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The Role of Cognitive Control and Approach-Avoidance Motivation in the Relationship between Stress and Aggression - A Psychophysiological Investigation

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General Abstract

The stress hormone cortisol as the end-product of the hypothalamic-pituitary-adrenal (HPA) axis has been found to play a crucial role in the release of aggressive behavior (Kruk et al., 2004; Böhnke et al., 2010). In order to further explore potential mechanisms underlying the relationship between stress and aggression, such as changes in (social) information processing, we conducted two experimental studies that are presented in this thesis. In both studies, acute stress was induced by means of the Socially Evaluated Cold Pressor Test (SECP) designed by Schwabe et al. (2008). Stressed participants were classified as either cortisol responders or nonresponders depending on their rise in cortisol following the stressor. Moreover, basal HPA axis activity was measured prior to the experimental sessions and EEG was recorded throughout the experiments.

The first study dealt with the influence of acute stress on cognitive control processes. 41 healthy male participants were assigned to either the stress condition or the non-stressful control procedure of the SECP. Before as well as after the stress induction, all participants performed a cued task-switching paradigm in order to measure cognitive control processes. Results revealed a significant influence of acute and basal cortisol levels, respectively, on the motor preparation of the upcoming behavioral response, that was reflected in changes in the magnitude of the terminal Contingent Negative Variation (CNV).

In the second study, the effect of acute stress and subsequent social provocation on approach-avoidance motivation was examined. 72 healthy students (36 males, 36 females) took part in the study. They performed an approach-avoidance task, using emotional facial expressions as stimuli, before as well as after the experimental manipulation of acute stress (again via the SECP) and social provocation realized by means of the Taylor Aggression Paradigm (Taylor, 1967). Additionally to salivary cortisol, testosterone samples were collected at several points in time during the experimental session. Results indicated a positive relationship between acute testosterone levels and the motivation to approach social threat stimuli in highly provoked cortisol responders. Similar results were found when the testosterone-to-cortisol ratio at baseline was taken into account instead of acute testosterone levels. Moreover, brain activity during the approach-avoidance task was significantly influenced by acute stress and social provocation, as reflected in reductions of early (P2) as well as of later (P3) ERP components in highly provoked cortisol responders. This may indicate a less accurate, rapid processing of socially relevant stimuli due to an acute increase in cortisol and subsequent social provocation.

In conclusion, the two studies presented in this thesis provide evidence for significant changes in information processing due to acute stress, basal cortisol levels and social provocation, suggesting an enhanced preparation for a rapid behavioral response in the sense of a fight-or-flight reaction. These results confirm the model of Kruk et al. (2004) proposing a mediating role of changed information processes in the stress-aggression-link.

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Chapter I

General Introduction and Outline of the Thesis

1.1 Introduction

One evening in May 2010, a 16 year-old boy named Elias knifed another adolescent named Mel who was unknown to him, without any reason. Later, the offender states that he had been mad at his younger brother with whom he had quarrelled just before the deathly meeting with Mel. When he then saw Mel laughing with a friend, he felt provoked by their amusement and killed him by knifing him in the chest (Rückert, 2011). According to a report of the World Health Organization (WHO, 2007), approximately 1.6 million human beings world-wide die every year due to violence that is self-inflicted, interpersonal, or collective. In case of Elias and Mel, Elias perceived Mel's laughter as a further provocation in addition to previous events that have led to a high arousal in Elias. This example of a true story illustrates that the perpetrator would probably have reacted aggressively regardless of the behavior of the victim, and it raises the question of how information processing is changed in such an aggressive and aroused/stressed state. This thesis deals with the investigation of altered cognitive processes, in order to elucidate some of the accountable mechanisms that enhance aggressive behavior in the context of stress.

Both stress and aggression are concepts with primarily negative connotations. Stress is one of the most important contributors to many disorders (Chrousos, 2009; Chrousos & Gold, 1992; Sapolsky, 2000) and is generally tried to be avoided as best as possible. Aggressive behavior is of equally bad reputation and is socially condemned. However, they both also fulfil adaptive and essential functions for survival. For instance, in certain situations, such as a threat by a dangerous animal or person, it is vital to have a stress response and, if necessary, to defend oneself in an aggressive manner. However, if stress responses are elicited chronically or in an inappropriate manner (too strong or too weak reactions) and if aggressive behavior occurs without a proper cause, this can turn into a problem.

Given the high relevance of the topic, this thesis aims to further elucidate contributing factors that increase the likelihood of aggressive behavior in stressful contexts. In the following, the first chapter will give a general introduction to the topics of stress, aggressive behavior, and the relationship between them. Also, the role of social information processing and neural underpinnings of aggression will be outlined. This chapter will address previous research in animals as well as in humans. Chapters II and III comprise two event-related potential (ERP) studies investigating the influence of acute stress on cognitive control processes (study 1, chapter II) and the influence of acute stress and social provocation on approach-avoidance motivation (study 2, chapter III). Finally, in chapter IV, the results of both studies will be

discussed and integrated into a broader framework, and conclusions for future research will be drawn.

1.2 Stress

1.2.1 Definition

Although there exists a variety of different definitions of stress, what most of them have in common is the concept of homeostasis. One of the more recent definitions was given by Ulrich-Lai & Herman (2009) who broadly define stress as "[...] an actual or anticipated disruption of homeostasis or an anticipated threat to well-being" (p. 397). This challenge of homeostasis results in adaptive responses of the individual, in order to re-establish the internal balance (Levine, 2005). Stress includes the following components: Firstly, there is an aversive stimulus (stressor), which can be internal or external, as well as physiological (e.g., extreme forms of cold) or psychological (e.g., an aversive situation, such as public speech). Secondly, the stressor is evaluated subjectively by the individual, and thirdly, the organism reacts with a stress response manifesting itself on a physiological, behavioral, cognitive, and emotional level (Steckler, 2005). Since the focus of this thesis lies on the psychophysiology of the stress-aggression-link, the physiological part of the stress response will be further explained in the following.

1.2.2 The Physiological Stress Response

The physiological stress response manifests itself on two stress axes: On the one hand, a stressor leads to a rapid activation of the sympathetic nervous system (SNS), involving the release of noradrenaline and adrenaline (de Kloet, Joels & Holsboer, 2005) as well as an increase of heart rate, skin conductance, blood pressure, and muscle activity (Birbaumer & Schmidt, 2006, pp. 141-156). On the other hand, with the onset of a stressor, the hypothalamic-pituitary-adrenal (HPA) axis is activated, leading to an increased concentration of glucocorticoid hormones. These steroid hormones are produced in the adrenal glands - primarily cortisol in humans and corticosterone in rodents (de Kloet et al., 2005). As displayed in Figure 1, the hormonal cascade begins with a stimulation of the hypothalamus, a brain structure importantly involved in the regulation of homeostasis (Sapolsky, 2000). Thereupon, the hypothalamus secretes the corticotropin-releasing hormone (CRH), which further stimulates the pituitary, resulting in the secretion of the adrenocorticotrophic hormone (ACTH). This leads to a release of the end product cortisol by the adrenal glands (Kirschbaum

& Hellhammer, 1999). The crucial function of the so-called stress hormone cortisol is the mobilization of energy reserves via gluconeogenesis, in order to prepare the organism for coping with the challenging situation (Sapolsky, Romero & Munck, 2000). In order to facilitate recovery, the released cortisol inhibits further activity of the HPA axis via a negative feedback loop. The interplay of the activation of the SNS and the HPA axis following a stressor acts on the organism to prepare rapid behavioral reactions, such as fight-or-flight responses (de Kloet et al., 2005).

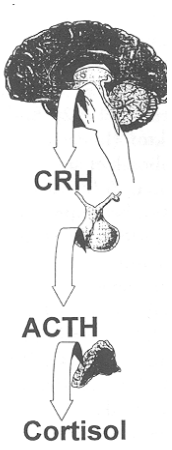

Figure 1 Illustration of the HPA axis (adapted from Kirschbaum & Hellhammer, 1999, p. 92).

Psychological stress particularly leads to a strong activation of the HPA axis, if the situation is perceived as uncontrollable or characterized by social-evaluated threat (Dickerson & Kemeny, 2004). One has to distinguish between acute and basal HPA axis activity or reactivity, respectively. In this thesis, the term "activity" of the HPA axis refers to the total level of cortisol concentrations, whereas "reactivity" applies to changes in the activity due to any external (e.g., stressors) or internal (e.g., cortisol awakening response) challenges (see Clow, Thorn, Evans & Hucklebridge, 2004). Furthermore, the term "basal" refers to trait-like aspects of HPA axis (re)activity, whereas "acute" corresponds to state (situational) aspects of HPA axis (re)activity. Under basal conditions, the HPA axis has a circadian rhythm. The cortisol secretion reaches its peak in the morning, shortly before or after awakening (cortisol awakening response, CAR) and declines slowly in the afternoon, until it reaches the circadian minimum during the night. After the first few hours of sleep, cortisol concentrations increase again (Kirschbaum & Hellhammer, 1999).

According to Hellhammer et al. (2007), reliable measures of basal HPA axis activity and reactivity require several assessments of the CAR over consecutive days. Specifically, in order to reliably assess trait aspects of basal cortisol secretion, the total area under the curve

(AUC total) or, equivalently used, the area under the curve with respect to ground (AUC_G), which both indicate the overall cortisol level, should be measured on at least two consecutive days. To get a reliable assessment of basal HPA axis *re*activity, however, the increase of cortisol after awakening (absolute or mean AUC increase) should be measured on at least six consecutive days (Hellhammer et al., 2007). Figure 2 displays the typical shape of a cortisol awakening response measured within one hour after awakening with the illustration of AUC total (HPA axis activity) and AUC increase (reactivity). Hellhammer et al. (2007) conclude that measures of AUC increase may be more appropriate for investigating state or situational effects, whereas AUC_G/AUC total may rather reflect trait aspects of HPA axis activity.

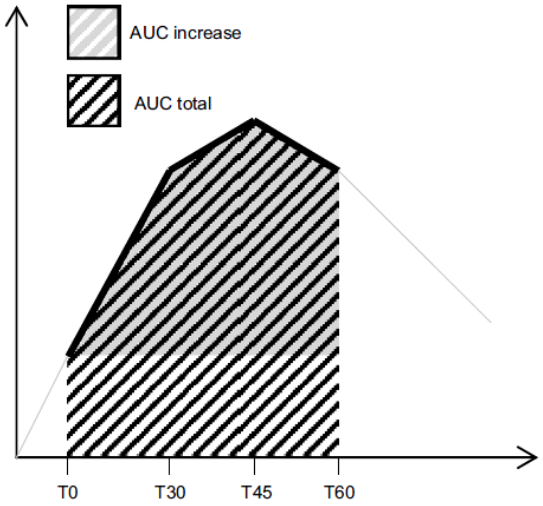

Figure 2 Illustration of AUC total and AUC increase as measures of the cortisol awakening response (Hellhammer et al., 2007, p. 81).

1.3 Aggression

1.3.1 Definition and Classifications

Aggressive behavior can be defined as "[...] any form of behavior directed toward the goal of harming or injuring another living being who is motivated to avoid such treatment" (Baron & Richardson, 1994, p. 7). This definition implies that an accidental harm is not aggression, as it is not aimed by the harm-doer. Moreover, injuring behavior as a concomitant of a benevolent action (e.g., in case of medical procedures that cause pain) is not aggressive behavior, either, since the actor intends to help and the target person is not motivated to avoid the treatment because of a long-term benefit. Furthermore, a distinction is made between aggression and

violence. The latter is the extreme form of aggressive behavior with the goal of severe harm, e.g., death (Anderson & Bushman, 2002).

Traditionally, aggression is dichotomized into categories, such as affective versus instrumental aggression or, often equivalently used, reactive versus proactive aggression. While the terms "affective" and "reactive" aggression, respectively, refer to aggressive behavior that is driven by anger and occurs in response to perceived provocation, "instrumental" or "proactive" aggression is conceived as a means to achieve a goal different from harming others (e.g. Geen, 2001), "[...] resulting from cold calculation rather than hot affect" (Anderson & Huesmann, 2003, p. 298). However, since such dichotomies have difficulties in integrating mixed motive aggression, Anderson & Huesmann (2003) argue for a dimensional approach instead of dichotomies. According to them, there are four dimensions along which aggressive behavior can be characterized: degree of hostile affect present, magnitude of automaticity, extent to which the primary or ultimate goal is to harm the victim versus to profit from the aggressive act, and amount of contemplation of consequences (Anderson & Huesmann, 2003).

1.3.2 An Integrative Framework for Theories on Aggression - The General Aggression Model

In order to consider and integrate existing theories of smaller domains of aggression, Anderson & Bushman (2002) have postulated the General Aggression Model (GAM). As depicted in Figure 3, this model focuses, amongst others, on person as well as situational factors that have an interactional influence on aggressive behavior. Person factors contain the following aspects that can have an important impact on the preparedness to an individual's aggression: traits (e.g., certain types of high self-esteem, such as in narcissists; Baumeister, Smart & Boden, 1996), sex (e.g., male and female aggression differences depending on type of provocation; Bettencourt & Miller, 1996), beliefs (e.g., self-efficacy and outcome efficacy; Bandura, 1977), attitudes towards violence, values (ethical or moral beliefs), long-term goals, and scripts of social situations. Situational factors involve the presence of aggressive cues (e.g., weapons effect, Berkowitz & LePage, 1967), provocation (e.g., insults, slights, physical attacks), frustration, pain and discomfort, drugs, and incentives (as triggers of instrumental aggression). These person and situational factors influence the behavioral outcome through cognitive, affective, and arousal routes that form the present internal state of the individual. For instance, concerning cognition, the accessibility of hostile thoughts can be increased by the person and situational factors. Also, the affect (mood and emotion) as well as the arousal

of the individual can be swayed by these person and situational factors. Arousal, in turn, has an impact on aggressive behavior by enforcing the dominant action tendency, such as aggressive motivation. Moreover, arousal elicited by other (irrelevant) sources can be transferred and attributed misleadingly to ambiguous situations (excitation transfer), thus producing anger and aggressive behavior. Finally, the GAM postulates complex information processes that influence the type of outcome - either thoughtful or impulsive behavior. These information processes include immediate appraisals as well as reappraisals of the present internal state. Depending on these (re)appraisals, on the importance of the outcome, and on the availability of resources, the resulting action is impulsive/aggressive or not (Anderson & Bushman, 2002).

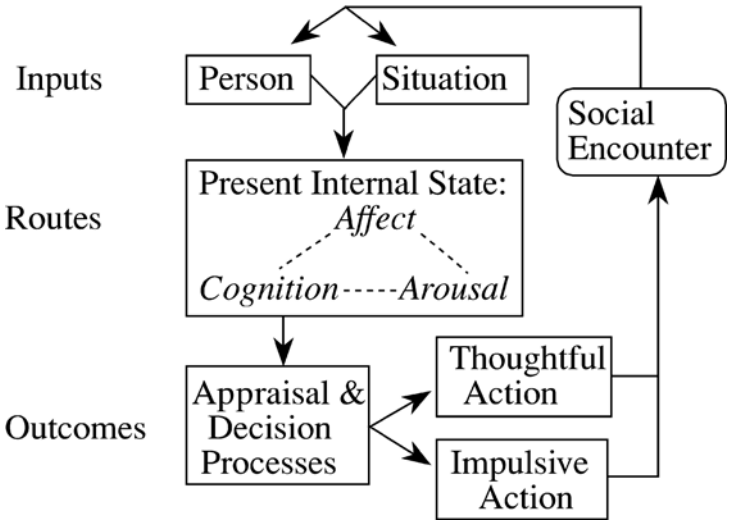

Figure 3 The General Aggression Model (Anderson & Bushman, 2002, p. 34).

To summarize, according to the GAM, aggressive behavior is a result of multiple interacting causes within the person and in the situation that influence complex decision and information processes through cognition, affect, and arousal of the individual (Anderson & Bushman, 2002).

However, the GAM does not refer to the physiological and neural mechanisms underlying the mental processes that trigger aggressive behavior. Since this is a crucial topic for this thesis, the next section deals with social information processing before, during, and after aggressive behavior.

1.3.3 Social Information Processing and Aggression

Crick & Dodge (1994) have postulated a Model of Social Information Processes, which involves several steps of cognitive processes that *antecede* aggressive behavior: (1) selective attention to and encoding of signals that are associated with hostility; (2) hostile attribution to the intention of the other person; (3) selection of hostile goals; (4) generation of aggressive action possibilities as a response to provocation; (5) anticipation of a beneficial outcome due to the aggressive response; and (6) behaving aggressively. Such a model of social information processing rather fits to reactive or affective aggression, as the impulsive behavior is driven by intense anger and perceived threat. Although this model has been formulated for children, it also gives useful indications for the social information processing in adults, as it points out the relevance of increased attention to and encoding of threat related signals (processing bias for hostile stimuli) before behaving aggressively.

Krämer, Büttner, Roth & Münte (2008) used psychophysiological methods, such as electroencephalography (EEG), to assess social information processes *during* an aggressive encounter in a laboratory setting. They found altered cortical activity – specifically, enhanced frontal negativities – in adult participants with high trait aggressiveness when they were highly provoked. This enhanced negativity was positively correlated with the inhibition of their aggressive behavior despite being provoked. This was interpreted as a greater requirement for inhibitory processes in highly aggressive subjects to control their behavior in case of provocation. Furthermore, Bertsch, Böhnke, Kruk & Naumann (2009) found evidence for changes in early as well as in later stages of information processing during an emotional Stroop task *after* experimentally provoked aggressive behavior in healthy subjects. Specifically, high provocation was related to increased positive event-related potential (ERP) amplitudes in response to emotional facial expressions compared to low provocation. Concerning the early positivity, the provocation effect was particularly strong for threat-related facial expressions (Bertsch et al., 2009).

To summarize, there is increasing evidence for altered information processes that precede, accompany, and follow aggressive behavior. Neural circuits that can be influenced by those changes in information processing and therefore trigger aggression will be the subject of the next section.

1.3.4 The Prefrontal Cortex as a Core Neural Substrate in the Context of Aggression

The prefrontal cortex (PFC) is a brain region that is, among other things, crucially involved in cognitive control processes (Miller & Cohen, 2001). Moreover, the top-down control systems of the prefrontal cortex play an important role in the context of aggression and violence. According to Davidson, Putman & Larson (2000), impulsive aggression results from a failure of emotion regulation. They postulate a neural circuit including the prefrontal cortex (particularly its orbitofrontal and ventromedial subdivisions), the anterior cingulate cortex (ACC) and subcortical-limbic structures, such as the amygdala and the hypothalamus. More precisely, they suggest that the subcortical-limbic brain structures promote impulsive/aggressive behavior and receive inhibitory input from the frontal cortex, and that a dysfunction of this central circuitry promotes impulsive, affective aggression and violence (Davidson et al., 2000).

In line with this theory, many studies have referred to a relationship between aggressive behavior and brain damage to the frontal cortex (e.g., Anderson, Bechara, Damasio, Tranel & Damasio, 1999). This is further underlined by Coccaro, McCloskey, Fitzgerald & Phan (2007) who provided evidence for a link between increased amygdala reactivity and diminished activation of the orbitofrontal cortex (OFC) in response to angry facial expressions in individuals diagnosed with a disorder characterized by affective aggression, intermittent explosive disorder. Supporting evidence also came from several studies using positron emission tomography (PET) reporting dysfunctions of the prefrontal brain in violent individuals compared to non-violent ones (for a review, see Patrick & Verona, 2007).

In his review, Siever (2008) further specifies possible factors that may have an impact on the neural circuit of aggression. According to him, a provocative stimulus is processed by sensory processing centers, which can be influenced by drugs, alcohol, or metabolic disturbances, resulting in incomplete or distorted sensory impressions. This can promote a threatening or provocative perception of the stimulus. Subsequently, early information processing, which can be affected by cultural and social factors as well as by cognitive impairments, leads to an appraisal of the stimulus. Finally, processing in the amygdala and related limbic regions prompt the "drive" (Siever, 2008, p. 431) to aggressive behavior, whereas top-down control processes in the orbital frontal cortex and anterior cingular gyrus inhibit impulsive behavior. If there is an imbalance between the limbic triggers of aggression and the prefrontal control processes, the likeliness of an impulsive/aggressive act increases (Siever, 2008).

According to Siever (2008), several neuromodulators may contribute to abnormalities in this neural circuitry. Specifically, reduced serotonergic, gabaminergic, and oxytocin activity, as well as increased vasopressin activity enhances aggression. Also briefly mentioned in this review is the association between low cortisol concentrations and high aggressiveness in different populations (Coccaro & Siever, 1995, as cited in Siever, 2008). Beside cortisol, another neuromodulator of the neural circuit is the neurosteroid testosterone (Siever, 2008). The role of testosterone (also in combination with cortisol) in the release of aggression is reported in detail in section 3.1 (introduction to the second study).

1.4 Stress and Aggression

The next section provides an overview of past research reporting evidence that the HPA axis activity - and especially its end product cortisol - is one of the most important promoters of aggressive behavior.

1.4.1 Research in Animals

Strong evidence supporting a mutual relationship between aggressive behavior and the activation of the adrenocortical stress response in animals has been provided by Kruk, Halász, Meelis & Haller (2004) who have conducted a series of experiments in rats. On the one hand, the researchers stimulated the hypothalamic attack area, a brain region that is involved in the neural circuitry of territorial aggression in rodents (Halász, Liposits, Kruk & Haller, 2002), and found strong activation of the HPA axis due to this stimulation. On the other hand, they removed the adrenal glands of the rats and implanted low release pellets of corticosterone, in order to keep the endogenous corticosterone production low. When an acute surge of corticosterone through injections was then induced in the rats, aggressive behavior (measured in terms of the attack threshold when stimulating the hypothalamic attack area) was facilitated. This effect, however, was only apparent if the corticosterone surge was administered 10 minutes before assessing the attack threshold, whereas it was absent if corticosterone injections were administered 60 or 240 minutes beforehand. This finding implies rapid, non-genomic (without gene transcription) effects of corticosterone on aggressive behavior. The researchers proposed a fast positive feedback loop containing a "[...] mutual stimulatory interaction between brain mechanisms involved in attack and the stress response" (Kruk et al., 2004, p. 1066). As displayed in Figure 4, Kruk et al. suggest a "vicious

circle" (p. 1068) of stress and aggression. Specifically, acute activation of the HPA axis leads to high circulating levels of glucocorticoids that facilitate the activation of the hypothalamic attack area. This in turn further activates the HPA axis (Kruk et al., 2004).

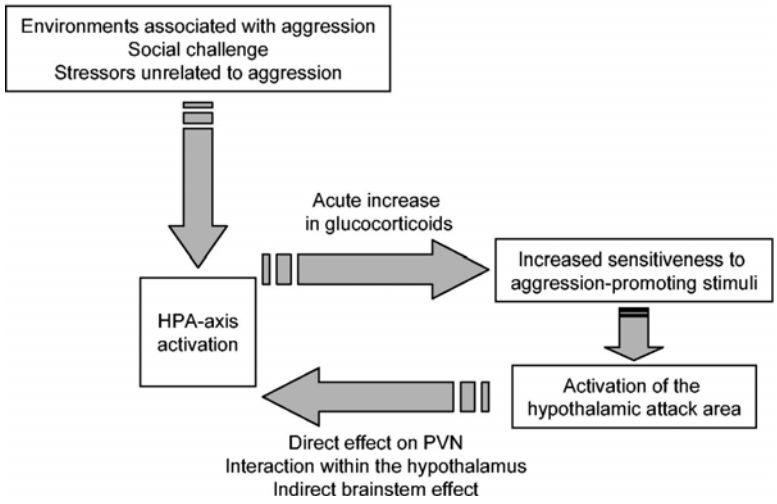

Figure 4 Illustration of the fast positive feedback loop between HPA axis activation and activation of the hypothalamic attack area (Kruk et al., 2004, p. 1067). PVN: paraventricular nucleus of the hypothalamus.

This model allows possible explanations for the escalation of aggression as, according to this fast positive feedback loop, aggressive behavior is hard to stop once it is elicited. As a possible mediator for the effect of acute HPA axis activity on aggressive behavior, the authors propose an enhanced sensitivity to threat related stimuli (Kruk et al., 2004). Since information processes in rats is difficult to assess, Bertsch, Böhnke, Kruk, Richter & Naumann (2011) have examined the influence of an acute dose of cortisol and provocation on social information processing after an aggressive encounter in humans. In the next section, their methods and findings will be reported in detail.

1.4.2 Research in Healthy Humans

There is also increasing evidence for a strong relationship between stress (or HPA axis activity, respectively) and aggressive behavior in healthy humans. In a series of experiments, the research group around Verona (Verona, Joiner, Johnson & Bender, 2006; Verona & Kilmer, 2007; Verona, Reed, Curtin & Pole, 2007) found effects of an acute physical stressor (air blast) on subsequent aggressive behavior of healthy men and women, measured as shock delivery to an alleged employee (confederate). They found different gender effects, with men responding more aggressively after high stress exposure compared to low stress, and women

reacting less aggressively after high compared to low stress exposure (Verona & Kilmer, 2007). However, they also found gender differences with a distinct pattern (Verona et al., 2007). Since Verona and colleagues did not measure cortisol to validate their stress induction, their "stress effects" on aggression cannot be definitely attributed to the HPA axis activity.

Böhnke, Bertsch, Kruk & Naumann (2010a) have investigated the relationship between acute as well as basal HPA axis activity and aggression in healthy young men and women. In order to assess basal HPA axis activity, they measured the cortisol awakening response prior to the experimental session. Several samples of acute cortisol were collected during the experiment. They used a competitive reaction time task against an alleged opponent in order to highly or mildly provoke the subjects and to measure their aggressive behavior. They found a negative correlation between basal HPA axis activity and aggressive behavior in highly provoked subjects. Moreover, the high provocation group showed higher acute cortisol levels after the aggressive encounter compared to the low provocation group, if differences in baseline levels were taken into account (Böhnke et al., 2010a).

In a continuative study, Böhnke, Bertsch, Kruk, Richter & Naumann (2010b) pharmacologically increased the cortisol levels of healthy men and women by 20mg of hydrocortisone (cortisol condition) or administered a placebo (placebo condition). Subsequently, they again provoked the subjects either highly or mildly and measured their aggressive behavior. They found different effects of cortisol on aggression in males and females, opposite to the gender differences reported by Verona & Kilmer (2007) mentioned above. More precisely, in the study of Böhnke et al. (2010b), only women behaved more aggressively after cortisol administration compared to the placebo condition, whereas in men there was no difference between the two groups (cortisol vs. placebo). Thus, the gender difference in the placebo group - more aggressive behavior in men than in women - disappeared after cortisol administration due to the aggression enhancing effect of cortisol in women. Additionally, they could replicate the negative correlation between basal HPA axis activity and aggressive behavior, but it was only apparent in women and particularly strong in the placebo group (Böhnke et al., 2010b).

Within the same research group, Bertsch et al. (2011) examined changes in social information processing during an emotional Stroop task after cortisol administration and provocation. They measured reaction times as well as event-related potentials during the processing of several emotional facial expressions (happy, angry, fearful, and neutral). Results showed that exogenous cortisol enhancement together with high provocation led to faster responses towards all facial expressions. Concerning the ERPs, cortisol and provocation

influenced early as well as later stages of information processing, but these effects were independent from each other. Specifically, high provocation led to increases of the P1 amplitude and the posterior late positive potential (LPP), whereas it had no impact on the P2 amplitude. On the contrary, exogenous cortisol reduced the P2 amplitude in response to all facial expressions, but especially for angry faces, while it had no effect on the P1 and the LPP (Bertsch et al., 2011). These findings support the model of Kruk and colleagues (2004) by strengthening the assumption of significantly changed social information processes as a mediator between HPA axis activity and aggressive behavior.

1.5 Open Research Questions and Outline of the Thesis

Stress involves more than the mere increase of cortisol, since it also activates the sympathetic nervous system, as described earlier. Therefore, it is important to elucidate the influence of the whole stress system as a natural and dynamical physiological response to a stressor on aggressive behavior. The activation of the sympathetic nervous system may be a critical component in moderating the effects of cortisol on aggression reported by the previous studies of Bertsch et al. (2011) and Böhnke et al. (2010b). Therefore, in the present thesis, a psychophysiological stressor is used in two different studies in order to acutely induce stress and thereby to enhance the cortisol level endogenously together with all other components of a "real" stress response.

Furthermore, despite first indications of changed social information processing in the context of stress and aggression (Bertsch et al., 2009; Bertsch et al., 2011), little is known yet about the exact aspects or components of information processing that are influenced by stress and/or provocation. Therefore, the present thesis seeks to examine different constructs of information processing, such as (1) cognitive/inhibitory control processes and (2) approach-avoidance tendencies that are assumed to play an important role in the release of aggression.

Specifically, according to the model of Davidson and colleagues (2000), cognitive or inhibitory control processes may be diminished during aggressive behavior, as they are controlled by the PFC, the activity of which is then in imbalance with the impulsive drive from limbic structures. Since cortisol is one of the most important promoters of aggression and may cause a loss of cognitive control, the effects of an acute (endogenous) stress induction and basal HPA axis activity on cognitive control processes is the subject of the first study presented in this thesis. This is intended as a first step towards a further understanding of the foundations of the stress-aggression-link.

