysis was performed using R (version 3.5.2) and RStudio (version 1.2.1335). Two-tailed tests with a significance level of $p < .05$ were used to test the hypotheses. For all our analyses, we resorted to either robust methods using a trimmed mean of 10% provided by the R package "WRS2" or non-parametric methods provided by the R package "mediation". This approach was necessary since across all measures the assumptions of normal distribution of the data and normal distribution of error variance were violated according to the Shapiro-Wilk test.

We ran robust one-way analyses of variance (ANOVAs) with repeated measures. Each of the ANOVAs included the three conditions as the between-subjects factor (CONDITION) and six repeated measures as within-subjects factors (TIME). Post-hoc tests were performed on significant main effects of CONDITION and significant interaction effects of CONDITION × TIME. These tests were corrected using the Hochberg method to prevent the accumulation of the type 1 error. The results of standard one-way repeated measure ANOVAs including the report of effect sizes (i.e., partial η^2) and correction for sphericity violations with the Greenhouse-Geisser method can be found in the appendix. The results of the robust and standard analyses were congruent (see Tables A.2, A.3, A.4).

We also conducted robust and standard one-way ANOVAs followed by Hochberg-corrected post-hoc tests in order to determine group differences regarding the PASA subscales. Furthermore, we ran separate exploratory, non-parametric mediation analyses with the stress conditions (dummy coding: SC1 = 0, SC2 = 1) as the independent variable and $MDMQ_{good\ mood}$, $MDMQ_{calmness}$, $MDMQ_{wakefulness}$, perceived stress item, HR, RMSSD, sAA and salivary cortisol as dependent variables and threat appraisal as a mediator. For each dependent variable we computed peak reactivity as changes from the initial resting phase to the psychosocial stress response. Peak reactivity for cortisol was computed between t1 and t5 and not between t1 and t4. We computed recovery as changes from the psychosocial stress response to the recovery phase 2. Delta values were used to determine peak reactivity and recovery for psychological and cardiac measures. Cortisol and sAA changes in output were determined with the area under the curve with respect to increase ($AUCi_{peakreactivity}$, $AUCi_{recovery}$; Pruessner et al., 2003). The bias-corrected and accelerated bootstrap confidence intervals (BCa CI) were calculated based on 1000 simulations and a sample size of either N = 59 or N = 60 depending on the measure. The effect size for indirect effects υ was calculated with the R package "MBESS".

3. Results

3.1. Sample characteristics

Ninety healthy individuals (46 male, 44 female) with a mean age of 23.11 (SD = 3.80) took part in the study. Participants had an average body mass index (BMI) of 21.76 (SD = 2.70) and were mainly non-smokers (N = 82; 91%). The smokers reported smoking ≤ 5 cigarettes a day. About half (N = 43; 48%) of the participants reported being physically active for ≤ 5 hours per week, while the other half reported being physically active for > 5 hours per week.

We conducted a series of robust one-way ANOVAs to investigate whether our randomization protocol was successful. Here, we compared the means of age, BMI, levels of chronic stress, vital exhaustion and depression as well as the resting values for self-reported psychological state and biochemical measures and cardiac activity measures across the three conditions, cf. Table 1 and bar charts in Fig. 3. Our results revealed no significant differences between the three conditions (all $p's > .05$), except for age ($p = .02$). According to self-report, 19 women were currently in the follicular phase and 25 in the luteal phase. A χ^2-test revealed no significant differences in menstrual cycle phases between conditions, $\chi^2(2, 44) = 5.36$, $p = .07$.

3.2. Effect of office stress on psychobiological measures

3.2.1. Psychological measures

Results of the robust ANOVAs revealed a significant main effect of the within-subjects factor TIME for $MDMQ_{good\ mood}$, $F(5, 49.57) = 6.38$, $p < .001$, $MDMQ_{calmness}$, $F(5, 51.21) = 12.02$, $p < .001$, $MDMQ_{wakefulness}$, $F(5, 51.17) = 11.57$, $p < .001$, and the perceived stress item, $F(5, 51.67) = 11.29$, $p < .001$. There were no main effects of the between-subjects factor CONDITION, all $F's < 0.51$, all $p's > .05$. We found significant interaction effects of CONDITION × TIME for $MDMQ_{good\ mood}$, $F(10, 44.92) = 3.22$, $p = .003$, $MDMQ_{calmness}$, $F(10, 45.27) = 3.86$, $p < .001$, and perceived stress, $F(10, 45.54) = 3.46$, $p = .002$, but not for $MDMQ_{wakefulness}$, $F(10, 45.16) = 0.85$, $p = .59$. Results of the SAM are consistent with these results and are reported in the appendix (see Table A.1).

Hochberg-corrected robust post-hoc comparisons of the significant interactions of CONDITION × TIME revealed significant differences between the CC and SC1 at t4 for $MDMQ_{good\ mood}$, $\hat{\psi} = 2.67$ [0.25, 5.08], $p = .027$, for $MDMQ_{calmness}$, $\hat{\psi} = 3.83$ [1.49, 6.17], $p < .001$, and for perceived stress, $\hat{\psi} = -0.92$ [−1.70, −0.13], $p = .02$, as well as significant differences between the CC and SC2 at t4 for $MDMQ_{calmness}$ and perceived stress, $\hat{\psi} = 2.42$ [0.02, 4.81], $p = .03$, and $\hat{\psi} = -0.83$ [−1.59, −0.08], $p = .02$ (see Fig. 3a-d).

