Stress effects on endogenous and exogenous visuospatial attention.

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December 8, 2021

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Abstract

In this study we investigated the influence of stress on covert visual attention. Current research yields mixed results concerning their relationship with findings suggesting supportive as well as diminishing stress effects on attention. A framework by Hermans et al. (2011, 2014) proposes a stress induced network shift that promotes bottom-up at the cost of top-down mechanisms approximately for one hour following stress exposure. Participants were exposed to bilateral feet Cold Pressor Tests (CPT) repeatedly triggering activation of the sympathetic adrenomedullary system (SAM) and the hypothalamus-pituitary-adrenal axis (HPA) supposedly initiating the proposed network shift. Following the CPTs, participants performed a variant of the Posner cueing task in which central and peripheral cues were combined to assess endogenous and exogenous attention processes and their interaction. Behavioral results showed no differences between stress and control group regarding endogenous attention processes. This was supported by means of the EEG, where stressed participants showed no difference to controls in posterior alpha lateralization following endogenous cues. However, the stress group showed worse behavioral performance in trial conditions associated with exogenous processes respectively in trials where endogenous and exogenous cues interacted. In relation to Hermans framework, the behavioral and EEG evidence found in this study suggests stress effects that point away from a pure impairment of endogenous attention processes. Rather, the behavioral measures indicate that the stress response affects the interaction of exogenous and endogenous attentional mechanisms.

Keywords: Stress, Covert Visual Attention, Posner Task, Endogenous, Exogenous. CPT, HPA, SAM, EEG.

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Introduction

Stress is part of everyday life for many people and has widespread acute and chronic consequences. While acute effects of stress are considered adaptive in an evolutionary context, stress can also have a detrimental effect, i.e., in safety relevant fields of work, when unforeseen situations with potentially harmful consequences require error-free actions. Indeed, such observations mark the beginning of stress research on cognition. After the second world war it was discovered that even highly skilled pilots made erroneous decisions during stressful situations (Broadbent, 1971 as cited in Arnsten, 2009; Hermans et al., 2014). More recently, research has uncovered how physiological changes induced by stress can affect brain functioning and thereby modulate information processing and higher-order cognition. This thesis will focus on how stress affects a particular cognitive function that lies at the heart of volitional thought and behavior, namely selective attention.

In the following chapters, attentional processes will be elaborated in more detail first. Subsequently, stress and the physiological stress response will be described. Finally, stress effects in the brain and especially on mechanisms associated with attentional processing will be outlined.

Attention

Attention enables us to selectively process the large amount of information that continuously impinges on our sensory organs. This selection process is necessary to enable goal-oriented behavior in a changing environment but presumably also arises from the energy cost of neuronal activity and the limited amount of energy available to the brain (Carrasco, 2011; Chica et al., 2013). When looking at a visual scene, two basic phenomena occur while processing it. First, not all of the visual input can be processed simultaneously, indicating some form of limited capacity. Second, while processing a specific aspect of the visual scene, unwanted information within the scene is filtered out, showing a selection process (Beck & Kastner, 2009). Based on these two phenomena, Desimone and Duncan (1995) derived their biased competition theory of selective attention. Their proposition that limited processing capacity results in competition for representation of visual information in the visual cortex is supported by many behavioral (e.g. Duncan, 1984), single-cell recording (e.g. Reynolds et al., 1999), and neuroimaging studies (e.g. Beck & Kastner, 2005). This competition between visual information is thought to be influenced by the interaction of two independent attentional mechanisms, endogenous or top-down attention, and exogenous or bottom-up stimulus-driven attention (Chica et al., 2013; Desimone & Duncan, 1995). Both endogenous and exogenous

attention produce comparable behavioral effects in detection or discrimination tasks for example in the form of lower reaction times (RT) and increased accuracy for attended compared to unattended targets. However, these two attentional mechanisms are associated with two overlapping, but partially segregated brain networks (Chica et al., 2013; Corbetta & Shulman, 2002; Muller & Rabbitt, 1989) and differ in the way they are triggered.

Endogenous spatial attention can be oriented voluntarily and is evoked by cognitive task demands, for example when an observer expects a relevant stimulus to appear at a specific location (Beck & Kastner, 2009). In neuroimaging studies it was shown that when deploying endogenous attention to a specific location, baseline activity in all visual areas with a representation of the attended location is enhanced even before any visual stimulus is presented there (Kastner et al., 1999). In another fMRI experiment, Kastner et al. (1998) showed that deploying endogenous attention to a specific stimulus within a visual quadrant of competing stimuli counteracts the usually observed suppressive influence of the competing stimuli. In contrast to the endogenous deployed attention, the basis of exogenous attention lies in the stimulus itself. Exogenous attention reduces the suppressive interactions between visual stimuli, based on the stimulus' salience within its visual context, which depends on visual features like brightness, contrast, orientation, and speed (Beck & Kastner, 2005, 2009). Another important difference is their time course. Endogenous attention has a slower onset than exogenous orientation (approx. 300 ms vs. 100 ms) but can be deployed longer whereby exogenous attention effects are not sustained over a longer time period and start to vanish after approximately 300 ms (Chica et al., 2013).

Exogenous attention is associated with a ventral fronto-parietal network that detects salient and behaviorally relevant stimuli in the environment (Chica et al., 2013; Corbetta et al., 2008; Corbetta & Shulman, 2002). The core regions of this network include the temporo-parietal junction, the ventral frontal cortex together with parts of the middle frontal gyrus, the inferior frontal gyrus, the frontal operculum, and the anterior insula. Endogenous attention is associated with a dorsal fronto-parietal network whose core regions include the dorsal parietal cortex, particularly the intraparietal sulcus and superior parietal lobe, and the dorsal frontal cortex along the precentral sulcus, near or at the frontal eye field (Chica et al., 2013; Corbetta et al., 2008; Corbetta & Shulman, 2002). Preparatory activation of this network, for example when expecting to see an object at a specific location, is extended to the visual cortex, presumably indicating top-down modulation of sensory representations.

Using electroencephalography, deployment of endogenous attention has been linked to α-Band activity (~8-12 Hz) over the occipital cortex (Thut et al., 2006; Worden et al., 2000). When endogenous attention is focused at a specific location in the left or right hemifield, posterior alpha power decreases over the contralateral hemisphere, while it increases over ipsilateral sites (α-lateralization). The level of α-lateralization has been related to behavioral measures, for example it was shown that increased α-lateralization is accompanied by increased speed of target detection (Thut et al., 2006). While in the past it was assumed that decreased alpha power represents signal enhancement of visual information, today alpha activity is commonly believed to enable spatial selection through suppression of irrelevant information in the unattended hemifield (e.g. Foster & Awh, 2019; Pfurtscheller et al., 1996; Thut et al., 2006). However recent evidence indicates independent mechanisms for distractor suppression and target facilitation (Noonan et al., 2016). In a variant of the Posner paradigm, Noonan and colleagues either cued the location of the target, the distractor, or provided no predictive information. While the reduction of α-band activity corresponded to the location where targets were cued, they could not relate α-band activity to the distractor location (in trials with predictive information about distractor location), indicating that alpha-band activity is associated with spatial selection, but not with distractor suppression.

Stress

The human stress response is triggered when a stimulus – a stressor – is perceived to be an anticipated or actual threat to the homeostasis (Ulrich-Lai & Herman, 2009). In response to a stressor, multiple interacting stress mediators are released, targeting peripheral organs as well as specific neuronal populations that launch downstream effects aiming to provide resources for a rapid and appropriate response to the stressor (Joels & Baram, 2009).

The paraventricular nucleus of the hypothalamus (PVN) is the central actor in the stress response as it integrates ascending information about body functions from the brainstem (e.g., information about physical stressors like extreme cold) and descending information from the limbic system that may forward information regarding psychological stressors (Ulrich-Lai & Herman, 2009). The PVN triggers activation of the two main branches of the stress response, the sympathetic nervous (SNS) and sympathetic adrenomedullary systems (SAM) and the hypothalamic-pituitary-adrenal axis (HPA) that interact through complementary actions throughout the body (Marin et al., 2019).

The stress response of the SAM starts within seconds after the onset of a stressor (Ulrich-Lai & Herman, 2009). When exposed to a stressor, preganglionic sympathetic neurons

in the intermediolateral cell column of the thoracolumbar spinal cord are activated. These preganglionic sympathetic neurons activate chromaffin cells in the adrenal medulla (via pre- or paravertebral ganglia). After activation, the chromaffin cells secrete the catecholamines adrenaline (ADR) and noradrenalin (NA) which cause widespread effects at different target sites e.g., increasing the heart rate and its force of contraction. The stress response of the SAM is ephemeral though and is counteracted by reflex parasympathetic activation and rarely outlasts the duration of stressor exposure (Joels & Baram, 2009; Ulrich-Lai & Herman, 2009).

Besides the catecholaminergic stress response of the SAM, the hypothalamus starts a second, slower stress response via the HPA axis (Marin et al., 2019; Tsigos & Chrousos, 2002). Parvocellular neurons of the PVN release the peptide corticotropin-releasing-hormone (CRH) that binds to receptors in the anterior pituitary, which then releases adrenocorticotropic hormone (ACTH) into the bloodstream. Circulating ACTH binds to receptors on the cortex of the adrenal glands which then release glucocorticoids (GCs), mainly cortisol in humans approximately 10 minutes after the stressor. The level of corticosteroids released through the HPA axis is downregulated by multiple mechanisms to avoid excessive GC concentration and terminate the stress response.

Regarding stress effects in the brain, NA and cortisol are the most relevant mediators for the effects explored in this study. While the effect of NA is spatially restricted through its available pathways and binds to adrenergic α- and β-receptors, unbound cortisol can produce effects throughout the whole brain after it passed the blood-brain-barrier. The characteristics and timing of cortisol effects depend on the distribution of its two main receptor types – glucocorticoid (GR) and mineralocorticoid (MR) (Joels, 2018; Joels & Baram, 2009). GR and MR receptors show high density in multiple brain areas especially the hippocampus, the amygdala, and the PFC (Maggio, 2019). While for some time it was assumed that cortisol only produces slow gene mediated effects (genomic effects) via protein synthesis through nuclear receptors, it has recently been demonstrated that they also exert rapid nongenomic effects via membrane receptors (Joels, 2018; Maggio, 2019). In the next chapter specific mechanisms of NA and cortisol will be outlined regarding their potential influence on attentional processes and recently proposed effects on large scale neural network reconfiguration.

Stress and Attention

Especially in dangerous and stressful situations, attentional mechanisms have an important role in focusing on stimuli that are relevant for successfully handling the occurring circumstances. There are various studies indicating stress alters prefrontal brain activity (e.g. Qin et al., 2009;

Weerda et al., 2010). One group of researchers proposed that the release of catecholamines and cortisol triggers a shift to salience-based processing, while inhibiting top-down processes (Arnsten, 2009; Arnsten et al., 2015; Hermans et al., 2014). This upregulation of the salience network and downregulation of the executive control network is proposed to be carried by rapid catecholaminergic and nongenomic cortisol effects, while genomic cortisol effects reverse the network organization to enhance executive control functions following the stressor.

Fundamental to the proposition of this large scale network shift, are two fMRI experiments by Hermans et al. (2011). While participants were exposed to highly aversive video material that successful triggered a physiological stress response (as seen in elevated salivary cortisol and alpha amylase), participants showed increased neural activity in regions normally being activated by salient stimuli. Furthermore they found that the topology of these regions showed a large overlap with the ventral fronto-parietal attention network associated with exogenous attention by Corbetta et al. (2008). The functional connectivity of this salience network also positively correlated with cortisol and alpha amylase indicating that the network shift is also dependent on the strength of the stress response. To investigate which neuromodulators drive this network shift, in a second experiment participants received either a β-adrenergic receptor blocker, a cortisol synthesis blocker, or a placebo. They successfully isolated noradrenergic and cortisol effects, as seen in saliva measures, and found that functional connectivity strength was significantly reduced in the group that received the β-adrenergic receptor blocker compared to the cortisol-blocker and control group, indicating noradrenaline as the major modulator of the salience network. As physiological mechanisms carrying this network shift, they proposed stress induced altered activity of the locus coeruleus (LC), and multiple effects impairing PFC functioning (Arnsten, 2009, 2015; Hermans et al., 2014; Hermans et al., 2011).

The LC is closely linked with the ventral attention system, and LC neurons exhibit both tonic and phasic activity modes (Corbetta et al., 2008). It was proposed that CRH released during the stress response from the central amygdala, shifts LC activity from a phasic mode that is associated with focused task execution, to a high tonic mode that releases large concentrations of NA and is related to distractibility and hypervigilance (Hermans et al., 2011).

Furthermore, it is proposed that the network switch is carried by NA and cortisol impairing the PFC (Arnsten, 2009; Hermans et al., 2014). In the primate dorsolateral PFC (dlPFC), NA bind to α1-adrenoceptor that activate protein kinase C signaling, which reduces delay-cell firing in the dlPFC where mental representations are generated needed for working

memory and top-down control. Glucocorticoids can potentially accentuate the effects of NA in the PFC by blocking glia plasma membrane transporters that normally remove catecholamines from the extracellular space thus coordinating and amplifying the network switch (Arnsten, 2015).