On the other hand, when examining social information processes in the context of aggression, it might be even more appropriate to choose a paradigm with an "active" component such as the so-called approach-avoidance paradigm. With this paradigm, the motivation to approach or avoid certain (affective) stimuli can be measured. Assuming that aggressive behavior implicates a pronounced approach motivation towards the victim, the role of the tendency to approach affective and socially relevant stimuli appears to be important in the investigation of potential promoting factors of aggressive behavior. Thus, in the second study, the impact of acute stress together with subsequent provocation on approach-avoidance tendencies is investigated. Furthermore, salivary testosterone as another important hormone involved in the release of aggression was assessed during the experimental session.

In both studies presented in this thesis, endocrinological (cortisol, and in the second study also testosterone), behavioral (reaction times), as well as electrophysiological (ERPs) data is collected. The advantages of ERPs are, firstly, the high temporal resolution, secondly, the non-invasiveness, and thirdly, the online registration of information processes (Hillyard & Kutas, 1983).

To conclude, this thesis seeks to further elucidate the moderating role of changed social information processing in the stress-aggression-link. Therefore, two studies were conducted: The first study deals with the influence of stress on cognitive control processes, whereas the second study is concerned with the effects of acute stress and subsequent social provocation on approach-avoidance tendencies, including the assessment of testosterone as a further moderating variable.

Chapter II

The Influence of Acute Stress on Cognitive Control Processes

2.1. Introduction

Cognitive control plays an important role in the release of impulsive aggression: An imbalance between subcortical-limbic brain structures, which promote impulsive behavior, and the top-down (inhibitory) control processes of the prefrontal cortex (PFC) and the anterior cingulate cortex (ACC) increases the likeliness of aggressive behavior (Davidson et al., 2000; Siever, 2008). In line with this, a relationship between dysfunctions of the PFC and aggression was consistently found in several studies (e.g., Anderson et al., 1999; Coccaro et al., 2007; for a review, see Patrick & Verona, 2007). Cognitive control is essential in everyday life, as one of its most crucial aspects is "the ability to select a weaker, task-relevant response (or source of information) in the face of competition from an otherwise stronger, but task-irrelevant one" (Miller & Cohen, 2001, p. 170). This also implies the ability to withstand a strong (impulsive) reaction tendency in favour of a more appropriate or socially accepted behavior. Thus, cognitive control could function as a possible mediator in the link between stress and aggression postulated by Kruk et al. (2004). According to their model, the activation of the HPA axis, especially the release of its end-product cortisol, leads to a lower threshold for aggressive behavior, which in turn further stimulates the HPA axis (Kruk et al., 2004). A recent study by Sprague, Verona, Kalkhoff & Kilmer (2011) found evidence for a moderating influence of executive function (including inhibitory control) on the relationship between stress and aggression. Specifically, the level of perceived stress was strongly related to aggressive behavior in subjects with low executive function. Hence, the authors consider executive function as an important ability to control one's behavior and to regulate emotions in an appropriate way after the exposure to stress (Sprague et al., 2011).

To summarize, stress might attenuate cognitive control and thereby enhance the probability of impulsive acts, presumably by inducing an imbalance between the drive of limbic structures and PFC control as postulated by Davidson et al. (2000) and Siever (2008).

A well established tool to examine cognitive control processes in an experimental context is the so-called task-switching paradigm, during which subjects have to switch frequently between two tasks with respect to the same set of stimuli. Each task requires the categorization of the current stimulus regarding a distinct feature. That is, for instance, to classify a digit either as even vs. odd (task 1) or as smaller vs. larger than a reference digit (task 2). In case of a task-cueing paradigm, the order of the two tasks is unpredictable for the subject, but a precue (e.g., circle vs. triangle) occurs prior to the target stimulus, in order to inform the subject which task is to be performed on the following target stimulus. Usually, the reaction times and error rates are higher in trials when the current task is different from the

previous trial (switch trial) compared to when the current task is the same as in the previous trial (repeat trial). This loss in performance is called "switch costs" and is assumed to reflect cognitive control processes. Another robust finding is the so-called preparation effect, a reduction of these switch costs when the interval between the precue and the target stimulus (Cue Target Interval, CTI) is increased (for reviews, see Kiesel et al., 2010; Monsell, 2003).

So far, only a few studies addressed the relationship between stress and cognitive control, with rather inconsistent results. Two studies concerning the influence of chronic stress on cognitive control processes led to contradictory findings: On the one hand, Kofman, Meiran, Greenberg, Balas & Cohen (2006) have investigated the performance of students in a task switching paradigm either before (no stress condition) or during an examination period (stress condition). They reported reduced switch costs during chronic stress compared to the non-stressful phase (Kofman et al., 2006). On the other hand, however, Liston, McEwen & Casey (2009) found larger switch costs in chronically stressed subjects (during an examination period) than in a non-stressed control group. Steinhauser, Maier & Hübner (2007), in contrast, induced either high or low acute stress in their subjects by means of an intelligence task with either high or low degrees of difficulty, time pressure and threat to self-esteem. Subsequently, subjects performed a task-switching paradigm with different lengths of the CTI (either long or short). The authors reported the usual preparation effect (reduction of switch costs after long CTIs) in the low-stress group, whereas this facilitation effect was not observed in the high-stress group. They interpreted this finding as a change in processing strategies under high levels of acute stress (Steinhauser et al., 2007).

The findings of these previous studies, however, have to be interpreted with care, since no physiological validation of the stress induction or the chronic stress level, respectively, was realized. This may have contributed to the inconsistency of the results.

Additionally, not only acute stress, indexed by acute cortisol rise, seems to have significant influences on cognitive control processes, but also basal HPA axis activity as a trait measure may play a role in changing cognitive or inhibitory control. The research group around Böhnke (2010a), for instance, found evidence for a negative relationship between basal morning cortisol and aggressive behavior following social provocation, which potentially implies reduced cognitive/inhibitory control during an aggressive encounter if basal cortisol levels are low. Hence, when investigating the influence of HPA axis activity on cognitive control, it seems promising to take basal cortisol levels into account, in addition to acute cortisol changes - particularly, since this has not yet been a subject of research.

Moreover, previous studies concerning the relationship between stress and cognitive control addressed changes in information processing only on a behavioral level - although there is extensive literature on event-related potentials (ERPs) in task-switching paradigms. Especially the Contingent Negative Variation (CNV), a negative-going slow wave which typically occurs between the precue and the target stimulus and which has been related to motor preparation for the upcoming task (Walter, Cooper, Aldridge, McCallum & Winter, 1964), has been of interest in task-switching experiments (Brass, Ullsperger, Knoesche, von Cramon & Phillips, 2005; Gajewski et al., 2010; Gladwin, Lindsen & de Jong, 2006; Hsieh & Cheng, 2006). Furthermore, slow negative brain potentials are considered particularly suitable for the investigation of information processing since they can be directly related to resource allocation, in the sense of larger slow wave amplitudes reflecting the allocation of more resources when performing a task (Rösler, Heil & Röder, 1997).

The aim of the present study was to investigate the influence of acute stress as well as of basal cortisol levels on cognitive control processes. Acute stress induction was realized by means of the Socially Evaluated Cold Pressor Test (SECP, Schwabe, Haddad & Schächinger, 2008) and validated by several salivary cortisol measurements during the experiment. Cognitive control processes were measured using a cued task-switching paradigm, which was performed by the participants before as well as after stress induction. In addition to measurements of acute cortisol levels, basal HPA axis activity was assessed prior to the experimental session. In order to get insights into changes in information processing due to basal and/or acute cortisol levels, ERPs during the task-switching paradigm were analyzed in addition to behavioral data.

Acute stress, especially the rise of cortisol, was expected to reduce cognitive control, which should be reflected in larger switch costs after stress induction compared to a non-stressed control group. Furthermore, the basal cortisol level should also influence the amount of switch costs via a negative association, although the investigation of this relationship was rather explorative. Both the influences of acute and of basal cortisol levels should be reflected in changes within information processing as measured with ERPs - specifically, in the magnitude of the terminal CNV. The study was designed as a rather explorative first step towards elucidating the role of cognitive control processes in the stress-aggression link.

2.2 Methods

2.2.1 Participants

41 male students (mean age = 23.51 years, SD = 2.74 years) of the University of Trier, Germany, took part in the study. However, nine subjects had to be removed from analysis due to reasons that are discussed in section *2.2.8*. Thus, data of 32 participants (mean age = 23.44 years, SD = 2.71 years) will be reported here. Inclusion criteria were as follows: no acute or chronic medical disease, no psychiatric disorder, no use of medication, normal weight (mean $BMI_{N=32}$ = 23.28, $SD_{N=32}$ = 2.47), non-smoking, right-handedness, and German as mother tongue. The study was conducted in accordance with the Declaration of Helsinki and has been approved by the ethics committee of the University of Trier. All participants provided a written informed consent and received 35 Euros for their participation.

2.2.2 General Procedure

The experimental sessions started at 12:00 a.m., 14:30 p.m., or 17:00 p.m., respectively, in order to examine the subjects at a time when, due to the circadian rhythm of HPA axis activity, the endogenous cortisol level is relatively low (Kirschbaum & Hellhammer, 1999; Schreiber et al., 2006). All subjects were randomly assigned to the time of experiment and the experimental conditions (stress induction vs. non-stressful control procedure). Due to later categorization of stressed subjects into cortisol responders and nonresponders, two thirds were subjected to the stress condition and one third to the non-stress condition. All participants were examined individually, and the experimental procedure was the same for all of them. Subjects were told that the study was dealing with the relationship between stress and cognitive functioning. The experiment took place in a sound-attenuated EEG-laboratory where the light was dimmed. Subjects were seated (at a 1 m distance) in front of a computer screen, and the EEG was applied. After this, the experimenters left the room and the subject received all instructions via the computer. The experimental procedure was as follows: Participants had to perform a Task Switching Paradigm and a Go-Nogo Task (explained and analyzed elsewhere) in a randomized order, for a first time (block 1). Afterwards, the stress induction (or non-stressful control procedure) was applied by means of the Socially Evaluated Cold Pressor Test (SECP, Schwabe et al., 2008). Subsequently, subjects performed the Task Switching Paradigm (and the Go-Nogo Task) for a second time (block 2), again in a balanced order. Four orders of the two tasks were realized: ABAB, ABBA, BABA, and BAAB, with "A" representing the Task Switching Paradigm and "B" the Go-Nogo Task. At the end of the

experimental session, all subjects were extensively debriefed, thanked, and paid for their participation. The entire experiment took about 90 minutes.

During the experimental session, cortisol samples were collected at the following time points[1]: At the beginning of the experimental session, following the application of the EEG device (C0); after the first block of the Task Switching Paradigm (C1) and of the Go-Nogo Task (C2, directly before SECP); after the SECP (C3); after the second block of the Task Switching Paradigm (C4) and the Go-Nogo Task (C5); and finally, at the end of the experiment, just before the participants were debriefed and left the laboratory (C6). For the cortisol samples, salivettes (Sarstedt, Nümbrecht, Germany) were used. Subjects were instructed to chew gently on these salivettes for a duration of one minute. Immediately after the experimental session, the cortisol samples were frozen for biochemical analysis. For analysis, a time-resolved immunoassay with fluorescence detection was used as described in detail elsewhere (Dressendörfer, Kirschbaum, Rohde, Stahl & Strasburger, 1992). The intra- and inter-assay variability was below 10% and 12%, respectively.

2.2.3 Materials

For the presentation of experimental stimuli and recording of participants reactions, E-Prime© experiment presentation software (Version 2.0, Psychology Software Tools Inc., Pittsburgh, PA) was used (Schneider, Eschman & Zuccolotto, 2002).

2.2.4 Task Switching Paradigm

In order to measure cognitive control processes, a Task Switching Paradigm was used. At the beginning of each trial, a fixation cross appeared at the center of the computer screen. Subsequently, one of two possible precues (either a circle or a triangle) was presented and lasted for either 200 ms (short Cue Target Interval, CTI) or 1200 ms (long CTI) until the target stimulus appeared (see Astle, Jackson & Swainson, 2008). As target stimuli, lowercase (a, e, i, u, g, b, t, d) or uppercase (A, E, I, U, G, B, T, D) vowels or consonants were used. The presentation of the target stimuli was randomized on condition that the same letter must not occur on two consecutive trials. The task of the participant was to categorize the target letter as correctly and as quickly as possible with respect to two different attributes: (1) lowercase vs. uppercase letter, or (2) vowel vs. consonant, respectively. Which of these two attributes of the target the subjects had to pay attention to was signalized by the precue: The circle

[1] Exemplary for one of the four realized orders (ABAB) of Task Switching Paradigm and Go-Nogo Task

represented the discrimination of uppercase versus lowercase letters, whereas the triangle signalized the distinction between vowel and consonant. The participants responded via pressing the left or right arrow key, depending on the task. The mapping of left and right arrow key to either vowel and uppercase letter or to consonant and lowercase letter was counterbalanced across participants, but it was kept constant within each participant over the two blocks. The inter trial interval was kept variable in order to ensure that each trial had a total length of 3500 ms.

Similar to a study of Kieffaber & Hetrick (2005), the switch of the two tasks (1. uppercase vs. lowercase or 2. vowel vs. consonant categorization) was varied as follows: In 26 cases of each task (52 times in total), subjects had to switch to the other task after one trial (X - Y). In 13 cases of each task (26 times in total), subjects had to switch to the other task after two trials of the same task, i.e., after one repeat trial (X - X - Y). Also, in 13 cases of each task (26 times in total), subjects had to switch to the other task after four trials of the same task, that is after three repeat trials (X - X - X - X - Y). The order of these different switch rules between tasks was completely randomized. Astle and colleagues (2008) found significant switch costs (= longer reaction times after a task switch than after a task repetition) after short CTIs, but not after long CTIs. Therefore, short CTIs were realized in order to elucidate stress effects on behavioral performance. However, for the analysis of ERP components, such as the CNV, long CTIs were more appropriate. Thus, similar to Astle et al. (2008), the CTIs were short (200 ms) in 20 % of the trials and long (1200 ms) in 80 % of the trials. The probabilities of switch and repeat trials (50% each) were equal for short and long CTIs. Figure 5 displays an example of two possible consecutive trials of the Task Switching Paradigm. At the beginning of the first block, 32 practice trials were realized. Then, 416 experimental trials followed, 208 in block 1 (before SECP) and 208 in block 2 (after SECP).

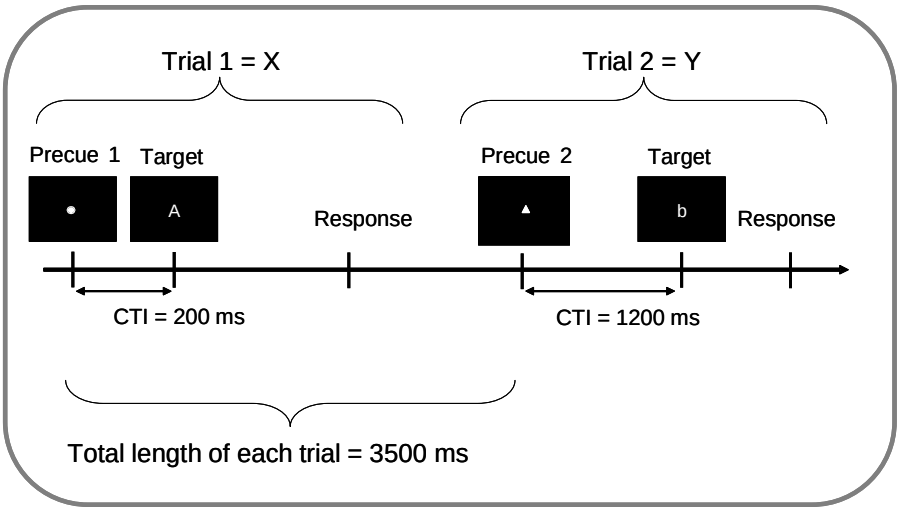

Figure 5 Example of two subsequent trials of the Task Switching Paradigm.

2.2.5 The Socially Evaluated Cold Pressor Test

In order to induce acute stress, the SECP (Schwabe et al., 2008) was used. Under the stress condition, subjects had to immerse their left hand for 3 minutes into ice-cold water of 1-3 °C. At the same time, they were videotaped and observed by an investigator of the opposite sex (female). In order to increase the psychosocial stressor component, they were led to believe that their mimics and gestures during the task would be analyzed by the investigator. The non-stressful control procedure (warm water group) was exactly the same, except that the water was at body temperature (36-38 °C). Since the stressed participants would be classified into cortisol responders and nonresponders depending on their cortisol rise due to the SECP, for later analyses, two-thirds of the sample were randomly assigned to the stress condition and one third to the warm water condition.

2.2.6 Basal HPA Axis Activity

According to Hellhammer and colleagues (2007), measurements on at least two consecutive days are necessary to obtain a reliable trait measure of the HPA axis activity. Similar to studies of Böhnke and colleagues (2010a, 2010b), all participants collected samples of salivary cortisol on three consecutive weekdays prior to the experimental session. Since time of awakening strongly influences the cortisol awakening response (Kudielka & Kirschbaum, 2003), all participants had to awake between 6:00 and 8:00 a.m., whereas the exact awakening time, which had to be the same on all three days, was arranged individually with each participant. Moreover, the participants were asked to abstain from eating, drinking anything but water, brushing their teeth, and exercising while collecting the saliva samples. Subjects had to collect four samples within one hour after awakening (+0, +30, +45, and +60 min., see Hellhammer et al., 2007) and to store them in the refrigerator until returning them to the experimenters at the beginning of the experimental session. To assess a trait measure of the basal HPA axis activity, the area under the curve with respect to ground (AUC_G) was calculated (see, Hellhammer et al., 2007) using the formula of Pruessner, Kirschbaum, Meinlschmid & Hellhammer (2003). To get one value of the basal cortisol level after awakening for each participant, the AUC_G was computed for each day and then averaged over the three consecutive sampling days for each participant individually. Regarding the 32 participants included in statistical analyses, the AUC_G correlated significantly between the three sampling days ($.504 < r\text{'s} < .604$) indicating a high intra-individual stability of this measure.

2.2.7 EEG Recording and Quantification

EEG was recorded from 27 electrode positions plus the mastoids in accordance with the 10-10 electrode reference system (Chatrian, Lettich & Nelson, 1988) with the Easy-Cap electrode system (Falk Minow Services). All sites were referenced to FCz and a bipolar horizontal as well as vertical electrooculogram (EOG) was recorded. Ag/AgCl electrodes were used, and the impedances of the EEG electrodes were below 5 kΩ. EEG and EOG were amplified by means of a 32-channel BrainAmp amplifier (input impedance: 10 MΩ; Brain Products, GmbH) in AC mode. The pass-band was put to 0.016 to 499 Hz (-12 dB/octave rolloff). Signals were digitalized at 1000 Hz.

For EEG editing and quantification, Analyzer 2.0 (Brain Products GmbH) was used. For the analyses, the EEG was re-referenced to linked mastoids. Using the algorithm of Gratton, Coles & Donchin (1983) EEG was corrected semi-automatically for eye movements. Segments with non-physiological artifacts were excluded from analysis by means of the semi-automatic artifact rejection. The sampling rate was set to 200 Hz. EEG was segmented off-line into periods of 2700 ms, starting 200 ms prior to precue onset and ending 2500 ms after precue onset, or 1300 ms after target onset, respectively. The signal was baseline-corrected using the 200 ms prior to precue onset as reference. A digital low pass filter of 12 Hz (-48 dB) was applied. Separate average amplitudes were calculated for each electrode, individual, and each subtrial (switch vs. repeat trial), but only for the experimental trials with correct responses and only for long CTIs (1200ms). The terminal CNV was quantified by calculating the mean activity of the time interval of the last 200 ms prior to target onset (1000-1200 ms after precue onset). The following midline electrodes were included in statistical analyses: Fz, Cz, and Pz.

2.2.8 Statistical Analyses

To disentangle general stress effects from specific cortisol effects, the subjects of the stress condition (cold water condition of the SECP) were categorized into cortisol responders and nonresponders depending on their cortisol rise due to the stressor. Since cortisol peaks in saliva can be expected at about 20 minutes after a stressor (Kirschbaum & Hellhammer, 1989), the difference between cortisol samples C3 (immediately after the SECP) and C4 (after the second block of Task Switching Paradigm or Go-Nogo Task, respectively, approximately 20 minutes after SECP) was used as an index of the cortisol increase following the stressor. By means of a median-split, subjects with an increase of at least 0,98 nmol/l of cortisol were

classified as cortisol responders, and as nonresponders otherwise. This resulted in a mean cortisol increase of 4,36 nmol/l for the cortisol responders and a mean cortisol decline of -0,16 nmol/l for nonresponders following the stress induction[2].

Nine subjects had to be excluded from statistical analyses, resulting in a total number of 32 analyzed participants. Four subjects (one of the non-stressed warm water group, two of the nonresponders, and one of the cortisol responders) had missing values of basal morning cortisol on all three days of ambulant cortisol assessment and therefore could not be included in analyses. Two subjects (one of the warm water group, one of the cortisol responders) had to be removed from analyses due to technical problems during EEG recording. Moreover, one subject (warm water group) had to be discarded because of too many artifacts in the EEG. Another subject (cortisol responder) was removed from analyses because of a cortisol rise following the SECP that was more than 3 SD above the mean cortisol rise of the stressed group. Finally, in order to keep the number of subjects in the three stress groups as equal as possible, one subject (cortisol responder) was excluded who had a cortisol rise after the SECP that exactly equaled the median of the stressed group and thus could not be definitively classified as a nonresponder or cortisol responder. Of the remaining 32 subjects, 10 were in the warm water group, 11 were nonresponders, and 11 were cortisol responders.

Behavioral data. The reaction times for each subject were adjusted by removing individual outlier values, and only trials with correct responses were analyzed. In order to analyze the influence of acute cortisol rise on cognitive control processes during the task switching paradigm, an analysis of variance (ANOVA) was conducted, including block (1 vs. 2; repeated measures), CTI (short vs. long; repeated measures), subtrial (switch vs. repeat trial; repeated measures), and stress group (warm water group, nonresponder, cortisol responder; between-subject) as independent variables and the median of the reaction times as the dependent variable.

In order to investigate the influence of the basal HPA axis activity as well, the z-standardized AUC_G was added as a continuous predictor (between-subject) into the analysis (see Aiken & West, 1991).

Since error rates were too low (mean error rates in switch trials: < 3 % in block 1 and < 2 % in block 2; in repeat trials: < 2 % in block 1 and < 1% in block 2), only reaction times in correct trials were analyzed.

[2] These cortisol values refer to the remaining 32 subjects after the exclusion of nine participants due to reasons discussed below.

Electrophysiological data. A three-way ANOVA was performed for the terminal CNV (1000-1200ms after precue onset, the last 200ms prior to target onset, respectively) including the factors electrode position (Fz, Cz, Pz, repeated measures), subtrial (switch vs. repeat trial; repeated measures), and stress group (warm water condition, nonresponder, cortisol responder; between-subject). These ANOVAs were calculated for long CTIs only and separately for block 1 and block 2. The mean average CNV magnitude was used as the dependent variable.

In order to check for main effects and interactions of basal HPA axis activity, the same ANOVA was calculated with AUC_G as a continuous predictor. For that purpose, the AUC_G was z-standardized in advance (Aiken & West, 1991).

In cases where the assumption of sphericity was violated, the degrees of freedom were Huynh-Feldt corrected (Huynh & Feldt, 1976). The statistical significance level alpha was set to 0.05 (two-tailed), and significant results are reported, with η^2 (partial eta squared) as the effect size measure. As post-hoc tests, Dunn's Multiple Comparison Procedure or Pearson correlations, respectively, were used. All statistical analyses were conducted using SPSS for Windows (Version 17.0, SPSS Inc.).

2.3 Results

2.3.1. Manipulation Check of Stress Induction

In order to check if the stress induction was successful, an ANOVA with the factors time of cortisol sample (C0, C1, C2, C3, C4, C5, C6; repeated measures) and stress group (warm water group, nonresponder, cortisol responder; between-subject) was conducted.[3] The analysis revealed a significant interaction of time and stress group ($F(12,162) = 4.171$, $p = .003$, $\eta^2 = .236$), which is displayed in Figure 6. Post-hoc tests revealed that cortisol responders differed significantly from nonresponders and the warm water group at time points C4 and C5, indicating that the cortisol levels of the responders were well above those of the nonresponders and of the warm water group after the SECP. To conclude, the stress induction via the SECP was successful.

[3] Since two subjects of the warm water group had one missing cortisol value each during the experimental session, this analysis includes the cortisol values of 30 subjects.

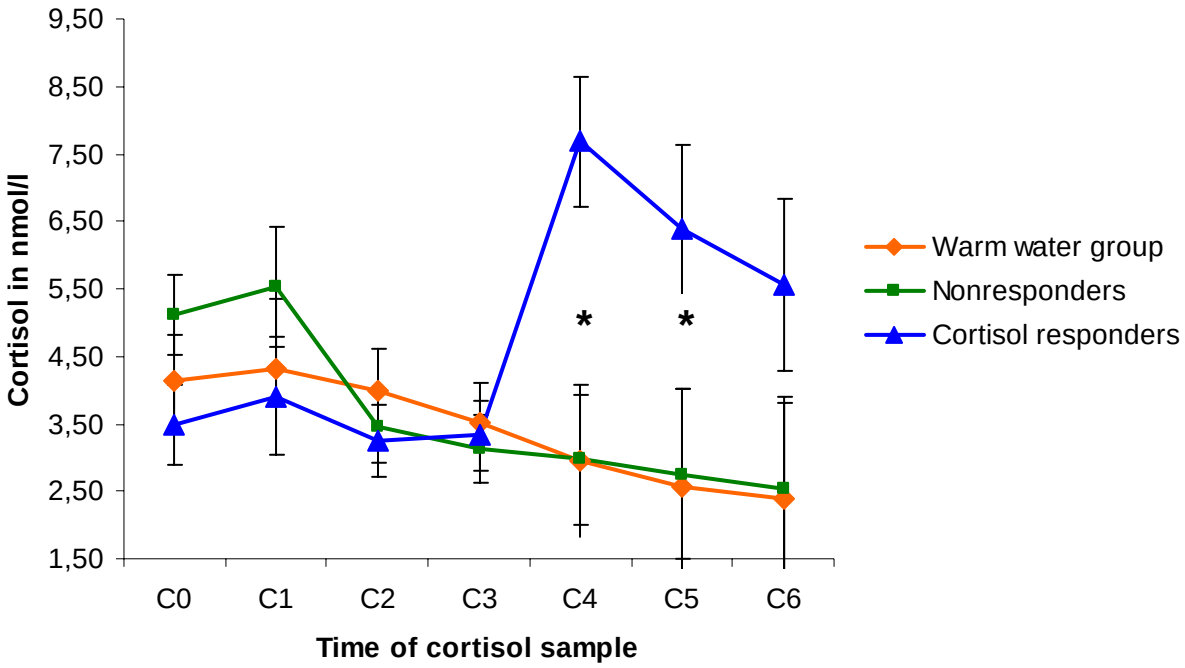

Figure 6 Cortisol course during the experimental session for the warm water group, cortisol responders, and nonresponders. * = significant difference according to post-hoc test.

2.3.2 Influence of Acute Stress on Cognitive Control

The ANOVA revealed significant main effects of block ($F(1,29) = 19.264$, $p = .000$, $\eta^2 = .399$), CTI ($F(1,29) = 141.035$, $p = .000$, $\eta^2 = .829$), and subtrial ($F(1,29) = 47.522$, $p = .000$, $\eta^2 = .621$), as well interactions of block and stress group ($F(2,29) = 4.256$, $p = .024$, $\eta^2 = .227$) and of CTI and subtrial ($F(1,29) = 34.723$, $p = .000$, $\eta^2 = .545$). These effects were further qualified by a four-way interaction of block, CTI, subtrial, and stress group ($F(2,29) = 3.720$, $p = .036$, $\eta^2 = .204$). As displayed in Figures 7a) and 7b), post-hoc tests indicated significant switch costs (= longer reaction times in switch trials than in repeat trials) for all groups when CTIs were short, whereas switch costs were diminished and not significant after long CTIs. Moreover, both nonresponders and cortisol responders had faster reaction times in block 2 compared to block 1, especially when CTIs were short. Specifically, nonresponders became significantly faster in switch trials after stress induction, whereas cortisol responders became significantly faster in repeat trials following stress induction. On the contrary, the non-stressed warm water group did not change with respect to the reaction times, neither in switch trials nor in repeat trials. Regarding the amount of switch costs after short CTIs, nonresponders had significantly reduced switch costs in block 2 (77 ms) compared to block 1

(188 ms), whereas the switch costs of neither the warm water group (block 1: 118 ms; block 2: 120 ms) nor of cortisol responders (block 1: 98 ms; block 2: 108 ms) changed significantly.