3.2.2. Biochemical measures

Results of the robust ANOVAs revealed a significant main effect of TIME for salivary cortisol, $F(5, 41.01) = 16.72$, $p < .001$, and sAA, $F(5, 51.51) = 4.61$, $p = .002$. The main effect of CONDITION was significant for cortisol, $F(2, 44.04) = 4.12$, $p = .02$, but not for sAA, $F(2, 45.28) = 0.09$, $p = .92$. The results also revealed significant interaction effects of CONDITION × TIME for cortisol, $F(10, 41.92) = 4.21$, $p < .001$, and sAA, $F(10, 45.28) = 2.61$, $p = .01$.

Hochberg-corrected robust post-hoc tests of the significant main effect of CONDITION for cortisol revealed significant differences between the CC and SC1, $\hat{\psi} = -1.25$ [−1.79, −0.71], $p < .001$, the CC and SC2, $\hat{\psi} = -0.55$ [−0.95, −0.14], $p = .003$, and SC1 and SC2, $\hat{\psi} = 0.70$ [0.13, 1.28], $p = .004$.

Regarding interaction effects for cortisol, the Hochberg-corrected post-hoc tests revealed significant differences between the CC and SC1 at t5, $\hat{\psi} = -2.87$ [−4.70, −1.03], $p < .001$, and at t6, $\hat{\psi} = -1.96$ [−3.29, −0.62], $p = .002$, as well as between the CC and SC2 at t5, $\hat{\psi} = -2.63$ [−4.17, −1.09], $p = .001$, and at t6, $\hat{\psi} = -1.74$ [−2.89, −0.59], $p = .002$. Significant differences for sAA were lost after adjusting for multiple comparisons with the Hochberg correction (see Fig. 3e-f).

3.2.3. Cardiac measures

Results of the robust ANOVAs revealed a significant main effect of TIME for HR, $F(5, 50.82) = 27.11$, $p < .001$, and RMSSD, $F(5, 50.96) = 2.47$, $p = .04$. The main effect of CONDITION was significant for HR, $F(2, 46.34) = 4.78$, $p = .01$, but not for RMSSD, $F(2, 47.41) = 1.52$, $p = .23$. In addition, we found significant interaction effects of CONDITION × TIME for HR, $F(10, 45.95) = 7.44$, $p < .001$, and RMSSD, $F(10, 47.01) = 2.86$, $p = .007$. Results of the frequency domain HRV measures HF, LF and LF/HF are consistent with the RMSSD results with the exception of the lack of a main effect of TIME for LF/HF and are reported in the appendix (see Table A.5).

For HR, the Hochberg-corrected post-hoc tests of the significant main effect of CONDITION revealed significant differences between the CC and SC1, $\hat{\psi} = -6.65$ [−9.50, −3.80], $p < .001$, as well as between the CC and SC2, $\hat{\psi} = -5.03$ [−7.49, −2.57], $p < .001$. No significant difference was found between SC1 and SC2, $\hat{\psi} = 1.61$ [−1.52, 4.74], $p = .22$.

