Katja Bertsch

Stress, Aggression, and Social Information Processing

A Psychophysiolocial Investigation

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Stress, Aggression, and Social Information Processing

A Psychophysiological Investigation

Doctoral thesis

by

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Introduction and General Outline of the Thesis

Aggression is a very common social behavior. Basically everyone is in some way affected by aggression: as a target of it, by engaging in it themselves, or in being charged with observing and controlling it in others (Renfrew, 1997). The omnipresence of aggression in everyday life and its enormous social and economic costs worldwide (e.g., 1.6 million lives in 2000; Krug, Dahlberg, Mercy, Zwi, & Lozano, 2002) highlight the relevance of studying potential causes and consequences of aggression.

Although the investigation of aggression has a long research tradition in psychology, still little is known about the neural underpinnings and biological correlates of (normal) aggression. Modern psychosocial models, such as the General Aggression Model (GAM; Anderson & Bushman, 2002), assume that aggression is influenced by multiple person and situation variables mediated by affect, cognition, and arousal. Among others, stress has been identified as one of the most important promoting factors for aggression. However, evidence is mostly based on clinical and animal studies (see, for instance, Kruk, Halász, Meelis, & Haller, 2004; van Goozen, Fairchild, Snoek, & Harold, 2007), whereas the relationship between stress and aggression in healthy humans has hardly been investigated yet. Interestingly, there is considerable overlap in brain areas involved in the generation of an appropriate stress response as well as the regulation of aggression. These brain areas, for instance, the amygdala, the hypothalamus, and prefrontal regions, are part of a neural network which is engaged in the processing and regulation of emotions (Davidson, Putman, & Larson, 2000). It has therefore been hypothesized that the processing of social signals as well as the regulation of emotions may play an important role in the context of stress and aggression (Kruk et al., 2004). In fact, acutely elevated levels of the stress hormone cortisol as well as high levels of trait anger or aggression influence the processing of threat-related social information (e.g., Putman, Hermans, & van Honk, 2007; van Honk, Tuiten, van den Hout et al., 2001). Nevertheless, the role of social information processing in the relationship of stress and aggression still remains unclear.

The present work therefore aims to investigate (1) the association of stress and aggression, (2) effects of both stress and aggression on social information processing, and (3) the temporal dynamics of stress and aggression effects on information processing in healthy individuals. To do so, the present work combines psychosocial, psychophysiological, and endocrine methods and measures.

In the following Chapter I, theoretical approaches and empirical evidence concerning human aggression will be outlined. Starting with a definition and an integrative model of aggression, the General Aggression Model (Anderson & Bushman, 2002),

Chapter I will then turn to the neural underpinnings of aggression as well as the role of emotion regulation and social information processing in pathological and normal aggression. This chapter will also give an overview of the stress system and the empirical evidence of an association between stress and aggression. The Chapters II and III comprise two event-related potential (ERP) studies in which the influence of experimentally induced aggression (Chapter II) as well as of exogenously elevated cortisol levels *and* experimentally induced aggression (Chapter III) on social information processing were investigated. Chapter IV will regard the influence of ostracism, a very strong social stressor including the experience of being ignored and excluded, *and* experimentally induced aggression on aggressive behavior and social information processing in a behavioral study. Finally, the results of the three studies and their implications for future research on aggression will be discussed in Chapter V.

Chapter I

Stress, Aggression, and Social Information Processing

1.1. Aggression

Aggression and violence occur all around the world and in all segments of society (Krug et al., 2002). Seemingly random acts of violence, e.g., school shootings, gang riots, murders, or rapes occur everywhere, again and again, day after day. While these could be considered the most extreme examples of aggression, less spectacular incidences of aggression take place in families, school classes, peer groups, or workgroups on a daily basis. Acts of aggression not only cause suffering to one or more individuals but also increase the likelihood for further aggression (Berkowitz, 1993). Besides the negative effects of aggression and violence, it should be noted that aggression is also necessary for human survival (Renfrew, 1997). It serves important purposes of allowing individuals to effectively compete for limited resources and to establish and maintain their position in society. The omnipresence of aggression and its impact on our everyday lives highlight the relevance of investigating potential causes *and* consequences of (normal) aggression.