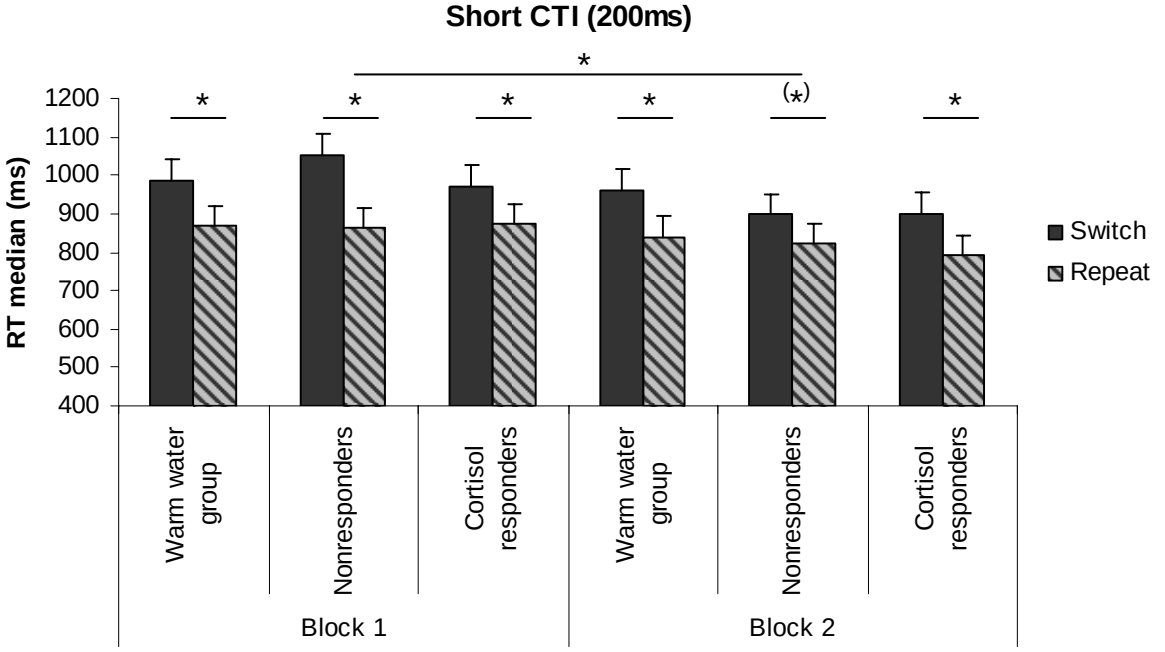

Figure 7a) Reaction times of the three stress groups in switch and repeat trials after *short* CTIs, separately for block 1 and block 2. Values are means and SEM. * = significant difference, (*) = marginally significant difference according to post-hoc test.

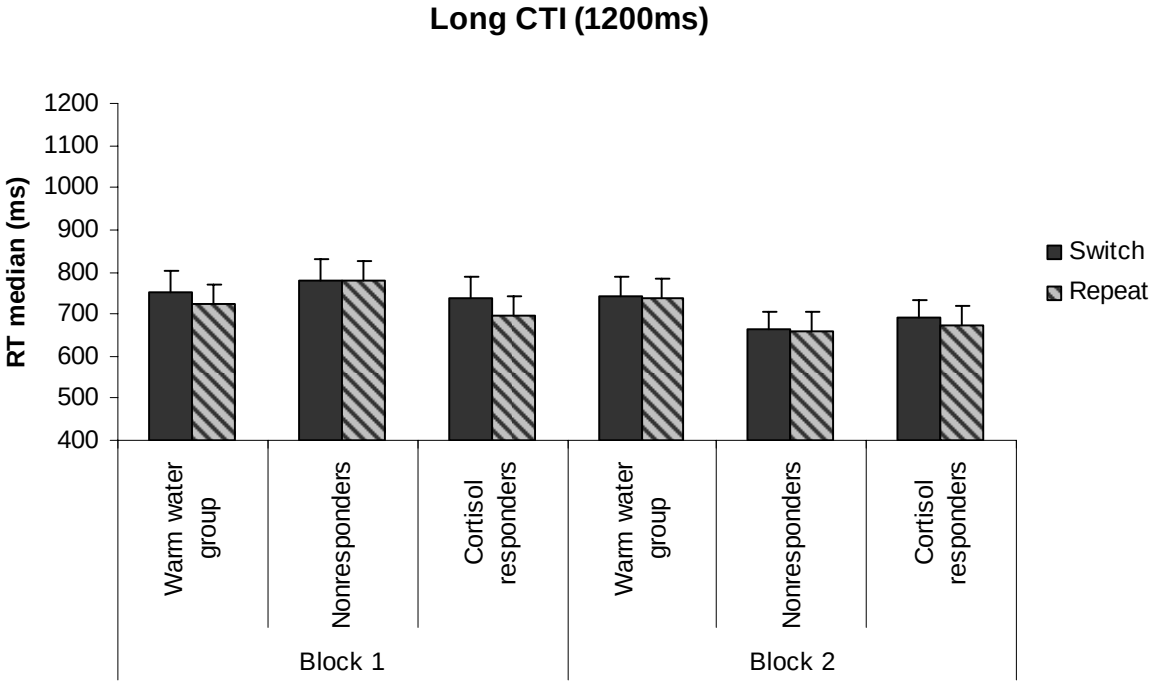

Figure 7b) Reaction times of the three stress groups in switch and repeat trials after *long* CTIs, separately for block 1 and block 2. Values are means and SEM.

No further main effects or interactions were significant (all *F*'s < 3.191, all *p*'s > .084).

2.3.3 Influence of Basal HPA Axis Activity on Cognitive Control

Neither the main effect of AUC_G nor any interactions involving AUC_G reached significance (all *F*'s < 2.315, all *p*'s > .14).

2.3.4 Electrophysiological Data

Overall, the general morphology of the ERP waveforms at midline electrodes in response to the precue involved an early positive peak at 260 ms (P2), a second positive peak at 405 ms (P3), followed by a negative-going slow wave (CNV) lasting until the onset of the target stimulus, that was 1200 ms after onset of the precue. In response to the target, the ERP waves again included two positive peaks at 1480 ms and at 1780 ms after precue onset - that is, 280 ms (P2) or 580 ms (P3) after target onset, respectively. Figure 8 displays the general morphology at Cz, as this electrode position is most representative for the analyzed midline electrodes Fz, Cz, and Pz.

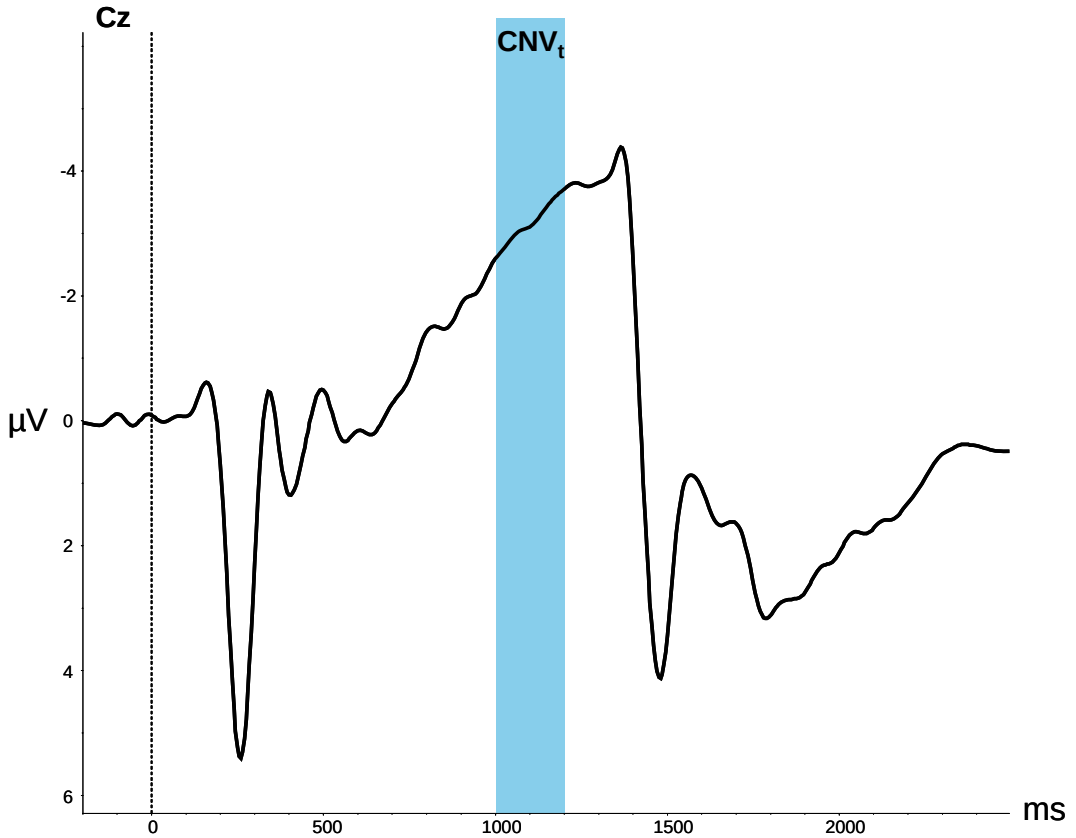

Figure 8 General morphology of the ERP waveforms at Cz in block 2 averaged across all experimental conditions. Precue onset was at time point 0ms, target onset was at 1200ms after precue onset.

2.3.4.1 Influence of **Acute Stress** on the CNV Magnitude

Overall, the CNV was most pronounced at central and frontal electrode sites and least pronounced at the parietal site ($F(2,58) = 24.078$, $p = .000$, $\eta^2 = .454$). Moreover, the CNV was significantly reduced in switch trials compared to repeat trials ($F(1,29) = 8.456$, $p = .007$, $\eta^2 = .226$). This main effect of subtrial was further qualified by a significant interaction of subtrial and stress group ($F(2,29) = 4.708$, $p = .017$, $\eta^2 = .245$)[4], which was not significant in block 1 ($F(2,29)<1$). Post-hoc tests indicated that the difference between switch and repeat trials (CNV switch costs; reduced CNV in switch trials compared to repeat trials) was only significant in the non-stressed warm water group. Concerning the stressed participants, however, both nonresponders and cortisol responders showed no significant switch costs regarding their CNV. Especially the nonresponders had diminished CNV switch costs with a difference of only -0.21µV between switch and repeat trials (-2.568µV and -2.357µV, respectively). This interaction is displayed in Figure 9.

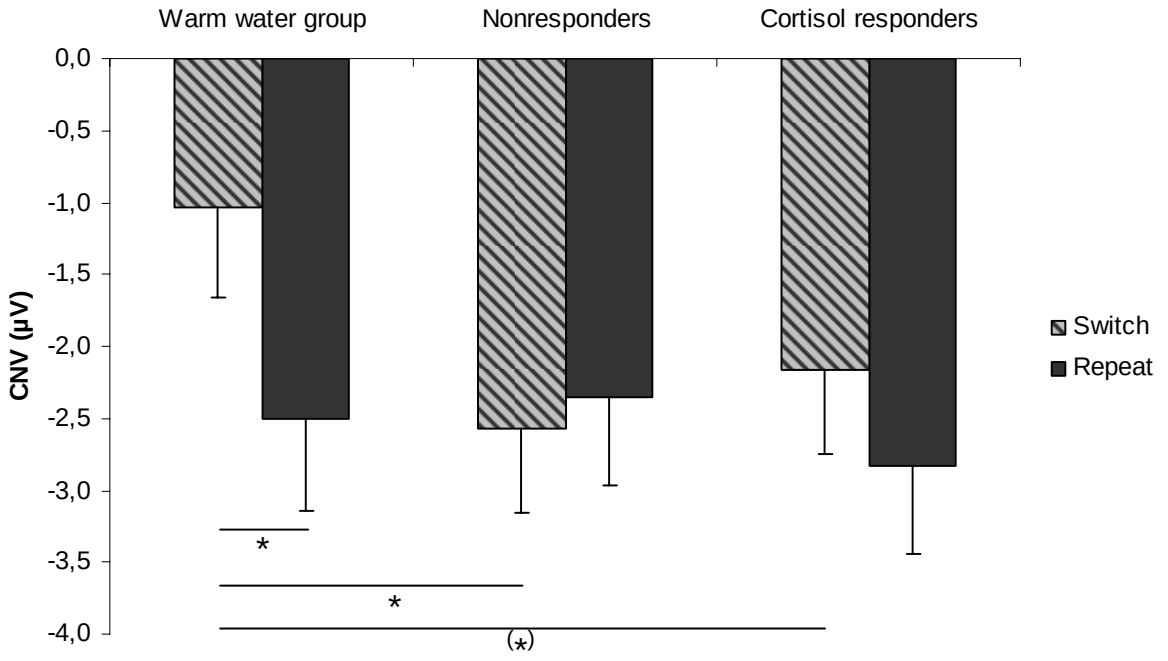

Figure 9 Mean CNV magnitude (µV) in switch and repeat trials for each stress group averaged across electrode sites. Values are means and SEM. * = significant difference, (*) = marginally significant difference according to post-hoc test.

There were no other significant main or interaction effects (all F's < 1).

[4] Since this interaction did not include electrode position as a factor, separate ANOVAs for each electrode position were conducted, in order to demonstrate that this was no overall effect over the whole scalp. ANOVAs showed that the interaction of stress group and subtrial was stronger at Fz ($F(2,29)=3.133$, $p=.059$) and Cz ($F(2,29)=2.640$, $p=.088$) than at Pz ($F(2,29)=1.876$, $p=.171$).

2.3.4.2 Influence of **Basal HPA Axis Activity** on the CNV Magnitude

In order to investigate the influence of basal cortisol levels on cognitive control processes, the z-standardized AUC_G was included as a continuous predictor into the analysis. Again, the main effects of electrode position ($F(2,52) = 22.378$, $p = .000$, $\eta^2 = .463$) and subtrial ($F(1,26) = 11.038$, $p = .003$, $\eta^2 = .298$) were significant. The interaction of subtrial and stress group was only marginally significant ($F(2,26) = 2.717$, $p = .085$, $\eta^2 = .173$). However, subtrial interacted significantly with AUC_G ($F(1,26) = 10.765$, $p = .003$, $\eta^2 = .293$). Post-hoc Pearson correlations at each electrode site indicated that the size of the terminal CNV in switch trials was negatively correlated with AUC_G, but this correlation was only significant at the frontal electrode site ($r = -.428$, $p = .014$), whereas it was negative, but not significant, at central ($r = -.112$, $p = .542$) and parietal ($r = -.027$, $p = .884$) electrode sites. In repeat trials, the correlations were positive, but not significant, regardless of the electrode position (Fz: $r = .131$, $p = .475$; Cz: $r = .195$, $p = .284$; Pz: $r = .125$, $p = .495$). In other words, in subjects with low AUC_G, the CNV distinguished between switch and repeat trials, in that the CNV was less pronounced in switch trials compared to repeat trials. On the other hand, in subjects with high AUC_G, the CNV in switch trials was as large as in repeat trials. To summarize, the higher the AUC_G, the more pronounced was the CNV in switch trials, resulting in a diminished difference between switch and repeat trials in individuals with high AUC_G. This interaction of subtrial and AUC_G (exemplary at Fz) is displayed in Figure 10.

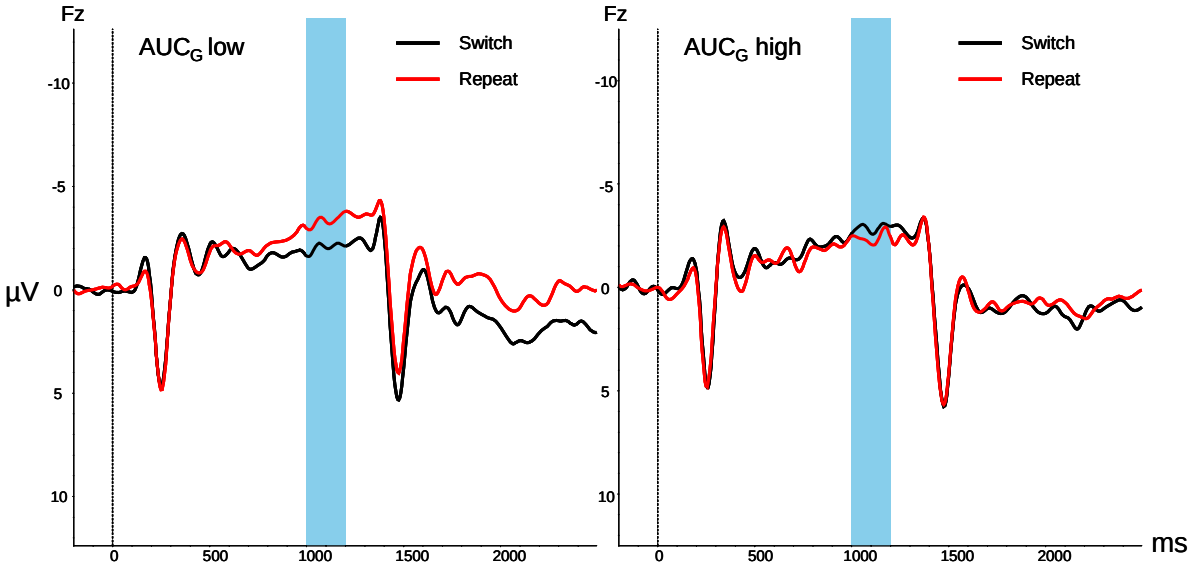

Figure 10 Grand average ERP waveforms at Fz for switch and repeat trials, for illustration separately for subjects with low and high AUC_G.

No other significant main effects or interactions were found (all F's < 1.792, all p's > .178).

2.4 Discussion

The aim of the present study was to investigate the influence of acute stress as well as of basal cortisol levels on cognitive control processes, in order to get further insights into mechanisms that may contribute to the link between stress and aggressive behavior (Kruk et al., 2004). In doing so, behavioral as well as electrophysiological data was analyzed. In the following, results will be discussed in relation to prior research.

2.4.1 Behavioral Results

Acute stress induction led to significantly faster reaction times, whereas the non-stressed control group did not change over the two blocks. More precisely, nonresponders reacted faster especially in switch trials after stress induction, while cortisol responders, on the other hand, became faster primarily in repeat trials following stress. Additionally, contrary to expectations, in the case of nonresponders switch costs were significantly reduced after stress induction compared to block 1, whereas both the warm water group and cortisol responders did not change regarding their switch costs.

In a very recent study, Plessow, Kiesel & Kirschbaum (2012) have also investigated stress effects (using the Trier Social Stress Test, TSST; Kirschbaum, Pirke & Hellhammer, 1993) on performance in a task-switching paradigm. The authors reported larger switch costs in error rates due to high levels of cortisol, but they did not find stress effects on switch costs within reaction times. They interpreted their findings as an impairment of cognitive control processes due to acute stress (Plessow et al., 2012). In the present study, error rates were too low for analysis (below 3 %), and thus this finding of Plessow et al. (2012) could not be replicated. Contrary to the study presented here, the research group around Plessow (2012) did not differentiate between cortisol responders and nonresponders. Hence, the specific reduction of switch costs after stress induction in nonresponders as reported in the study presented here, could not be analyzed in the study of Plessow et al. (2012). Nevertheless, the *in*crease of switch costs within error rates in stressed subjects found by Plessow et al. (2012) contradicts the *de*crease in switch costs within the reaction times in nonresponders reported here. One possible explanation for this discrepancy of results could be a speed-accuracy trade-off due to stress (e.g., see Hockey & Hamilton, 1983), i.e., reduced switch costs in reaction times to the detriment of larger switch costs in error rates. According to Hockey (1997), adverse conditions sometimes lead to "[...] a faster but less accurate mode of responding" (p. 85).

Furthermore, contrary effects of acute cortisol rise and the sole activation of the sympathetic nervous system without an HPA axis response following a stressor (as in nonresponders) have been reported by earlier studies concerning memory performance. More precisely, there is evidence for detrimental effects of cortisol increase on memory performance, but for no impairments - or even beneficial effects of stress, either - if the HPA axis activation is absent (see e.g., Buchanan & Tranel, 2008; Elzinga & Roelofs, 2005). This is in line with the reduction of switch costs in nonresponders as reported here. However, in the present study, no detrimental effects, such as larger switch costs, in cortisol responders compared to the nonresponders and the non-stressed control group were found. It is possible that the rise in cortisol due to the SECP in the present study was not sufficient to induce impairing effects on cognitive control processes. Furthermore, the performance of humans can be satisfactory even despite adverse or stressful conditions because of a further allocation of cognitive resources to the task as proposed by the regulatory-control model (Hockey, 1997). According to this model, performance stability can be maintained despite challenging circumstances by an active control of the individual which requires the allocation of further cognitive resources and mental effort (Hockey, 1997). Hence, potentially in healthy young humans, a further situational demand such as an aggressive encounter with social provocation may be necessary to impair cognitive/inhibitory control under stress. Further research is required to test this assumption.

As a rather explorative investigation, in the present study, basal cortisol levels were included in the analysis. However, against prior expectations, basal HPA axis activity did not affect performance in the task-switching paradigm, neither the absolute reaction times nor switch costs. Moreover, basal cortisol levels did not interact with an acute rise in cortisol.

Böhnke et al. (2010a, 2010b) found strong negative correlations between basal cortisol levels and aggressive behavior during an aggressive encounter with another alleged participant, but this association was only found in women, not in men. In the present study, however, firstly, all subjects were males, and secondly, only stress induction was realized, without any experimental manipulation of social provocation, which might explain absent significant effects of basal cortisol levels in the present study. Again, in healthy young humans, cognitive control may possibly not be affected by basal HPA axis activity unless an additional situational trigger, such as a social conflict or provocation, occurs. Nevertheless, even if overt behavior was not affected, the underlying information processing as measured with EEG was influenced by basal cortisol levels, as discussed in the next section.

2.4.2 Electrophysiological Results

In addition to the investigation of overt behavior as an indirect measurement of information processing, another aim of the present study was to examine information processing via measuring brain activity directly during cognitive control processes. An ERP component which has been of interest in the investigation of cognitive control using task-switching paradigms is the terminal CNV, which has been associated with the motor preparation for an upcoming task (Gaillard, 1977; Walter et al., 1964). In the current study, the magnitude of the terminal CNV was reduced in switch trials compared to repeat trials, which is in line with previous literature (e.g., Hsieh & Cheng, 2006), although there are also studies reporting an increased CNV in switch trials compared to repeat trials (e.g., Lorist et al., 2000; Rushworth, Passingham & Nobre, 2002). Differences in paradigms and experimental designs may have contributed to the inconsistency of the results. Slow negative brain potentials such as the CNV are believed to reflect the allocation of neural resources (Rösler et al., 1997). Thus, an attenuation of the CNV in switch trials as reported here might indicate less available processing resources for the preparation of the upcoming task due to the fact that it is more difficult to switch between tasks than to repeat the previous task. This is consistent with interpretations by Gajewski and colleagues (2010) who state that "the reduced CNV may imply problems in keeping track of the task sequence or impairments of the allocation of task-relevant resources" (p.196). Furthermore, an enhancement of the CNV was found to reflect effort allocation, coinciding with an improved performance (Falkenstein, Hoormann, Hohnsbein & Kleinsorge, 2003).

Thus, the difference of the CNV magnitude between switch and repeat trials reported here, which was found only in the non-stressed control group, might reflect electrophysiological switch costs, corresponding to the behavioral switch costs. In case of acute stress, however, these CNV switch costs were diminished, particularly if the activation of the HPA axis was absent like in nonresponders. This matches the behavioral results, since nonresponders were the only group with decreased switch costs in reaction times after stress induction. Nevertheless, cortisol responders also had reduced CNV switch costs after stress compared to the non-stressed warm water group. These differences between the stressed and the non-stressed subjects were primarily caused by an increase in the CNV magnitude in switch trials. Hence, the acute stress induction might have led to the suppression of the switch-related lack of resource allocation for task preparation, which is indexed by a pronounced CNV not only in repeat trials but also in switch trials. In other words, the present findings suggest that acute stress, especially if only the sympathetic nervous system is

activated without an HPA axis response, may improve the motor preparation for a rapid behavioral response. This could be interpreted in the sense of an adaptive cognitive preparation for a fast fight-or-flight reaction.

On a more explorative level, basal HPA axis activity was also expected to influence the CNV during task-switching. In fact, basal morning cortisol levels significantly correlated with the CNV magnitude, but only in switch trials. More precisely, subjects with low levels of basal cortisol showed CNV switch costs; i.e., a reduced CNV in switch trials compared to repeat trials. Subjects with high basal cortisol levels, on the contrary, did not differentiate between switch and repeat trials, since the CNV in switch trials was as pronounced as in repeat trials. Thus, high levels of basal morning cortisol might compensate for the lack of resource allocation in switch trials, leading to a motor preparation just as well as in repeat trials. This may indicate an optimization of the preparation for a rapid behavioral response when basal cortisol concentrations are high.

The influences of acute as well as of basal HPA axis activity were strongest at the frontal electrode site. Prior studies suggest that the prefrontal cortex (PFC) is crucially involved in the generation of the CNV (Basile, Brunder, Tarkka & Papanicolaou, 1997; Rosahl & Knight, 1995). Moreover, there is evidence that the PFC is not only an important regulator (Cerqueira, Almeida & Sousa, 2008) but also a key target (Cerqueira, Mailliet, Almeida, Jay & Sousa, 2007) of stress. Glucocorticoids bind to two types of receptors in the brain, the mineralcorticoid (MR) and the glucocorticoid (GR) receptors, with a preferential distribution of the latter in the PFC (for a review, see Lupien, Maheu, Tu, Fiocco & Schramek, 2007). According to de Kloet, Oitzl & Joels (1999), stress can have adaptive or maladaptive effects on cognition depending on the ratio of occupied MRs and GRs through glucocorticoids. As reflected by an inverted U-shaped dose-response function, cognition can be improved if most of the MRs and only a portion of the GRs are occupied by glucocorticoids. However, at the extremes of the inverted U-shaped curve (sole activation of MRs without GR activation or both receptor subtypes fully occupied by glucocorticoids), cognitive functioning can be impaired (de Kloet et al., 1999; Lupien et al., 2007). Accordingly, the reduction of the CNV switch costs in the stressed participants as found in the present study might be caused by an optimal level of circulating cortisol elicited by the cold-pressor test. If the elevation of endogenous cortisol levels in cortisol responders had been stronger or if cortisol levels were enhanced exogenously by hydrocortisone administration, behavioral as well as CNV switch costs might have been increased instead of reduced.

To summarize, in the present study both elevated levels of acute cortisol and relatively high basal HPA axis activity were associated with an increase of the CNV in switch trials, indicating enhanced motor preparation for the upcoming behavioral response.

2.4.3 Limitations and Strengths of the Study

Some limitations of the present study should be addressed: First, the investigated sample involved only healthy male students of a young age and a high educational status. Thus, the reported findings cannot be generalized to women or to men of a different population. Nevertheless, this study was designed as a first step towards evidence for the importance of cognitive control as a factor in the relationship between stress and aggression. Therefore, the primary goal of the experiment was to examine the influence of acute stress on cognitive control in a feasible sample of subjects. Second, the classification of stressed participants into cortisol responders and nonresponders establishes a quasi-experimental design, as the assignment of the subjects to these two groups was not randomized but depended on their HPA axis response due to the stressor. As a consequence, it is yet unknown which personality factors distinguish these two groups that may have influenced the results. Moreover, little is known about the extent to which the cortisol reaction in response to the (socially evaluated) cold pressor test is stable over time within each individual and whether it would be the same in response to other stressor types, e.g. exposure to the TSST. This should be subject of future studies using endogenous cortisol elevation and has to be kept in mind when interpreting the present results regarding the differences between these stress groups. Third, the stimuli used in the task-switching paradigm were exclusively non-emotional. However, in the context of acute stress as a highly arousing and emotional state of an individual, the performance of subjects in a task with emotional content might have been different to the findings presented here, as the test situation would be in closer relation to the stress-causing event.

On the other hand, several strengths of the current study should be noted as well: First, cognitive control as the variable of interest was measured not only after the experimental manipulation of stress but also beforehand. Hence, due to this within-subject design, differences between stress groups that were apparent after stress induction but not before could be clearly attributed to the experimental manipulation. Second, cortisol concentrations were elevated endogenously through a real stress situation. Thus, the entire natural stress system of the organism was activated, thereby improving ecological validity, which differs from experiments with exogenous cortisol administration that only manipulate one factor of the complex endocrinological and psychophysiological stress system. Third, not only acute

HPA axis activity was assessed, but also basal morning cortisol levels. This allowed us to investigate the influence of both more state- and more trait-like aspects of the stress system.

2.4.4 Conclusion

The present study has given insights into the role of cognitive control within the relationship between stress and aggression. Using behavioral as well as electrophysiological measurements of cognitive control processes, the reported findings suggest that not only acute stress induction but also high basal cortisol concentrations optimize the motor preparation for a rapid behavioral response.