Genomic effects of cortisol that take at least one hour to initiate are proposed to oppose the first wave to of the stress response and enhance PFC function subsequent to the stressor (Hermans et al., 2014). This proposition is based on findings in rats where GR-mediated genomic stress effects enhance PFC function and working memory (Karst & Joëls, 2005).

Connecting this stress induced network shift with attentional mechanisms, current evidence indicate mixed results ranging from heightened distractibility by salient stimuli (Sänger et al., 2014) to lowered distractibility (Hoskin et al., 2014; Plessow et al., 2011), reduced attentional blink effects (Schwabe & Wolf, 2010) and no stress effects on inhibition of return (Larra et al., 2016). This shows that more work is needed to understand the specific stress effects on top-down and bottom-up attentional mechanisms. While Sänger et al. (2014) specifically looked at these attentional mechanisms in their experimental paradigm, the timing of the stress induction potentially mixes nongenomic and genomic stress effects which could exert opposing effects on attentional mechanisms.

Aim of this study

In order to further sharpen the impact of stress on attention and isolate potentially opposing stress effects, this study was aimed at nongenomic stress effects on covert spatial attention. In order to do this, stress and spatial attention were manipulated using a bilateral feet cold pressor test (CPT) (Larra et al., 2015) and a variant of the Posner paradigm (Posner et al., 1980).

The CPT is a method to reliably induce HPA axis activation, starting the above described nongenomic and genomic stress effects. HPA axis activation should show in significantly increased saliva cortisol, heart rate (HR), and blood pressure (BP) in the experimental group compared to the control group. In the control condition, a warm water feet bath was used to ensure comparability between the groups but not induce stress effects, as has been done reliably in the past (e.g. Larra et al., 2016). Furthermore, subjective markers e.g., perceived stress, arousal, and pain were measured using visual analogue scales (VAS). To isolate catecholaminergic and nongenomic from genomic stress effects, the duration of the experimental paradigm was limited to one hour, the earliest point in time at which genomic stress effects start to show (e.g. Hermans et al., 2014). To ensure participants were stressed throughout the duration of the whole experimental paradigm, and catecholaminergic and

nongenomic stress effects do not fade out, the CPT procedure is repeated prior to each experimental block, i.e., approximately every 20 minutes.

The Posner paradigm allows to manipulate endogenous and exogenous attention by presenting different stimuli to the participants (Chica et al., 2013). To induce endogenous attention, central predictive cues are used that reliably indicate the location of a target stimulus. Exogenous attention is deployed by showing peripheral nonpredictive cues that correspond to the target location in 50% of the trials. In this study, directional and neutral central cues, and peripheral non-predictive cues were presented together in trials, making it possible to investigate interactions between endogenous and exogenous attention. To operationalize stress effects on attention, behavioral and EEG measures will be used. As described above, deployed attention in the Posner paradigm shows in decreased RT and improved detection accuracy of target stimuli. Regarding EEG measures, deployment of endogenous attention will be examined using the lateralization index of posterior alpha power (Thut et al., 2006).

The research question of this study is whether *spatial covert attention is modulated by catecholaminergic and nongenomic stress effects in a bottom-up promotion order.* Based on the proposed network shift by Hermans and colleagues (2014), in the experimental group bottom-up mechanisms were expected to improve, while top-down mechanisms would be impaired. This would show in improved RT and response accuracy for exogenous trials, and in reduced behavioral performance in endogenous trials as well as in reduced lateralization of alpha power in the experimental group compared to the control group.

Methods

Sample

48 healthy, right-handed men and women (N female = 28) aged between 18 and 39 years (M_{age} = 22.5 years, SD_{age} = 3.5 years) participated in the experiment. They were randomly assigned to either the stress (N = 23, female = 12) or control condition (N = 25, female = 16). Sex was balanced in the whole sample and within experimental conditions. 7 participants were excluded from the final analysis due to non-compliance with instructions (i.e., eye movements in >35% of trials), reducing the final sample size to N = 41 (N = 22 control, female = 14). Seven other subjects were excluded only from EEG analyses due to heavy artifact contamination. Three participants were excluded only from cortisol analyses (2 due to missing saliva measurements, one due to extreme outliers ($\sigma > 5$). Eight participants were excluded from analysis of cardiovascular measures due to missing values.

Participants recruited through social media and a student subject database thus were mostly students from the Technical University of Dortmund. They were compensated for their participation with either 50€ or test subject credits. Data were collected at Leibniz Research Centre for Working Environment and Human Factors in Dortmund, Germany from 2nd September 2020 to 28th May 2021.

In a preliminary screening interview, it was ensured that participants met all criteria for inclusion in the study. Participants were included if they were between 18 and 35 years old, right-handed, had (corrected to) normal visual acuity (less than 4 diopters, no astigmatism), and had normal weight (Body Mass Index between 19 and 25). Also, it was checked whether their body size allowed them to sit in the laboratory setup (height between 1.55 and 2 meters, maximum shoe size of 47 (EU)). Participants were excluded if they abused drugs or other substances, used medication or psychotropic substances (e.g. Ritalin) except for occasional use of non-prescription medication (e.g. Aspirin) until two days prior to the experiment, showed any evidence of acute or chronic diseases (Raynaud's disease, arterial hypertension, history of fainting, acute or anamnestic dermatosis), had a family history of arterial hypertension, and cerebral or aortic aneurisms, or had injured feet (open wounds, burns, infections). Furthermore, participants were excluded if they smoked more than five cigarettes per day, had an increased sensitivity to cold, or participated in pharmaceutical studies within the last three months. None of the participants had been involved in an experiment with a cold-pressor-test before. Caffeinated and alcoholic drinks, physical exercise, and meals were not permitted in the two hours immediately preceding the experiment.

The study was approved by the ethics committee of the IfADo and complies with the Declaration of Helsinki. After being informed about the procedure and their right to stop the experiment, participants gave their written informed consent (Appendix I). During the experiment, it was ensured that a physician was available in case of any medical incidents.

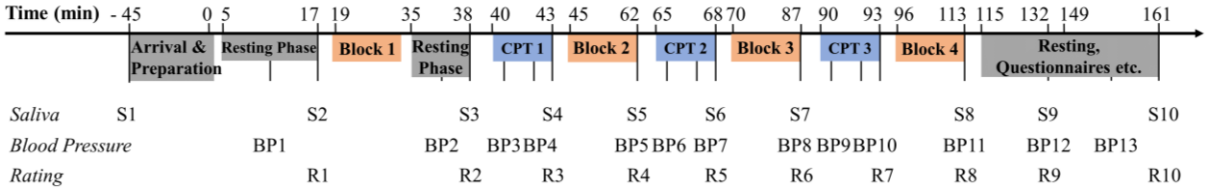
Procedure

Upon arrival of the participants, a 10-minute screening interview was conducted first, to ensure that they met inclusion criteria and inform them about the study. Participants were not told which experimental condition they would be allocated to. Participants were instructed on how to collect their saliva samples, and after giving their informed consent, collected their first saliva sample (0:45 min before beginning of the experiment). Participants were then seated in a dimly lit EEG laboratory, with their bare feet placed in two floor-mounted foot basins, which were still empty at this time. During the preparation of the physiological instruments (EEG,

ECG, hEOG, blood pressure), participants completed a general sociodemographic questionnaire (Appendix II) including the 12-item Edinburg Handiness Inventory (EHI) (Oldfield, 1971) to verify that they were right-handed.

Figure 1

Experimental procedure, timing of measurements and laboratory setup.



Note. CPT = bilateral feet Cold Pressor Test; S = Salivette, BP = Heart Rate and Blood Pressure, R = Rating Scale.

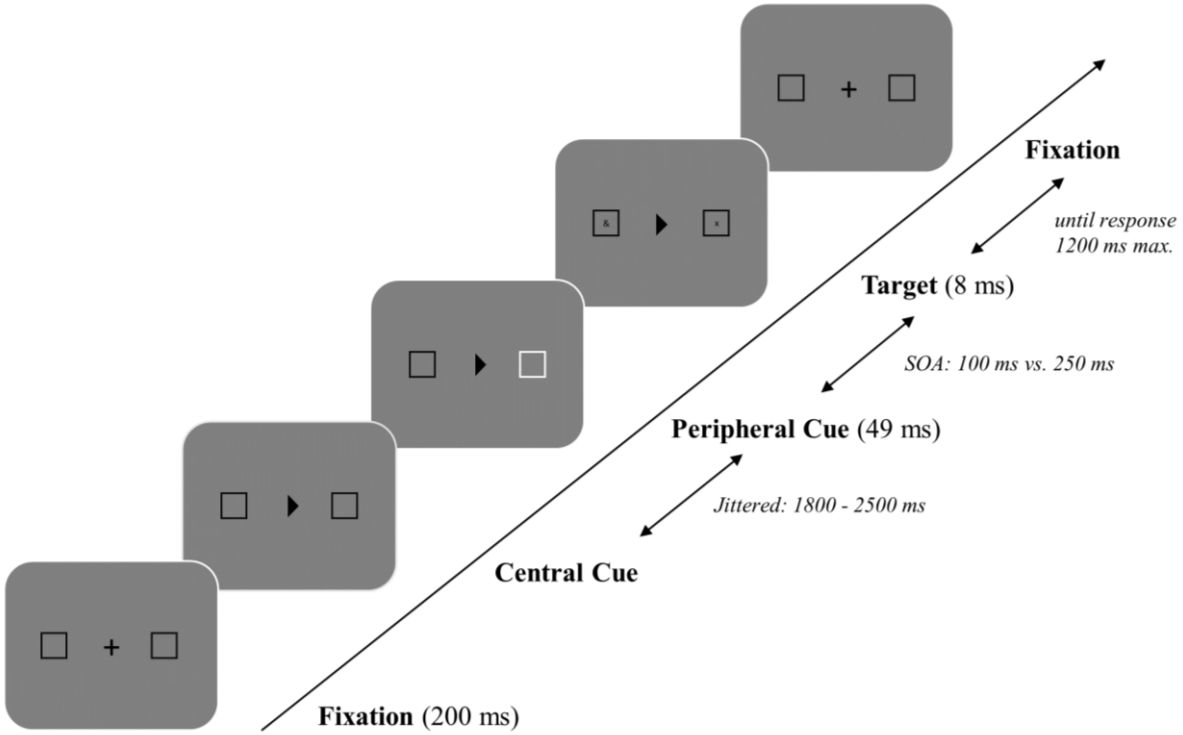
The experiment started with a training block of the below described paradigm. After completing the training block, a first 12 min resting phase started, during which blood pressure measurements were taken, and after which participants gave their second saliva sample. Subsequently, the first of four experimental blocks began lasting approximately 17 minutes as all of the experimental blocks. The first block was followed by a 3 min resting phase during which baseline blood pressure and heart rate were taken, and saliva sample 3 was conducted. Following this resting phase, the first CPT or warm-water control procedure started during which blood pressure was measured and after which participants collected saliva sample 4. Experimental block 2 followed at which end blood pressure and saliva sample 5 were conducted. This procedure was repeated for another two times (CPT 2 – Block 3; CPT 3 – Block 4). Finally, another 12 min resting phase completed the experiment after which participants filled out additional questionnaires (data presented elsewhere) and provided the last saliva samples.

Stimuli and Paradigm

The main task during the experiment was a variant of the Posner et al. (1980) cueing paradigm combining central and peripheral cues. The first, central cue was an arrow which was followed by a peripheral cue in form of a luminance change after which the target was presented that had to be discriminated from a similar stimulus (see Figure 2).

Figure 2

Experimental paradigm and sequence of events during each trial.



Note. Each trial contained a central and a peripheral cue, and a target. The central cue (arrow) was either directional so pointed to the target location, or neutral so did not provide any spatial information about the target location. After a jittered interval of 1800-2500 ms, the peripheral cue was displayed in form of one of the target boxes turning white. The target was displayed after different SOAs, either 100 or 250 ms after the peripheral cue. This resulted in six trial conditions: central cue (directional, neutral) * peripheral cue (compatible, incompatible) * SOA (100, 250 ms SOA).

Each trial, a default display was presented consisting of a central, black fixation cross (RGB: 0-0-0, size: 0.7° x 0.7°) and two open, black squares (size: 1.7° x 1.7°) in the left and right visual field with equal eccentricity from the center (3.3°) displayed within a light gray background (RGB: 128-128-128). After 200 ms, the fixation cross was replaced by the central cue, a centered black arrow. In directional trials it pointed either to the right or the left square indicating the target location with 100% probability. In neutral trials it pointed both to the left and right direction. Following the central cue, the peripheral cue was displayed consisting in one of the black squares turning white (RGB: 255-255-255) for a duration of 49 ms resulting in a flash with a 50% validity to correspond to the later target location (compatible vs. incompatible). After different SOAs of either 100 or 250 ms, the target was displayed in one of the black squares. Targets consisted of a black “x” (size: 0.23° x 0.23°) or “+”and were displayed for 7 ms with no mask following. They were accompanied by a distractor stimulus (size: 0.23° x 0.23°, two vertical black lines containing an equal amount of black and grey pixels

as the targets) in the respective other square. Participants were instructed to press the arrow-up key (Right hand middle finger) or the arrow-down key (Right hand index finger) corresponding to the target shown, with the keyboard mapping being counterbalanced within the experimental conditions. Participants were instructed to keep their eyes directed at the center of the screen during the whole trial. The training block consisted of 48 trials, while each experimental blocks consisted of 288 trials, totaling 1152 trials over all 4 blocks. No feedback on performance was given at any point of the experiment. Frequencies of the different trial conditions were evenly balanced within the experimental blocks.