1.1.1. Definition and Classification of Aggression

When defining aggression, an often quoted statement by Justice Potter Stewart (1964), "I know it when I see it," comes to mind. Almost everyone thinks he or she knows what aggression is, and consequently there have been numerous definitions of aggression. In the present work, a definition by Baron and Richardson (1994) will be used which describes aggression as "any behavior directed towards the goal of harming or injuring another living being that is motivated to avoid such treatment" (p. 7). The main strength of this definition is that it includes many different forms of aggression, while excluding related concepts that are not necessarily part of aggression, such as negative emotions, attitudes, or motives, accidental harm-doing as well as damaging inanimate objects (Baron & Richardson, 1994; Giancola & Chermack, 1998).

Typical classifications of aggression distinguish between *hostile* aggression and *instrumental* aggression (Berkowitz, 1993; Bushman & Anderson, 2001). Hostile aggression is an impulsive, affective, reactive behavior which is motivated by a desire to hurt someone, while instrumental aggression is a premeditated, calculated, proactive behavior which is motivated by some other goal, e.g., to obtain money (Bushman & Anderson, 2001). Anderson and Bushman (2002; Bushman & Anderson, 2001) have admonished that a dichotomous classification of aggression risks an oversimplification of a

complex behavior because dichotomous theories are not suited for the known interactions between automatic and controlled aspects of information processing. Moreover, many observed types of aggression involve mixed or multiple motives and thus do not fit well with ideal types of aggression (Bushman & Anderson, 2001). Despite these substantial difficulties of the dichotomous classification of aggression, the (neuro-)biological literature largely continues to draw this distinction (see, for instance, Chapter 1.3.3).

1.1.2. A Psychosocial Approach to Aggression: the General Aggression Model

The General Aggression Model (GAM; Anderson & Bushman, 2002) is an attempt to integrate several rather specific theories on aggression into a broader framework. According to this model, person and situation variables influence aggressive behavior through the mediating effects of affect, cognition, and arousal (see Figure 1).

All important features of a *situation*, for instance, the presence of aggressive cues, provocation, frustration, or social rejection, may influence aggression. Interpersonal provocation probably constitutes the most important single cause of aggression (Anderson & Bushman, 2002; Berkowitz, 1993). Most of the currently used aggression paradigms, i.e., the Taylor Aggression Paradigm (TAP; Taylor, 1967) or the Point Subtraction Aggression Paradigm (PSAP; Cherek, 1981), are built on forms of interpersonal provocation to induce aggression. According to Berkowitz (1990), stressful events of all kinds (including physical discomfort) can prime the initiation of escape and attack behavior. Laboratory studies have confirmed that aversive stimulation does not need to involve an anger-specific event to prime aggression. Hot rooms (Anderson, Anderson, & Deuser, 1996), painful cold water immersion (Berkowitz, Cochran, & Embree, 1981), or the exposure to an air blast stressor (Verona, Patrick, & Lang, 2002), for instance, have been found to increase the likelihood for aggressive behavior in humans.

Besides these situation factors, *person factors* such as traits, attitudes, and beliefs as well as genetic predispositions and sex influence an individual's preparedness to aggress. For instance, certain traits, such as narcissism or self-esteem affect an individual's proneness to aggressive behavior (Baumeister, Boden, & Smart, 1996; Bushman & Baumeister, 1998; Kirkpatrick, Waugh, Valencia, & Webster, 2002). Moreover, people with high trait aggression are susceptible to hostile attribution, perception, and cognitive biases (van Honk, Tuiten, De Haan, van den Hout, & Stam, 2001a; van Honk et al., 2001b) and are thus more likely to aggress particularly under high provocation (Cohen, Eckhardt,

& Schagat, 1998; Eckhardt & Cohen, 1997). Sex differences in aggression have been reported in several studies with males showing more (direct) aggression and violent crimes (Archer, 2004; Krug et al., 2002). Provocation, however, diminishes sex differences in physical aggression (Bettencourt & Miller, 1996) although qualitative distinctions may still be observable, i.e., while males show more direct aggression, females prefer indirect forms of aggression (Oesterman et al., 1998).