Chapter III

The Influence of Acute Stress and Social Provocation on Approach-Avoidance Motivation

3.1 Introduction

Stress is one of the most important contributors to aggressive behavior, even in healthy individuals (e.g., Böhnke et al., 2010b; Verona & Kilmer, 2007; Verona et al., 2007). Böhnke and colleagues (2010b), for instance, found enhanced aggressive behavior in women who exogenously received 20 mg of hydrocortisone, which is a pharmacological equivalent to the stress hormone cortisol. According to the model of Kruk and colleagues (2004), an activation of the HPA axis leads to distorted information processing in the sense of increased sensitivity towards social conflict signals, which in turn results in a lower threshold for aggressive behavior. Work of the research group around Bertsch (2011) provided evidence for altered information processing in an emotional Stroop paradigm after the administration of exogenous cortisol and subsequent provocation during an aggressive encounter. More precisely, they found faster reactions in response to all facial expressions (angry, happy, fearful, and neutral) after cortisol administration and provocation. Furthermore, provocation resulted in increased early as well as later positive posterior ERP components. Cortisol, on the other hand, led to a reduction of an early frontocentral bias for angry faces (Bertsch et al., 2011). However, it remains unclear if endogenously elevated cortisol levels in combination with the activation of the sympathetic nervous system (a crucial part of the natural stress response) would lead to equivalent effects, or if the dynamic aspect of a real stress response would even reinforce the association between cortisol, changes in social information processing, and aggression.

Moreover, there are paradigms for assessing social information processing that seem particularly suitable in the context of aggression, for instance, tasks with active components such as the so-called approach-avoidance paradigms that measure the motivation to approach or avoid certain stimuli. Approach and avoidance tendencies are said to be a direct consequence of the automatic classification of a stimulus as appetitive/positive or aversive/negative. While - under normal circumstances - a positive evaluation of a stimulus leads to a predisposition for an immediate approach tendency, negative evaluations produce an immediate tendency to avoid the stimulus (e.g., Chen & Bargh, 1999). Nevertheless, these approach-avoidance tendencies can be affected, for instance, by the emotional state of the individual. Especially anger is an emotion that is discussed controversially within the literature with respect to the question of whether it is related to approach or avoidance tendencies. Anger is usually experienced as a negative and unpleasant emotion, which should lead to avoidance tendencies. However, there is also evidence for an association between

anger and approach behavior, i.e., the tendency to approach in order to resolve the anger-producing stimulus/event (for a detailed review, see Carver & Harmon-Jones, 2009). Since anger is a critical component of reactive or affective aggression (Geen, 2001), which clearly involves approach behavior, experimental provocation leading to anger should thus be associated with faster approach than avoidance reactions.

Furthermore, there is some evidence for cortisol effects on approach-avoidance behavior. The research group around Roelofs (Roelofs, Elzinga & Rotteveel, 2005), for instance, showed that endogenously elevated cortisol levels led to a reduction of approach-avoidance behavior during a stressful situation. Specifically, subjects with a high cortisol increase following the stressor showed the normal congruency effect in response to stimuli representing facial expressions (faster reactions in congruent conditions - that is, approaching happy faces and avoiding angry faces - than in incongruent conditions - when forced to approach angry faces and to avoid happy faces) prior to the stressor, but a diminished congruency effect in a social stress-context. The authors interpreted this as a possible consequence of a hyperactive ACC conflict detection system reflecting a higher need for cognitive control processes by the PFC in response to all potentially hostile stimuli due to the stress exposure (Roelofs et al., 2005). On the other hand, there is also evidence for enhanced congruency effects in response to angry faces (faster avoidance than approach) after cortisol administration, but only in highly avoidant individuals (van Peer et al., 2007). In healthy individuals, exogenously administered cortisol was found to increase the likeliness of risky decision making in case of a potentially big reward. This was interpreted as an anxiolytic-like effect of cortisol resulting in an approach-motivated reduced sensitiveness to punishment and enhanced sensitiveness towards reward promising stimuli (Putman, Antypa, Crysovergi & van der Does, 2010). Putman & Roelofs (2011) summarize these findings by suggesting that "[...] cortisol may immediately facilitate goal-directed motivational behavior" (p. 443).

First findings suggesting a mediating role of approach-avoidance motivation in the stress-aggression-link have been provided by a recent study of Verona, Sadeh & Curtin (2009), who measured behavioral approach motivation by frontal EEG-alpha asymmetry after stress exposure and related the lateralized brain activity to stress-induced aggression. Results showed that stress induction (compared to no stress exposure) led to more left than right frontal EEG-activity, which in turn in the stress condition was associated with increased subsequent aggressive behavior in an employee-supervisor task. This was not the case for the no-stress condition, in which frontal asymmetry predicted decreased subsequent aggressive behavior (Verona et al., 2009). However, these results have to be interpreted with care, as no

cortisol measurements were used to validate the stress induction. Moreover, important aspects remain that need to be further investigated and that will be subject of the present study:

First, the mediating role of approach tendencies in the relationship between stress and aggression should be further examined by using more direct measures of approach (and avoidance) motivation, such as reaction time paradigms. In joystick tasks, for instance, subjects have to evaluate the valence of emotional stimuli by executing arm movements (e.g., pushing or pulling a lever, thereby making approaching or avoiding arm movements), that are either congruent or incongruent to their spontaneous/implicit action tendencies (see, e.g., Chen & Bargh, 1999; Marsh, Ambady & Kleck, 2005; Roelofs et al., 2010). Another often used and well validated (Krieglmeyer & Deutsch, 2010) more direct measure of approach-avoidance motivation is the so-called Manikin Task designed by De Houwer, Crombez, Baeyens & Hermans (2001), during which subjects have to move a small figure either towards or away from a presented stimulus by pressing a key on the keyboard (see, e.g., Krieglmeyer, Deutsch, De Houwer & De Raedt, 2010). In the current study, an adapted version of the Manikin Task (with emotional facial expressions as stimuli) was used in order to measure approach-avoidance tendencies.

Second, little is known yet about the underlying changes in brain processes during approach (or avoidance) reactions as the link between stress and aggression. Thus, besides reaction times, EEG measures such as ERPs should be examined, as they are well suited for the assessment of social information processing such as emotional face processing (Eimer & Holmes, 2007). Van Peer and colleagues (2007; van Peer, Spinhoven, van Dijk & Roelofs, 2009) provided evidence for significant increases of early (P150) as well as late (P3) positive ERP amplitudes during the avoidance of angry facial expressions after exogenous cortisol administration. However, these cortisol effects were only true for highly avoidant individuals (van Peer et al., 2007) and patients with social phobia (van Peer et al., 2009) but not for healthy subjects. On the other hand, effects of endogenously elevated cortisol levels in combination with subsequent provocation on emotional face processing during an approach-avoidance task in healthy individuals has not been subject of research, yet.

Third, in addition to cortisol, another steroid hormone said to be crucially involved in the release of aggression that should therefore be taken into account, is testosterone (for a review, see Archer, 2006). According to a recent study by Mehta & Beer (2010), testosterone impacts on aggressive behavior by reducing the activity of the medial orbitofrontal cortex, thus leading to diminished impulse control and self-regulation. However, recent studies suggest a modulation of testosterone effects on aggression by cortisol. Specifically, a positive

correlation between testosterone levels and aggressive behavior was only found in individuals with low cortisol concentrations, whereas there were no testosterone effects on aggressive behavior in subjects with high cortisol levels (Popma et al., 2007). According to a review by Terburg, Morgan & van Honk (2009), the balance between testosterone and cortisol concentrations can be seen as the biological equivalent to the psychological balance between the behavioral activation system (BAS, approach motivation) and the behavioral inhibition system (BIS, avoidance motivation). Thus, a high testosterone-to-cortisol ratio (i.e., high testosterone levels and low cortisol levels) seems to predispose for approach motivation towards threat, often resulting in impulsive aggression (Terburg et al., 2009). However, both the influence of the basal testosterone-to-cortisol ratio and the interaction of acutely (endogenously) elevated cortisol levels with acute testosterone concentrations on approach-avoidance motivation has not been studied, yet.

Therefore, the aims of the present study were (1) to elucidate main and interaction effects of acutely endogenously increased cortisol levels and acute testosterone concentrations following stress induction and social provocation on approach-avoidance tendencies that may have an influence on the stress-aggression link; (2) to examine the role of the baseline testosterone-to-cortisol ratio on approach-avoidance tendencies after acute stress induction and provocation; and (3) to investigate changes in social information processing using ERP technique during an approach-avoidance task caused by acute stress induction and subsequent social provocation. Angry and happy facial expressions served as stimuli in the approach-avoidance task, since facial expressions are presumed to be the most important and most direct social signals for humans (Le Doux, 1998; Öhman, Flykt & Lundqvist, 2000). Early (P2) as well as late (P3) positive ERP components were found to be involved in emotional face processing and seem to be sensitive to the influence of cortisol as well as of provocation (Bertsch et al., 2009; Bertsch et al., 2011; van Peer et al., 2007; van Peer, Spinhoven & Roelofs, 2010). Thus, in the current study, the ERP analyses focused on these two positive amplitudes.

Healthy female and male participants were subjected either to an acute stress induction using the Socially Evaluated Cold Pressor Test (SECP; Schwabe et al., 2008), or to a non-stressful control procedure. Subsequently, the subjects were either highly or mildly provoked in a competitive reaction time task (Taylor Aggression Paradigm, TAP; Taylor, 1967). Prior to these experimental manipulations of stress (SECP) and provocation (TAP), and also afterwards, all participants had to perform an adapted version of the Manikin Task with angry and happy facial expressions, in order to measure their approach-avoidance tendencies. At the

end of the experimental session, subjects performed a non-specific task with emotional pictures that is described and analyzed elsewhere (Dierolf, in prep.).

In the current study, it was expected that the acute rise in cortisol levels and provocation significantly influence reaction times and information processing during the Manikin Task. Specifically, we hypothesized that the acute cortisol increase in combination with subsequent high provocation would lead to greater approach tendencies (faster reaction times when approaching the faces), especially in response to angry faces. Moreover, this effect should be further influenced by acute testosterone levels as well as by the basal testosterone-to-cortisol ratio. Regarding social information processing measured with EEG, it was supposed that high social provocation would cause an enhancement of early as well as of later positive ERP amplitudes, whereas cortisol should decrease these ERP components (Bertsch et al., 2009; Bertsch et al., 2011). Additionally, the interaction of high provocation and increased cortisol levels was expected to influence both amplitudes, as well. Since earlier findings concerning gender differences in the stress-aggression link are inconsistent (Böhnke et al., 2010b; Verona & Kilmer, 2007; Verona et al., 2007), there were no specific hypotheses regarding differential effects of cortisol, testosterone and provocation on males and females.

3.2 Methods and Material

3.2.1 Participants

72 healthy students (36 female, 36 male; mean age = 23.96 years, SD = 2.27 years) of the University of Trier in Germany, took part in the study. Inclusion criteria were: right-handedness, non-smoking, no acute or chronic medical disease, no psychiatric disorder, no regular medication (except oral contraceptives), no color blindness, no hearing problems, normal weight (mean BMI = 22.77, SD = 2.54), and German as mother tongue. In addition, students taking courses in psychology were not allowed to take part in the study because of potential previous knowledge that may have led to biased behavior during the experiment. Moreover, we only included women who were taking hormonal contraceptives, in order to keep the hormonal status equal. Because of possible influences on endogenous cortisol concentrations, the following contraceptive brands were excluded: Yasmine, Yasminelle, Petibelle, Aida, Yaz, and Angeliq. The study was conducted in accordance with the Declaration of Helsinki and was approved by the ethics committee of the University of Trier.

All participants provided written informed consent and received 45 Euros for their participation.

3.2.2 General Procedure

The experimental sessions started at 13:30, 15:30, or 17:30 p.m., to make sure that subjects were examined when the endogenous cortisol level is relatively low due to the circadian rhythm of the HPA axis activity (Kirschbaum & Hellhammer, 1999; Schreiber et al., 2006). All subjects were randomly assigned to the experimental conditions (two thirds to the stress condition, one third to the non-stress condition, one half to the high provocation group, and the other half to the low provocation group). Due to later categorization of stressed subjects into cortisol responders and nonresponders (see section *3.2.9*), the six experimental conditions were as follows: (1) no stress / low provocation, (2) no stress / high provocation, (3) nonresponders / low provocation, (4) nonresponders / high provocation, (5) cortisol responders / low provocation, (6) cortisol responders / high provocation. Sex was balanced across groups. All participants were investigated individually, and the experimental procedure was the same for all of them. Subjects were told that the study dealt with the influence of stress on the reaction times and on cognitive functioning. At the beginning of the experimental session, subjects were introduced to an alleged other participant (same sex), who in fact was a confederate of the investigators. The experiment took place in a sound-attenuated EEG-laboratory, where the light was dimmed. Subjects were seated (at a 1 m distance) in front of a computer screen, and the EEG was applied. After this, the experimenters left the subjects alone in the room, instructing them via computer. As depicted in Figure 11, the procedure was as follows: Participants had to perform the Approach-Avoidance Task (Manikin Task) for a first time (block 1), followed by the stress induction (or non-stressful control procedure) by means of the SECP (see Schwabe et al., 2008). Subsequently, subjects were either highly or mildly provoked by using a modified version of the Taylor Aggression Paradigm (TAP, Taylor, 1967). After this, subjects performed the Approach-Avoidance Task for a second time (block 2). Finally, there was an unspecific IAPS Task, which is explained and analyzed elsewhere (Dierolf, in prep.). At the end of the experimental session, all subjects were extensively debriefed, thanked, and paid for their participation. The entire experiment lasted for about 90 minutes.

During the experimental session, subjects gave salivary cortisol and testosterone samples at several points in time, as displayed in Figure 11. Cortisol samples were collected at

the following points in time: At the beginning of the experimental session after the EEG device was applied to the subjects (C0); after the first block of the Manikin Task, directly before the SECP (C1); after the SECP (C2); after the TAP (C3); after the second block of the Manikin Task (C4); and finally, at the end of the experimental session, after the nonspecific IAPS Task (C5). For the cortisol samples, salivettes (Sarstedt, Nümbrecht, Germany) were used. Subjects were instructed to chew gently on these salivettes for a duration of 1 minute.

Salivary testosterone samples were collected in 2ml reaction tubes (Sarstedt, Nümbrecht, Germany) and were taken at the beginning of the experiment (TA); after the SECP (TB); after the TAP (TC); after the second block of the Manikin Task (TD), and finally, at the end of the experiment (TE). In case of cortisol and testosterone samples being taken at the same points in time, subjects always had to give the cortisol sample first, and the testosterone sample afterwards, as the chewing of the salivettes stimulates the saliva production and thus facilitates the filling of the reaction tubes for the testosterone samples.

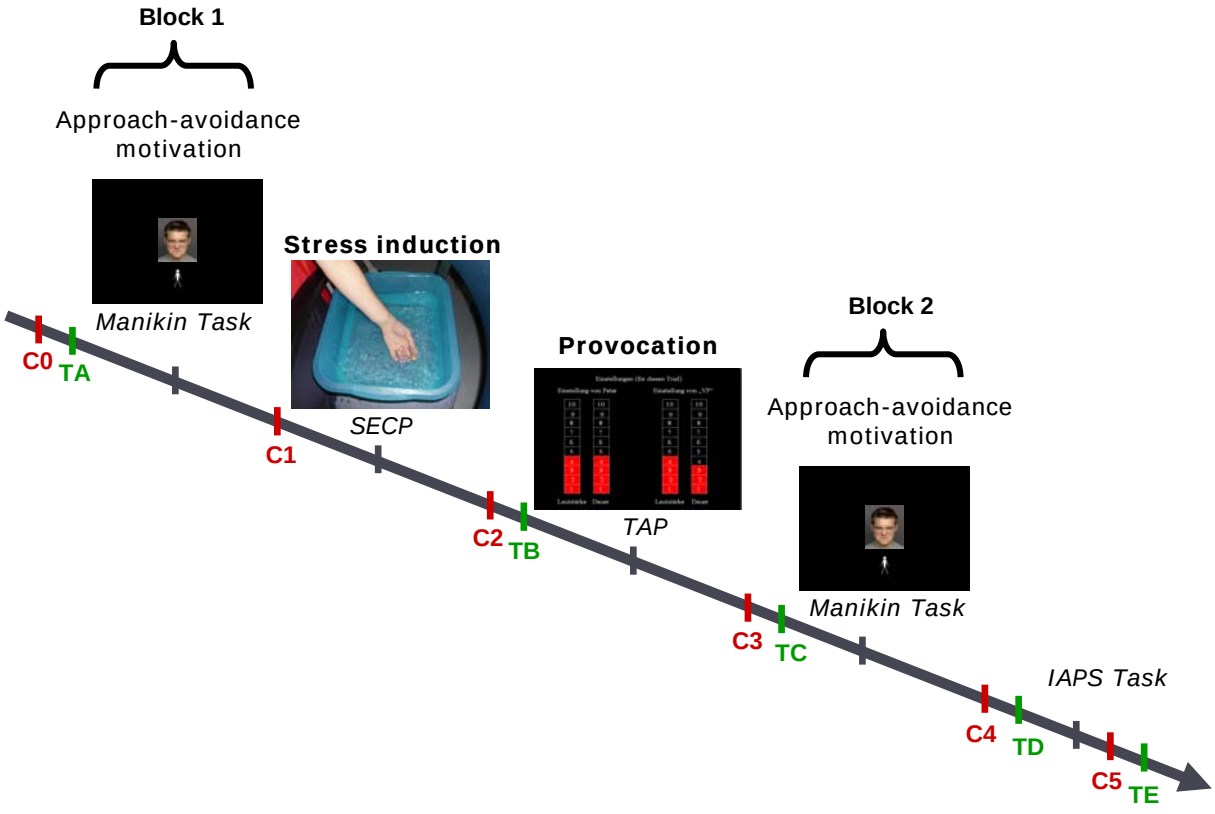

Figure 11 Timeline of the experimental session. C0-C5 = cortisol samples, TA-TE = testosterone samples.

3.2.3 Materials

For the presentation of experimental stimuli and the recording of participants reactions, E-Prime© experiment presentation software (Version 2.0, Psychology Software Tools Inc., Pittsburgh, PA) was used (Schneider et al., 2002).

3.2.4 The Socially Evaluated Cold Pressor Test

In order to induce acute stress, the SECP (Schwabe et al., 2008) was used. Under the stress condition, subjects had to immerse their left hand for 3 minutes into ice-cold water of 1-3°C. At the same time, they were videotaped and observed by an investigator of the opposite sex who was present in the room. In order to increase the psychosocial stressor component, they were led to believe that their mimics and gestures during the task would be analyzed by the investigator. The non-stressful control procedure (warm water group) was exactly the same, except that the water was at body temperature, i.e., 36-38°C. Since for analyses the stressed participants would be classified into cortisol responders and nonresponders depending on their cortisol rise following the SECP, two thirds of the sample were randomly assigned to the stress condition and one third to the warm water condition.

3.2.5 The Taylor Aggression Paradigm

As in previous studies by Böhnke et al. (2010a, 2010b) and Bertsch et al. (2009, 2011), the provocation of the subjects was realized by means of a modified version of the Taylor Aggression Paradigm (Taylor, 1967), which is a well validated method to provoke aggressive behavior in a laboratory setting (Anderson & Bushman, 1997; Phillips, 2011). Subjects were led to believe that they were playing a reaction time task against an alleged other participant (of the same sex) whom they had met at the beginning of the experiment (but who, in fact, was a confederate of the investigators). During this task, participants had to react as quickly as possible by pressing a key when a green square appeared on the computer screen. They were told that whoever was slower and therefore lost the trial would be confronted with a blast of noise. Before each trial, the participant was asked to set the volume as well as the duration of the noise for the alleged opponent for those cases in which the latter lost the trial. The duration could vary between level 0 (0 seconds) and level 10 (5 seconds) in increments of 0.5 seconds, while the volume varied between level 0 (0 dB) and level 10 (105 dB) in increments of 5 dB - volume level 1 equated to 60 dB. At the end of each trial, participants received

feedback about whether they had won or lost the trial. Subsequently, the own volume and duration settings and those of the opponent were presented on the computer screen. In case of having lost the trial, the participant received the blast of noise. Since the alleged opponent was no real competitor, participants won and lost half of the trials regardless of their reaction times, and noise volume and duration were set by the computer. The task consisted of three experimental blocks with 10 trials each. During the first block, the given noises were gentle (volume: M = 62.5 dB, range 0-70 dB; duration: M = 0.75s, range 0-1.5s) for all participants. In the high provocation group the volume and duration of the noises increased during the second (volume: M = 82.5 dB, range 75-90 dB; duration: M = 2.75 s, range 2-3.5 s) and especially during the third block (volume: M = 99 dB, range 90-105 dB; duration: M = 4.4 s, range 3.5-5 s). On the other hand, the low provocation group received short and gentle noises as in block 1 throughout all three blocks. The volume and duration settings of each participant for his alleged opponent were recorded and averaged for each trial. All trials of each block were averaged for each participant in order to calculate the mean aggressive behavior within the distinct blocks. These values of mean aggressive behavior within each block served as the dependent variable "aggressive behavior" in the subsequent statistical analyses. Overall, the task took about 10 minutes.

3.2.6 The Manikin Task

At the beginning of the experiment as well as after the manipulation of acute stress and provocation (see Figure 11), all subjects had to perform an adapted version of the Manikin Task designed by De Houwer et al. (2001), a well validated approach-avoidance paradigm (Krieglmeyer & Deutsch, 2010). During this task, subjects had to move a small figure - the manikin - either towards (approach) or away from (avoid) a picture depicting an emotional facial expression, which was either angry or happy. They were instructed to imagine that they were the manikin themselves.

As illustrated in Figure 12, each trial began with a fixation cross being displayed in the center of the screen, which lasted for 500ms. Subsequently, the manikin appeared in the middle of either the upper or lower third of the screen for a duration of 750ms. Then the picture with the facial expression appeared in the center of the screen. The participant had to react as correctly and quickly as possible by pressing upwards or downwards arrow keys, in order to move the manikin either towards the facial expression or away from it. Two blocked conditions were realized: In the congruent condition the instruction was to move the manikin

towards happy faces and away from angry faces, whereas in the incongruent condition it was the opposite (i.e., to move it towards angry faces and away from happy faces). To move the manikin, subjects had to take three steps by pressing the arrow keys three times. Thus, depending on the movement direction and the primary position of the manikin, it ended up either close to the facial expression or at the edge of the screen. The initial position of the manikin (above or below the face, respectively) was determined in a randomized fashion, resulting in an equally frequent occurrence of both positions. 500ms after the third step all stimuli disappeared, and the black screen lasted for a variable inter-stimulus interval, resulting in a fixed total trial length of 4.5 seconds. An exception occurred if subjects took the first step later than 1400ms after the face onset. In this case, the total trial length was set to 5 seconds. Subjects were told to respond by pressing the first key as fast as possible. Whenever they took too long (more than 1500ms) to take the first manikin step, a message appeared with the instruction to respond faster. In case of an error, that is, moving the figure into the wrong direction, an error message appeared directly after the first wrong key press with a duration of 500 ms. After this, the next trial started immediately without filling up the 4.5 seconds of total trial length. The time between the presentation of the face and the first key press in correct trials was used as the dependent variable "reaction time" (RT) in later statistical analyses.

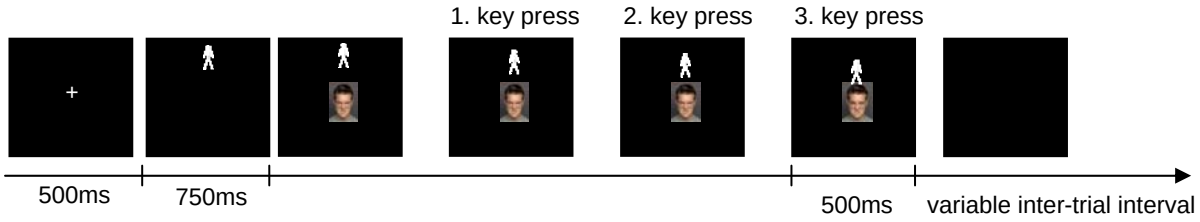

Figure 12 Timeline of a trial of the Manikin Task, exemplary for an incongruent trial.

Both in block 1 (before the manipulation of stress and provocation) and in block 2 (after manipulation of stress and provocation), all subjects performed both congruent and incongruent conditions in a blocked design. The order of these two congruency blocks (congruent vs. incongruent) were randomly assigned to the subjects. However, within each subject the order of the congruency blocks was kept constant for block 1 and block 2. That is, subjects who began with the congruent condition followed by the incongruent condition in block 1 got the same order (congruent - incongruent) in block 2, and vice versa. The order of congruency conditions was balanced across all experimental conditions.

Each (in)congruency block contained six practice trials and 56 experimental trials, resulting in 112 experimental trials before and 112 experimental trials after the manipulation of stress and provocation. Thus, the total number of experimental trials was 224. In each congruency block, half of the facial expressions were happy, half of them were angry.

The facial stimuli were taken from Ebner, Riediger & Lindenberger (2010). For the experimental trials, pictures of 28 male and 28 female models of the young age pool from the database ($M_{N=58}$ = 24.2 years, $SD_{N=58}$ = 3.4 years, see Ebner et al., 2010) were used. Each model displayed two slightly different versions of both angry and happy expressions (which were balanced across block 1 and block 2), resulting in a total number of 224 facial expressions. The picture sizes were 2.835 x 3.543 pixels (JPEG format). The manikin figure was gender-free, white colored, and 2.8 cm tall. With each step, the manikin moved 38 pixels downwards or upwards, respectively. The faces were presented in a pseudo-randomized fashion; that is, no more than three pictures of the same emotion appeared in succession.

3.2.7 Cortisol and Testosterone Analyses

Immediately after the experimental session, the cortisol and testosterone samples were frozen for biochemical analysis. For the analysis of the salivary cortisol, a time-resolved immunoassay with fluorescence detection was used as described in detail elsewhere (Dressendörfer et al., 1992). The intra- and inter-assay variability was below 10% and 12%, respectively. Salivary testosterone was measured by ELISA (Salimetrics, Newmarket, UK) with a minimum detection limit of <1.0 pg/ml, and intra- and inter-assay coefficients of variation of 2.5% and 5.6%, respectively, in males, and of 6.7% and 14.05% respectively, in females.

3.2.8 EEG Recording and Quantification

The EEG was recorded from 27 electrode positions plus the mastoids in accordance with the 10-10 electrode reference system (Chatrian et al., 1988) with the Easy-Cap electrode system (Falk Minow Services). All sites were referenced to FCz, and the bipolar horizontal as well as vertical electrooculogram (EOG) was recorded. Ag/AgCl electrodes were used, and the impedances of the EEG electrodes were below 5kΩ. EEG and EOG were amplified by means of a 32-channel BrainAmp amplifier (input impedance: 10 MΩ; Brain Products, GmbH) in AC mode. The pass-band was put to 0.016 to 499 Hz (-12 dB/octave rolloff). Signals were digitalized at 1000 Hz.

Analyzer 2.0 (Brain Products GmbH) was used for the editing and quantification of the EEG. For analyses, the EEG was re-referenced to linked mastoids. Using the algorithm of Gratton, Coles & Donchin (1983) EEG was corrected semi-automatically for eye movements. Segments with non-physiological artifacts were excluded from analysis by means of the semiautomatic artifact rejection. The sampling rate was set to 200Hz. EEG was segmented off-line into periods of 2200ms, starting at 200ms prior to face onset and ending 2000ms after face onset. The signal was baseline-corrected using the 200ms prior to stimulus onset as reference. A digital low pass filter of 12Hz (-48dB) was applied. Separate average amplitudes were calculated for each electrode, individual, and each combination of Emotion (angry vs. happy face) and Movement (approach vs. avoidance), but only for those experimental trials with correct responses. Based on visual inspection as well as on the topography of grand average ERPs averaged across all participants, the stimulus (face)-locked P2 and P3 components were quantified by calculating the mean activity of the time intervals between 160-200ms (P2) and of 550-750ms (P3) after face onset. Similar to previous studies using approach-avoidance tasks (van Peer et al., 2007; van Peer et al., 2010; van Peer et al., 2009), the following midline electrodes were included in statistical analyses: Fz, Cz, and Pz.

3.2.9 Statistical Analyses

Due to outlier values of cortisol, one subject (female, warm water condition, high provocation) was excluded from all statistical analyses, resulting in a total number of 71 analyzed participants.

To disentangle general stress effects from specific cortisol effects, subjects of the stress condition (cold water condition of the SECP) were categorized into cortisol responders and nonresponders depending on their cortisol rise following the stressor. Since cortisol peaks in saliva can be expected at about 20 minutes after a stressor (Kirschbaum & Hellhammer, 1989), the difference between cortisol samples C2 and C3 (see Figure 11) was used as an index of cortisol increase following the stressor. By means of a median-split, subjects with an increase of at least 0.79nmol/l of cortisol were classified as cortisol responders, otherwise subjects were classified as nonresponders. This resulted in a mean cortisol increase of 4.14nmol/l (equivalent to a rise of 97% in the present sample) for the cortisol responders and a mean cortisol decline of -0.45nmol/l for nonresonders.