Automated bilateral feet cold pressor test

Timing, duration, and temperature of the bilateral feet CPT (Larra et al., 2015) were automated using MATLAB 2020a (9.8.0.1721703, Update 7). The water used for the CPT was brought to the desired temperature in two boilers in an adjacent room. For the CPT condition this was 3-4 °C, for the warm water condition 37-38 °C. The 35 x 20 x 19 cm rectangular food basins were filled via a pipe and pump system and to avoid the formation of stable temperature layers next to the skin, the water around the feet was floating permanently. Water level and temperature were continuously monitored using several sensors in the foot basins and cameras pointing at the basins were used to check if the participants kept their feet in the water.

The cold and warm water exposure lasted for three minutes; however, participants could terminate the procedure at their discretion. After 0.5 and 2.5 min participants were asked to either close or open their eyes, the sequential order being counterbalanced. During the CPT, participants were not given any information about the remaining duration of the water exposure.

Apparatus and measurements

Laboratory setup

The experiment was performed on a 24-inch LCD monitor (144 Hz, 1920 x 1080 px) with a viewing distance of 80 cm. The experiment was realized in E-Prime (Psychology Software Tools, Inc., Pittsburgh, PA, USA) running on a Windows 7 PC, connected to a standard keyboard.

EEG

EEG was recorded using a 64 active electrode cap (actiCAP; Brain Products GmbH; Munich, Germany) with the extended 10/20 system at 1 kHz. FCz was used as an online reference and AFz as ground. All electrode impedances were reduced until below 10 kΩ. The

horizontal electrooculogram (EOG) was measured using electrodes located on the outer canthi of the eyes. The EEG signals were acquired via a BRAINAMP DC and EOG signals via a BRAINAMP ExG amplifier (Brain Products GmbH; Munich, Germany).

Cardiovascular measures

Heart rate and blood pressure were assessed using the Dinamap Pro 300V2 (Critikon; Tampa, Florida, USA). Stress values for heart rate and blood pressure during the CPT were measured at 0.5 and 2.5 min after the start of the CPT, baseline values were derived from measurements taken in 3-minute intervals during the resting phase. Blood pressure measurements were not performed during epochs designated for EEG analysis.

Cortisol

Saliva samples were collected using Salivettes (Sarstedt, Nürnbrecht, Germany). Participants were instructed not to touch the cotton swab, and to use their tongue to move it inside their mouth without chewing the cotton swab. After collecting the saliva, the Salivettes were stored in a fridge until centrifugation after the session, after which the samples were stored at -80 C° until analysis. After thawing for biochemical analysis, the fraction of free cortisol in saliva (salivary cortisol) was determined using a time-resolved immunoassay with fluorometric detection, as described in detail in Dressendorfer et al. (1992). Due to missing samples, two subjects were excluded from statistics on cortisol data.

Subjective Ratings

Stress and arousal ratings were collected before and after each CPT/control exposure, along with retrospective pain ratings given after each intervention. Participants were asked to rate their current stress and arousal levels as well as the level of pain experienced during exposure on visual analogue scales (VAS) ranging from 0 (“not at all stressed/aroused/painful”) to 100 (“extremely stressed/aroused/painful”). The scales were displayed on-screen, and participants were asked to provide their rating by placing a cursor along the scale using keyboard arrow-buttons.

Data Preprocessing & Statistical Analysis

Behavioral Data

Trials containing responses faster than 150 ms (premature responses) were excluded from further analysis and responses slower than 1200 ms were counted as misses. Trials with

horizontal eye movements during the endogenous cueing period were removed (*M* = 9.21 %) using a step function adapted from ERPLab (Lopez-Calderon & Luck, 2014). Step functions resemble the voltage steps found in saccadic eye movements resulting in removal of trials with step like voltage increases larger than the set threshold of 50 μV (50 μV steps, 400 ms window and 10 ms steps). Average reaction time (RT) and proportion of correct responses (Pcorrect, %), missed responses (Pmisses, %), and false responses (Perrors, %) were computed per subject, block, and type of trial.

EEG preprocessing and analysis

EEG data was preprocessed and analyzed using the EEGLAB toolbox v2020.0 (Delorme & Makeig, 2004) implemented in MATLAB R2020a. First, EEG data was re-referenced to average, and band-pass filtered from 1 Hz to 30 Hz. Subsequently, bad channels were removed using kurtosis (*SD* = 5, norm = on) and probability (*SD* = 5, norm = on) criteria. Following that, data was down-sampled to 250 Hz. In the next step, the continuous data were segmented into 5 s data epochs relative to central cue onset (-1000 ms to 4000 ms). Epochs containing artifacts were rejected, using an automated trial rejection function (pop_autorej, threshold limit for detection of fluctuations: 500 μV, probability threshold: 5 *SD*, maximum percentage of rejected trials per iteration: 5 %). Next, an ICA was performed to remove components that had a non-cortical origin (Infomax algorithm with option for detection of sub-gaussian sources activated, Principal Component Analysis to correct number of ICs to data rank). ICs were classified using ICLabel (Pion-Tonachini et al., 2019) to remove components with a probability of less than 50 % to reflect brain activity. On average 28.29 components were rejected per participant. Finally, epochs were again checked for remaining artefacts (see criteria above). On average 7.02 % trials were rejected.

With the preprocessed data, time-frequency decomposition was performed using the EEGLAB newtimef function (Delorme & Makeig, 2004). The segmented data was convolved with complex Morlet wavelets from 3 Hz to 20 Hz in 69 linear steps. The number of cycles started with 3 cycles at the lowest frequency and increased by 0.5 per frequency to 8.5 cycles at the highest frequency (20 Hz), with each epoch containing 500 timepoints (between -1000 ms and 3500 ms). After time-frequency decomposition, epochs were pruned to 3.5 s, -500 to 3000 ms relative to central cue onset.

To compare alpha band activity, lateralized alpha power (LAP) was calculated for directional and neutral central cues. To retrieve LAP, the alpha power at a specific time point of two symmetrical electrodes above the left and right hemisphere (e.g. PO7 and PO8) is used

to calculate the power difference between the electrodes, and scale it by the sum of their power (Thut et al., 2006; Van der Lubbe & Utzerath, 2013). If ipsilateral power is subtracted from contralateral power, this results in a LPS index which values range from -1 to +1, with negative values indicating that alpha power at time point *t* is larger above the ipsilateral hemisphere than the contralateral hemisphere, and vice versa regarding positive values. Left and right cued trials were computed separately and averaged subsequently. Trials with neutral central cues were computed dependent on the target location (left vs. right) and were then averaged as well. LAP was calculated and averaged for P3/4, P7/8 and PO7/8 and binned in 13-time windows with a 250 ms baseline (-250 to 0 ms) and twelve 150 ms time windows reaching from 0 – 1800 ms.

Stress Response Parameters

To analyze the cardiovascular stress response, deltas for heart rate (HR) and mean arterial pressure (MAP) were calculated using resting phase 1 as baseline. For analysis, separate repeated-measures ANOVAs were calculated for ΔHR and ΔMAP, with GROUP (stress vs. control) as between-subject factors, and TIME (measurement time, depending on respective measure) as within-subject factors.

The saliva sample after the resting phase preceding the first CPT was used as a baseline to calculate Δ Cortisol. VAS values were baseline corrected as well using rating 1 after the resting phase to build deltas for subjective stress, and arousal.

Statistical Analyses

Data were analyzed with mixed model ANOVAs comprising GROUP (stress vs. control) as a between subject-factors and TIME (measurement time, depending on respective measure; for EEG = BIN) as within-subject factors. For behavioral data, within-subject factors CENTRAL CUE, PERIPHERAL CUE, and SOA were added. For analyses of EEG data CENTRAL CUE and BIN were used as within-subject factors. If not otherwise stated Greenhouse-Geisser correction was used to adjust for lack of sphericity. In case of significant main or interaction effects, Fishers LSD tests were used for pairwise comparisons. In other cases, Bonferroni corrected pairwise comparisons were used for further analyses. For all statistical analyses, SPSS 24 (IBM Corp.; Armonk, New York, USA) was used.

Results

Response to the CPT

The stress induction produced robust increases in all assessed response parameters. This is described in detail below for cortisol, cardiovascular measurements, and subjective ratings. There were no significant baseline differences between groups.

Cortisol

A repeated measures ANOVA with Greenhouse-Geisser correction was conducted on Δ Cortisol with GROUP as between subject factors and TIME (7-level) as within-subject factor (see Figure 3). It revealed a significant main effect for TIME ($F(6,222) = 3.2$, $p = .006$, $\eta_p^2 = .078$, $\varepsilon = .39$) indicating significant changes of Δ Cortisol over the course of the experiment. There was a significant main effect of GROUP indicating increased Δ Cortisol for the stress group ($M = 0.94$, $SD = 0.48$) as compared to the control group ($M = -1.18$, $SD = 0.57$) ($F(1,37) = 8.59$, $p = .006$, $\varepsilon = .188$). Finally, the two-way interaction of TIME * GROUP revealed a significant interaction effect ($F(6,222) = 5.04$, $p = .003$, $\eta_p^2 = .12$, $\varepsilon = .39$). Post-hoc tests revealed significant higher levels in Δ Cortisol for the stress group from 62 min to 87 min (62 min: Stress: $M = 2.74$, $SD = 0.62$, Control: $M = -1.64$, $SD = 0.69$, $t(37) = -4.71$, $p < .001$, $d = 6.67$; 68 min: Stress: $M = 2.0$, $SD = 0.61$; Control: $M = -1.67$, $SD = 0.67$, ; $t(37) = -4.05$, $p < .001$, $d = 5.73$; 87 min: Stress: $M = 1.40$, $SD = 0.70$, Control: $M = -0.90$, $SD = 0.77$, $t(37) = -2.21$, $p = .033$, $d = 3.13$).

Heart rate and blood pressure

Separate repeated measures ANOVAs with Greenhouse-Geisser correction were conducted for Δ HR and Δ MAP with GROUP as between subject factors and TIME (13-level) as within-subject factor.

There was a significant main effect of TIME for Δ HR ($F(11, 330) = 4.36$, $p < .001$, $\eta_p^2 = .127$, $\varepsilon = .54$) and Δ MAP ($F(1, 32) = 6.423$, $p < .001$, $\eta_p^2 = .167$, $\varepsilon = .40$) indicating significant changes in Δ HR and Δ MAP over the course of the experiment. GROUP had a significant effect on Δ MAP ($F(1, 32) = 17.43$, $p < .001$, $\eta_p^2 = .35$) showing significantly increased Δ MAP for the stress group ($M = 4.47$, $SD = 0.83$) compared to the control group ($M = -.079$, $SD = .95$). The main effect of GROUP was not significant for Δ HR ($F(1, 30) = 3.24$, $p = .082$, $\eta_p^2 = .097$).

For Δ MAP and Δ HR there was a significant interaction of GROUP * TIME (Δ HR ($F(11, 330) = 4.16$, $p = .001$, $\eta_p^2 = .122$, $\varepsilon = .54$; Δ MAP ($F(1, 352) = 9.15$, $p < .001$, $\eta_p^2 = .22$,

Ɛ = .40). Post-hoc comparisons showed significant increases of Δ HR in the stress group during the second CPT (Stress: M = 7.83, SD = 2.5; Control: M = -1.67, SD = 2.69; t(30) = -2.6, p = .014, d = 3.65). Δ MAP significantly increased in the stress group compared to the control group during the three CPTs and shortly before the third CPT.

Subjective Responses

Separate repeated measures ANOVAs with Greenhouse-Geisser correction for the subjective measures Δ Stress, Δ Arousal, and Pain with GROUP as between subject factors and TIME (8-level for stress and arousal, 3-level for pain) as within-subject factor were conducted.