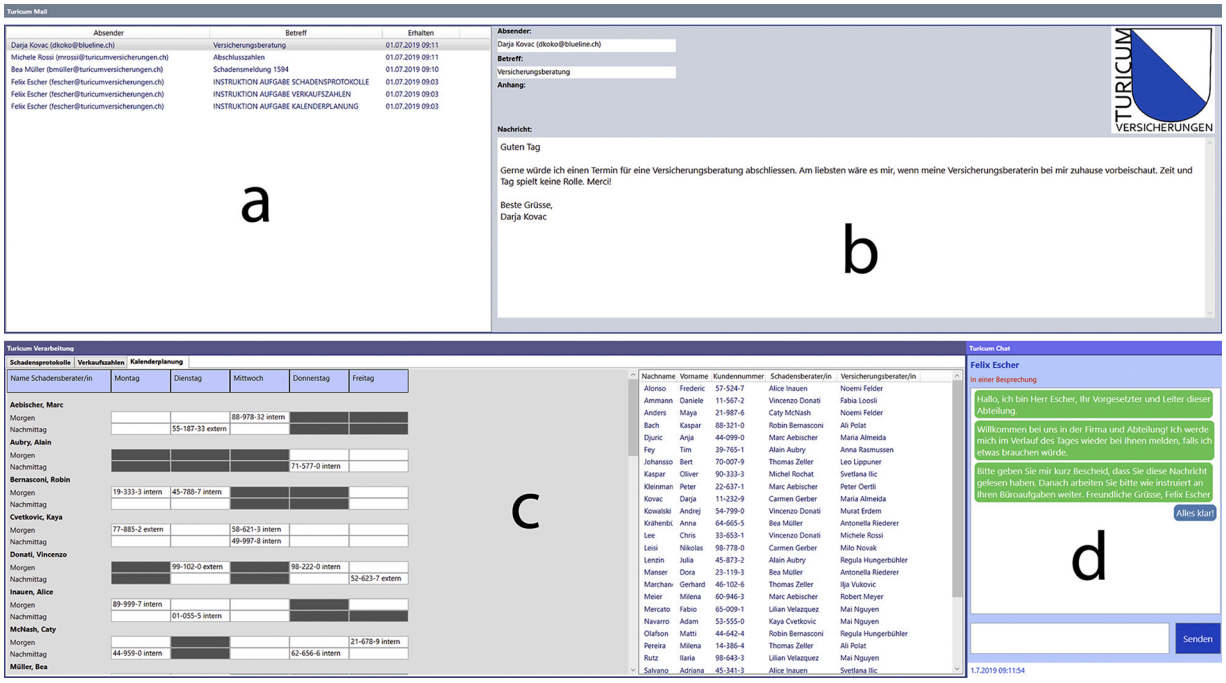

Fig. A.1. Screenshot of the software used for the office tasks. The top left section (a) of the screen presented a list of incoming emails. If an email in the list was selected, the email was displayed in the top right section (b). On the bottom left section (c) participants could switch between three different tabs, depending on the task (i.e., scheduling meetings, digitizing scans, aggregating sales numbers). In the first task, they were asked to copy information from handwritten and scanned claims forms into corresponding digital forms (transcribing task). In the second task, participants received emails with sales numbers from various company employees and were tasked with inserting them in their correct category in a table and calculating the sales per employee (numeric task). In the third task (shown in this screenshot), participants received emails from clients requesting meetings with their respective insurance agents. Here, participants had to identify the responsible agent from a list and schedule their appointment in a calendar (scheduling task). Lastly, the chat window, in which they received messages from their line manager, was located on the bottom right section (d). Note that all names and email addresses were fictitious.

Table A.1
Robust and standard one-way repeated measures analysis of variance of the Self-Assessment Manikin.

source	robust ANOVA			standard ANOVA			
	df1, df2	*F*	*p*	*df1, df2*	*F*	*p*	η^2
$SAM_{valence}$							
Between-subjects effects							
(Intercept)				(1, 87)	837.69	.00	0.86
Condition	(2, 45.99)	0.02	.98	(2, 87)	0.11	.90	0.00
Within-subjects effects							
Time†	(5, 50.76)	9.81	.00	(4.27, 371.79)	11.56	.00	0.05
Between-within-subjects effects							
Condition × time†	(10, 45.41)	1.16	.34	(8.55, 371.79)	1.57	.13	0.01
$SAM_{arousal}$							
Between-subjects effects							
(Intercept)				(1, 87)	1776.15	.00	0.93
Condition	(2, 45.81)	2.51	.09	(2, 87)	2.58	.08	0.04
Within-subjects effects							
Time†	(5, 51.14)	11.04	.00	(3.55, 308.68)	10.61	.00	0.04
Between-within-subjects effects							
Condition × time†	(10, 45.32)	5.33	.00	(7.10, 308.68)	4.92	.00	0.04
$SAM_{dominance}$							
Between-subjects effects							
(Intercept)				(1, 87)	1127.58	.00	0.91
Condition	(2, 45.95)	0.03	.97	(2, 87)	0.07	.93	0.00
Within-subjects effects							
Time†	(5, 51.57)	2.57	.04	(3.96, 344.43)	3.14	.02	0.01
Between-within-subjects effects							
Condition × time†	(10, 45.46)	1.14	.36	(7.92, 344.43)	1.52	.15	0.01

Abbreviations: ANOVA = Analysis of variance; *df* = Degrees of freedom; *F* = F-statistics; *p* = Probability value; η^2 = Partial eta squared; SAM = Self-Assessment Manikin. † = Degrees of freedom and *p*-values of the standard ANOVA are Greenhouse-Geisser corrected.

Table A.2
Standard one-way repeated measures analysis of variance of the Multidimensional Mood Questionnaire and perceived stress.

source	standard ANOVA			
	df1, df2	*F*	*p*	η^2
$MDMQ_{good\ mood}$				
Between-subjects effects				
(Intercept)	(1, 87)	4289.42	.00	0.97
Condition	(2, 87)	0.17	.85	0.00
Within-subjects effects				
Time†	(3.96, 344.09)	10.20	.00	0.03
Within-between subjects effects				
Condition × time†	(7.91, 344.09)	4.06	.00	0.03
$MDMQ_{calmness}$				
Between-subjects effects				
(Intercept)	(1, 87)	2447.56	.00	0.95
Condition	(2, 87)	0.31	.74	0.00
Within-subjects effects				
Time†	(3.91, 340.11)	11.62	.00	0.04
Within-between subjects effects				
Condition × time†	(7.82, 340.11)	6.30	.00	0.05
$MDMQ_{wakefulness}$				
Between-subjects effects				
(Intercept)	(1, 87)	2712.16	.00	0.96
Condition	(2, 87)	0.38	.69	0.01
Within-subjects effects				
Time†	(3.77, 328.00)	18.34	.00	0.04
Within-between subjects effects				
Condition × time†	(7.54, 328.00)	0.77	.62	0.00
Perceived stress				
Between-subjects effects				
(Intercept)	(1, 87)	683.84	.00	0.83
Condition	(2, 87)	0.55	.58	0.01
Within-subjects effects				
Time	(5, 435)	13.15	.00	.05
Within-between subjects effects				
Condition × time	(10, 435)	4.26	.00	0.04

Abbreviations: ANOVA = Analysis of variance; *df* = Degrees of freedom; *F* = F-statistics; *p* = Probability value; η^2 = Partial eta squared; MDMQ = Multidimensional Mood Questionnaire. † = Degrees of freedom and *p*-values are Greenhouse-Geisser corrected.