Behavioral data. In order to elucidate cortisol effects, a five-way analysis of variance (ANOVA) was conducted for the median of the first reaction time (dependent variable) including the factors emotion (angry vs. happy faces; repeated measures), movement (approach vs. avoidance of the facial expression; repeated measures), stress group (warm water condition, cortisol responder, nonresponder; between-subject), provocation (high vs. low provocation; between-subject), and gender as independent variables.

To investigate the influence of the acute testosterone level as well as of the interaction of acute testosterone and cortisol on approach avoidance motivation, the z-standardized testosterone level of sample TC (after TAP, see Figure 11) was additionally included as a continuous predictor (between-subject) into the analysis (see Aiken & West, 1991). The testosterone scores were z-standardized separately for males and females (as in Mehta & Beer, 2010). Sample TC was chosen because of the time delay of testosterone peaks in saliva. Thus, with this sample we could check for the effects of testosterone levels that include changes due to the stressor.

In order to elucidate the influence of the baseline testosterone and cortisol levels, the z-standardized baseline testosterone-to-cortisol ratio (between-subject) was included as a continuous predictor in a separate ANOVA. To get a more reliable value of baseline cortisol and testosterone, the mean of the two first testosterone samples (TA and TB) and the mean of the three first cortisol samples (C0, C1, and C2) were used for assessing a baseline value of each hormone. The baseline testosterone-to-cortisol ratio score for each subject was calculated as in Hermans, Ramsey & van Honk (2008).

Electrophysiological data. Separate six-way ANOVAs were performed for the P2 (160-200 ms after face onset) and P3 (550-750 ms) components including the factors emotion (angry vs. happy faces; repeated measures), movement (approach vs. avoidance; repeated measures), stress group (warm water condition, nonresponder, cortisol responder; between-subject), provocation (high vs. low provocation; between-subject), gender, and electrode position (Fz, Cz, Pz, repeated measures). For each component, mean average ERP amplitudes were used as the dependent variable.

Since effects of stress and provocation should only be apparent in block 2 (after the experimental manipulation of both), for testing the hypotheses involving cortisol and provocation, separate ANOVAs were conducted for each block. This applies to the analysis of both the behavioral and the electrophysiological data. In the following, only significant effects in block 2 will be reported. Results in block 1 will only be referred to when it is to show that

the relevant effects were solely caused by the experimental manipulation of stress and provocation and that they did not occur beforehand.

Only in case of the manipulation check of the Manikin Task (in order to check, if the expected congruency effect was apparent), an ANOVA including both blocks was conducted.

In case the assumption of sphericity was violated, the degrees of freedom were Huynh-Feldt corrected (Huynh & Feldt, 1976). The statistical significance level alpha was set to 0.05 (two-tailed), and significant results are reported with η^2 (partial eta squared) as the effect size measure. As post-hoc tests, Dunn's Multiple Comparison Procedure or Pearson correlations, respectively, were used. All statistical analyses were conducted by means of SPSS for Windows (Version 17.0, SPSS Inc.).

3.3 Results

3.3.1. Manipulation Checks

3.3.1.1 Stress Induction

To check if the stress induction was successful, an ANOVA was performed including the factors time of cortisol sample (C0, C1, C2, C3, C4, C5; repeated measures) and stress induction (stress / cold water vs. no stress / warm water condition; between-subject) as independent variables and the salivary cortisol concentration as the dependent variable. The main effect of time was significant ($F(5,345) = 3.526$, $p = .028$, $\eta^2 = .049$) and interacted marginally significantly with stress $F(5,345) = 2.891$, $p = .053$, $\eta^2 = .040$).

Since it we expected that about 50% of the stressed participants would show a significant increase of cortisol following the SECP, the stressed subjects were divided into cortisol responders and nonresponders by means of a median split. Again, an ANOVA with the factors time of cortisol sample (C0, C1, C2, C3, C4, C5, repeated measures) and stress group (warm water group, nonresponders, cortisol responders; between-subject) was conducted. The analysis revealed not only a significant main effect of Time ($F(5,340) = 5.312$, $p = .004$, $\eta^2 = .072$), but also a significant interaction of time and stress group ($F(10,340) = 7.969$, $p = .000$, $\eta^2 = .190$), which is displayed in Figure 13. Post-hoc tests revealed that cortisol responders differed significantly from nonresponders and from the warm water group at time points C3, C4, and C5, indicating that the cortisol levels of the responders

were well above those of the nonresponders and of the warm water group after the SECP. To conclude, the stress induction via the SECP was successful.

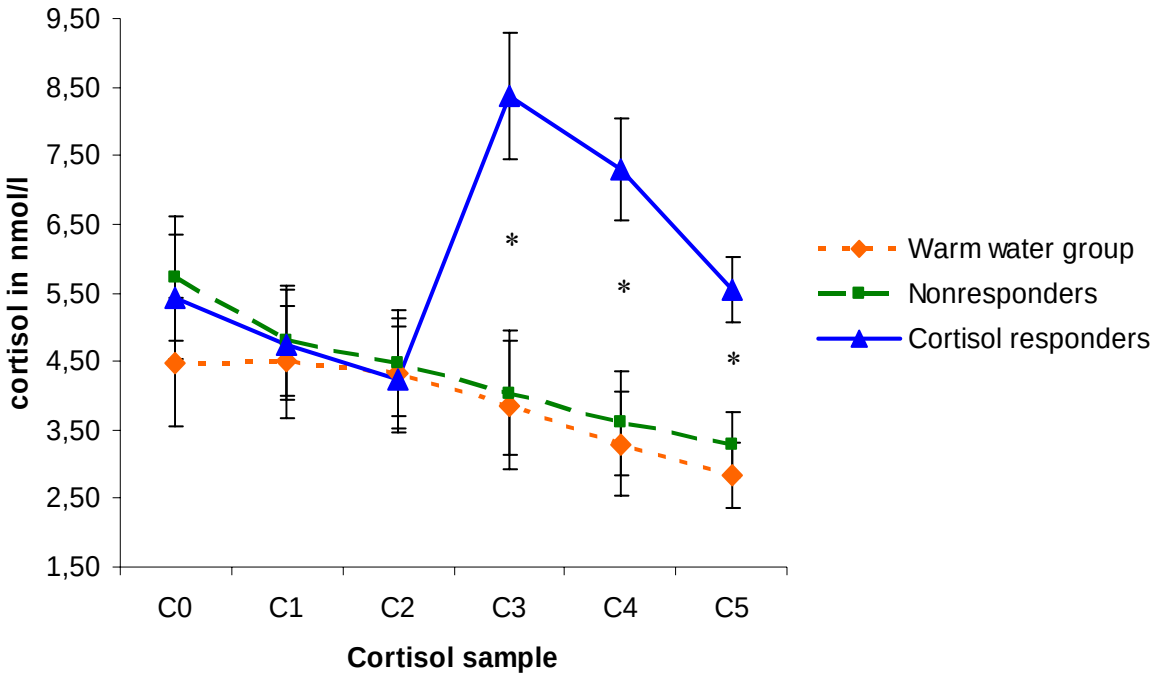

Figure 13 Cortisol levels during the experimental session, separately for the warm water group, cortisol responders and nonresponders. * = significant difference according to post-hoc test.

3.3.1.2 Social Provocation

An ANOVA including the factors block of the TAP (block 1, block 2, block 3; repeated measures) and provocation (high vs. low provocation; between-subject) revealed significant main effects of TAP-Block ($F(2,138) = 17.653$, $p = .000$, $\eta^2 = .204$) and provocation ($F(1,69) = 15.104$, $p = .000$, $\eta^2 = .180$) as well as a significant interaction of both factors ($F(2,138) = 29.658$, $p = .000$, $\eta^2 = .301$). Post-hoc tests demonstrated that the two groups did not differ in the first block, but in the second and third block of the TAP, with the high provocation group behaving more aggressively than the low provocation group (see Figure 14). Thus, the provocation procedure via the TAP did successfully elicit aggressive behavior in the high provocation group.

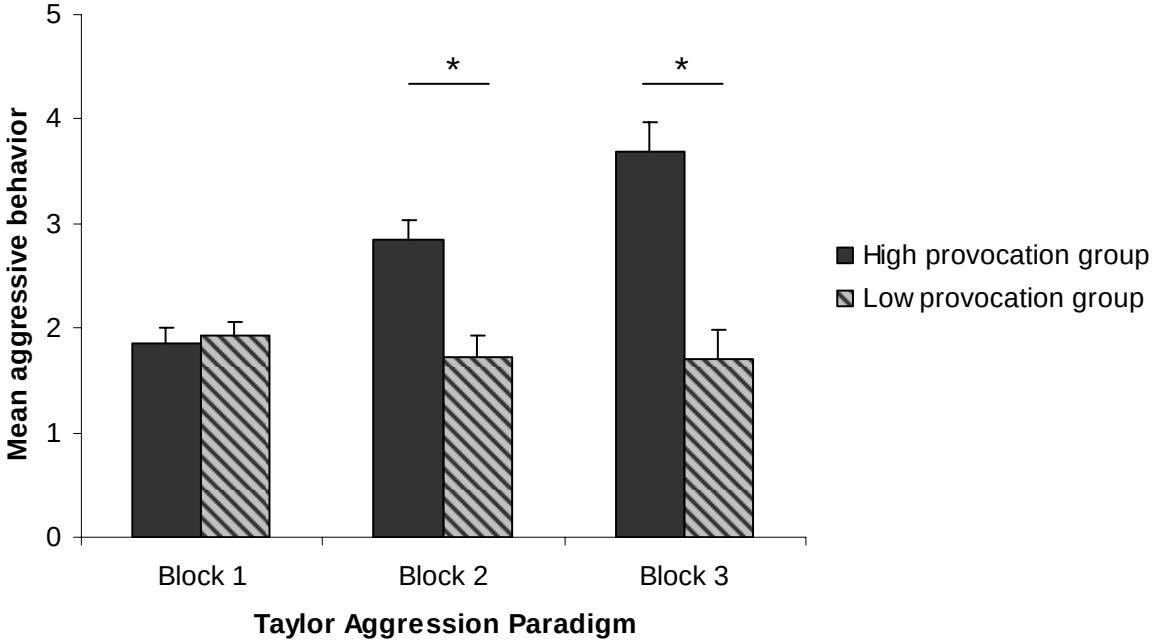

Figure 14 Mean aggressive behavior of the high and low provocation groups during the three blocks of the Taylor Aggression Paradigm. Values are means and SEM. * = significant difference according to post-hoc test.

3.3.1.3 Manikin Task

Only trials with correct responses were analyzed. Since the subjects had to react within a time frame of 1500ms after the onset of the face presentation, no reaction times longer than 1500ms had to be excluded from analysis.

The main effect of emotion ($F(1,59) = 17.545$, $p = .000$, $\eta^2 = .229$) as well as the main effect of movement ($F(1,59) = 107.805$, $p = .000$, $\eta^2 = .646$) reached significance; reaction times in response to happy faces (M = 748.20, SEM = 11.18) were significantly faster than in response to angry faces (M = 759.54, SEM = 10.72). Furthermore, subjects were faster in approaching (M = 732.70, SEM = 11.07) than in avoiding (M = 775.04, SEM = 11.04) the faces.

As expected, these main effects were qualified by a significant interaction of emotion and movement ($F(1,59) = 71.599$, $p = .000$, $\eta^2 = .548$), indicating the congruency effect (see Figure 15). Specifically, reaction times were faster in congruent trials (when having to approach happy faces and to avoid angry faces) than in incongruent trials (when having to avoid happy faces and to approach angry faces). Post-hoc tests revealed that this congruency effect was especially strong for happy faces, with faster reaction times when approaching (M = 694.61, SEM = 11.80) compared to when avoiding (M = 801.79, SEM = 12.25) happy

faces. The difference between approaching angry (M = 770.79, SEM = 12.00) and happy (M = 694.61, SEM = 11.80) faces as well as the difference between avoiding angry (M = 748.28, SEM = 11.02) and happy (M = 801.79, SEM = 12.25) faces were significant as well. For angry faces, the difference between approaching and avoiding was not significant.

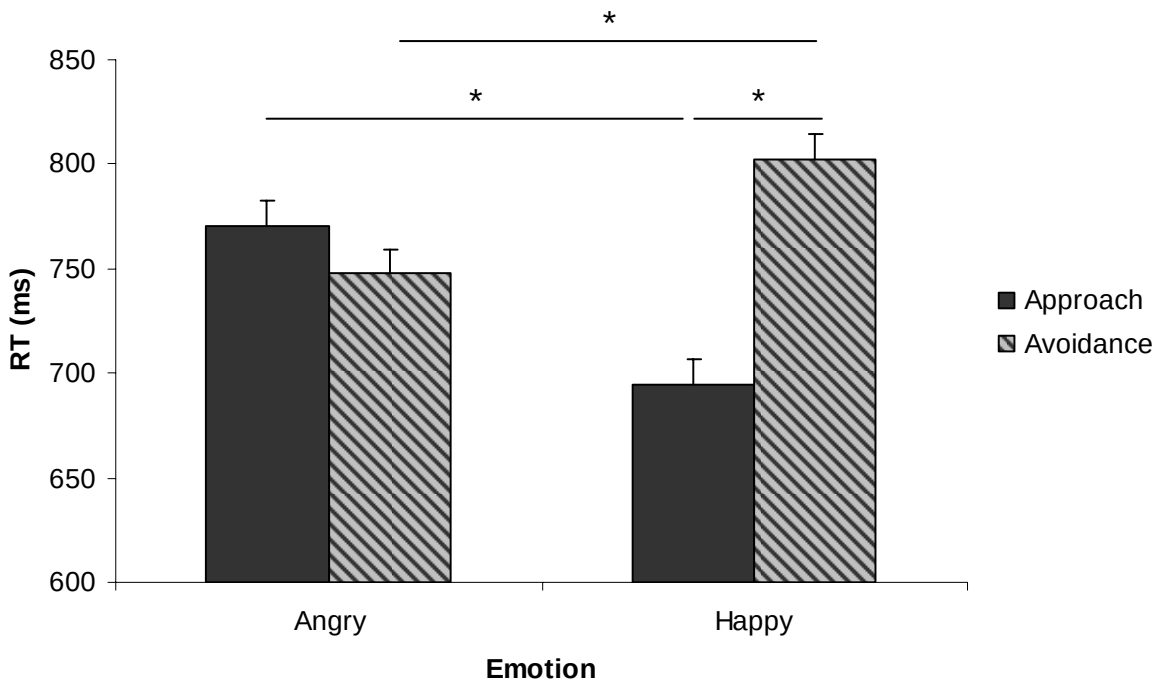

Figure 15 Congruency effect. Reaction times for the approach and avoidance of happy and angry faces, averaged across all groups. Values are means and SEM. * = significant difference according to post-hoc test.

3.3.2 Influence of Acute Stress on Aggressive Behavior

An ANOVA including the factors TAP-Block, provocation, stress group, and gender revealed only a marginally significant interaction of stress group and provocation ($F(2,59) = 2.391$, $p = .100$, $\eta^2 = .075$). As depicted in Figure 16, the provocation effect (= more aggressive behavior after high provocation compared to low provocation) was only apparent for the warm water group and the cortisol responders, whereas for the nonresponders there was no difference between high and low provocation.

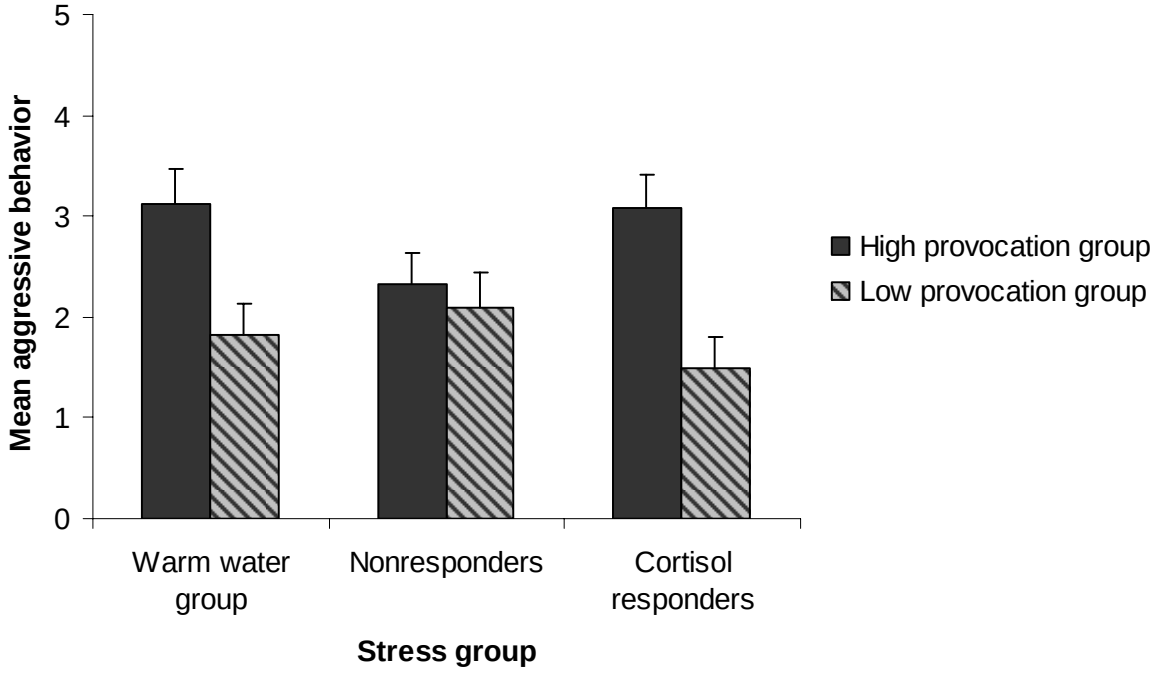

Figure 16 Mean aggressive behavior over all TAP-blocks for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders.

There were no further significant interactions including the factor stress group (all F's < 1).

*3.3.3 Effects of Acute **Cortisol** Levels and Provocation on Approach-Avoidance Motivation*

Relevant for the first hypothesis (i.e., greater approach tendency, particularly in response to angry faces, following acute cortisol increase and subsequent high provocation) was the interaction of movement, emotion, stress group, and provocation, which did not reach significance in block 2 ($F(2,59) < 1$).

However, the interaction of movement, stress group, provocation, and gender was significant in block 2 ($F(2,59) = 5.591$, $p = .006$, $\eta^2 = .159$), but not in block 1 ($F(2,59) < 1$). Subjects in general faster approached than avoided the faces. However, in men this difference was diminished when they got stressed *and* highly provoked (see Figure 17a). Descriptively, highly provoked male cortisol responders had the fastest reactions both in approach and in avoidance trials. In women, on the other hand, the difference between approach and avoidance was only significant in highly provoked nonresponders who were particularly fast in approaching the faces, whereas all other groups showed similar reaction times in approach and avoidance trials (see Figure 17b). Females in general had longer reaction times compared to males ($F(1,59) = 8.827$, $p = .004$, $\eta^2 = .130$).

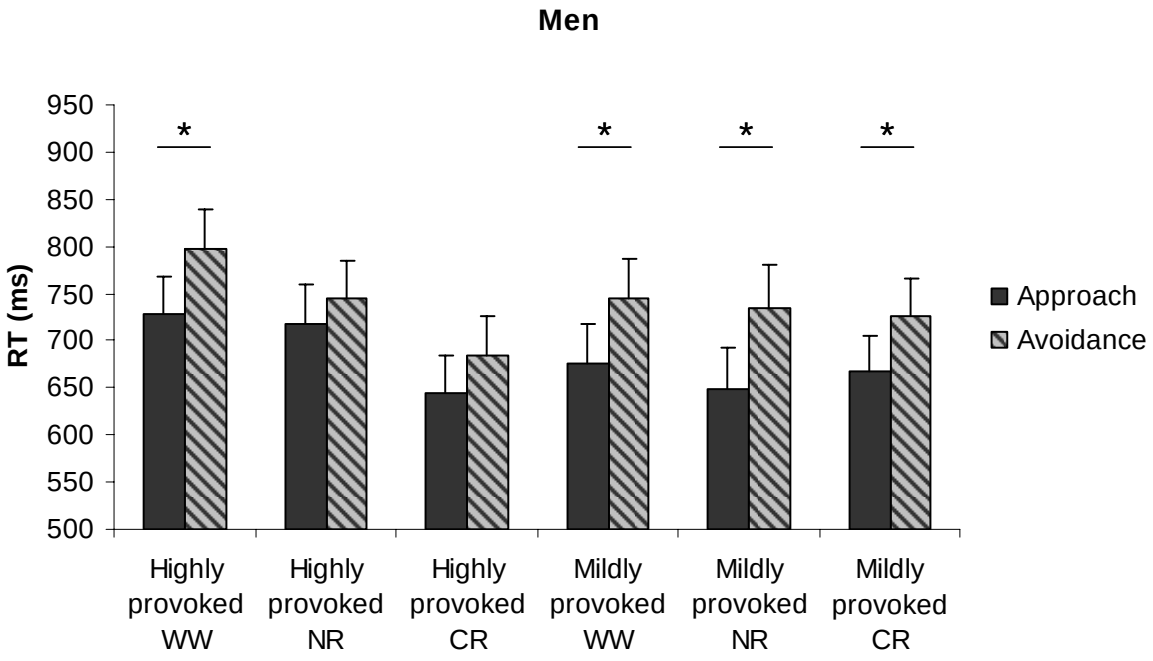

Figure 17a) Reaction times in approach and avoidance trials for *male* cortisol responders, nonresponders, and the warm water group in the high and low provocation groups. Values are means and SEM. WW = Warm water group, NR = nonresponders, CR = cortisol responders. * = significant difference according to post-hoc test.

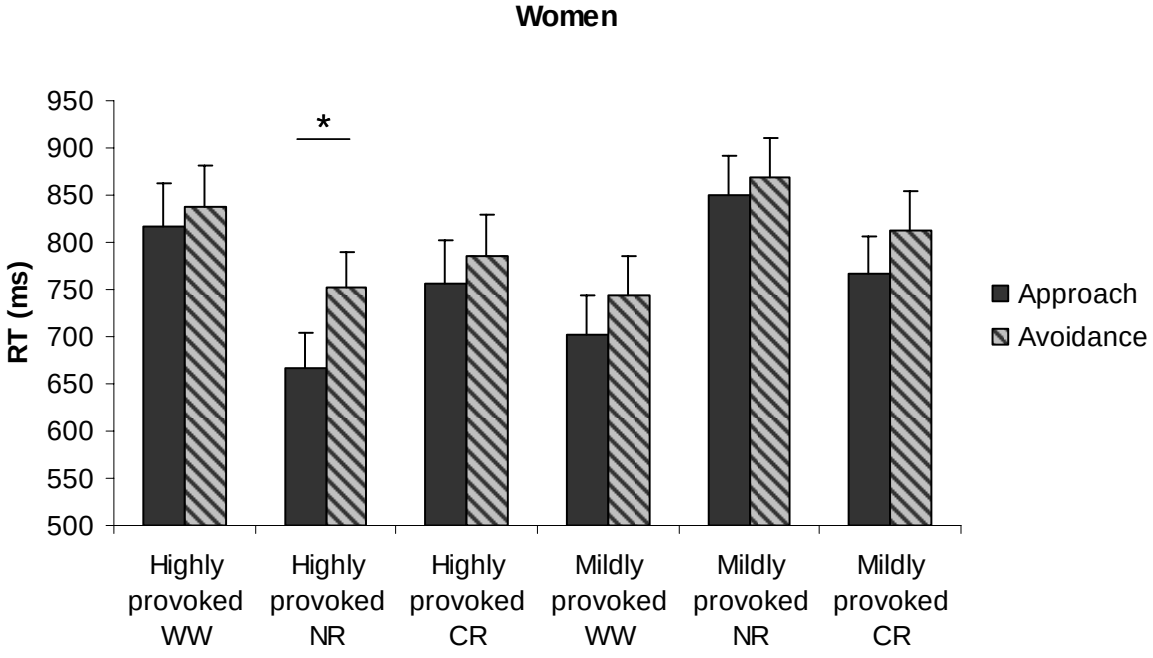

Figure 17b) Reaction times in approach and avoidance trials for *female* cortisol responders, nonresponders, and the warm water group in the high and low provocation groups. Values are means and SEM. WW = Warm water group, NR = nonresponders, CR = cortisol responders. * = significant difference according to post-hoc test.

*3.3.4 Interaction of **Acute Cortisol** and **Testosterone** Levels on Approach-Avoidance Motivation following Provocation*

In order to investigate the influence of the interaction of acute levels of cortisol *and* testosterone on approach-avoidance motivation following provocation, the z-standardized testosterone level (standardized separately for men and women) after stress induction and provocation (Testosterone sample TC) was included in the analysis as a continuous predictor.

The ANOVA revealed that the congruency effect was qualified by the interaction of emotion, movement, provocation, stress group, and testosterone level, which was not significant for block 1 ($F(2,47) = 1.463$, $p = .242$), but for block 2 ($F(2,47) = 3.855$, $p = .028$, $\eta^2 = .141$). Post-hoc Pearson correlations for this interaction in block 2 revealed that groups differed significantly in their relationship between the testosterone level and reaction times, with partially contrary directions of this association (see Table 1).

Table 1 Pearson correlations r between the z-standardized testosterone level TC and the reaction times for the approach and avoidance of happy and angry faces.

		Warm water group		Nonresponders		Cortisol responders	
		High provocation	Low provocation	High provocation	Low provocation	High provocation	Low provocation
Angry	Approach	.63	-.28	.30	.27	**-.45**	-.05
	Avoidance	.38	-.25	.26	.56	-.03	.09
Happy	Approach	.59	-.29	.22	.50	.06	.01
	Avoidance	.33	-.32	.33	.17	**-.41**	.26

More precisely, there were substantial *negative* correlations for highly provoked *cortisol responders*, when they had to approach angry faces and to avoid happy faces, whereas there were no or only small correlations in mildly provoked cortisol responders. Concerning the congruent conditions (avoiding of angry faces and approaching happy faces), all correlations were around zero in cortisol responders, regardless of the degree of provocation.

Nonresponders, on the other hand, showed *positive* correlations in all conditions, but these were strongest in congruent conditions and when they were only mildly provoked.

In case of the *warm water group*, there were moderate to strong *positive* correlations in the highly provoked subjects, with strongest correlations in approach trials. On the other hand, in the mildly provoked warm water group there were relatively small negative correlations for all conditions. This interaction of emotion, movement, provocation, stress group, and testosterone level is displayed in Figures 18a)-d).

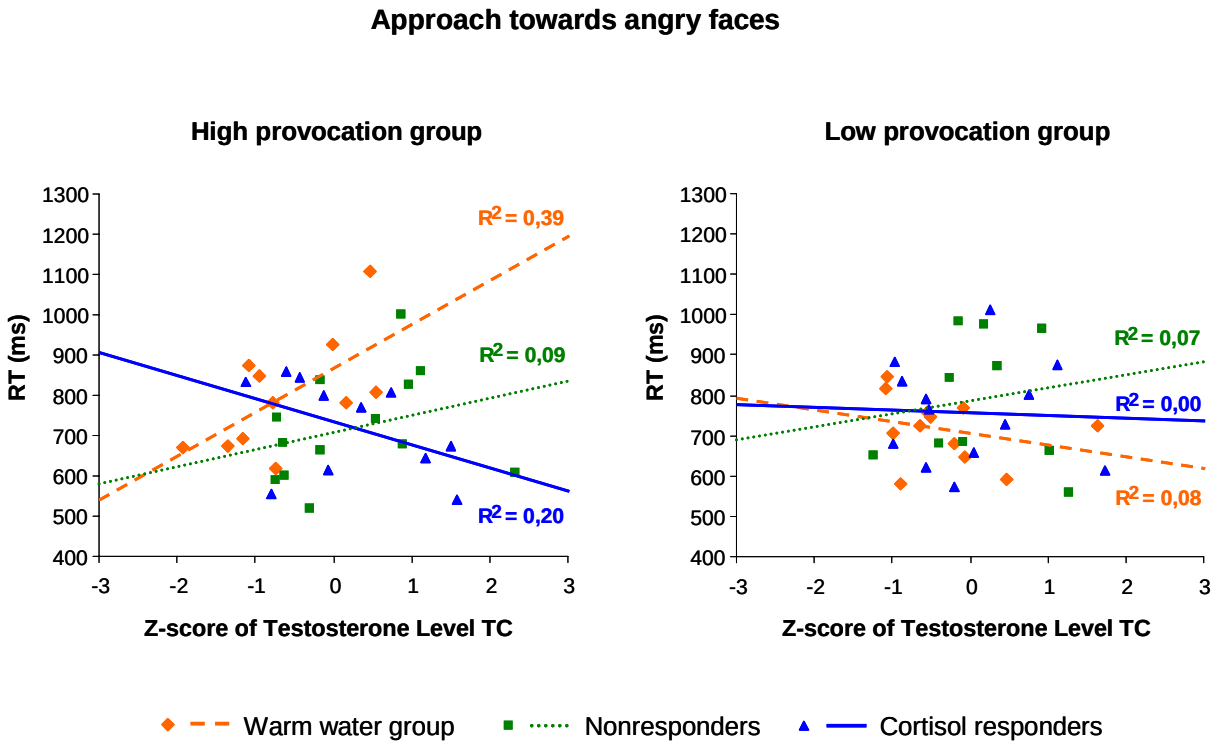

Figure 18a) Post-hoc Pearson correlations between the z-standardized testosterone level TC and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *approach angry* faces.