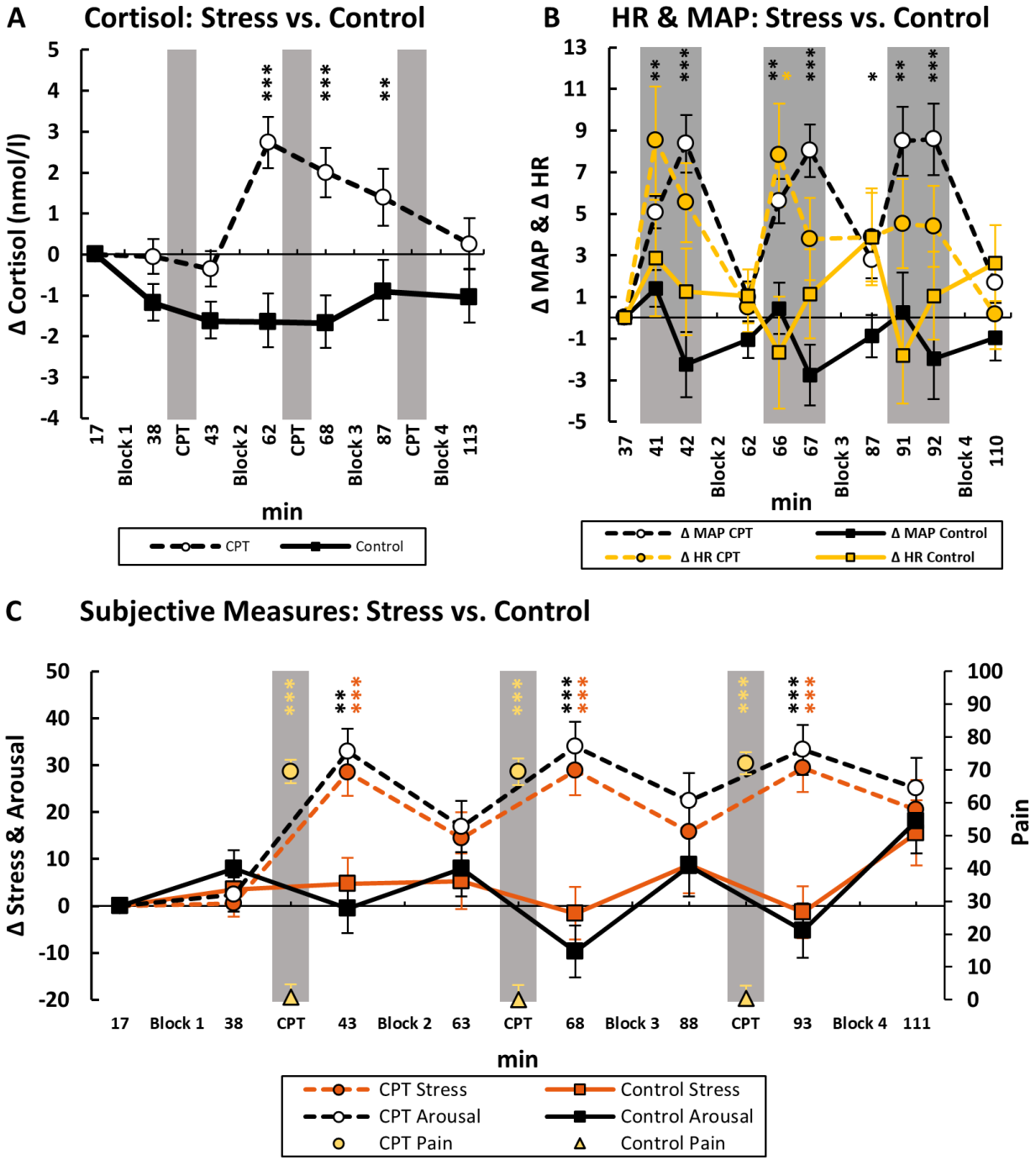
There was a significant main effect of TIME on Δ Stress (F(7, 273) = 3.82, p = .001, η_p^2 = .09, Ɛ = .59) and Δ Arousal: F(7, 273) = 2.8, p = 0.008, η_p^2 = .067, Ɛ = .51) indicating significant changes in Δ HR and Δ MAP over the course of the experiment. TIME did not have a significant main effect on Pain (F(2,78) = .27, p = .764, η_p^2 = .01, Ɛ = .76).

GROUP had a significant main effect on all subjective measures (Δ Stress: F(1, 39) = 4.27, p = .045, η_p^2 = .099; Δ Arousal: F(1,39) = 8.143, p = 0.007, η_p^2 = .173; Pain: F(1,39) = 206.11, p < .001, η_p^2 = .841) indicating significantly higher Δ Stress (Stress group: M = 18.28, SD = 4.12; Control: M = 5.87, SD = 4.4), Δ Arousal (Stress group: M = 22.09, SD = 4.12; Control: M = 4.82, SD = 4.45), and Pain (Stress: M = 70.31, SD = 3.3; Control: M = 0.48, SD = 3.57) for the stress group.

There was a significant interaction effect of GROUP * TIME on Δ Stress (F(7, 273) = 7.41, p < .001, η_p^2 = .16, Ɛ = .59) and Δ Arousal (F(7,273) = 11.08, p < .001, η_p^2 = .22, Ɛ = .51) but not Pain (F(1,39) = 206.11, p < .001, η_p^2 = .841). For Δ Stress and Δ Arousal, post-hoc comparisons showed significant differences between groups immediately after the all of the CPTs e.g. after 43 minutes (Δ Stress: Stress group: M = 28.51, SD = 5.03; Control: M = 4.78, SD = 5.44, t(39) = -3.21, p = .003, d = 4.53; Δ Arousal: Stress group: M = 52.36, SD = 4.47; Control: M = 18.74, SD = 4.84, t(39) = -4.69, p = .003, d = 7.22), 68 minutes (Δ Stress: Stress group: M = 28.81, SD = 5.16; Control: M = -1.54, SD = 5.61, t(39) = -3.97, p < .001, d = 5.63; Δ Arousal: Stress group: M = 53.48, SD = 4.31; Control: M = 9.61, SD = 4.66, t(39) = -5.76, p < .001, d = 9.77), and 93 minutes (Δ Stress: Stress group: M = 29.41, SD = 5.12; Control: M = -1.32, SD = 5.54, t(39) = -4.07, p < .001, d = 5.76; Δ Arousal: Stress group: M = 52.71, SD = 4.28; Control: M = 14.02, SD = 4.63, t(39) = -4.88, p < .001, d = 8.67).

Figure 3

*Responses to the CPT protocol per experimental group. **A**. Changes in cortisol compared to baseline (Δ Cortisol). **B**. Baseline differences in Heart rate (Δ HR) and mean arterial blood pressure Δ MAP. **C.** Differences in subjective measures stress, arousal, pain.*



Note. Grey areas represent the 3min CPT. Error bars show standard errors. **A.** Changes in Δ Cortisol between stress and control group over the course of the experiment. Measurements at 62, 68, 87 minutes show significant differences in cortisol level between the groups. **B.** Comparison of changes in Δ MAP and Δ HR between stress and control group. Δ MAP is significantly increased in the stress group during the CPTs and at 87 minutes. Δ HR is significantly increased in the beginning of the second CPT. **C.** Subjective pain is significantly higher during the CPT than the control procedure. Arousal and stress are significantly increased in the stress group immediately after the CPT procedure but not after the experimental blocks. * $p \leq 0.05$ ** $p \leq 0.01$ *** $p \leq 0.001$.

Behavioral data

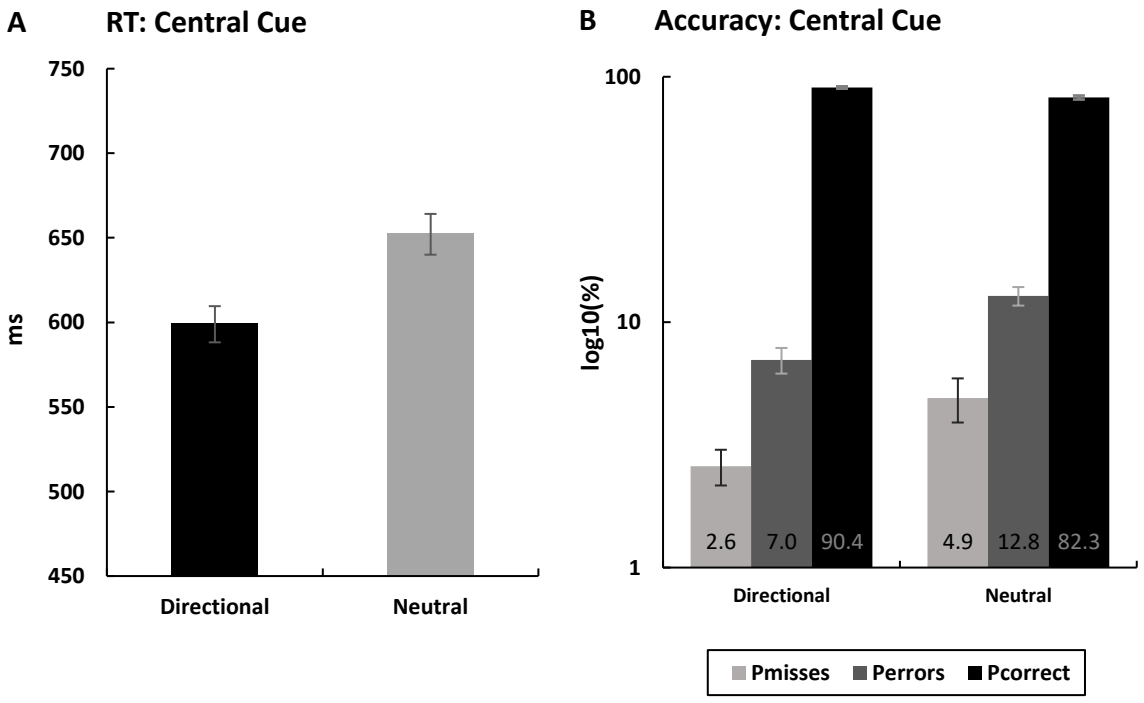
For the purpose of clarity, results are structured by main and interaction effects on all dependent variables although they were analyzed in separate mixed model ANOVAs (CENTRAL CUE * PERIPHERAL CUE * SOA * GROUP). There were no significant baseline differences between groups.

Posner Paradigm

Cueing had a substantial influence on behavioral performance in terms of both response speed and accuracy measures (Pcorrect, Pmisses, Perrors). CENTRAL CUE had a significant effect on performance as indicated by RT ($F(1,39) = 41.78$, $p < .001$, $\eta_p^2 = .52$), Pcorrect ($F(1,39) = 30.37$, $p < .001$, $\eta_p^2 = .44$), Pmisses ($F(1,39) = 10.55$, $p = .002$, $\eta_p^2 = .21$), and Perrors ($F(1,39) = 26.77$, $p < .001$, $\eta_p^2 = .41$). Performance was better for directional (RT: $M = 599.40$ ms, $SD = 10.17$ ms; Pcorrect: $M = 90.4$ %, $SD = 0.97$ %; Pmisses: $M = 2.59$ %, $SD = 0.43$ %; Perrors: $M = 7.01$ %, $SD = 0.84$ %) than for neutral central cues (RT: $M = 652.32$ ms, $SD = 11.81$ ms; Pcorrect: $M = 82.31$ %, $SD = 1.54$ %; Pmisses: $M = 4.9$ %, $SD = 1.0$ %; Perrors: $M = 12.79$ %, $SD = 1.11$ %).

Figure 4

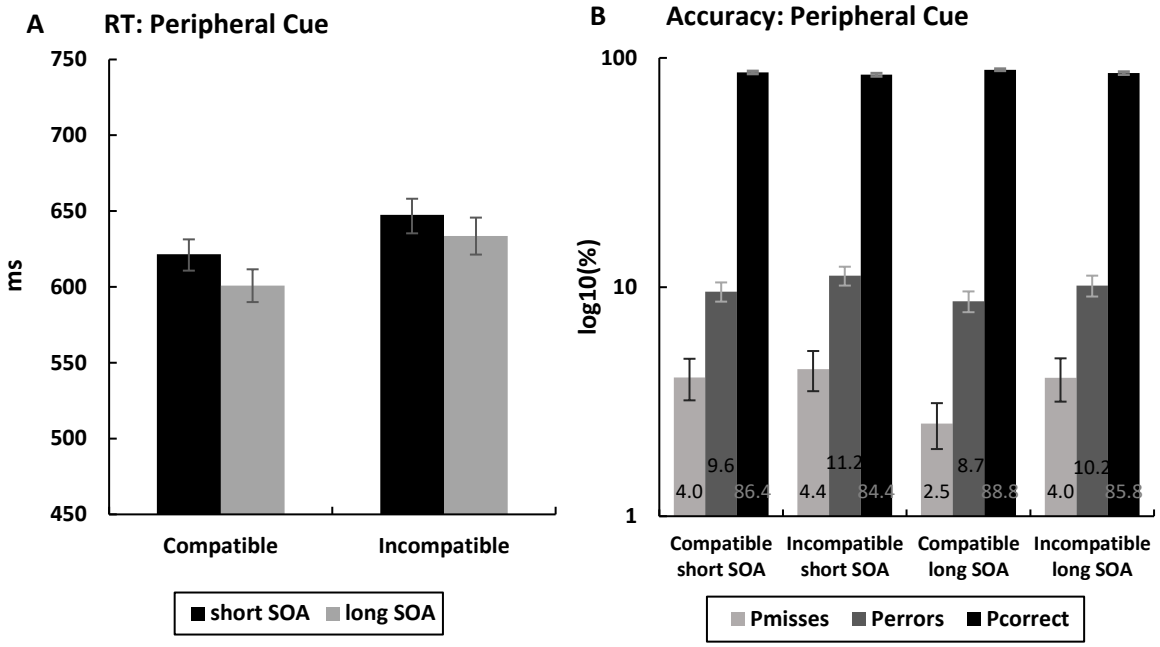
Paradigm check: RT (A) and Accuracy (B) for type of central cue.