Regarding the interaction effects, significant differences were found between the CC and SC1 in block 4 for HR, $\widehat{\psi} = -17.09$ [−25.44, −8.75], $p < .001$, and RMSSD, $\widehat{\psi} = 18.75$ [1.27, 36.24], $p = .02$, and in block 6 for HR, $\widehat{\psi} = -6.24$ [−12.60, 0.11], $p = .04$. Moreover, significant differences were found between the CC and SC2 in block 4 for HR, $\widehat{\psi} = -15.33$ [−23.78, −6.87], $p < .001$, and RMSSD, $\widehat{\psi} = 19.16$ [4.07, 34.25], $p = .009$, and in block 6 for HR, $\widehat{\psi} = -6.35$ [−11.57, −1.13], $p = .01$ (see Fig. 3g-h). Mean changes of raw cardiac activity and biochemical measures over time can be found in the appendices A.6 and A.7.

3.3. Effect of office stress on cognitive stress appraisal

Group comparisons regarding the two PASA subscales revealed significant differences between conditions for *threat*, $F(2, 45.93) = 6.91$, $p = .002$, and *challenge*, $F(2, 44.70) = 7.02$, $p = .002$.

For the *threat* subscale, Hochberg corrected post-hoc tests showed that there were significant differences between the CC and SC1, $\widehat{\psi} = -1.21$ [−2.01, −0.41], $p = .002$, and between SC1 and SC2, $\widehat{\psi} = 0.73$ [−0.05, 1.51], $p = .04987$, but not between the CC and SC2, $\widehat{\psi} = -0.48$ [−1.25, 0.29], $p = .13$. Regarding the *challenge* subscale, results revealed differences between the CC and SC1, $\widehat{\psi} = -1.05$ [−1.77, −0.33], $p = .002$, and between the CC and SC2, $\widehat{\psi} = -0.81$ [−1.46, −0.16], $p = .007$. There were no significant differences between the two stress conditions, $\widehat{\psi} = 0.24$ [−0.37, 0.85], $p = .33$. Here again, the standard ANOVAs were in line with the robust results (*threat* subscale: $F(2, 87) = 7.63$, $p < .001$; *challenge* subscale: $F(2, 87) = 8.27$, $p < .001$).

3.3.1. Indirect effects of cognitive stress appraisal

In order to further investigate the role of threat appraisal within the stress conditions and their respective psychobiological stress responses, we ran exploratory non-parametric mediation analyses. Results showed that participants in SC2 scored significantly lower on the PASA *threat* subscale than participants in SC1 (for psychological and biochemical measures with N = 60: $b = -0.66$, $p = .03$; for cardiac measures with N = 59: $b = -0.68$, $p = .02$). Furthermore, our analyses revealed a series of significant indirect effects as indicated by the confidence intervals (see Table 2), albeit with relatively small effect sizes. In particular, there was an indirect effect of stress condition on the peak reactivity and recovery of all psychological measures (i.e., $MDMQ_{good\ mood}$, $MDMQ_{calmness}$, $MDMQ_{wakefulness}$, perceived stress) through threat appraisal.

Compared to experiencing both psychosocial stress and interruptions, experiencing only psychosocial stress led to a stronger increase of bad mood, calmness, wakefulness and perceived stress due to the indirect effect of higher levels of threat appraisal. Similarly, higher levels of threat appraisal indirectly caused participants exposed to psychosocial stress without interruptions to experience a stronger recovery of mood, calmness, wakefulness and perceived stress than those who experienced psychosocial stress and interruptions. There were no significant indirect effects regarding the biological measures (i.e., cortisol, sAA, HR and RMSSD) for either phase of the stress response.

4. Discussion

In this paper, we set out to make four contributions. To begin with, we successfully measured the psychobiological stress responses to realistic office stressors in a controlled group office environment. In particular, we investigated the psychobiological effects of psychosocial stress and work interruptions. We also explored the role of cognitive stress appraisal as a mediating variable between experiencing psychosocial stress with or without prior work interruptions and the psychobiological stress response.