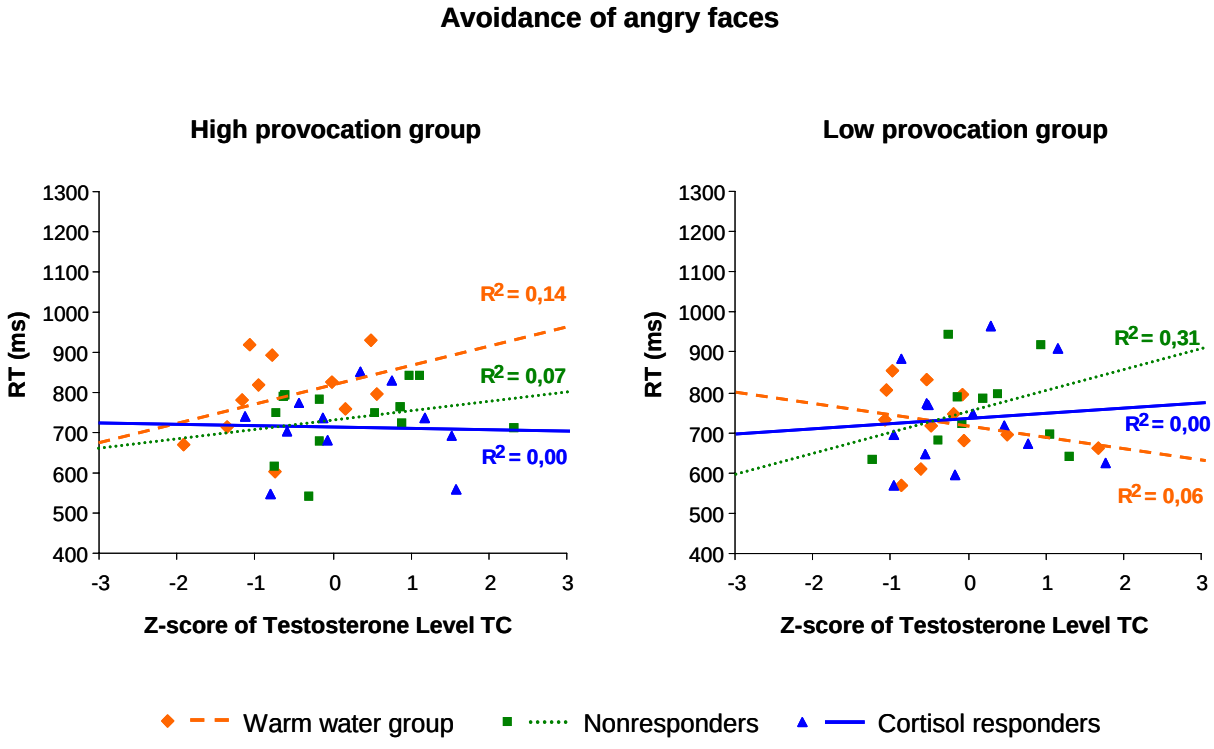

Figure 18b) Post-hoc Pearson correlations between the z-standardized testosterone level TC and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *avoid angry* faces.

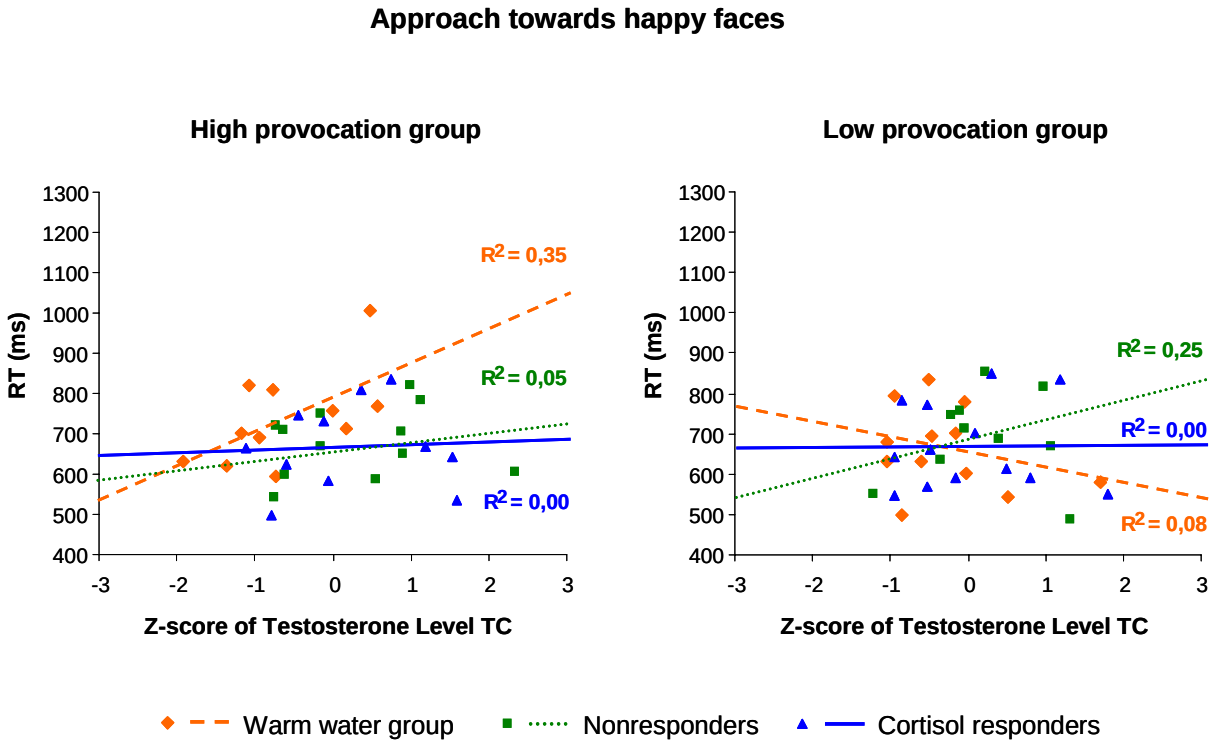

Figure 18c) Post-hoc Pearson correlations between the z-standardized testosterone level TC and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *approach happy* faces.

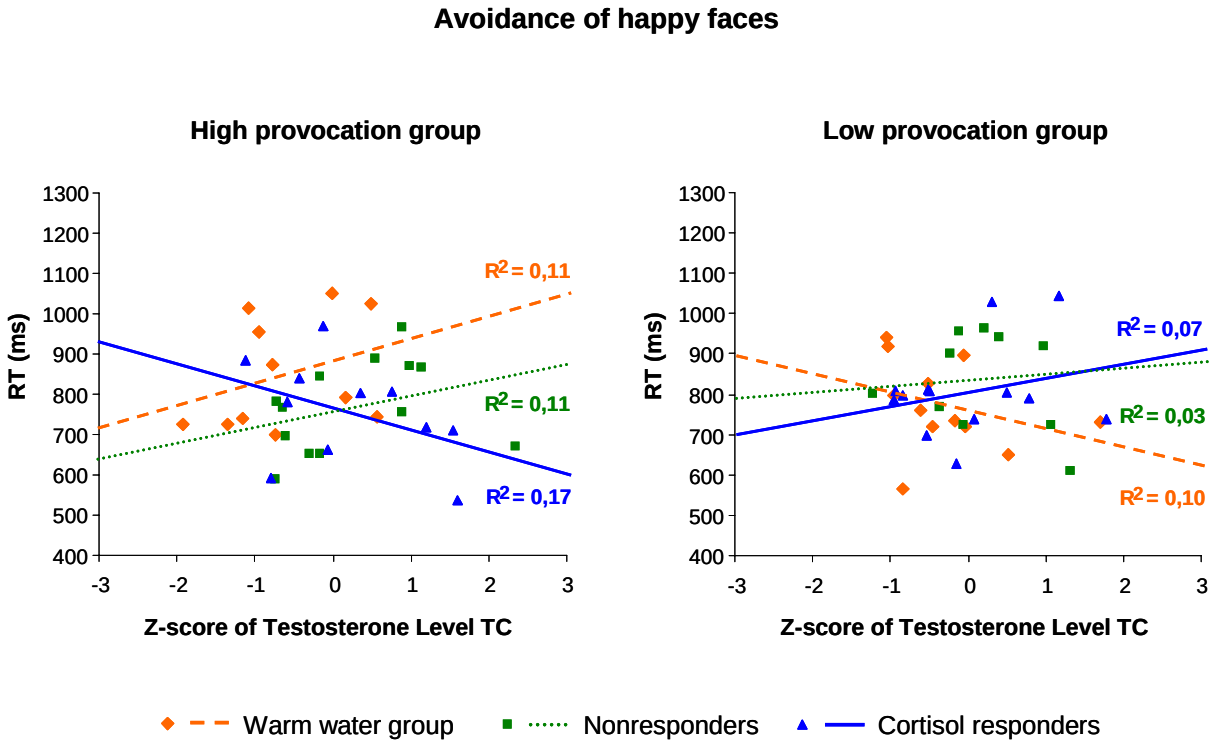

Figure 18d) Post-hoc Pearson correlations between the z-standardized testosterone level TC and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *avoid happy* faces.

Taken together, the higher the testosterone levels in highly provoked cortisol responders, the faster were the reaction times when they had to approach angry faces and avoid happy faces. In other words, the combination of increased cortisol and increased testosterone levels led to a faster approach towards angry faces and a faster avoidance of happy faces, in case of previous high provocation. On the other hand, within the non-stressed warm water group, the higher the testosterone levels, the larger were the reaction times after high provocation, especially in approach trials. In nonresponders, however, the pattern "the higher the testosterone levels, the higher the reaction times" was only apparent for congruent conditions and if they were only mildly provoked.

Furthermore, in block 2, the acute testosterone level interacted significantly with movement and gender ($F(1,47) = 5.411$, $p = .024$, $\eta^2 = .103$) as well as with movement and stress group ($F(2,47) = 5.575$, $p = .007$, $\eta^2 = .192$). Both interactions were further qualified by a four way interaction including testosterone level, movement and both gender and stress group ($F(2,47) = 6.105$, $p = .004$, $\eta^2 = .206$). However, the latter interaction was also significant in block 1 ($F(2,47) = 5.103$, $p = .010$, $\eta^2 = .178$), and therefore will not be discussed any further.[5]

*3.3.5 Influence of **Baseline Testosterone-to-Cortisol Ratio** on Approach-Avoidance Motivation following Stress and Provocation*

In order to analyze the effects of the individual baseline testosterone-to-cortisol ratio (T-C-Ratio) on approach-avoidance motivation after being stressed and provoked, this z-standardized ratio was included in the analysis as a between-subject continuous predictor.

The main effect of T-C-Ratio ($F(1,47) = 5.471$, $p = .024$, $\eta^2 = .104$) was significant, but it was further qualified by several interactions. The two interactions of the highest order were the interaction of provocation, stress group, movement, gender, and T-C-Ratio ($F(2,47) = 4.990$, $p = .011$, $\eta^2 = .175$), on the one hand, and the interaction of the congruency effect (emotion by movement) with provocation, stress group, T-C-Ratio ($F(2,47) = 3.692$, $p = .032$, $\eta^2 = .136$), on the other hand. Neither of those interactions reached significance in block 1 ($Fs < 1$). Regarding the hypotheses presented above, the crucial interaction was the latter one.

[5] There were also significant effects in block 2 that did not include the acute testosterone level. The interaction of the highest order was a four way interaction including movement, gender, stress group, and provocation ($F(2,47) = 5.843$, $p = .005$, $\eta^2 = .199$), which was not significant for block 1 ($F(2,47) = 1.383$, $p = .261$). This interaction had a pattern very similar to the interaction using the same factors in the ANOVA without testosterone level as a continuous predictor (see section *3.3.3*).

Similar to the results involving the acute testosterone levels TC following the TAP, post-hoc correlations between the T-C-Ratio at baseline and reaction times in block 2 revealed *negative* relationships in highly provoked *cortisol responders* for incongruent conditions, but correlations around zero for the congruent conditions (see Table 2). On the other hand, highly provoked *nonresponders* showed *positive* correlations in congruent as well as in incongruent conditions. Both nonresponders and cortisol responders had negative correlations in all conditions, when they were only mildly provoked previously. Within the *warm water group*, however, there were only very small correlations in all conditions.

Table 2 Pearson correlations r between the z-standardized testosterone-to-cortisol ratio at baseline and the reaction times for the approach and avoidance of happy and angry faces.

		Warm water group		Nonresponders		Cortisol responders	
		High provocation	Low provocation	High provocation	Low provocation	High provocation	Low provocation
Angry	Approach	.06	.09	.45	-.62	**-.39**	-.34
	Avoidance	.22	.14	.29	-.32	.03	-.20
Happy	Approach	.11	.01	.43	-.53	.09	-.21
	Avoidance	.23	.10	.45	-.59	**-.37**	-.15

Scatterplots of these correlations between the z-standardized T-C-Ratio at baseline and reaction times in block 2 are displayed in Figures 19a)-d).

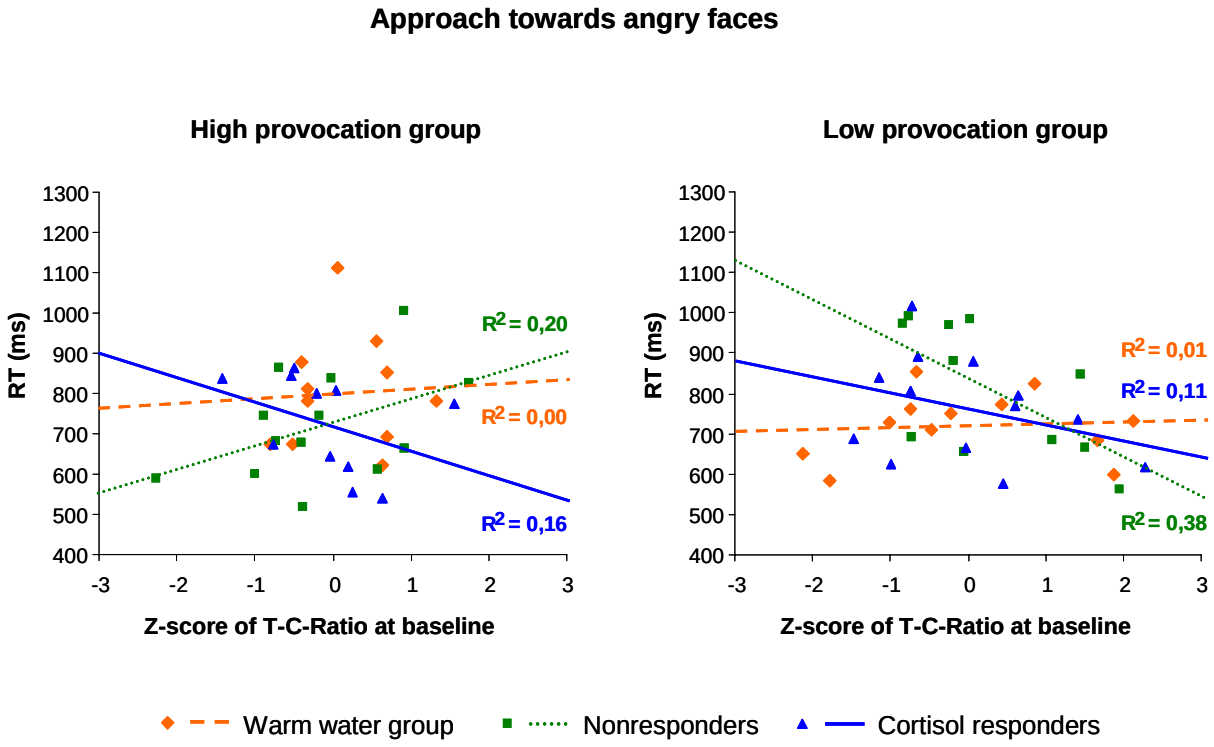

Figure 19a) Post-hoc Pearson correlations between the z-standardized testosterone-to-cortisol ratio at baseline and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *approach angry* faces.

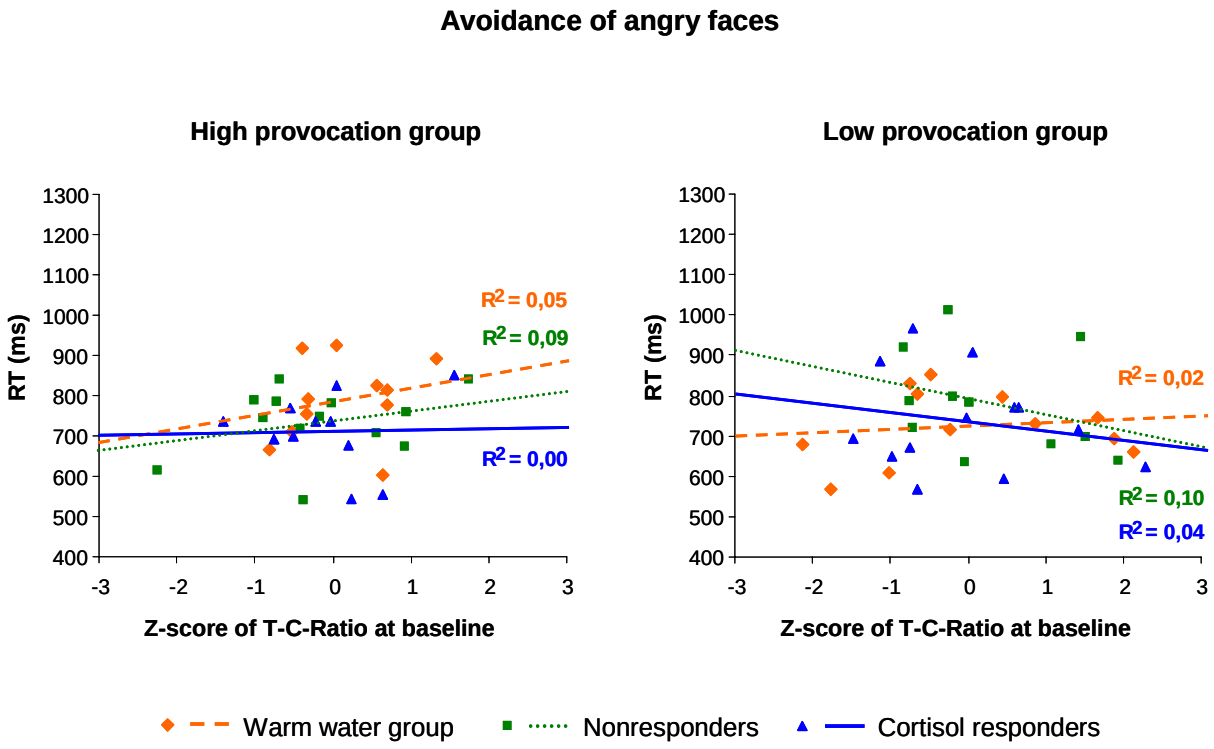

Figure 19b) Post-hoc Pearson correlations between the z-standardized testosterone-to-cortisol ratio at baseline and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *avoid angry* faces.

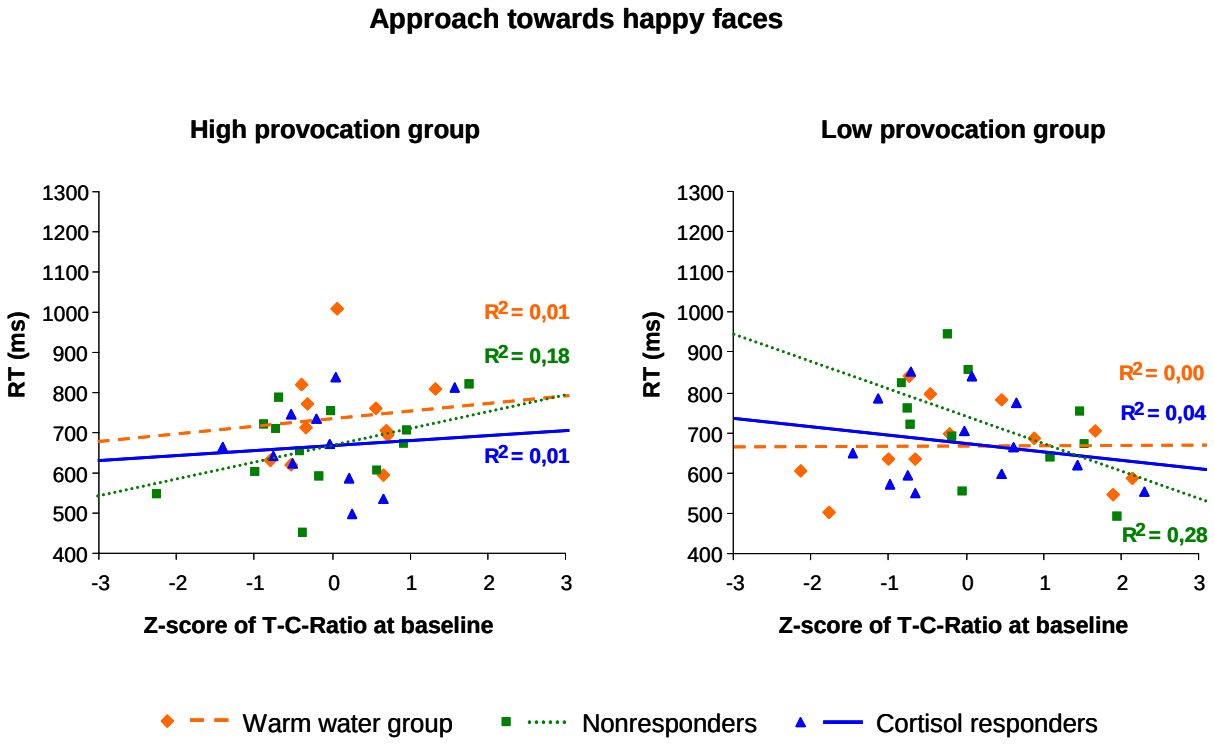

Figure 19c) Post-hoc Pearson correlations between the z-standardized testosterone-to-cortisol ratio at baseline and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *approach happy* faces.

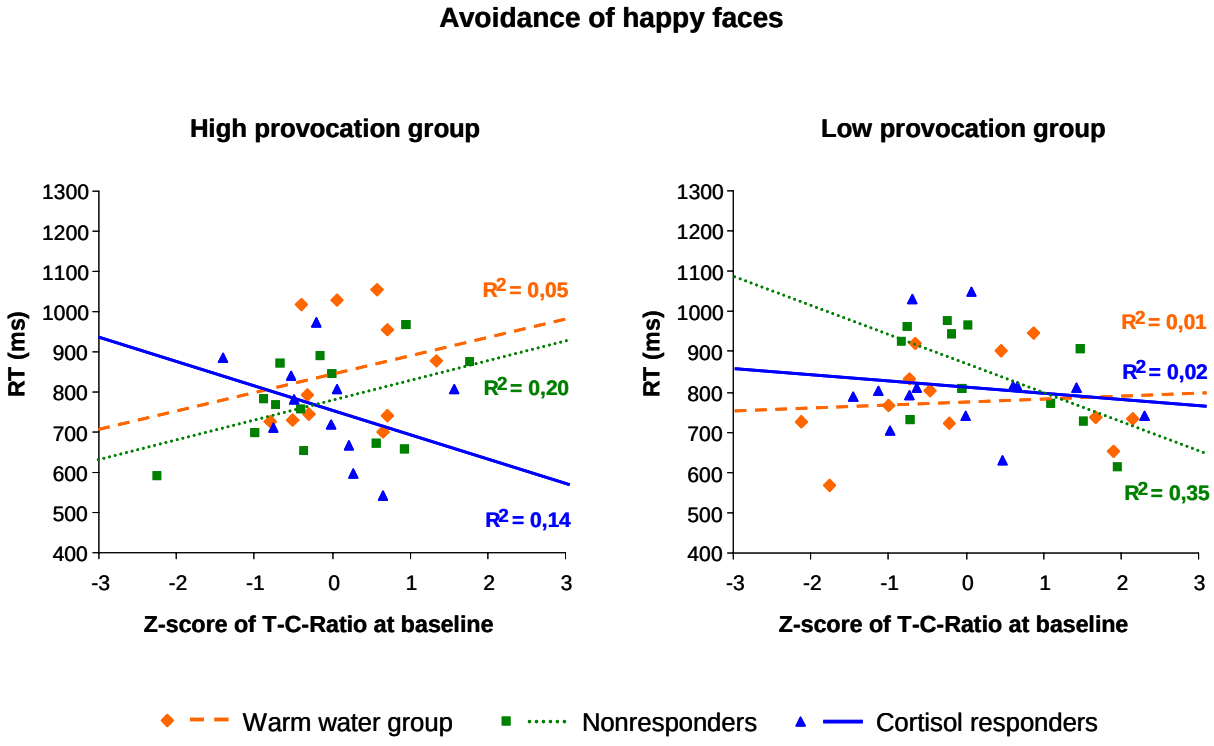

Figure 19d) Post-hoc Pearson correlations between the z-standardized testosterone-to-cortisol ratio at baseline and reaction times, shown separately for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders when they had to *avoid happy* faces.

In summary, the T-C-Ratio at baseline had strong effects on the stressed participants, but not on the non-stressed control group: In highly provoked cortisol responders, the higher the T-C-Ratio at baseline, the faster were the reaction times when they had to approach angry faces or to avoid happy faces, respectively. Among nonresponders, the relationships between reaction times and T-C-Ratio were reversed for the high provocation group (positive correlations) and the low provocation group (negative correlations), regardless of the (in)congruency of trials.

3.3.6 Electrophysiological Data

Overall, the general morphology of the ERP waveforms at midline electrodes included an early negative peak at 130ms (N1), followed by a positive peak at 180ms (P2), a second negative peak at 280ms (N2), and a late positive peak at 650ms (P3) after face onset (0ms). Since the electrode position of Cz is most representative for Fz, Cz, and Pz, the general morphology at Cz is displayed in Figure 20.

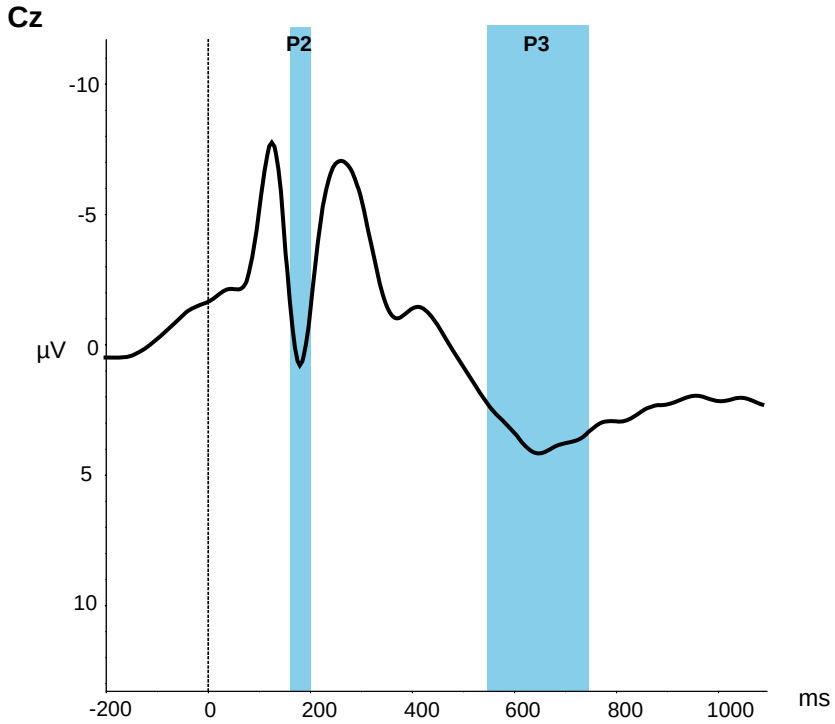

Figure 20 General morphology of ERP waveforms at Cz in block 2 averaged across all experimental conditions. Face onset was at 0ms.

3.3.6.1 The **P2** Amplitude (160-200ms)

Overall, the P2 amplitude had its maximum at the parietal electrode site, resulting in a significant main effect of electrode position ($F(2,118) = 19.794$, $p = .000$, $\eta^2 = .251$), which interacted (marginally) significantly with emotion ($F(2,118) = 3.257$, $p = .051$, $\eta^2 = .052$). Specifically, the P2 amplitude was smaller in response to angry faces compared to happy faces, and this was only apparent for Cz and Pz, but not for Fz. Overall, the main effect of emotion (smaller P2 amplitude in response to angry faces compared to happy faces) reached significance as well ($F(1,59) = 7.555$, $p = .008$, $\eta^2 = .114$).

The main effect of provocation was significant as well ($F(1,59) = 4.638$, $p = .035$, $\eta^2 = .073$), with the high provocation group having an enhanced P2 amplitude compared to the low provocation group. This main effect was qualified by electrode position ($F(2,118) = 4.751$, $p = .018$, $\eta^2 = .075$), indicating that the effect of provocation was largest at the parietal electrode site. This interaction was not significant in block 1 ($F(2,118) < 1$). ERPs at each electrode site for the high and low provocation groups and difference maps for the P2 amplitude in block 2 are displayed in Figure 21 and 22.