There was a significant main effect of PERIPHERAL CUE on performance seen in RT ($F(1,39) = 37.28$, $p < .001$, $\eta_p^2 = .49$), Pcorrect ($F(1,39) = 15.61$, $p < .001$, $\eta_p^2 = .29$), Pmisses ($F(1,39) = 7.69$, $p = .008$, $\eta_p^2 = .17$), and Perrors ($F(1,39) = 8.39$, $p = .006$, $\eta_p^2 = .18$). Performance was better for compatible (RT: $M = 611.18$ ms, $SD = 10.0$ ms; Pcorrect: $M = 87.60$ %, $SD = 0.91$ %; Pmisses: $M = 3.29$ %, $SD = 0.63$ %; Perrors: $M = 9.11$%, $SD = 0.79$ %) than for incompatible trials (RT: $M = 640.53$ ms, $SD = 10.99$ ms; Pcorrect: $M = 85.11$%, $SD = 1.21$ %; Pmisses: $M = 4.21$ %, $SD = 0.77$ %; Perrors: $M = 10.68$, $SD = 0.92$).

Figure 5

Paradigm Check: RT (A) and Accuracy (B) for type of peripheral cue and SOA.



SOA had a significant effect on performance seen in RT ($F(1,39) = 22.32$, $p < .001$, $\eta_p^2 = .36$) and Pcorrect ($F(1,39) = 6.16$, $p = .018$, $\eta_p^2 = 17$). Performance was decreased for short SOAs (RT: $M = 634.55$ ms, $SD = 9.84$ ms; Pcorrect: $M = 85.41$ %, $SD = 1.23$ %) compared to long SOAs (RT: $M = 617.17$ ms, $SD = 10.92$ ms; Pcorrect: $M = 87.31$ %, $SD = 1.02$ %). The main effect of SOA was not significant for Pmisses ($F(1,39) = 3.294$, $p = .077$, $\eta_p^2 = .08$) and Perrors ($F(1,39) = 1.719$, $p = .197$, $\eta_p^2 = .04$).

There were no significant interactions of CENTRAL CUE, PERIPHERAL CUE, or SOA for RT, Pcorrect, Pmisses, and Perrors. Detailed results for their interaction effects are shown in Appendix III.

Stress Effects

The main effect of GROUP was not significant indicating no general differences between the stress and control group regarding RT ($F(1,39) = 2.12$, $p = .147$, $\eta_p^2 = .05$), Pcorrect ($F(1,39) = 1.27$, $p = .266$, $\eta_p^2 = .03$), Pmisses ($F(1,39) = 0.37$, $p = .547$, $\eta_p^2 = .09$), and Perrors ($F(1,39) = 0.92$, $p = .342$, $\eta_p^2 = .02$).

There was a significant ordinal interaction effect of GROUP * PERIPHERAL CUE on Pmisses ($F(1,39) = 4.32$, $p = .044$, $\eta_p^2 = .10$). Post-hoc Fishers LSD tests showed that in the stress group, compatible ($M = 4.05$ %, $SD = 4.9$ %) and incompatible peripheral cues ($M = 4.28$ %, $SD = 4.65$ %) did not significantly differ ($t(18) = -0.47$, $p = .639$, $d_z = -.05$) while in the control group compatible peripheral cues ($M = 2.53$ %, $SD = 3.37$ %) produced significantly less errors than incompatible ones ($M = 4.13$ %, $SD = 5.17$ %; $t(21) = -3.56$, $p = .001$, $d_z = -.36$).

There was a significant three-way interaction of CENTRAL CUE * PERIPHERAL CUE * GROUP for Pmisses ($F(1,39) = 4.26$, $p = .046$, $\eta_p^2 = .10$). Separate ANOVAs for the stress and the control group revealed different two-way interaction effects of CENTRAL CUE * PERIPHERAL CUE. For the stress group, the effect of CENTRAL CUE * PERIPHERAL CUE on Pmisses was not significant ($F(1, 18) = 0.63$, $p = .437$, $\eta_p^2 = .034$). Fisher´s LSD test showed that independently from the type of central cue, errors did not differ significantly between compatible (Directional: $M = 2.36$ %, $SD = 0.51$ %; Neutral: $M = 5.73$%, $SD = 1.66$ %) and incompatible peripheral cues (Directional: $M = 3.14$, $SD = 0.74$ %; Neutral: $M = 5.42$ %, $SD = 1.57$ %; Compatible: $t(18) = -1.46$, $p = .158$, $d_z = -0.34$; Incompatible: $t(18) = 0.31$, $p = .758$, $d_z = 0.07$). For the control group, the ordinal two-way interaction of CENTRAL CUE * PERIPHERAL CUE was significant ($F(1, 21) = 5.15$, $p = .034$, $\eta_p^2 = .20$) where the type of central cue influenced the effect of peripheral cue. For directional central cues, there was no significant difference in compatible ($M = 2.24$ %, $SD = 0.71$ %) and incompatible peripheral cues ($M = 2.60$ %, $SD = 0.61$ %; $t(21) = -0.61$, $p = .546$, $d_z = -0.13$). However, for neutral central cues, compatible peripheral cues ($M = 2.81$ %, $SD = 1.09$%) showed significantly lower errors than incompatible ones ($M = 5.66$ %, $SD = 1.65$ %; $t(21) = -2.94$, $p = .008$, $d_z = -0.63$).

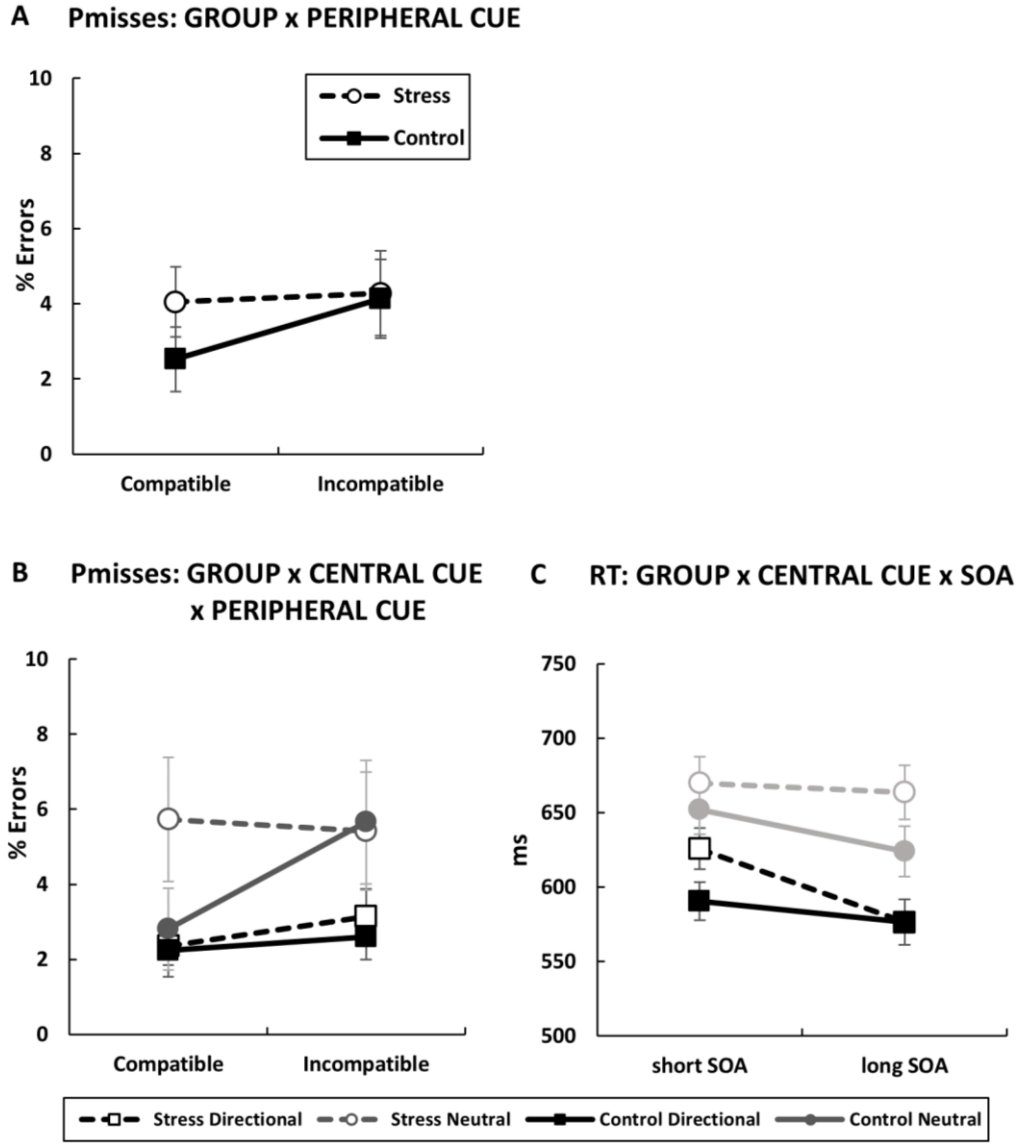
The three-way interaction of CENTRAL CUE * SOA * GROUP had a significant effect on RT ($F(1,39) = 5.18$, $p = .028$, $\eta_p^2 = .18$). Separate ANOVAs for SOA * GROUP for directional and neutral central cues did not reveal significant two-level interactions (directional: $F(1,39) = .702$, $p = .407$, $\eta_p^2 = .02$; neutral: $F(1,39) = 3.78$, $p = .059$, $\eta_p^2 = .09$). However, post-hoc Fishers LSD tests of the three-way-interaction showed that the type of central cue

modulated RT differences between short and long SOAs in the stress group. In the control group, RT for short SOAs (directional: M = 590.68 ms, SD = 12.82 ms; neutral: M = 651.98 ms, SD = 16.29 ms), is significantly higher than for long SOAs (directional: M = 576.28 ms, SD = 15.27 ms; neutral: M = 623.93 ms, SD = 16.76 ms) independently of type of central cue (directional: $t(21) = 2.74$, $p = .009$, $d_z = 0.58$; neutral: $t(21) = 3.67$, $p = .001$, $d_z = 0.78$).

However, in the stress group only directional central cues produce significantly higher RTs in short SOAs (M = 625.79 ms, SD = 13.8 ms) than long SOAs (M = 576.28 ms, SD = 15.26 ms) (t(18) = 3.69, $p = 0.001$, $d_z = 0.85$). For neutral central cues, RTs during short SOAs (M = 669.80 ms, SD = 17.52 ms) and long SOAs (M = 663.58 ms, SD = 18.04 ms) did not differ significantly ($t(18) = 0.76$, $p = .454$, $d = 0.17$). There were no further significant two- or three-way interactions including GROUP with RT, or any accuracy measure (see Appendix III).

Figure 6

Interaction Effects of GROUP on Pmisses (A & B) and RT (C).