In regard to our first and second contributions, our results confirm that we elicited a psychobiological stress response by simultaneously exposing groups of participants to psychosocial office stress. In

Table A.3
Standard one-way repeated measures analysis of variance of salivary cortisol and salivary alpha-amylase.

source	Salivary cortisol				Salivary alpha-amylase			
	df1, df2	*F*	*p*	η^2	*df1, df2*	*F*	*p*	η^2
Between-subjects effects								
(Intercept)	(1, 87)	355.84	.00	0.72	(1, 87)	262.13	.00	0.64
Condition	(2, 87)	5.82	.00	0.08	(2, 87)	0.05	.95	0.00
Within-subjects effects								
Time†	(2.11, 184.00)	14.69	.00	0.06	(4.05, 352.67)	3.40	.01	0.02
Within-between subjects effects								
Condition × time†	(4.23, 184.00)	7.56	.00	0.06	(8.11, 352.67)	2.56	.01	0.02

Abbreviations: *df* = Degrees of freedom; *F* = F-statistics; *p* = Probability value; η^2 = Partial eta squared. † = Degrees of freedom and *p*-values are Greenhouse-Geisser corrected.

Table A.4
Standard one-way repeated measures analysis of variance of heart rate and heart rate variability.

source	HR				RMSSD			
	df1, df2	*F*	*p*	η^2	*df1, df2*	*F*	*p*	η^2
Between-subjects effects								
(Intercept)	(1, 85)	4780.95	.00	0.98	(1, 85)	281.51	.00	0.76
Condition	(2, 85)	3.77	.03	0.07	(2, 85)	1.09	.34	0.02
Within-subjects effects								
Time†	(1.60, 135.68)	56.62	.00	0.08	(3.15, 268.10)	4.10	.01	0.00
Within-between subjects effects								
Condition × time†	(3.19, 135.68)	14.55	.00	0.04	(6.31, 268.10)	3.38	.00	0.00

Abbreviations: *df* = Degrees of freedom; *F* = F-statistics; *p* = Probability value; η^2 = Partial eta squared; HR = Heart rate; RMSSD = Root mean square of successive differences. † = Degrees of freedom and *p*-values are Greenhouse-Geisser corrected.

Table A.5
Robust and standard one-way repeated measures analysis of variance of frequency domain HRV measures.

source	robust ANOVA			standard ANOVA			
	df1, df2	*F*	*p*	*df1, df2*	*F*	*p*	η^2
HF							
Between-subjects effects							
(Intercept)				(1, 85)	67.06	.00	0.42
Condition	(2, 47.04)	0.62	.54	(2, 85)	0.06	.94	0.00
Within-subjects effects							
Time†	(5, 50.80)	3.14	.02	(2.98, 253.68)	3.87	.01	0.00
Between-within-subjects effects							
Condition × time†	(10, 47.13)	2.36	.02	(5.97, 253.68)	2.30	.04	0.00
LF							
Between-subjects effects							
(Intercept)				(1, 85)	218.70	.00	0.69
Condition	(2, 47.86)	1.48	.24	(2, 85)	1.15	.32	0.02
Within-subjects effects							
Time†	(5, 53.81)	10.59	.00	(3.15, 267.57)	10.93	.00	0.02
Between-within-subjects effects							
Condition × time†	(10, 47.42)	4.26	.00	(6.30, 267.57)	6.51	.00	0.02
LF/HF							
Between-subjects effects							
(Intercept)				(1, 85)	163.37	.00	0.62
Condition	(2, 46.88)	1.59	.21	(2, 85)	1.08	.34	0.02
Within-subjects effects							
Time†	(5, 50.75)	.85	.52	(3.71, 315.47)	0.42	.78	0.00
Between-within-subjects effects							
Condition × time†	(10, 46.97)	2.44	.02	(7.42, 315.47)	2.74	.01	0.01

Abbreviations: ANOVA = Analysis of variance; *df* = Degrees of freedom; *F* = F-statistics; *p* = Probability value; η^2 = Partial eta squared; HRV = Heart rate variability; HF = power in the high frequency range (0.15-0.4 Hz) of the power spectral density of the normal-to-normal heartbeat time series; LF = power in the low frequency range (0.04-0.15 Hz) of the power spectral density of the normal-to-normal heartbeat time series; LF/HF = LF to HF ratio; † = Degrees of freedom and *p*-values of the standard ANOVA are Greenhouse-Geisser corrected.

comparison, the participants subjected to a social interaction without socio-evaluative component showed no increase of stress-related measures. We extend earlier work (e.g., von Dawans et al., 2011; Wekenborg et al., 2019; Boesch et al., 2014) by demonstrating that an adapted version of the TSST-G in a controlled but realistic office environment is capable of eliciting psychobiological stress responses, whereas a more friendly interaction in the CC shows no such activation (cf. Wiemers et al., 2013).

We believe that the extent of the stress-induced changes over time we have observed may be compared to studies using the TSST in a group setting (von Dawans et al., 2011; Boesch et al., 2014; Childs et al., 2006; Domes et al., 2019; Wekenborg et al., 2019). In line with these previous studies, we found significantly higher levels of perceived stress, greater decreases of calmness as well as a significant worsening of mood in the stress conditions compared to the CC. These changes were accompanied by increased levels of salivary cortisol at t5 and t6. However, the salivary cortisol increases in our stress conditions were less pronounced than in other TSST-G studies and closer to changes observed in Childs et al.'s (2006) single TSST setting. The differences in the TSST-G protocols (i.e., length of phases, content, free speech time, number of participants and type of control condition, sitting or standing during the TSST-G, additional work interruptions) may have affected the extent of induced changes, which is why comparisons for all measures should be drawn with caution. HR levels in the stress conditions also increased significantly compared to the control condition and even remained significantly increased in block 6. The heart rate reactivity observed in our study is situated between the changes reported by Boesch et al. (2014) and von Dawans et al. (2011). The stress conditions also showed significantly lower RMSSD values during the psychosocial stress test, comparable to the observations of Boesch and colleagues (2014). The relatively small decrease in RMSSD for SC1 and SC2 has to be considered in relation to a strong increase of RMSSD in the CC during the social interaction. This HRV increase in the control condition during the friendly version of the psychosocial stress test is, to the best of our knowledge, a novel observation, since previous TSST-G studies have either lacked a comparable control condition or not assessed any HRV measures. We argue that the HRV increase in the CC might be attributed to synchronized breathing from reading out loud (Bernardi et al., 2000).