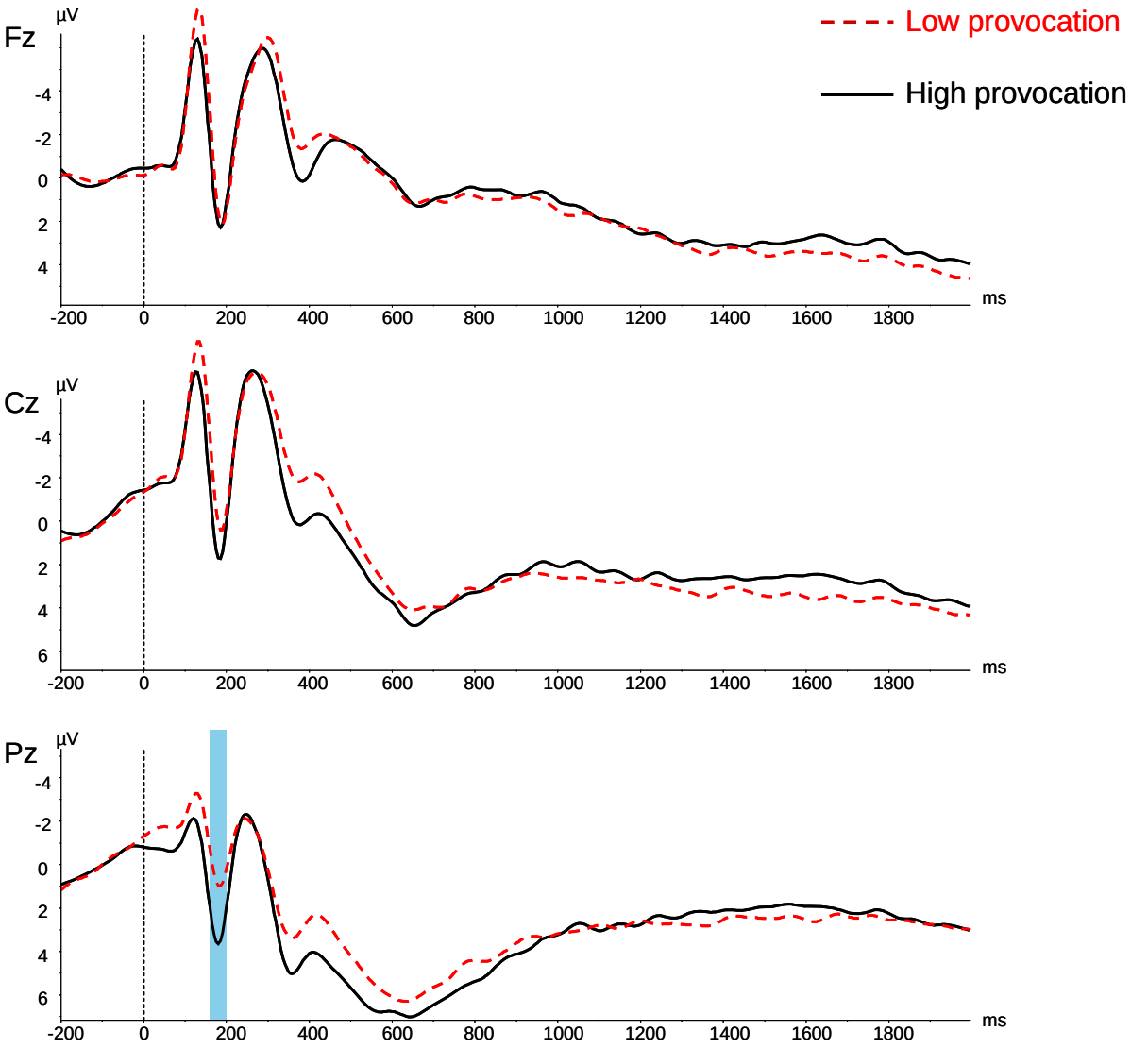

Figure 21 Grand average ERP waveforms for the time domain of the P2 (160-200 ms) in block 2 for the high and low provocation groups, averaged over both facial emotions (angry, happy), the two movements (approach, avoidance), the three stress groups (warm water condition, nonresponders, cortisol responders), and gender of the subject.

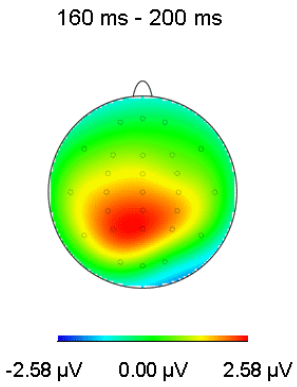

Figure 22 Difference map of high provocation group minus low provocation group.

Furthermore, there was a significant interaction of electrode position and stress group ($F(4,118) = 2.889$, $p = .039$, $\eta^2 = .089$). Post-hoc tests showed that at Fz and Cz, the stressed subjects had a reduced P2 amplitude compared to the non-stressed warm water condition. This reduction was most pronounced for cortisol responders, who showed the smallest P2 amplitude. On the other hand, there were no significant stress group differences at Pz. This interaction is displayed in Figures 23 (ERP waveforms) and 24 (difference maps). This interaction did not reach significance in block 1 ($F(4,118) = 1.636$, $p = .187$).

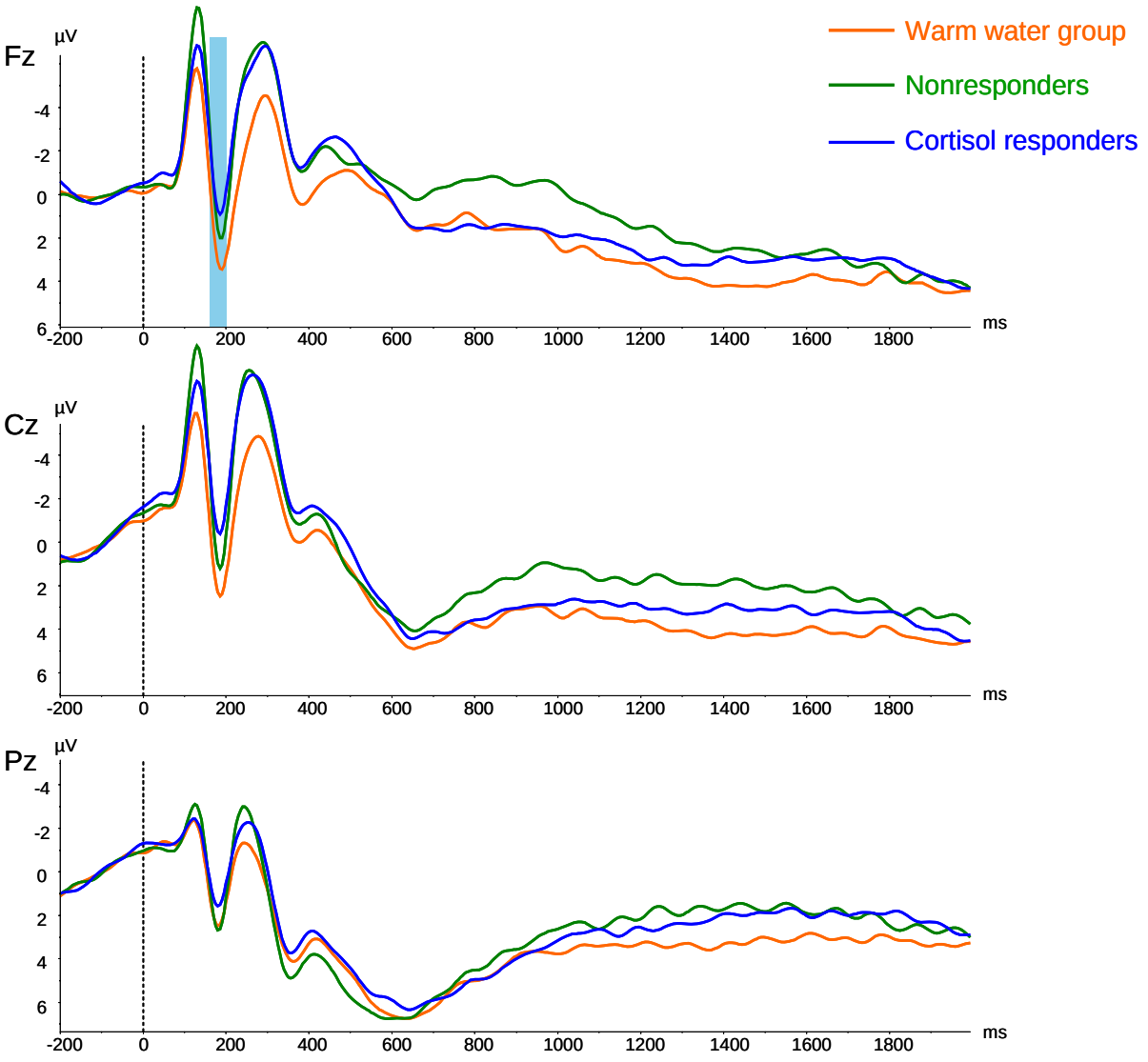

Figure 23 Grand average ERP waveforms for the time domain of the P2 (160-200 ms) in block 2 for the warm water group, the nonresponders, and the cortisol responders, averaged over both facial emotions (angry, happy), the two movements (approach, avoidance), the two provocation groups (high, low provocation), and gender of the subject.

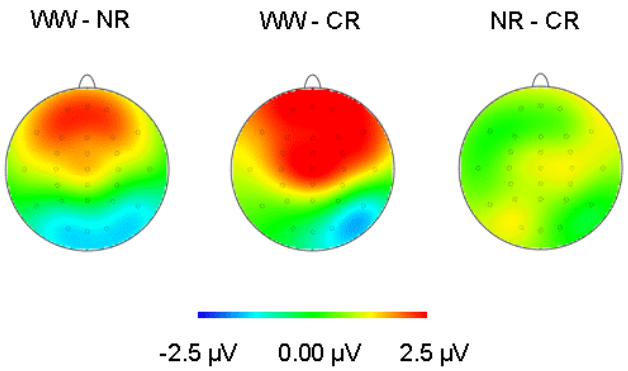

Figure 24 Difference maps of the three stress conditions for the time domain of the P2 (160-200ms); Left: warm water group vs. nonresponders, middle: warm water vs. cortisol responders, right: nonresponders vs. cortisol responders.

Both interactions were further qualified by a significant interaction of electrode position, stress group, provocation, and gender ($F(4,118) = 3.187$, $p = .026$, $\eta^2 = .098$). As can be seen in Figure 25a), women showed the provocation effect - larger P2 amplitude after high provocation compared to low provocation - regardless of the stress group. However, men only showed the provocation effect if they were not stressed (warm water condition) or if they were nonresponders - although the differences were smaller compared to women (see Figure 25b). In the case of male cortisol responders, the provocation effect was reversed: cortisol responders had a *smaller* P2 amplitude after high provocation compared to when they were only mildly provoked. This effect was located at frontal (and central) electrode positions but not at the parietal site. Again, this interaction was not significant for block 1 ($F(4,118) = 2.271$, $p = .085$).

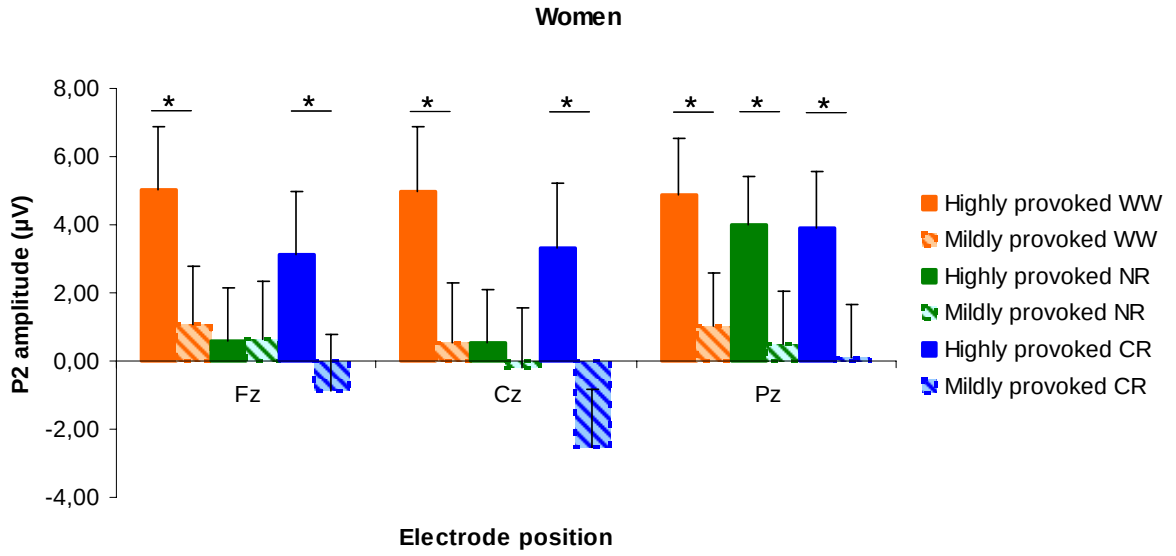

Figure 25a) Mean P2 amplitude (µV) at Fz, Cz, and Pz, for the highly and mildly provoked *female* warm water groups, nonresponders and cortisol responders. Values are means and SEM. * = significant difference according to post-hoc test.

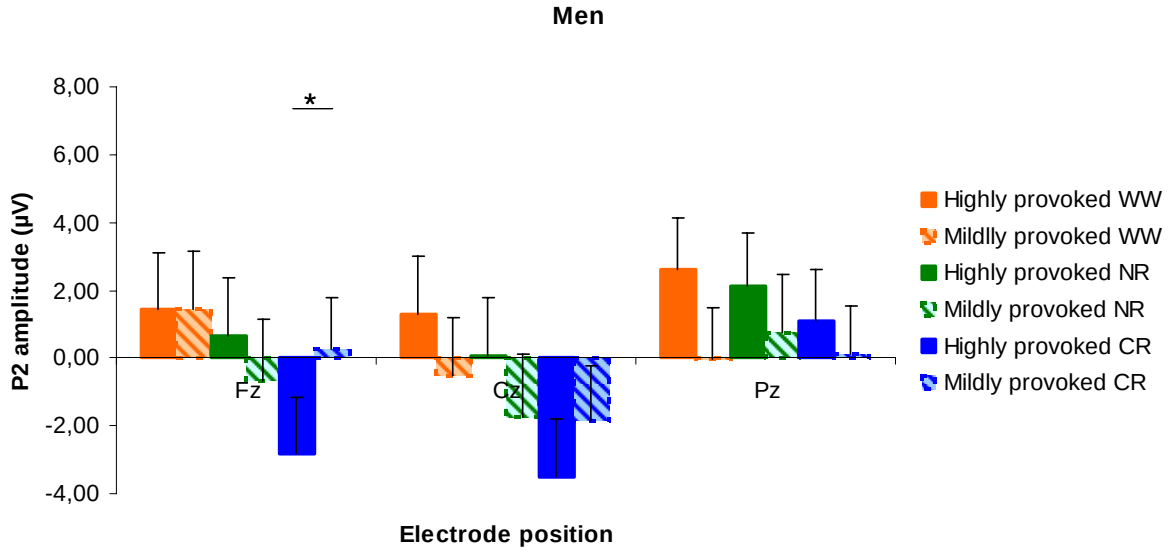

Figure 25b) Mean P2 amplitude (µV) at Fz, Cz, and Pz, for the highly and mildly provoked *male* warm water groups, nonresponders and cortisol responders. Values are means and SEM. * = significant difference according to post-hoc test.

Additionally, the interaction of emotion, provocation and gender reached significance ($F(1,59) = 4.702$, $p = .034$, $\eta^2 = .074$). Post-hoc tests indicated that the provocation effect was more pronounced in women than in men and that for men the influence of provocation on the P2 amplitude was only significant for happy faces, not for angry faces. Regarding block 1, this interaction did not reach significance ($F(1,59) < 1$).

Finally, stress group interacted significantly with movement, gender, and electrode position ($F(4,118) = 3.429$, $p = .011$, $\eta^2 = .104$). However, post-hoc tests did not find any approach-avoidance differences, neither in men nor in women, at any electrode site. Moreover, this interaction nearly reached significance in block 1 ($F(4,118) = 2.427$, $p = .052$, $\eta^2 = .076$).

No further effects were significant in block 2 (all F's < 2.369, all p's > .063).

3.3.6.2 The **P3** Amplitude (550-750ms)

Overall, the P3 amplitude had a parietal distribution, resulting in a main effect of electrode position ($F(2,118) = 107.324$, $p = .000$, $\eta^2 = .645$). Moreover, there was a significant main effect of emotion ($F(1,59) = 4.908$, $p = .031$, $\eta^2 = .077$) with a greater P3 amplitude in response to angry faces compared to happy faces. The interaction of electrode position and emotion was significant as well ($F(2,118) = 3.936$, $p = .022$, $\eta^2 = .063$). Furthermore, movement interacted significantly with electrode position ($F(2,118) = 9.441$, $p = .000$, $\eta^2 = .138$). This interaction was further qualified by a three-way interaction of electrode position, emotion and movement ($F(2,118) = 3.926$, $p = .022$, $\eta^2 = .062$). As a result of post-hoc tests, the difference between the approach and avoidance of faces (greater P3 amplitude in avoidance trials than in approach trials) was only significant in response to angry faces, not in response to happy ones, and this was only apparent at Cz and Pz, but not at Fz.

Concerning the influence of stress induction on the P3 amplitude, there were several significant interactions: First, stress group interacted significantly with electrode position and movement in block 2 ($F(4,118) = 2.499$, $p = .046$, $\eta^2 = .078$), but not in block 1 ($F(4,118) < 1$). Post-hoc tests indicated that in block 2, cortisol responders had a more pronounced P3 amplitude when avoiding the faces than when approaching them. This was especially the case at central and parietal electrode sites. Nonresponders showed the same effect at parietal site, but a reversed pattern at frontal electrode position. In case of the warm water group, the differences between approach and avoidance did not reach significance at any of the electrode positions.

Second, stress group interacted significantly with electrode position and emotion ($F(4,118) = 2.780$, $p = .030$, $\eta^2 = .086$), which was further qualified by a four-way interaction of stress group, electrode position, emotion, and gender ($F(4,118) = 4.515$, $p = .002$, $\eta^2 = .133$). Post-hoc tests indicated that among the men, only the nonresponders showed significantly greater P3 amplitudes at Cz and Pz in response to angry faces compared to happy

faces, whereas among the women - in addition to the differences in nonresponders similar to those of the males- the cortisol responders showed a reversed emotion pattern at Fz, that is a more pronounced P3 amplitude for happy than for angry faces. This interaction was not significant in block 1 ($F(4,118) < 1$).

Third, in block 2, stress group also interacted significantly with emotion and movement ($F(2,59) = 3.520$, $p = .036$, $\eta^2 = .107$), which was not the case in block 1 ($F(2,59) = 1.843$, $p = .167$). However, post-hoc tests did not find any significant approach-avoidance differences within any stress group and any emotion.

Fourth, there was a significant interaction of movement, stress group and gender in block 2 ($F(2,59) = 4.186$, $p = .020$, $\eta^2 = .124$), which was not significant in block 1($F(2,59) < 1$). Post-hoc tests, however, did not find any significant differences between the approach and avoidance of faces within female and male stress groups.

Finally, there was a significant interaction of stress group and provocation in block 2 ($F(2,59) = 3.806$, $p = .028$, $\eta^2 = .114$). As displayed in Figures 26 (ERP waveforms) and 27 (difference maps), within the warm water group, there were only very small differences between the high and the low provocation group, whereas according to post-hoc tests, the nonresponders had significantly greater P3 amplitudes after high provocation compared to low provocation. Post-hoc tests also revealed that the cortisol responders showed a reversed pattern: Highly provoked cortisol responders had a significantly reduced P3 amplitude compared to the mildly provoked cortisol responders.[6] This interaction was not significant in block 1 ($F(2,59) = 1.934$, $p = .154$).

[6] Since this interaction did not include electrode position as a factor, separate ANOVAs for each electrode position were conducted in order to demonstrate that this was not an overall effect over the whole scalp. ANOVAs showed that the interaction of stress group, provocation and emotion was only significant for Fz ($F(2,59)=3.294$, $p=.044$, $\eta^2=.100$) but not for Cz ($F(2,59)=1.207$, $p=.306$), and marginally significant at Pz ($F(2,59)=2.464$, $p=.094$).

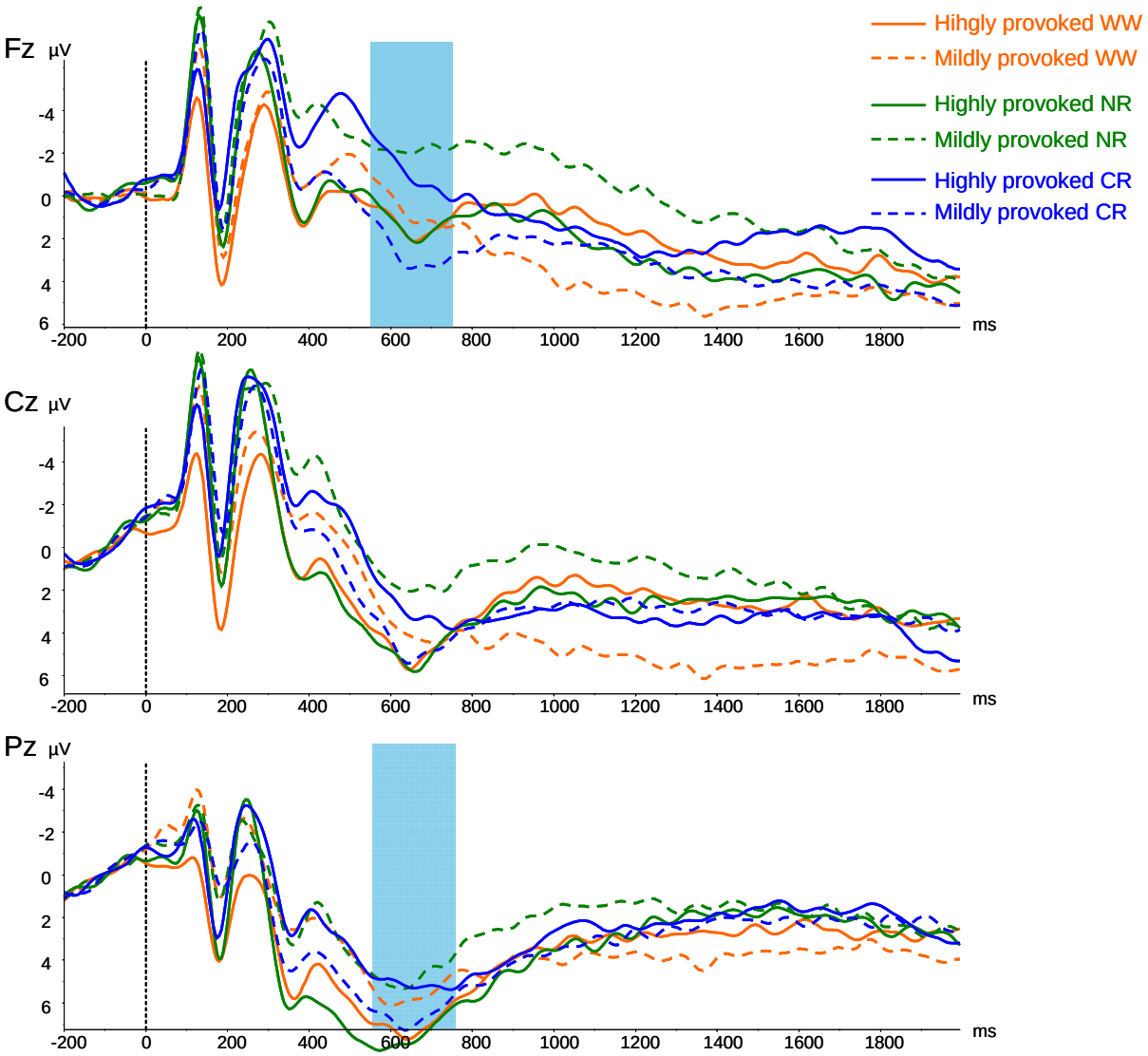

Figure 26 Grand average ERP waveforms for the time domain of the P3 (550-750 ms) in block 2 for the highly and mildly provoked warm water groups, nonresponders, and cortisol responders, averaged over both facial emotions (angry, happy), the two movements (approach, avoidance), and gender of the subject.

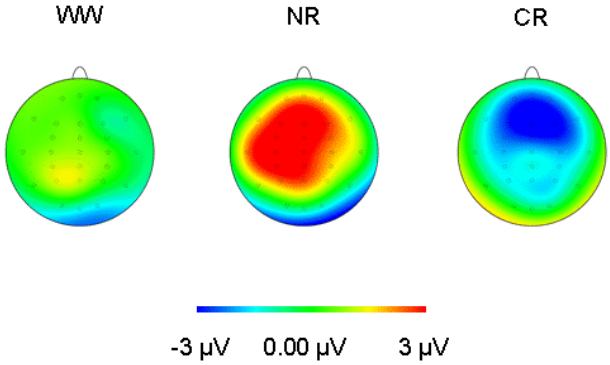

Figure 27 Difference maps (high minus low provocation) for all stress groups. WW = Warm water group, NR = nonresponders, CR = cortisol responders.

This interaction of stress group and provocation was further qualified by a marginally significant three-way interaction involving emotion as a further factor ($F(2,59) = 3.050$, $p = .055$, $\eta^2 = .094$). Both the differences in nonresponders and those in cortisol responders were more pronounced in response to angry faces than to happy faces, as displayed in Figure 28. This interaction was not significant in block 1 ($F(2,59) < 1$).

Figure 28 Mean P3 amplitude (µV) averaged across all electrode sites, for the highly and mildly provoked warm water groups, nonresponders and cortisol responders. Values are means and SEM. * = significant difference according to post-hoc test.

Regarding the mere influence of provocation (without stress effects) on the P3 amplitude, there was a significant interaction of emotion, movement, gender, and provocation in block 2 ($F(1,59) = 10.101$, $p = .002$, $\eta^2 = .146$) but not in block 1 ($F(1,59) < 1$). However, post-hoc tests revealed no significant differences between the approach and avoidance of angry or happy faces within male and female high and low provocation groups.

There were no other significant effects in block 2 (all F's < 2.642, all p's > .08).

3.4 Discussion

The present study sought to elucidate mechanisms underlying the stress-aggression link by investigating approach-avoidance motivation after acute stress induction and subsequent social provocation. As potential moderators of the cortisol effects, the acute testosterone level and the basal testosterone-to-cortisol ratio were included in the study. In order to investigate changes in concomitant brain activity, ERPs were analyzed focusing on two positive components (P2 and P3) which were found to be involved in emotional face processing.

3.4.1 Behavioral Results

The findings show that cortisol alone did not have the expected impact on reaction times during the approach-avoidance task: That is, cortisol did not lead to faster approaches towards angry faces, neither after low nor after high prior provocation. However, in case of prior high provocation, there was a relationship between acute testosterone levels and the tendency to approach angry faces among the cortisol responders (the higher the testosterone level, the faster the reaction times for approaching angry faces) which was neither apparent in nonresponders nor in the non-stressed subjects. In other words, the interaction of acute cortisol increase and relatively high acute testosterone levels resulted in a faster approach reaction towards a social threat. In this case, an angry face might be perceived as a provoking and threatening stimulus even more than under normal circumstances (Dimberg & Öhman, 1996) and therefore trigger an approaching reaction in order to relieve the pressure of the individual's aversive (aggressive) state. This would be in line with ideas by Harmon-Jones, Lueck, Fearn & Harmon-Jones (2006) who have found anger-induced increased activation of the left anterior frontal cortex (interpreted as an index of approach motivation) if an approach-related action (resolving the anger-inducing event) was possible. With regard to Cannon's concept of a rapid fight-or-flight response following a stressor (1914), individuals with acutely increased cortisol and high testosterone levels may develop a rapid fight tendency towards social threat.

The results presented here are in line with previous studies which found evidence for exogenous testosterone administration leading to enhanced autonomic responding and attentional vigilance towards social threat stimuli, such as angry faces (van Honk et al., 2001), and to attenuated autonomic responding and attentional vigilance towards fearful stimuli (Hermans, Putman, Baas, Koppeschaar & van Honk, 2006; van Honk, Peper & Schutter, 2005). These studies taken together indicate an increased approach and a decreased avoidance

motivation in response to social threat due to high levels of acute testosterone (for a recent review, see Montoya, Terburg, Bos & van Honk, 2012). Exogenous cortisol administration, on the other hand, was repeatedly found to decrease fearful withdrawal (see the review by Montoya et al., 2012). Specifically, in a series of various studies, cortisol administration was found to reduce fear in response to threatening stimuli (Oei, Tollenaar, Spinhoven & Elzinga, 2009; Putman, Hermans, Koppeschaar, van Schijndel & van Honk, 2007; van Peer et al., 2010). Furthermore, Böhnke and colleagues (2010b) reported increased aggressive behavior in women during an aggressive encounter following exogenous cortisol administration, which also indicates enhanced approach motivation due to high levels of acute cortisol. Investigating the underlying neural mechanisms of exogenous testosterone and cortisol effects, previous studies found evidence for enhanced neural responses to angry faces after testosterone administration (Hermans et al., 2008) and decreased reactivity of the amygdala in response to fearful and happy faces after cortisol administration (Henckens, van Wingen, Joels & Fernandez, 2010). Montoya et al. (2012) conclude that exogenous testosterone triggers approach motivation and diminishes avoidance motivation by tapping into anger as well as into fear. Exogenous cortisol, on the other hand, is thought to attenuate fearful withdrawal by anxiolytic effects (Montoya et al., 2012). Thus, according to Montoya et al. (2012), despite different underlying mechanisms, both high levels of acute testosterone as well as high levels of acute cortisol promote approach behavior.