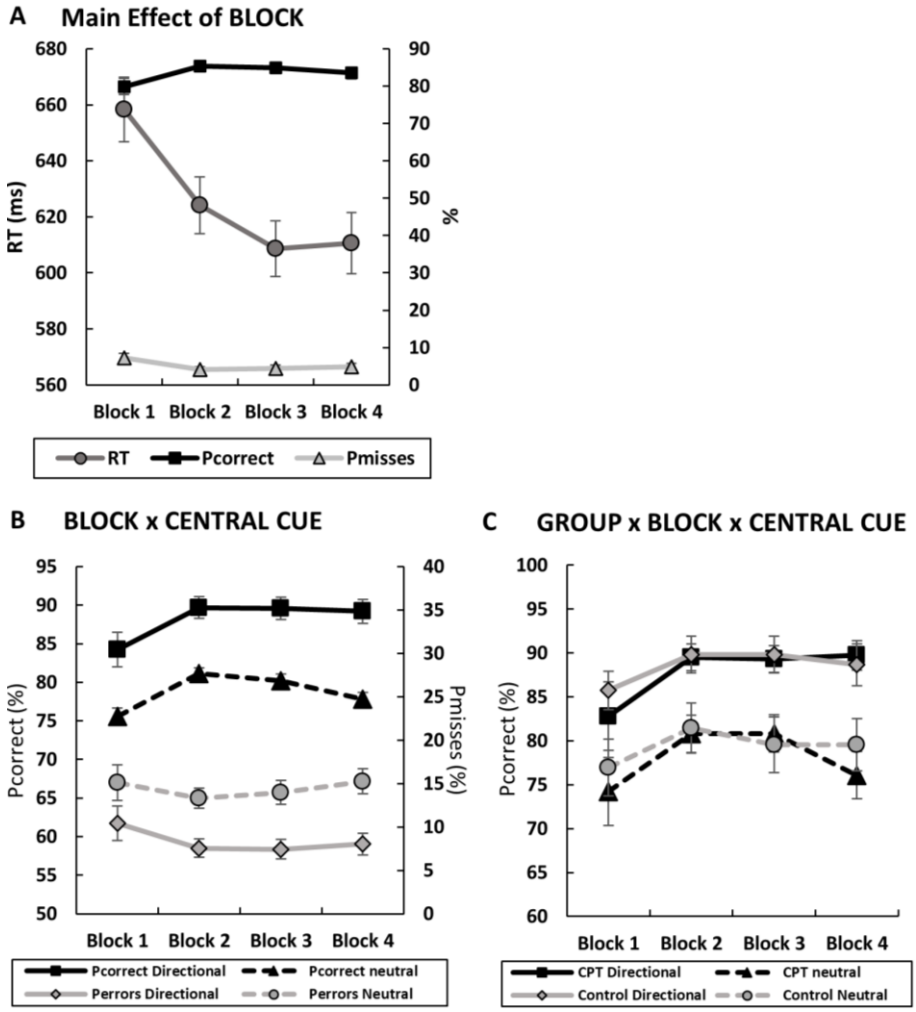
Exploratory Results: block effects

Behavioral performance differed significantly per block in terms of both response speed and accuracy measures (Pcorrect, Pmisses). BLOCK had a significant effect on performance as indicated by RT ($F(1,126) = 21.66$, $p < .001$, $\eta_p^2 = .34$, $\varepsilon = .67$), Pcorrect ($F(1,126) = 6.42$, $p = .008$, $\eta_{p^2} = .13$, $\varepsilon = .45$), Pmisses ($F(1,126) = 10.83$, $p < .001$, $\eta_p^2 = .21$, $\varepsilon = .79$). BLOCK did not have a significant effect on Perrors ($F(1,126) = 1.150$, $p = .332$, $\eta_p^2 = .03$, $\varepsilon = .4$). RT improved until block 3 after which it stayed at the same level. For the accuracy measures Pcorrect and Pmisses it improved only after block 1 after which it stayed constant (see Figure 7 A). There was a significant interaction effect of BLOCK*CENTRAL CUE for Pcorrect ($F(1,126) = 3.63$, $p = .021$, $\eta_p^2 = .09$, $\varepsilon = .45$) and Perrors ($F(1,126) = 3.129$, $p = .039$, $\eta_p^2 = .07$, $\varepsilon = .4$). For both Pcorrect and Perrors performance for directional and neutral central cues improved after block 2. However, for directional central cues proportion of correct responses stabilized after block 2, while for neutral central cues performance started to decrease after block 2 (see Figure 7 B).

There was a significant interaction effect of BLOCK*CENTRAL CUE * CONDITION on Pcorrect ($F(3,126) = 3.87$, $p = .016$, $\eta_p^2 = .09$, $\varepsilon = .45$). Separate ANOVAs for BLOCK * CENTRAL CUE for the experimental groups revealed a significant two-way interaction for the CPT group ($F(3,60) = 5.175$, $p = .013$, $\eta_p^2 = .21$, $\varepsilon = .59$) but not for the control group ($F(3,60) = 955$, $p = .42$, $\eta_p^2 = .04$, $\varepsilon = .86$). For the control group, performance in directional cued trials improved significantly from Block 1 to 2 (Block 1 $M =$ 85.7 %, $SD = 2.2$ %; Block 2: $M = 89.8$ %, $SD = 2.1$ %; $t(21) = 3.11$, $p = 0.04$, $d_z = 1.9$) and remained constant as it significantly differed in Block 1 and 3 (Block 3: $M =$ 89.8 %, $SD = 2.1$ %; $t(21) = 3.11$, $p = 0.04$, $d_z = 1.9$). However, there were no more significant differences between blocks. For neutral central cues, accuracy increased from Block 1 to Block 2 (Block 1: $M =$ 76.95 %, $SD = 3.2$ %; Block 2: $M = 81.45$ %, $SD = 2.84$ %; $t(21) = 2.86$, $p = 0.09$, $d_z = 1.49$) but did not significantly ($p > .05$) between other blocks. In the stress group, performance in directional cued trials did not significantly differ between any blocks ($p > .05$). However, during neutral cued trials performance decreased from block 3 to block 4 (Block 3: $M = 80.8$ %, $SD = 2.1$ %; Block 4: $M = 76.1$ %, $SD = 2.7$ %; $t(20) = 3.2$, $p = .003$, $d_z = 1.94$). No other blocks differed significantly. There were no further no further significant two- or three-way interactions including BLOCK with any accuracy measure.

Figure 7

Block effects on behavioural measures.



Note. RT = Reaction Time, Pcorrect = Proportion of correct responses, Pmisses = Proportion of missed responses.

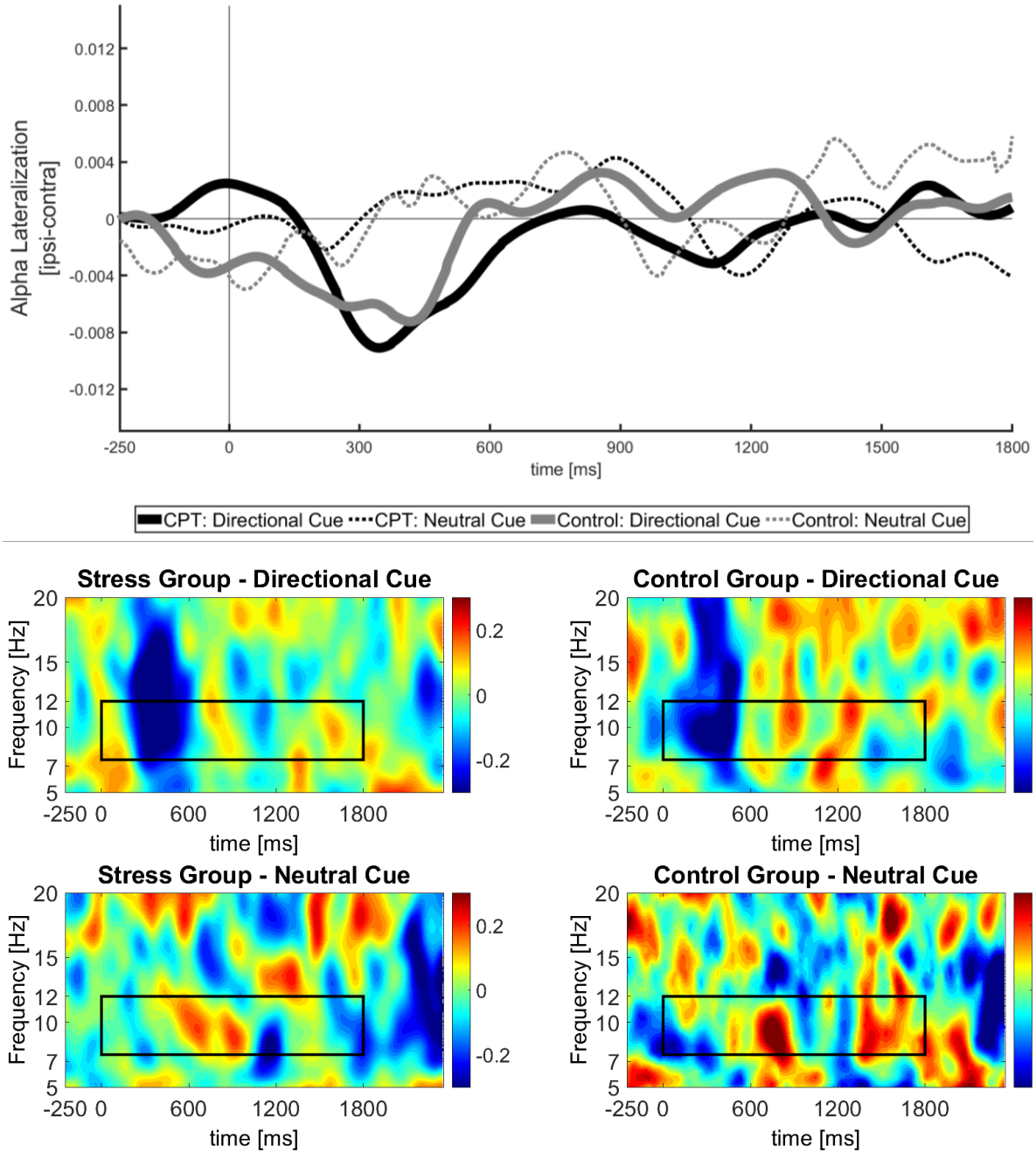
EEG data

The mixed model ANOVA revealed a significant main effect of BIN on LAP ($F(1,34) = 3.08$, $p = .009$, $\eta_p^2 = .08$, $\varepsilon = .45$) (see Figure 8). Furthermore, there was a significant interaction effect of CENTRAL CUE * BIN ($F(12, 408) = 2.33$, $p = .047$, $\eta_p^2 = .06$, $\varepsilon = .39$) indicating smaller LAP for directional compared to neutral cues. Pairwise comparisons indicated a clear temporal pattern in LAP with significant differences between cue conditions observable from 150 to 600 ms (150–300 ms: $t(34) = -2.15$, $p = .039$, $d_Z = 2.01$, Directional: $M = -.0042$, $SD = 0.001$, Neutral: $M = -.001$, $SD = 0.0012$; 300–450 ms: $t(34) = -4.16$, $p < .001$, $d_Z = 3.71$, Directional: $M = -.0066$, $SD = -0.0013$; Neutral: $M = .0004$, $SD = 0.0013$; 450–600 ms: $t(34) = -3.22$, $p = .003$, $d_Z = 2.68$, Directional: $M = -.0020$, $SD = 0.0013$, Neutral: $M = .0017$, $SD = 0.0011$) (compare Figure 7). The main effect of CENTRAL CUE was not significant ($F(1,34) = 1.34$, $p = .256$, $\eta_p^2 = .04$).

The main effect of GROUP was not significant, indicating no differences between the stress and control group regarding LPS ($F(1,34) = 0.18$, $p = .674$, $\eta_p^2 = .005$). No other interaction effect including GROUP was significant i.e., CENTRAL CUE * GROUP ($F(1,34) = 0.04$, $p = .847$, $\eta_p^2 = .001$), BIN * GROUP ($F(12,408) = .866$, $p = .512$, $\eta_p^2 = .025$, $\varepsilon = .451$), or CENTRAL CUE * BIN * GROUP ($F(12,408) = 0.841$, $p = .518$, $\eta_p^2 = .02$, $\varepsilon = .397$).

Figure 8

Top: Alpha Lateralization for type of central cue. Bottom: Time-Frequency-Plots for ipsi-contralateral difference in directional and neutral cues per experimental group.



Note. **Top:** LAP scores over both groups during the endogenous cueing period show a clear decrease in LAP scores for directional central cues. **Bottom:** Time Frequency Plots show ipsi-contralateral difference in power. Blue shows higher

ipsilateral power. Green shows balanced power. Red shows higher contralateral power. **Top TF-Plots:** For both experimental groups directional cues show increased ipsilateral power within the alpha range (7-12 Hz) until the first 600 ms after central cue onset. **Bottom TF-Plots:** In contrast to the directional cues, in neutral cues there is no clear trend in the ratio of ipsi- to contralateral power.

Discussion

The aim of this study was to explore the influence of nongenomic stress effects on covert visual attention, more specifically if stress modulates attention through promotion of bottom-up and impairing of top-down mechanisms as proposed by Hermans and colleagues (2014). This would show in improved RT and response accuracy for exogenous trials, and in reduced behavioral performance in endogenous trials as well as in reduced lateralization of posterior alpha power in the experimental group compared to the control group.

Response to the CPT

In order to test these hypotheses, a valid stress response was required, that fulfills the essential criterion of activating the HPA axis and the SAM system, that produce the nongenomic stress effects that are supposed to carry the proposed network shift. For this purpose, participants of the stress group were exposed to a bilateral CPT before each experimental block, while the control group went through a warm water control procedure.

The stress group showed increased blood pressure during all three of the CPTs and after the third block, while heart rate only increased significantly during the second CPT. Increases in blood pressure mainly originate from alpha-adrenergically mediated peripheral vasoconstriction, whilst heart rate responses are beta-adrenergically mediated (Nater & Rohleder, 2009). Usually, the bilateral CPTs shows to provoke both alpha- and beta-adrenergic activation reliably increasing both blood pressure and heart rate (Bachmann et al., 2018; Larra et al., 2015). The non-significant increase in heart rate could be explained by the baroreflex, which reduces the heart rate in reaction to strong blood pressure increases as caused by the CPT. Importantly however, the significant increases in blood pressure shows clear SAM axis activation during every CPT procedure (De Vente, 2003).

The stress group showed a clear increase in saliva cortisol about 20 minutes after the first CPT which is in line with earlier findings of cortisol response to bilateral feet CPTs (Bachmann et al., 2018; Larra et al., 2015). The decrease in cortisol during the experiment was observed before with repeated stressor exposure and activation of the HPA axis (e.g., Sänger et al., 2014). It can be explained by glucocorticoid actions to prevent stress-activated defense reactions from overshooting which would itself threaten homeostasis (Sapolsky et al., 2000).