For the remaining measures, we found no significant group differences. Wakefulness decreased gradually for all conditions over the course of the experiment. We believe that this decrease may be ascribed to a fatiguing experimental setting and a dominant circadian rhythm (Beersma and Gordijn, 2007). With regard to sAA, comparisons between the control and the stress conditions after the social interaction did not survive statistical correction for multiple comparisons. Similarly, Wiemers et al. (2013) found no differences in sAA between the TSST and a friendly version, revealing an SNS but not an HPA axis activation in the non-threatening CC. Differences in salivary cortisol and sAA output are also partly owed to their inherent diurnal pattern (Nater et al., 2007).

With respect to our third contribution, we found that the effect of work interruptions on the psychobiological stress response could not be discerned conclusively. While participants in SC2 had a significantly

Table A.6
Means (standard deviations) per experiment condition and block for raw cardiac activity measures.

	Block 1		Block 2		Block 3		Block 4		Block 5		Block 6	
Mean of NN intervals [ms]												
CC	866.66	(97.39)	874.93	(93.48)	897.17	(101.48)	908.36	(94.45)	920.93	(99.53)	929.43	(95.37)
SC1	834.21	(143.00)	848.64	(140.89)	844.71	(148.20)	756.85	(157.85)	870.13	(138.15)	865.48	(139.30)
SC2	852.68	(129.48)	861.70	(130.96)	858.01	(125.56)	773.59	(160.08)	875.71	(140.89)	861.56	(135.39)
Standard deviation of NN intervals [ms]												
CC	69.66	(21.50)	69.05	(22.08)	74.12	(27.49)	105.46	(28.49)	75.20	(21.78)	79.76	(27.06)
SC1	65.03	(24.55)	61.72	(23.84)	67.55	(25.14)	105.25	(34.03)	73.92	(27.53)	69.15	(26.26)
SC2	71.37	(24.42)	70.06	(25.83)	73.73	(23.67)	108.96	(32.03)	76.76	(21.95)	73.47	(23.25)

Abbreviations: NN intervals = Time between filtered and corrected (i.e., Normal-to-Normal) heartbeats; Block 1 = Resting phase; Block 2 = Interruptions phase; Block 3 = Anticipation phase; Block 4 = Psychosocial stress phase; Block 5 = Recovery phase 1; Block 6 = Recovery phase 2; CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2.

Table A.7
Means (standard deviations) per condition and time point for biochemical measures.

	t1		t2		t3		t4		t5		t6	
Salivary cortisol (nmol/l)												
CC	3.70	(1.93)	3.12	(1.42)	2.63	(1.23)	2.38	(1.44)	2.20	(1.12)	2.08	(1.08)
SC1	4.58	(2.94)	3.59	(1.91)	3.52	(2.16)	3.70	(3.12)	5.57	(4.16)	4.16	(2.62)
SC2	3.46	(1.76)	2.75	(1.31)	2.39	(1.10)	2.58	(1.24)	4.95	(2.76)	3.89	(2.09)
Salivary alpha-amylase (U/ml)												
CC	164.36	(139.92)	201.37	(154.55)	133.10	(116.10)	150.02	(138.54)	162.28	(138.32)	204.50	(165.06)
SC1	149.70	(122.71)	140.06	(115.76)	157.29	(126.33)	208.85	(168.73)	187.69	(137.72)	200.24	(121.85)
SC2	173.90	(118.19)	148.84	(105.79)	165.27	(123.62)	230.27	(132.85)	174.40	(116.08)	173.79	(107.00)

Abbrevations: nmol/l = Nanomole per liter; U/ml = Units per milliliter; t1 = Resting phase; t2 = Interruptions phase; t3 = Anticipation phase; t4 = Psychosocial stress phase; t5 = Recovery phase 1; t6 = Recovery phase 2; CC = Control condition; SC1 = Stress condition 1; SC2 = Stress condition 2.

higher overall output of salivary cortisol compared to SC1, we did not find significant differences between the two stress conditions at any specific time point. The significant main effect indicates that being interrupted by additional office tasks during the anticipation of a stressful social situation intensifies the activation of the HPA axis. Previous studies found interruptions at work to be irritating and strenuous (Baethge and Rigotti, 2013). However, we were unable to infer a stress-eliciting effect of work interruptions independent of psychosocial stress (i.e., see t1 to t2 in Fig. 3). A possible explanation could be that interruptions were not sufficient to lead to overt changes of stress, both alone and in combination with the psychosocial stress. Indeed, some studies have shown that interruptions may not always elicit stress if their content is related to the main task (Czerwinski et al., 2000). The basic workload in all conditions might also have increased the overall stress of all participants, making it harder to detect an effect of interruptions.