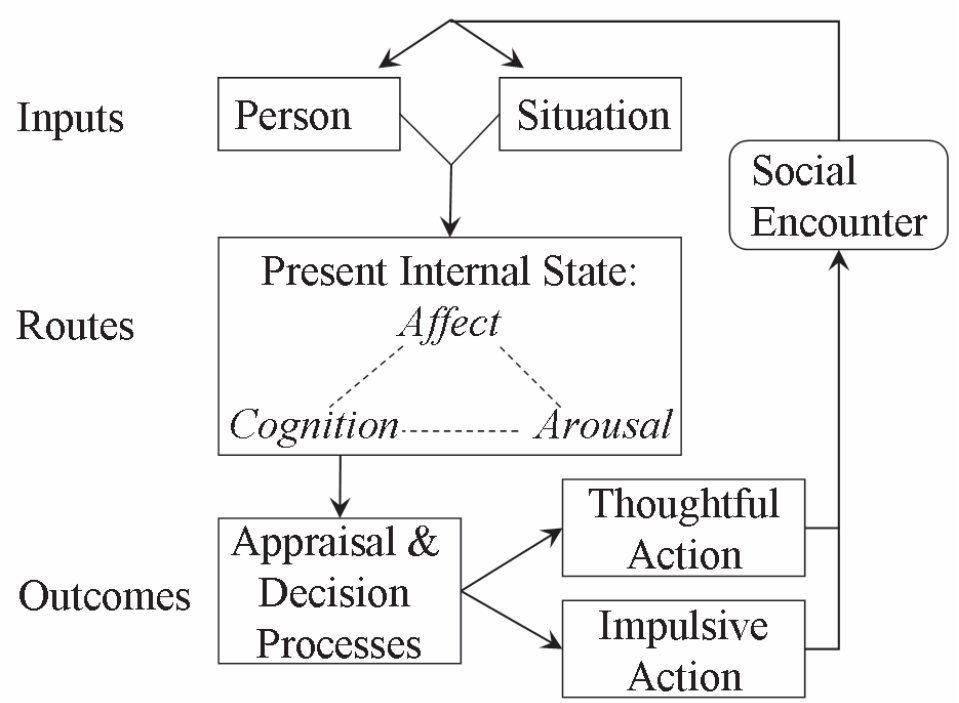

Figure 1. The General Aggression Model Episodic Processes (Anderson & Bushman, 2002, p. 34).

Both situation and person factors influence how a person behaves in a social encounter through this person's present internal state, in particular affective and cognitive processes as well as general arousal (see Figure 1). These processes lead to automatic as well as rather controlled (re)appraisal and decision processes, which then result in either thoughtful or impulsive actions and become a part of the input for the next person-situation episode. Thus, a person's (aggressive) behavior in one situation, along with the cognitive and affective processes involved in the generation of this behavior, affects this person's future evaluation of and behavior in social encounters.

Taken together, the GAM proposes that multiple causes may lead to various forms of aggressive behavior by influencing cognition, emotion, and general arousal. Predictions of the GAM have been tested and confirmed in several behavioral experiments (e.g., Carnagey & Anderson, 2005; Lindsay & Anderson, 2000). However, underlying neural mechanisms and substrates involved in these mental processes still remain unclear. As this

is of interest for the present work, studies investigating the neural underpinnings of aggression will be addressed in the next section.