Subjective reactions showed a clear increase of stress, arousal, and pain in the stress group compared to the control group. Stress and arousal increased immediately after every CPT procedure but did not differ between groups after the experimental blocks respectively immediately before the CPT procedures. Regarding subjective pain, there were clear differences between the groups, with the control group rating the warm water procedure as not painful, and the stress group perceiving the CPT as moderately to highly painful. In conclusion, stress induction in the stress group using the bilateral CPT was successful showing clear indication of SAM and HPA axis activation starting from the first CPT.

Cueing Effects

In order to observe stress effects on covert visual attention, the experimental paradigm should modulate endogenous and exogenous attention through the central and peripheral cues which should show in corresponding behavioral results.

Central cues showed clear cueing effects on behavioral performance. Directional central cues containing information about the target location produced significantly faster reaction times, increased proportion of correct responses, and decreased proportion of errors and misses in comparison to neutral central cues that provided no spatial information about the target location. Analogue, peripheral cues showed increased behavioral performance for compatible cues that correspond to the central cue and target location versus incompatible cues that corresponded to the opposite direction. As for central cues, the behavioral benefits of compatible peripheral cues showed in reaction times and all accuracy measures. For the different SOAs between the peripheral cue and the target, behavioral results showed improved performance for longer SOAs of 250 ms compared to short SOAs of 100 ms. However, performance increases of long SOAs only showed in reaction time and proportion of correct responses, while SOA did not significantly affect proportion of errors or misses. As no masks were used following target presentation, likely visual afterimages were present in participants lasting longer than the target presentation of 7 ms. However, this should not limit the results.

These results are in line with the original publication (Posner, 1980) and other variants of this paradigm (reviewed in Chica et al., 2013). Both the central cue and the peripheral cue show the expected validity effects associated with endogenous and exogenous deployment of attention. The reported SOA effect is also in line with the literature regarding discrimination tasks and the specific interval lengths used. In discrimination tasks, behavioral results for target compatible peripheral cues improve with increasing SOAs up to approximately 400 to 500 ms (Chica et al., 2006; Van der Lubbe et al., 2005). For longer SOAs decreased behavioral

performance is seen for target compatible cues, an effect known as inhibition of return (IOR) (Posner & Cohen, 1984). As in this experiment the long SOA is far below 400 ms, as expected no IOR effect is seen.

Following the first block, behavioral performance improved which is likely due to training effects. This might indicate that the training block was too short to get achieve a constant performance level of the participants. However, for RT results improved up until block 3 which would be an impractically long training block.

Concluding, both central cues and peripheral cues show the expected behavioral results reported in the literature. The results for the different SOAs used are also in line with earlier findings in discrimination tasks. Therefore, the paradigm shows that it successfully manipulates both endogenous and exogenous mechanisms of covert spatial attention, making it suitable for examining the research question.

EEG

The EEG results should be in line with the cueing effects associated with endogenous attentional orienting. As expected, the EEG showed changes in alpha-band activity (7 – 12 Hz) over left and right posterior electro sites (P3/4, P7/8, PO7/8) in response to the central cue. For the baseline period prior to the cue, LAP between directional and neutral central cues did not differ. Starting from ~150 ms until ~600 ms, directional cues produced significantly lower LAP than neutral central cues indicating higher ipsi- than contralateral alpha power at described electrode sites. This is mostly in line with earlier results. When endogenous attention is focused to a specific location in the left or right hemifield, posterior alpha power decreases over the contralateral hemisphere, while it increases over ipsilateral sites (Chica et al., 2013; Thut et al., 2006). This change in alpha-power is associated with the inferiorparietal lobule and the intraparietal sulcus of the dorsal fronto-parietal network which are activated proportionally to demands of endogenous visuospatial attention (Chica et al., 2013; Hopfinger et al., 2000). Whereby the direction and the spatial location of the effects found in this study are consistent with the literature, their onset is slightly different. While Thut et al. (2006) only reports differences in alpha lateralization between cues for the whole cue-target interval of 0 to 1300 ms after cue onset, other studies show differences in the onset of alpha lateralization after endogenous cues e.g. from 400 ms (Van der Lubbe et al., 2019), to 540 ms (Van der Lubbe & Utzerath, 2013) or as late as 700 ms (Grent-'t-Jong & Woldorff, 2007). With the onset of significant alpha lateralization as early as 150 ms found in this study, it is likely that not only endogenously induced but also exogenously evoked oscillatory activity is represented in the

EEG. This could be due to the arrows used as directional central cues. Verleger et al. (2000) showed that asymmetrical arrows elicit automatic orientation effects. However, if purely exogenously evoked, differences in alpha lateralization should not be seen as late as 600 ms after cue onset. This suggests a mixture of exogenously evoked and endogenously induced activity. Overall, in some parts the central cues produced the expected effects in the EEG signature of endogenous attention showing a difference in hemispherical alpha power in response to the cue. However, as the onset indicates not purely endogenously induced oscillatory power, EEG findings should be interpreted with caution.

Stress Effects on Attention

Behavioral results showed condition specific effects on RT and misses for blocks following the CPT procedure. Regarding misses, a specific difference between stress and control group was seen. First, for proportion of misses the two-way interaction of GROUP * PERIPHERAL CUE showed that peripheral cues did not produce a validity effect in the stress group. The significant three-way interaction of GROUP * CENTRAL CUE * PERIPHERAL CUE showed that this pattern depends on the type of central cue preceding the peripheral cue. For directional central cues neither group showed significant validity effects for the peripheral cue. For neutral central cues only the control group showed a significant validity effect for peripheral cues. Opposite to what would be expected, in the stress group errors after compatible and incompatible peripheral cues did not differ significantly. In other discrimination or detection tasks, misses are partly interpreted as lapses of sustained attention (Martel et al., 2014; O'Connell et al., 2009). When looking at the lacking validity effect of peripheral cues in the stress group, it is apparent that the stress group does not simply show more lapses, as this could not explain the missing validity effect, nor does to stress group shows more proportion of misses than the control group in other cue-conditions. Thus, is it reasonable to assume, that after stress induction groups differ in their ability to detect salience-based stimuli following neutral central cues. This is unlikely to be due to group differences in actual perceptual ability to recognize the target, since overall accuracy is very high and only two different target symbols were used.

Looking at the effect of GROUP * CENTRAL CUE * SOA on RT, a somewhat similar pattern shows. While for both groups in directionally central cued trials, longer SOAs produce shorter RTs, only the control group shows this SOA effect in neutral central cues. For the stress group, short and long SOAs do not produce different RTs in neutral central cues. This fits in with the described effects showing in proportion of misses. Both effects show in neutral cued trials either in spatial relation to the peripheral cue or in temporal relation to the interval of

peripheral cue to target. It is notably that the stress effects only show in proportion of errors and RTs and not in overall accuracy. However, in case of this paradigm, it seems plausible that differences in the time domain i.e., short, and long SOA show in reaction times, whereas spatial differences i.e., compatible versus incompatible peripheral cues show in accuracy measures.

The interaction effect of GROUP * BLOCK * CENTRAL CUE showed decreased accuracy of the stress group in neutral cued trials in the fourth compared to the third experimental block. This is the only group specific effect of block and specifically shows in neutral central cues comparable to the above-described group effects. As accuracy in neutral central trials is equally high during blocks 2 and 3, from a time perspective the sharply declining accuracy in block 3 might be explained by early onset of genomic stress effects. However, the lack of other significant behavioral effects regarding block (i.e., time course) renders this not very likely. Another reason could be a lack of motivation to attend neutral cues that offer no spatial information and more difficult to answer.

EEG analysis did not show any significant group differences regarding LAP. While both groups showed differences in LAP depending on the type of cue, stress and control group did not significantly differ in that regard.

As a preliminary conclusion, the results show little evidence for a general inhibition of endogenous attentional control. Neither the behavioral results nor the EEG results show group specific effects on central cues. Behavioral results point to group specific effects dependent on the peripheral cue. This is seen in significant effects on RT in temporal proximity (short and long SOAs) to the target, as well as in spatial proximity (target compatible and incompatible peripheral cues) to the target shown by significant effects on proportion of misses.

These results partly contrast with previous results. Sänger et al. (2014) found significant differences in a change detection-like task where participants had to respond to luminance changes of the stimuli and ignore salient orientation changes. After participants were exposed to a socially evaluated CPTs (SECPT), they showed higher error rates in trials that required top-down control to react to a less salient target instead of a more salient and spatially separated distractor. In the EEG this was reflected in a reduced N1pc which shows a reduced allocation to the relevant luminance change. The N2pc, which showed in trials where attention had to be re-allocated to the less salient luminance change, was decreased after the stress intervention. For their error rate, the authors aggregated both errors and misses. In a condition where participants have to detect unilateral luminance changes which largely addresses exogenous mechanisms, stress and control group significantly differ. Interestingly, in this condition errors

largely consisted of misses. This is comparable to the results found in this study, where the stress group also shows significantly more misses in compatible peripheral cues that address exogenous attention mechanisms. However, the lack of endogenous impairment found in this study does not fully align with decreased amplitude of the N2pc found in Sänger et al. (2014). As the target induced N2pc as an indicator of attentional reallocation methodologically cannot be completely isolated from bottom-up processes, stress induced effects on the interaction of bottom-up and top-down processes might be the underlying reasons for the different findings. Furthermore, as briefly addressed in the introduction, differences in the stress protocol could also contribute the varying results. In Sängers and colleagues' experiment, the last experimental block starts 100 minutes after the first SECPT whereby genomic stress effects could interfere with the initial non-genomic stress effects.

Recent evidence from Hu et al. (2021) and Broeders et al. (2021) point to a similar direction regarding the lack of stress effects on endogenous attention. After successful stress induction through the Trier Social Stress Test (TSST), Hu and colleagues monitored temporal dynamic changes of the large-scale brain networks associated with the salience respectively the executive control network. Stressed participants showed larger occurrences and coverage of the salience network which positively correlated with subjective stress. No significant stress effects on the executive control network were found. Furthermore, after acute stress salience and executive control network showed higher bidirectional transition probabilities which points to increased communication between the two networks. Broeders et al. (2021) found similar evidence with activity of the dorsal attention network only increasing approximately 90 minutes after exposure to the TSTT. Taken together, these findings are in line with our results that do not show an impairment of endogenous processes but indicate an interaction of exogenous and endogenous processes. Importantly, Broeders and colleagues' evidence is partly in line with the network shift proposed by Hermans et al. (2014) that suggests an upregulation of endogenous attention processes during the genomic stress effects that start approximately one hour after stress exposure.

Concluding, the behavioral and EEG evidence found in this study suggests stress effects that point away from a pure impairment of endogenous attention processes. This is supported by recent neuroimaging studies. Behavioral measures indicate complex stress effects on exogenous attentional mechanisms possibility in interaction with endogenous mechanisms. EEG data addressing exogenous cues and targets were not analyzed in this study but should give more insights on this interaction proposedly altered by stress effects. In their framework,

Hermans and colleagues (2014) have addressed that the resource allocation in reciprocal related networks is far from understood. The mechanism in which top-down and bottom-up processes interact with each other might offer an explanation for the stress effects found.

Limitations & Strengths

The study has different limitations. First, we cannot fully exclude early genomic effects that potentially cancel out non-genomic cortisol effects. As pointed out in Larra et al. (2016), only pharmacological studies could address this problem, which would in turn lack in other aspects of a full stress response. Another limitation concerns the arrows used as directional central cues. Verleger et al. (2000) showed that asymmetrical arrows elicit automatic orientation effects. Thereby the directional central cues cannot be considered as purely endogenous cues.

A strength of the study is that it isolates genomic, and catecholaminergic and non-genomic stress effects at the current state of research. In other studies, stress exposures were often timed in a way that opposing stress effects were analyzed collectively. Furthermore, it was shown that the repeated stress exposure activated an SAM response after every CPT, sustaining shorter lasting catecholaminergic effects, that are often ignored in other studies where the focus lies on cortisol effects in the one-hour interval post the stressor.

Finally, the combination of central and peripheral cues used in this paradigm proofed to work and showed the expected validity effects for central and peripheral cues. As found by Muller and Rabbitt (1989), it could also replicate the finding of endogenous attention enhancing exogenous attention when they indicate the same location as seen in improved behavioral performance for directional-compatible trials compared to neutral-compatible trials. Furthermore, it was replicated that incompatible peripheral cues during endogenously deployed attention can attract attention as seen in better performance for valid-valid than valid-incompatible trials (Theeuwes, 1991; van der Lubbe & Postma, 2005). Concluding, the version of the Posner cueing paradigm and the combination of behavioral and EEG measures allowed for a separate consideration of endogenous and exogenous attentional processes. This is a strength compared to previous studies in which bottom-up and top-down processes methodologically could not be considered absolutely separate.

Practical Implications and Future Research

Because the results do not offer a clear perspective on the complex stress influence on attentional mechanisms, practical implications are limited. However, the results point towards stress effects that do not impair top-down processes as has been assumed before. Should this

be supported by further results, endogenous cues, previously considered as limited, could be used to support attention allocation in potentially stressful, safety relevant working environments (e.g., air traffic controller, pilots, maritime transport). This is especially important as endogenously engaged attention enables more complex processes e.g., selecting one thing over another, following a specific rule etc. This allows for more complex assistance to be presented in stressful situations potentially caused by unfamiliar situations that need more guidance. However, future work should investigate other stimulus processing paradigms (e.g., Simon, Flanker task) to extend these indications. Furthermore, the additionally gathered data in this experiment should be analyzed. As EEG and behavioral measures indicated no straightforward stress effect on endogenous attention but an effect on peripheral cues or the interaction of endogenous and exogenous processes, EEG analysis time locked to the peripheral cue, target, and response should provide more insights on the underlying processes. Furthermore, behavioral and EEG measures should be related to stress measures to test whether group differences vary as a function of the stress response.