Regarding our fourth contribution, we found significant differences between SC1 and SC2 in appraisal of the psychosocial stress situation. Participants who experienced work interruptions appraised the upcoming socio-evaluative job interview as less threatening than participants who were not interrupted. Additionally, while both stress conditions viewed the social evaluation as significantly more challenging than the CC, we did not find significant differences between the two stress conditions. One explanation for the lower threat appraisal in SC2 might be that the chat interruptions increased the participants' sense of familiarity (cf. Tops et al., 2013) with their employer. This increased engagement with the content of their work and the social interaction via the chat function may have elevated feelings of certainty and control. Compatibly, Gaab et al. (2005) found decreased internal control expectancy to be linked to higher threat appraisal. A second explanation could be that the chat messages acted as a distraction from focusing on the upcoming job interview. Distractions have been previously linked to a reduced depressive mood in healthy subjects (Trask and Sigmon, 1999).

In order to better understand the association between work interruptions, cognitive appraisal and the psychobiological stress response, we performed exploratory mediation analyses. These analyses revealed significant indirect effects for $MDMQ_{good\ mood}$, $MDMQ_{calmness}$, $MDMQ_{wakefulness}$, and perceived stress through threat appraisal. A mediating variable can play an explanatory role even in the absence of a prior established association between the independent and dependent variables (Mathieu and Taylor, 2006). As such, these results are not contradictory to our findings from the ANOVAs. Instead, our results indicate that threat appraisal may be a linking mechanism (intervening variable) instead of a classical mediator (cf. Mathieu and Taylor, 2006). Indeed, our analysis showed that the lower threat appraisal of SC2 led to a lower peak reactivity of bad mood, nervousness, sleepiness and perceived stress, and to a weaker recovery of mood, calmness, wakefulness and perceived stress. These results seem to suggest a blunted psychological response in SC2, which is further supported by some descriptive evidence. While participants in both stress conditions experienced a worsening of mood throughout the experiment, this decrease was less acute for SC2. Similarly, participants in SC2 were less nervous during the psychosocial stress test compared to those in SC1 (see Fig. 3a-b). As for perceived stress, mean levels rose more strongly during the interruption and anticipation phase in SC2 compared to SC1 and the CC. Yet the peak response to the psychosocial stress in SC2 and SC1 was comparably high (but still below the high end of the scale). During recovery, perceived stress levels in SC2 remained higher than in SC1. Taken together, while there was an initial increase in SC2, the subsequent response of perceived stress also appears to be blunted (see Fig. 3d).

Results of our mediation analyses provided no evidence that threat appraisal predicted or mediated any biological response (i.e., cortisol, sAA, HR or RMSSD peak reactivity or recovery). Our findings regarding cortisol deviate somewhat from Gaab et al. (2005), who found that the PASA *threat* subscale explained 30% of the variance in cortisol reactivity. We believe that this discrepancy may be due to differences in the design and protocol of the two experiments. First, participants in our experiment went through a shorter version of the TSST-G. Here, while our adapted version was effective in eliciting stress, the stress response was not as pronounced compared to Gaab and colleagues. Second, the protocol used by Gaab et al. (2005) allowed for collection of cortisol over a longer recovery period.

To summarize, introducing work interruptions led to a main effect of HPA axis activation but of no other psychological or biological measure.

Mediation analyses revealed an indirect effect of work interruptions on all psychological measures through threat appraisal, albeit a blunting rather than a heightening effect. Contrary to the psychological and HPA axis responses, the ANS did not seem to be significantly affected by the additional presence of work interruptions. Overall, there seems to be a desynchrony between the two major stress systems but also discordant changes in the HPA axis and subjective indicators of stress. Indeed, Campbell and Ehlert (2012) pointed out that a certain desynchrony might exist regarding the stress reactivity of psychophysiological systems due to various mediating and moderating factors. Our results suggest cognitive stress appraisal to be one of these factors. We conclude that assessing biological and psychological stress responses in conjunction is essential for the further investigation of possible desynchronies between different measures. Such discordances could potentially be dangerous if they lead to some detrimental stress responses going unnoticed. Moreover, psychobiological desynchronies pose challenges regarding interpretation. In our case, the heightened HPA activation and lower threat appraisal accompanied by lower subjective stress levels could also reflect a state of eustress rather than distress. Thus, including moderating or mediating factors opens the way to a more holistic understanding of the mechanisms underlying an individual's response to office stress.