Taken together, previous research indicates that both high levels of acute testosterone as well as high levels of acute cortisol facilitate approach tendencies towards social threat, which may thereby lower the threshold for impulsive or aggressive behavior. However, to my knowledge, the study presented here is the first to show *combined* effects of high levels of acute testosterone and of endogenous cortisol elevation on approach tendencies towards social threat stimuli. In this study, high levels of acute testosterone alone (without increased cortisol levels, as it was the case for nonresponders and for the non-stressed warm water group) did not lead to faster approaches but rather resulted in slower approaches towards angry faces. Thus, acutely elevated cortisol levels may have functioned as a physiological trigger for eliciting testosterone effects on approach motivation towards social threat.

Moreover, in the present study high levels of social provocation were necessary for the combined testosterone and cortisol effect, indicating that situational factors also play an important role in generating approach motivation towards social threat. This is in concordance with the General Aggression Model of Anderson & Bushman (2002) which postulates an

interactional influence of both person and situational factors (such as social provocation) on impulsive or aggressive behavior.

To conclude, social provocation and acute stress (resulting in enhanced cortisol levels) in combination with also high acute testosterone levels trigger approach tendencies towards social threat.

In the present study, there was also a significant influence of the baseline testosterone-to-cortisol ratio on approach tendencies towards angry faces. More precisely, highly provoked cortisol responders, but not nonresponders nor the non-stressed subjects, showed a faster approach towards angry faces if the baseline ratio of testosterone to cortisol at the beginning of the experiment was relatively high (high testosterone/low cortisol). According to the testosterone-to-cortisol ratio hypothesis (Terburg et al., 2009; van Honk, Harmon-Jones, Morgan & Schutter, 2010), an imbalance of these two steroid hormones - i.e., high levels of basal testosterone in combination with low levels of basal cortisol - constitutes a risk factor or disposition for reactive aggression. Specifically, according to Terburg and colleagues (2009), a high ratio predisposes to an approach motivation and a reward sensitivity, which lowers the threshold to confront social threat, potentially resulting in impulsive behavior such as aggression. Hermans, Ramsey & van Honk (2008) investigated healthy women contemplating angry and happy facial expressions by means of functional magnetic resonance imaging (fMRI). They found a baseline hormonal profile of high testosterone and low cortisol to be correlated with enhanced activation of sub-cortical structures, such as the amygdala, hypothalamus and brainstem, which (besides the orbitofrontal cortex) are part of the neural circuitry of aggression (for a review of the neural mechanisms of aggression, see Nelson & Trainor, 2007). Terburg and colleagues (2009) conclude that when the testosterone-to-cortisol ratio is high, social threat stimuli such as angry faces activate this neural circuitry to an even greater extent, diminishing the connectivity with the orbitofrontal cortex so that inhibitory control is disturbed.

Similar to the effects of acute testosterone levels, the baseline testosterone-to-cortisol ratio only led to enhanced approach motivation towards social threat stimuli if the endogenous acute cortisol level was increased. Without a previous stressor (as for the warm water group), the testosterone-to-cortisol ratio had no impact on reaction times. In case of sole activation of the sympathetic nervous system without activation of the HPA axis (as for nonresponders), a high testosterone-to-cortisol ratio at baseline was positively related to approach motivation towards angry faces: The higher the ratio, the slower the reactions towards social threat. Thus,

again the acute activation of the HPA axis may have functioned as a precondition for establishing the relationship between a high baseline testosterone-to-cortisol ratio and a stronger approach motivation towards social threat. A possible explanation for the necessity of an acute rise in cortisol in the present study may be as follows: The testosterone-to-cortisol ratio hypothesis of Terburg et al. (2009) primarily refers to (sub-)clinical samples involving individuals with antisocial personality disorder or psychopathy, both characterized by high levels of trait aggressiveness. In contrast, the present sample consisted of healthy young students who may have developed efficient coping mechanisms against stress (as it is virtually omnipresent during academic studies) and who normally do not have problems with impulsive aggression in a clinical manner. Accordingly, the combination of several elicitors (such as social provocation during an aggressive encounter combined with an acute stressor) in addition to a predisposition for impulsive behavior (a high testosterone-to-cortisol ratio) may have been necessary to affect their impulsive behavior in a laboratory setting. In other words, under normal circumstances healthy individuals can control themselves rather well. If the HPA axis is acutely activated by stress, the cortisol rise may function as a trigger that inhibits control processes of the PFC, so that the influence of the basal testosterone-to-cortisol ratio can develop its impact, leading to stronger approach tendencies towards threat.

This would be in line with previous studies, which show that the PFC is a target structure of stress effects. Qin and colleagues (Qin, Hermans, van Marle, Luo & Fernandez, 2009), for instance, investigated healthy females via fMRI during a working memory task while inducing psychological stress. They report a reduced working memory related activation of the dorsolateral prefrontal cortex and less deactivation in the so-called default mode network following stress induction. The authors suggest: "Alongside rapid activation of autonomic and endocrine systems, excessive catecholamines released during acute stress may take prefrontal function 'offline' to facilitate more adaptive and habitual responses like the 'fight-or-flight' response" (Qin et al., 2009, p.30). According to the model of Davidson and colleagues (2000), the PFC is a core neural substrate in the context of aggression. Specifically, the PFC inhibits sub-cortical limbic structures that promote impulsive behavior. Regarding the findings in the present study, acute stress may lead to an attenuation of the PFC activity resulting in a reduced inhibitory control even in healthy individuals, so that the predisposition for impulsive behavior (a high testosterone-to-cortisol ratio) may affect the rapid fight-or-flight response in favor of a fight/approach tendency towards social threat. To validate this assumption, continuative studies could examine the activity of the PFC via fMRI during an approach-avoidance task following acute stress and provocation.

To conclude, not only do acute high levels of testosterone but also a high testosterone-to-cortisol ratio significantly influence approach tendencies towards stimuli that represent a social threat. But they do so only if the HPA axis is acutely activated and a social provocation occurs prior to that. In other words, high levels of endogenous cortisol in combination with high levels of endogenous testosterone or a high baseline testosterone-to-cortisol ratio, respectively, are associated with stronger approach motivation towards social threat following social provocation.

3.4.2 Electrophysiological Results

Besides analyzing behavioral data, another aim of this study was to investigate changes in brain processing associated with approach-avoidance motivation following acute stress and provocation. According to Eimer and Holmes (2007), "[...] ERPs represent a useful tool to study the time course and the functional properties of emotional face processing stages [...]" (p.16). Thus, the combination of behavioral and ERP data is suitable to reveal a more comprehensive understanding of possible changes in social information processing.

Based on previous findings by Bertsch et al. (2009), we expected an enhancement of early (P2) as well as later (P3) positive ERP components in the high provocation group compared to the low provocation group. This provocation effect could be replicated but was further qualified by acute stress, especially in case of an acute activation of the HPA axis. More precisely, concerning the P2 amplitude, the provocation effect (larger P2 after high provocation compared to low provocation) was apparent in females at each electrode site regardless of their stress level. In men, however, the provocation effect was only apparent in those with low cortisol levels, i.e., in the non-stressed warm water group and in the nonresponders. On the contrary, in male cortisol responders, the provocation effect was reversed. Highly provoked male cortisol responders had the smallest P2 amplitude compared to all other groups, especially at frontal and central electrode sites.

The P2 wave is believed to reflect an early, global evaluation of the affective significance of a stimulus and thus seems to be of importance for subsequent approach-avoidance behavior (Schapkin, Gusev & Kuhl, 2000). Hence, in the present study, the reduced P2 amplitude after high provocation in males with high cortisol concentrations may indicate a less precise rapid evaluation of the emotional quality of the faces. This may also explain the fact that the reduction of the P2 was independent of whether the facial expressions were angry

or happy. Moreover, regarding reaction times, compared to the other groups, the highly provoked male cortisol responders reacted particularly fast in response to the faces.

Bertsch et al. (2011) found a diminished early processing bias for angry faces after exogenous cortisol administration, which was independent of provocation. They interpreted this as a potentially decreased activity of the orbitofrontal cortex in response to social threat information due to high levels of cortisol, resulting in a rapid, less accurate processing of facial expressions. Provocation, on the other hand, modulated distinct stages of information processing. Specifically, they found increased potentials of very early (P1) and late (LPP) ERP components, independent of cortisol (Bertsch et al., 2011). In contrast to their findings, the present study revealed a combined effect of provocation and cortisol on one and the same ERP component. Thus, with regard to the interpretation of Bertsch et al. (2011), in the present study the reduced P2 amplitude in males due to activation of the HPA axis and subsequent provocation may indicate a relative attenuation of cognitive/inhibitory control of the orbitofrontal cortex in order to prepare the organism for a rapid fight-or-flight response following a stressor. This may also account for the faster reaction times found in the present study of highly provoked male cortisol responders in approach as well as in avoidance trials.

In females, however, the provocation effect seemed to be stronger than the influence of cortisol, since, regardless of their stress group, the highly provoked women had a more pronounced P2 amplitude compared to mildly provoked ones. An enhancement of the P2 amplitude has been associated with greater deployment of attentional resources (Carretie, Mercado, Tapia & Hinojosa, 2001). Thus, in females provocation may have led to a greater allocation of attention towards socially relevant stimuli, regardless of the emotional quality of the faces.

To summarize, in women, information processing was modulated mostly by the social provocation, in that socially relevant stimuli got more relevant/important. On the other hand, in men, the combination of high cortisol levels and high provocation led to altered social information processing, specifically, to a less precise early evaluation of social stimuli which might result in an initial preparedness for a rapid fight-or-flight response.

Furthermore, stress and provocation also interacted to alter later stages of information processing, namely the P3 amplitude. Specifically, similar to the P2 effects, those individuals with low cortisol levels (warm water group and nonresponders) showed the "normal" effect of provocation, i.e., a more pronounced P3 amplitude after high provocation compared to low provocation. This pattern was reversed in cortisol responders: Highly provoked cortisol

responders had a reduced P3 amplitude compared to mildly provoked cortisol responders. Contrary to the P2 effects, this was true both for males and females and more pronounced for angry than for happy faces.

A reduced P3 amplitude has been repeatedly and consistently associated with proneness to impulsive antisocial behavior. This was attributed to an inefficient allocation of neural resources for the processing of task-relevant information in individuals with antisocial behavior (for a meta-analysis, see Gao & Raine, 2009). The P3 is supposed to develop from an interaction of the frontal cortex and temporal/parietal brain regions. Enhanced P3 amplitudes are believed to indicate a greater allocation of cognitive resources towards important stimuli reflecting an inhibition of concurrent brain activity. The P3 thus seems to indicate attentional processes and memory operations (Polich, 2007). In relation to the results presented here, the attenuated P3 in highly provoked cortisol responders compared to mildly provoked cortisol responders may indicate less available cognitive resources because coping with induced stress and subsequent high levels of social provocation may have claimed resources that were needed for cognitive or inhibitory control. Thus, in case of sole provocation (high provocation group of non-stressed subjects and nonresponders), the relevance of social information stimuli may have been increased, reflected by an enhanced P3 amplitude, especially in response to angry faces. On the other hand, in case of sole activation of the HPA axis (low provocation group of cortisol responders), the cortisol increase may have led to enhanced alertness towards social information, again indexed by a more pronounced P3 amplitude. However, if acute cortisol levels (together with an activation of the sympathetic nervous system) coincide with an additional demanding situational factor such as social provocation, the requirement of resources for coping with this situation may reduce the amount of available task-relevant processing resources, reflected by a reduced P3 amplitude.

To my knowledge, this is the first study which has found an interactional influence of an endogenous cortisol increase and social provocation on early (P2) as well as later (P3) stages of social information processing. While the interaction of cortisol and social provocation significantly influenced the information processing on two distinct stages, the interaction only influenced behavioral approach behavior when testosterone was also taken into account. Since nowadays stress is a rather permanent state that healthy human beings must learn to cope with, it is plausible that a situational challenge such as an interpersonal provocation is able to affect information processing in the brain, while the actual behavior of the individual might still be adequate and socially acceptable.

3.4.3 Strengths and Limitations of the Study

The present study has several strengths: First, a within-subject design with respect to the measurement of approach-avoidance behavior and concomitant brain processing was realized, as both measures were taken before as well as after the experimental manipulation of acute stress and provocation. Hence, significant effects of cortisol and provocation could be ascribed exclusively to the experimental manipulations and not to baseline differences between groups. This sustains the internal validity of the study. Second, the present sample consisted of males and females. Thus, gender differences known to play an important role in the relation of stress and aggression (Verona & Kilmer, 2007; Verona et al., 2007) could be examined, strengthening the external validity of the study. Third, cortisol is only one component of the stress system of an organism (de Kloet et al., 2005). By using the SECP, the cortisol level could be elevated endogenously with concurrent activation of the sympathetic nervous system. Thus, an entirely natural stress response was induced, which may have different influences on aggressive behavior than an artificial high cortisol concentration induced by exogenous administration. This furthermore allowed us to separate specific effects of the hormone cortisol from effects of the sole activation of the sympathetic nervous system. Finally, two hormones that are known to play a crucial role in aggressive behavior, cortisol and testosterone, were measured at several points in time during the experiment. Thus, on the one hand, the success of the stress induction could be validated, and on the other hand, acute testosterone levels as well as the testosterone to cortisol ratio could be taken into account.

Some limitations of the present study should be noted as well: First, only females who took oral contraceptives ('the pill') were included into the study. Hence, the reported results can only be generalized to this specific female population. Moreover, different brands of these oral contraceptives might have distinct influences on endogenous testosterone levels that were not controlled for. Future studies should therefore include women not using oral contraceptives and control for their menstrual cycle. Second, comparing the effects of testosterone in males and females may be problematic because their endogenous testosterone levels are not equal and thus not comparable (Dabbs, 1990). Hence, gender differences concerning acute testosterone levels and the testosterone-to-cortisol ratio within the results should be interpreted with care. However, the focus of the present study did not lie on gender differences but was intended to provide first insights into the interactional influences of cortisol and testosterone on approach-avoidance motivation. Further studies are required in order to replicate the results. Third, although cortisol and testosterone play a dominant role in the release and enhancement of impulsive/aggressive behavior, the HPA axis (cortisol) and

the hypothalamic-pituitary-gonadal axis (testosterone) are only two components of a complex interactive hormonal system in the human body. According to the review of Montoya and colleagues (2012), serotonin is also a key regulator of social aggression. If possible, future research investigating aggressive behavior should include the analysis of testosterone, cortisol, and serotonin concentrations in one and the same study, in order to elucidate complex interactional influences of these different systems. Finally, as a statistical aspect, ANOVAs with continuous predictors (such as acute testosterone levels or testosterone-to-cortisol ratio in the present study) are not suitable to find non-linear relationships but only linear correlations of these continuous predictors with the dependent variable. This should be kept in mind when interpreting the results involving continuous predictors. In this case, however, the graphical displays of the correlations show that the significant relationships were indeed of a linear nature.

3.4.4 Conclusion

The results of the present study provide further insights in psychophysiological mechanisms, such as enhanced approach motivation and altered brain processing that underlie the relationship between stress and aggressive behavior. To my knowledge, this is the first study revealing (1) *interacting* effects of *acute* testosterone and *acute* cortisol levels as well as (2) interacting effects of the baseline testosterone-to-cortisol ratio and acute cortisol levels on approach-avoidance behavior after social provocation. Moreover, to my knowledge, this is also the first study that found *combined* effects of (endogenous) cortisol increase and provocation on early (P2) and on later (P3) stages of social information processing measured with EEG.

To conclude, (1) an acute rise in cortisol in combination with social provocation leads to increased approach motivation towards social threat if the acute testosterone level or the baseline testosterone-to-cortisol ratio is high; (2) an acute rise in cortisol followed by social provocation decreases ERP components involved in emotional face processing at an early as well as at a later stage. Taken together, these results support the notion of a complex interaction of psychophysiological mechanisms underlying the connection between stress and aggression.

Chapter IV

General Discussion

In the following, the results of the two studies presented in this thesis will be summarized and discussed in a wider context. Moreover, ideas for future studies concerning the topic of stress and aggression will be proposed.

4.1 Summary and Integration of Results

This thesis sought to elucidate mechanisms that potentially underlie the relationship between stress and aggression in a healthy population of young males and females. By means of both behavioral and electrophysiological data, the contributing role of changed information processing (cognitive control processes and approach-avoidance motivation, respectively) in the stress-aggression link was examined. Since stress is a complex and dynamical phenomenon that involves more than a mere increase of cortisol levels, a real stress situation was created in both studies by means of the socially evaluated cold pressor test designed by Schwabe and colleagues (2008).

Study 1 dealt with the influence of an acute psychophysiological stressor on cognitive control processes measured by means of a task-switching paradigm. Results provided evidence for an improvement of motor preparation for an upcoming behavioral response reflected by diminished CNV switch costs following acute stress. Moreover, high levels of basal HPA axis activity were also shown to enhance the motor preparation, indicated by a more pronounced CNV magnitude in switch trials compared to subjects with low basal HPA axis activity. Taken together, both acute stress and high basal cortisol concentrations led to an optimization of response preparation on an electrophysiological level. Influences of acute stress induction or basal cortisol levels on behavioral performance during the task-switching paradigm, however, were rather small or non-significant, respectively. Only the individuals who did not show an HPA axis response following the stressor (nonresponders) had significantly reduced switch costs after stress induction compared to prior to it. Assuming that switch costs reflect cognitive control processes (for reviews, see Kiesel et al., 2010; for reviews, see Monsell, 2003), individuals classified as nonresponders may have had improved cognitive control following stress induction. This is in line with a finding of the second study presented in this work: Although the effect was only marginally significant, merely the warm water group and the cortisol responders behaved more aggressively when facing high social provocation during the Taylor Aggression Paradigm. However, the nonresponders, on the other hand, showed less aggressive behavior compared to the other two groups although being highly provoked, resulting in similar levels of aggressive behavior in the high and low provocation groups of the nonresponders. This suppression of impulsive behavior again

suggests more efficient cognitive or inhibitory control processes of the nonresponders. These findings raise the question of what factors distinguish between individuals classified as cortisol responders or as nonresponders, besides their cortisol response following stress. Future research examining whether there exist differences in personality traits (for instance, resilience, self-control, or impulse control) between cortisol responders and nonresponders is necessary. Moreover, it is still unclear whether the reactivity of the HPA axis in response to a stressor is rather driven by state or by trait factors. In other words, is an individual once classified as a nonresponder also a nonresponder to other types of stressor, or does the acute HPA axis reactivity depend on situational factors? And if so, to what extent do personal and situational factors contribute to the acute HPA axis reactivity? Future studies addressing this subject are essential, since the categorization of stressed subjects into cortisol responders and nonresponders is widely-used in studies investigating stress effects.

Study 2 provided evidence for a complex interaction of the hormones cortisol and testosterone on approach-avoidance motivation in response to acute stress and social provocation: Within subjects with high cortisol levels, there was a positive relationship between high acute testosterone levels and the tendency to approach social threat stimuli, but only if they were socially provoked previously. A similar pattern occurred when the trait-like individual testosterone-to-cortisol ratio at baseline instead of acute testosterone was taken into account. A baseline hormonal profile of high testosterone and low cortisol levels was found to be associated with increased activation of certain subcortical brain structures - such as amygdala, hypothalamus, and brainstem - (Hermans et al., 2008) which are assumed to be involved in the neural circuitry of aggressive behavior (Nelson & Trainor, 2007). Hence, in the case of a stressful event eliciting an acute rise in cortisol, a predisposition of a high testosterone-to-cortisol ratio facilitates impulsive or aggressive acts, if the individual gets additionally provoked by another person.

These findings of the second study suggest that when investigating the underlying mechanisms of the stress-aggression link, cortisol is not the only hormone that is crucially involved in this relationship, but testosterone levels - acute and basal - have to also be taken into account. In addition to these two hormones, future studies should, if possible, also include the analysis of the neurotransmitter serotonin, since it also plays a crucial role in aggressive behavior. Specifically, it has been recently proposed that a high basal testosterone-to-cortisol ratio in combination with low serotonin concentrations in the brain increases the likeliness for impulsive aggression (Montoya et al., 2012; Terburg et al., 2009; van Honk et al., 2010). This further emphasizes the complexity of the biological mechanisms that

contribute to aggressive behavior. Thus, when investigating the neurophysiological mechanisms of aggressive behavior, it seems insufficient to include only one of these factors.

Study 2 also revealed significant influences of an endogenous cortisol increase in combination with subsequent social provocation on brain processing: Both early (P2) and later (P3) positive ERP components were reduced in highly provoked cortisol responders compared to mildly provoked ones, potentially indicating a less accurate, rapid processing of emotional and socially relevant stimuli. In line with the findings of study 1 suggesting improved response preparation in case of high (acute or basal) cortisol levels, the elevation of this stress hormone, and particularly in combination with social provocation, appears to affect social information processing in favour of a preparation of a rapid behavioral response in the sense of a fight-or-flight-reaction.

The results of the two studies presented here confirm the notion of Kruk et al. (2004) that information processing is significantly altered due to stress (and provocation), which is believed to thereby reduce the threshold for aggressive behavior (Kruk et al., 2004). Moreover, the presented findings confirm the assumptions of the General Aggression Model proposed by Anderson & Bushman (2002), in that not only situational but also personal factors contribute to the development of aggression. Regarding both cortisol and testosterone, not only acute levels (as a state-like or situational factor) but also basal concentrations (as a rather trait-like or personal factor) of these hormones affected information processing on the behavioral or on the electrophysiological level, respectively. The findings are highly relevant for future research on aggression, since according to the model of Anderson & Bushman (2002), information processes influence the present internal state of an individual which in turn affects the type of the behavioral outcome (either impulsive/aggressive or thoughtful).

In sum, the results underline the high complexity of the mechanisms involved in the relationship between stress and aggression. However, more research is needed to replicate and extend the presented findings. In the following, several suggestions for further studies are outlined.

4.2 Suggestions for Future Research

First, equivalently to the measurement of morning cortisol (the cortisol awakening response as an index of basal HPA axis (re)activity), the assessment of basal diurnal courses of testosterone concentrations as a measure for trait-like interindividual hypothalamic-pituitary-gonadal axis (re)activity could contribute to a deeper understanding of the complex interactions of cortisol and testosterone in the relationship between stress and aggression.

Second, studies including measurements of the peripheral physiology, such as heart rate and blood pressure, may provide significant information about the differences between cortisol responders and nonresponders following stress induction. Furthermore, a classification of stressed subjects into heart rate responders or heart rate nonresponders to the stressor, respectively, may be useful for elucidating further contributing physiological mechanisms that may release aggressive behavior in response to acute stress.

Third, another promising issue of future research may be the preselection of subjects with rather high or low basal HPA axis activity prior to the experimental session, in order to assign these two groups randomly to experimental conditions. Equivalently, subjects with high or low trait aggressiveness preselected beforehand could be examined regarding their social information processing in advance to acute aggressive behavior following stress induction.

Forth, when investigating approach-avoidance motivation in the context of stress and aggression, tasks that require whole body movements towards or away from emotional stimuli, as it was used for instance in a recent study of Stins et al. (2011), may be especially suitable for this topic of research. Particularly, when examining approach tendencies that may result in impulsive/aggressive behavior, such a task involving real body approaches is of superior ecological validity compared to joystick tasks or the Manikin Task that was used in the present work.

As a final suggestion for future studies and as a further step of the research presented in this thesis, one could separate temporally the manipulation of social provocation and the measurement of aggressive behavior using another paradigm than the Taylor Aggression Paradigm. Then it would be possible to measure approach-avoidance tendencies immediately after acute stress induction and social provocation, without the confounding with aggressive behavior, which in the case of study 2 of this thesis has been already acted out when approach-avoidance tendencies were measured. Thus, approach motivation should be assessed at a point in time, when the provoked subject is still in an aggressive and potentially aroused state, whereas otherwise this aversive/impulsive state of the individual may have been released when aggression could be acted out beforehand. Subsequent to the approach avoidance task, one could apply, for instance, the hot sauce paradigm in order to measure overt aggressive behavior. By this means, one could investigate more precisely, whether the link between stress and aggression is indeed mediated by approach tendencies.

4.3 Conclusion

Taken together, the two studies presented here suggest that acute stress as well as high basal cortisol levels lead to an improved motor preparation of a behavioral response, which may facilitate a rapid fight-or-flight reaction. In case of additional social provocation and elevated testosterone levels, this results in a tendency to approach social threat, which in turn may lower the threshold for impulsive acts, such as reactive aggressive behavior. The reported findings are in line with the assumption that changed social information processing plays an important role in the relationship between stress and aggression as proposed by Kruk et al. (2004).

The work presented in this thesis contributes to a more thorough understanding of the complex field of the stress-aggression-link. The presented results may also have implications for clinical psychology, as for instance one may consider the pharmacological reduction of acute cortisol levels in patients with disorders involving aggressive/impulsive behavior. Research examining the underlying psychophysiological mechanisms of stress and aggression is still highly relevant. The combination of behavioral, biological, and electrophysiological methods is promising in order to gain an elaborate understanding of the psychophysiological mechanisms underlying human aggression.

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List of Abbreviations

ACC	anterior cingulate cortex
ACTH	adrenocorticotrophic hormone
Ag/AgCl	silver/silver chloride
ANOVA	analysis of variance
AUC_G	area under the curve with respect to ground
AUC_t	total area under the curve
BAS	behavioral activation system
BIS	behavioral inhibition system
BMI	body mass index
CAR	cortisol awakening response
CNV	contingent negative variation
CR	cortisol responders
CRH	corticotrophin-releasing hormone
CTI	cue target interval
db	decibel
EEG	electroencephalography
EOG	electrooculography
ERP	event-related potential
fMRI	functional magnetic resonance imaging
GAM	General Aggression Model
GR	glucocorticoid receptor
HPA	hypothalamic-pituitary-adrenal
Hz	Hertz
LPP	late positive potential
M	mean
MR	mineralcorticoid receptor
nmol/l	nanomol per liter
N1	refers to first negative simulus-locked component of the event-related potential
N2	refers to second negative simulus-locked component of the event-related potential
NR	nonresponders
OFC	orbitofrontal cortex

P1	refers to first positive simulus-locked component of the event-related potential
P2	refers to second positive simulus-locked component of the event-related potential
P3	refers to third positive simulus-locked component of the event-related potential
PFC	prefrontal cortex
PET	positron emission tomography
SD	standard deviation
SE	standard error
SECP	Socially Evaluated Cold Pressor Test
SNS	sympathetic nervous system
TAP	Taylor Aggression Paradigm
TSST	Trier Social Stress Test
WW	warm water group

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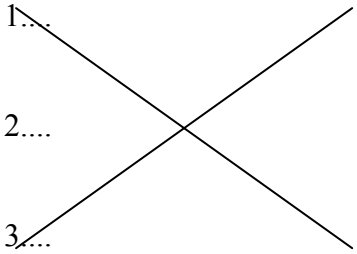
Erklärung

Ich erkläre hiermit, dass ich die vorliegende Arbeit ohne unzulässige Hilfe Dritter und ohne Benutzung anderer als der angegebenen Hilfsmittel angefertigt habe. Die aus anderen Quellen direkt oder indirekt übernommenen Daten und Konzepte sind unter Angabe der Quelle gekennzeichnet.

Bei der Auswahl und Auswertung folgenden Materials haben mir die nachstehend aufgeführten Personen in der jeweils beschriebenen Weise entgeltlich/unentgeltlich geholfen:



Weitere Personen waren an der inhaltlich-materiellen Erstellung der vorliegenden Arbeit nicht beteiligt. Insbesondere habe ich hierfür nicht die entgeltliche Hilfe von Vermittlungs- bzw. Beratungsdiensten (Promotionsberater oder andere Personen) in Anspruch genommen. Niemand hat von mir unmittelbar oder mittelbar geldwerte Leistungen für Arbeit erhalten, die im Zusammenhang mit dem Inhalt der vorliegenden Dissertation stehen.

Die Arbeit wurde bisher weder im In- noch im Ausland in gleicher oder ähnlicher Form einer anderen Prüfungsbehörde vorgelegt.

Ich versichere die Richtigkeit der vorangegangenen Erklärung und bin mir der strafrechtlichen Folgen einer Falschaussage bewusst.

Julia Fechtner