Hermans and colleague's framework and the recent evidence of Broeders et al. (2021) indicate that genomic stress effects might have beneficial effects on endogenous attention. To further sharpen the understanding of stress effects on attention, an adapted version of the experimental paradigm used could be to after onset of genomic stress effects.

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Appendix I – Informed Consent

Probandeninformation zur Studie „Effekte von Stress auf Informationsverarbeitungsprozesse: Bedeutung kardioafferenter Signale“

Sehr geehrte Dame, sehr geehrter Herr,

wir freuen uns über Ihr Interesse an unserer Studie. An dieser Stelle möchten wir Sie über die Ziele, Hintergründe und Abläufe des Forschungsvorhabens informieren. Wir tun dies in Form einiger Fragen, die Ihnen vielleicht bereits in den Sinn gekommen sind. Sollten Sie weitere Fragen haben, stellen Sie diese einfach dem Versuchsleiter.

Was wird gemacht?

Falls Sie sich zur Teilnahme an unserer Studie bereit erklären, passiert folgendes: Wir zeigen Ihnen am Computer verschiedene Reize. Dies können Buchstaben, Pfeile, laute oder leise Geräusche sowie leichte Luftstöße im Bereich der Schläfen sein. Ihre Aufgabe ist es, mittels Tastatur auf die Reize zu reagieren. Welche Tasten zu drücken sind, wird Ihnen zuvor am Bildschirm erklärt. Über den Versuch verteilt (insgesamt 3 Mal) erfolgt ein 3-minütiger Kaltwasser-Stresstest, bei dem beide Füße in eiskaltes Wasser getaucht werden (Wassertemperatur 4 °C), oder eine Kontrollprozedur. Die Zuteilung ist zufällig und weder durch Sie noch durch den Versuchsleiter zu beeinflussen. Während des Versuchs messen wir Ihre Herz-, Muskel- und Gehirnaktivität sowie die Cortisol- und Zytokinkonzentration im Speichel. Hierzu werden eine EEG Kappe am Kopf, Elektroden unterhalb der Augen und am Oberkörper, sowie eine Blutdruckmanschette am Arm angebracht. Wir bitten Sie, im Laufe des Versuchs einige Speichelproben mit Hilfe von Wattestäbchen zu sammeln.

Was ist das Ziel der Studie?

Es soll erforscht werden, ob die Erregung des Herz/Kreislauf-Systems eine Rolle für die Beeinflussung von Informationsverarbeitungsprozessen durch Stress spielt.

Welche Risiken ergeben sich durch eine Studienteilnahme, speziell durch den Kaltwasser-Stresstest?

Wenn Sie Kälte im Allgemeinen problemlos vertragen, ist bei der Durchführung des Kaltwasser-Stresstest ist nicht mit medizinisch relevanten und anhaltenden Komplikationen zu rechnen. Wichtig ist, dass bei Ihnen keine Raynaud-Erkrankung (kälteinduzierter Verschluss kleiner Blutgefäße) vorliegen darf und Sie nicht an durch Kälte hervorgerufenen Hautveränderungen leiden dürfen. Bei der Durchführung des Tests treten zum Teil deutliche Schmerzen auf. Dabei wird kein Gewebe anhaltend geschädigt. Die Schmerzen werden von fast allen Teilnehmerinnen und Teilnehmern ausgehalten, sie werden ganz sicher nach Beendigung des Tests rasch abklingen. Wir bitten Sie deshalb, den Test nicht abzubrechen. Falls Sie Schmerzen verspüren, die Sie nicht mehr aushalten, können Sie die Füße bereits vor Beendigung des Tests aus dem kalten Wasser ziehen. Falls kein Abbruch durch Sie erfolgt, wird der Versuchsleiter den Test beenden. Beim Test wird Ihr Blutdruck ansteigen. Normalerweise werden dabei Werte nicht überschritten, wie sie etwa auch bei Sport auftreten. Wenn Sie also, mindestens gelegentlich, ohne Probleme Sport treiben, bei Ihnen kein erhöhter Blutdruck bekannt ist und in Ihrer Familie keine Erkrankungen der Arterien (z.B. Gefäßaussackungen [Aneurysmen] der Gehirnarterien) vorliegen, stellt die zu erwartende Blutdruckerhöhung kein besonderes Risiko dar.

Im Fall einer (extrem unwahrscheinlichen) erheblichen Gesundheitsbeeinträchtigung durch den Versuch steht medizinische Hilfe durch das nahegelegene Klinikum Dortmund zur Verfügung. Für die Ersthilfe stehen Ärzte des IfADo verschiedener Fachrichtungen, insbesondere der Betriebsarzt Professor Dr. Golka, in ständiger Rufbereitschaft.

Welchen Nutzen hat eine Studienteilnahme für mich?

Das Ziel unserer Studie ist, ein tieferes Verständnis für grundlegende psychophysiologische Vorgänge im Körper zu gewinnen. Man spricht daher von Grundlagenforschung. Ihre Teilnahme dient somit dem wissenschaftlichen Erkenntnisgewinn, über den wir Sie nach Abschluss der Studie natürlich auch gerne informieren. Ein anderer Nutzen besteht für Sie nicht.

Kann ich an der Studie teilnehmen?

Wenn Sie gesund sind und keine Medikamente dauerhaft einnehmen (Ausnahme: Kontrazeptiva oder gelegentlich eine Kopfschmerztablette), dann können Sie an der Untersuchung teilnehmen. Wenn bei Ihnen durch Kälte Haut- oder andere Gewebeveränderun-

gen auftreten, dürfen Sie an der Studie nicht teilnehmen. Dies gilt auch, wenn andere Ausschlusskriterien (z.B. erhöhter Blutdruck) bestehen. Wenn Sie sich bezüglich möglicher Erkrankungen unsicher sind, fragen Sie bitte den Versuchsleiter.

Was geschieht mit meinen Daten?

Alle im Rahmen der Studie anfallenden Informationen werden vertraulich behandelt und aufbewahrt. Die Erhebung, Speicherung und Auswertung Ihrer Daten erfolgt pseudonymisiert (d.h. verschlüsselt, ohne Angabe von Name, Anschrift und ähnlichem). Die Weitergabe Ihrer Daten an Dritte (z.B. Publikation) erfolgt ausschließlich in anonymisierter Form. Allerdings haben Vertreter von Aufsichtsbehörden unter Umständen einen Anspruch darauf, Ihre Originaldaten einzusehen.

Beachten Sie bitte folgende Hinweise:

Ihre Teilnahme erfolgt freiwillig nach ausführlicher Information und Beantwortung aller Ihrer Fragen. Sie können jederzeit ohne Angabe von Gründen die Untersuchung abbrechen, ohne dass Ihnen daraus Nachteile entstehen würden. Die Berechnung der Aufwandsentschädigung (12 € pro Versuchsstunde oder entsprechende VP Stunden) erfolgt rein auf Basis der Versuchsdauer, dies gilt auch bei Abbruch des Versuchs.

Einwilligungserklärung

Hiermit bestätige ich, dass ich die ausführliche Probandeninformation sowie die Einwilligungserklärung erhalten, diese gelesen und verstanden habe. Insbesondere ist mir bekannt, dass die Durchführung des Kaltwasser-Stresstests vorgesehen ist, dass weiterhin die Teilnahme an der Studie freiwillig erfolgt und von mir jederzeit ohne Angabe von Gründen abgebrochen werden kann, ohne dass mir daraus Nachteile entstehen. Die Hinweise zum Datenschutz habe ich zur Kenntnis genommen. Einer Weitergabe anonymisierter Daten aus wissenschaftlichen Gründen stimme ich auf der Grundlage der Datenschutzerklärung zu. Ich kann mich zu jedem Zeitpunkt mit weiteren Fragen an die Versuchsleiter wenden.

Name, Proband:

Datum & Unterschrift, Proband:

Datum & Unterschrift, aufklärender Versuchsleiter:

Appendix II – General Questionnaire

Fragebogen 1

Geschlecht: ☐ w ☐ m

Alter: ____ Jahre

Höchster Schulabschluss: _____

Beruf/Studiengang: _____

Leiden Sie an neurologischen oder psychiatrischen Störungen? ☐ ja ☐ nein

Leiden Sie an Schlafstörungen? ☐ ja ☐ nein

Nehmen Sie regelmäßig Medikamente ein bzw. haben Sie heute Medikamente eingenommen? ☐ ja ☐ nein

Wenn ja, welche? _____

Nehmen Sie Hormone ein? ☐ ja ☐ nein

Wenn ja, welche? _____

Treiben Sie Sport? ☐ ja ☐ nein

Wenn ja, welchen Sport? _____

Wie viele Stunden pro Woche circa? _____

Rauchen Sie? ☐ ja ☐ nein

Wenn ja, wie viele Zigaretten pro Tag rauchen Sie normalerweise? ____

Wenn ja, haben Sie heute schon geraucht? ☐ ja ☐ nein

Wie viel haben Sie heute gefrühstückt?

☐ gar nicht ☐ wenig ☐ normal ☐ mehr als normalerweise

Haben Sie heute bereits Kaffee oder schwarzen Tee getrunken? ☐ ja ☐ nein

Wenn ja, wie viele Tassen? _____

Tragen Sie eine Brille oder Kontaktlinsen? ☐ ja ☐ nein

Wenn ja, wie viel Dioptrien? _____

Haben Sie schon einmal an einem Kaltwassertest teilgenommen? ☐ ja ☐nein

Wann sind Sie gestern Abend schlafen gegangen? ___ : ___ Uhr

Wann sind Sie heute Morgen aufgestanden? ___ : ___ Uhr

Wie viel Schlaf hatten Sie in etwa vergangene Nacht? ___ h, ___ m

Um wie viel Uhr gehen Sie normalerweise schlafen? ___ : ___ Uhr

Um wie viel Uhr stehen Sie normalerweise morgens auf? ___ : ___ Uhr

Wie lange schlafen Sie normalerweise pro Nacht? ___ h, ___ m

Appendix III – Other Behavioral Interaction Effects

Table 1

Paradigm: Interaction Effects for RT, PC, PE, PM.

Measure		RT			Pcorrect			Pmisses			Perrors		
	df	F	*p*	ηp^2	F	*p*	ηp^2	F	*p*	ηp^2	F	*p*	ηp^2
CENTRAL CUE * PERIPHERAL CUE	1,39[a]	1.02	.318	.03	0.18	.675	.005	0.64	.429	.02	1.08	.305	.03
CENTRAL CUE * SOA		0.01	.939	.01	0.68	.419	.04	1.27	.267	.03	3.51	.068	.08
PERIPHERAL CUE * SOA		0.60	.442	.02	0.26	.590	.008	1.20	.280	.03	0.2	.90	< .001
CENTRAL CUE * PERIPHERAL CUE * SOA		0.001	.947	.001	1.10	.300	.03	0.68	.412	.02	0.26	.62	.007

Note. Interaction effects with F values greater 1 are bold. [a] Holds for all analyses.

Table 2

Stress effects: Interaction Effect RT, PC, PE, PM.

Measure		RT			Pcorrect			Pmisses			Perrors		
	df	F	*p*	ηp^2	F	*p*	ηp^2	F	*P*	ηp^2	F	*p*	ηp^2
GROUP		**2.12**	**.147**	**.05**	**1.27**	**.266**	**.03**	0.37	.547	.09	0.92	.342	.02
CENTRAL CUE * GROUP		0.04	.850	.001	< .001	.993	< .001	0.50	.484	.01	0.22	.645	.01
PERIPHERAL CUE * GROUP	1,39[a]	0.02	.896	< .001	0.77	.394	0.2	**4.32**	**.044***	**.10**	0.73	.789	.002
SOA * GROUP		**1.1**	**.303**	**.03**	**1.61**	**.212**	**.04**	**2.73**	**.107**	**.07**	0.03	.865	.001
CENTRAL CUE *		0.064	.801	.002	1.81	.187	.04	**4.26**	**.046***	**.10**	0.02	.904	< .001

PERIPHERAL CUE * GROUP												
CENTRAL CUE * SOA * GROUP	**5.18**	**.028***	**.117**	2.07	.158	.05	**3.43**	**.071**	**.08**	0.08	.786	.002
PERIPHERAL CUE * SOA * GROUP	0.14	.710	.004	0.90	.349	.02	0.01	.925	< .001	**1.79**	**.188**	**.04**
CENTRAL CUE * PERIPHERAL CUE * SOA * GROUP	0.40	. 5 3 1	.010	0.02	.877	.001	**1.14**	**.291**	**.03**	0.80	.377	.02

Note. Significant Interaction effects with F values greater 1 are bold. [a] Holds for all effects.