There are a series of limitations associated with our experiment. Despite our best efforts to create a realistic office environment, the experiment was conducted in a laboratory and may not fully reflect work in a real office. First, participants took up the role of an office employee for a couple of hours, which may be insufficient to critically assess the effects of work stress. As such, this study consists of a single session, cross-sectional assessment of stressors and their effects. Second, the periodic collection of saliva and questionnaires interrupted the workflow, and may have reduced participants' immersion and identification as office employees. Third, the psychosocial stress situation depended on the actors' abilities to convincingly portray human resources personnel. Fourth, participants of this study were sampled from a young, educated, healthy and Caucasian subject pool and therefore caution should be taken when generalizing to older, less educated, clinical populations or other ethnicities. Fifth, the mediation analyses and respective effect sizes may be too small to discuss practical implications of the results. Sixth, the task of the CC might not reflect a real-world office scenario, as reading out in unison, especially in front of an HR employee, is usually not part of office work. Seventh, employees in a real office often have personal relationships with their co-workers, while we assume that participants did not know each other (though this was not explicitly assessed). Interpersonal factors such as the type of personal relationship and a shared social identity are known to affect the stress response (Frisch et al., 2015; Häusser et al., 2012). Eighth, participants were sampled from a population that consisted mainly of university students. Although their actual work experience was not assessed, it can be expected to be rather limited. Finally, we found comparably low levels of chronic stress, yet still in the range of other healthy samples (cf. Noser et al., 2018), but substantial levels of vital exhaustion (Kopp et al., 1998). Importantly, these two constructs are related yet still distinct from each other (Schoch et al., 2018). We believe the lower levels of chronic stress are owed to the rather work-specific items of the TICS which might apply less to students than to the more typical employee. The substantial levels of vital exhaustion may reflect their studying and examination period, which had been going on for about a month at the point of the experiment. The TICS (Schulz et al., 2004) on the other hand assesses chronic stress experienced during the last three months. Levels of depressive symptoms were comparable to norms of healthy students and adults in general (Franke, 2000), as were the basal values of the repeated psychobiological measures (Bradley and Lang, 1994; Backs et al., 2005; Handayani et al., 2015; Nater et al., 2007; Steyer et al., 1997; Hinz et al., 2012; La Marca et al., 2011; Nunan et al., 2010; von Dawans et al., 2011).

Future research could address some of these limitations. For example, experiments could be conducted in real-world offices where participants regularly face a variety of stressors. These experiments would have the advantage of generating longitudinal data that can be used to answer questions regarding long-term effects of stress on various other outcomes including different measures of chronic stress, sleep quality, well-being and quality of life but also organizational outcomes such as absenteeism, presenteeism and productivity. They might also be able to capture the effects of interpersonal relationships between co-workers on their stress response. The advent of new technologies including unobtrusive wearables and smartphone applications makes these types of studies more feasible. Nonetheless, experiments in laboratory settings can provide noteworthy contributions by offering the necessary control to isolate more specific effects of stress. Here, future studies should consider extending the recovery phase, in order to properly capture the timecourse of stress. In general, laboratory research on office stress would benefit from a standardized realistic stress elicitation protocol, for example by further validation of our adapted version of the TSST-G. We also see great value in extending laboratory research on acute to chronic work-related stress by including office employees at risk of or suffering from chronic stress-related mental and physical illnesses and disorders.

5. Conclusion

This study is the first to investigate psychobiological stress responses to different factors of office stress in a controlled yet realistic group office environment. We found that an office version of the TSST-G induced significant increases in the psychological, biochemical and cardiac stress responses. We also found that additional work interruptions led to an even greater HPA axis activation. In contrast, the psychological stress response seemed to be blunted by work interruptions. The heightened HPA axis and blunted psychological response suggest a stronger physiological mobilization, providing the organism with extra energy and therefore increasing emotional but also cognitive resources. This interpretation is supported by the observed lower levels of cognitive threat appraisal that partially explained this psychobiological discordance. Our results stress the importance of studying different types of office stressors and the differential effects they can have on various stress-relevant systems. Overall, our study demonstrates the feasibility of a large-scale and realistic stress provocation experiment in the context of work stress. The range of assessed measures and various types of work stressors allow for the investigation of numerous interacting factors and variables. Future research may adopt this setting to address similar questions in different age groups of the workforce or in high risks groups with high levels of clinically relevant symptoms of work stress-related diseases and disorders.

CRediT authorship contribution statement

Jasmine I. Kerr: Methodology, Validation, Formal analysis, Investigation, Resources, Data curation, Writing - original draft, Visualization, Project administration. **Mara Naegelin:** Methodology, Validation, Formal analysis, Investigation, Resources, Data curation, Writing - original draft, Visualization, Project administration. **Raphael P. Weibel:** Methodology, Software, Validation, Investigation, Data curation, Writing - original draft, Visualization, Project administration. **Andrea Ferrario:** Conceptualization, Methodology, Investigation, Writing - review & editing, Supervision, Project administration, Funding acquisition. **Roberto La Marca:** Conceptualization, Methodology, Resources, Writing - review & editing, Supervision, Funding acquisition. **Florian von Wangenheim:** Investigation, Writing - review & editing, Supervision, Funding acquisition. **Christoph Hoelscher:** Writing - review & editing, Funding acquisition. **Victor R. Schinazi:** Conceptualization, Methodology, Writing - original draft, Writing - review & editing, Supervision, Project administration, Funding acquisition.

Declaration of Competing Interest

The authors report no declarations of interest.

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Appendix A

Figure A.1
Tables A.1–A.7

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