1.1.3. The Neural Circuits of Aggression: the Role of Emotion Regulation and Social Information Processing

Converging evidence from animal and human studies indicates that an entire neural network is involved in aggression. This network includes the prefrontal cortex, in particular its orbitofrontal and medialfrontal subdivisions, and subcortico-limbic structures, namely, the amygdala, the hypothalamus, and the periaqueductal gray (Blair, 2004; Davidson et al., 2000; Kruk et al., 1998; Lee & Coccaro, 2007; Nelson & Trainor, 2007; Patrick & Verona, 2007).

In animals, a circuit running from the medial amygdala to the hypothalamus via the stria terminalis and from there to the periaqueductal grey (PAG) subserves aggression (Blair, 2004; Siegel & Victoroff, 2009). This system is organized in a hierarchical manner in that aggression evoked by the amygdala depends on the functional integrity of the hypothalamus and the PAG, but not vice versa. Evidence for the role of the hypothalamus and the PAG in aggression has primarily been established in animal studies, but has been confirmed in non-human primates (Lipp & Hunsperger, 1978) and in patients with neurological disorders (e.g., Berkovic et al., 1988; Kuhn et al., 2008; Tonkonogy & Geller, 1992). Lesioning these structures resulted in reduced aggression, whereas their stimulation elicited aggression and rage in cats and rodents (Kruk et al., 2004; Siegel, 2005; Siegel & Victoroff, 2009). The 'hypothalamic attack area'[1] (e.g., Halász et al., 2002; Kruk et al., 2004) is supposed to integrate afferents from limbic and prefrontal structures that regulate aggression and thus to provide a coordinated excitatory efferent output to the PAG, on the way to motor efferents (Summers & Winberg, 2006).

The amygdala and the frontal cortex are supposed to modulate this aggression-mediating neural circuitry (Blair, 2004; Davidson et al., 2000; Lee & Coccaro, 2007). Evidence suggests that a fronto-limbic system, primarily consisting of the amygdala, prefrontal areas, and anterior cingulate cortex (ACC), is important for emotion regulation, that is, the rapid appraisal of emotional material, the production of affective states, and the automatic regulation of autonomic responses to emotional stimuli (Phillips, Drevets, Rauch, & Lane, 2003). While the amygdala plays a key role in the activation of emotional states,

[1] In rodents, the hypothalamic attack area largely coincides with the intermediate hypothalamic area and the ventro-lateral pole of the ventromedical nucleus of the hypothalamus (Kruk, 1991; Siegel, Roeling, Gregg, & Kruk, 1999)

the ACC and prefrontal areas operate to detect circumstances under which affective control is needed and to implement control processes, respectively (Davidson et al., 2000). There are extensive interconnections between prefrontal regions, particularly the orbitofrontal cortex (OFC), and limbic brain structures (Kringelbach & Rolls, 2004) such as the amygdala and the rostral ACC (which is also regarded to be a part of the medial frontal cortex). As structures of this fronto-limbic network project their axons either directly or indirectly to the hypothalamus and/or the midbrain PAG, this endows the fronto-limbic system with the capacity to modulate and control the functions associated with the hypothalamus and the PAG, including aggression and rage behavior (Siegel & Victoroff, 2009). It has therefore been suggested that dysfunctions in this fronto-limbic network regulating emotional responses may be responsible for an increased susceptibility for impulsive aggression and violence (Davidson et al., 2000).

Neuroimaging studies have yielded convincing evidence for structural and functional abnormalities in prefrontal regions, in particular the OFC and the ventromedial prefrontal cortex (VMPFC), in pathologically aggressive individuals (for reviews, see Lee & Coccaro, 2007; Patrick & Verona, 2007; Strüber, Lück, & Roth, 2008). Overall, the current findings suggest an OFC hypofunction as a common risk factor for *impulsive aggression* (Lee & Coccaro, 2007; Strüber et al., 2008). Together with a hyperreactivity of the amygdala to emotional stimuli, the risk of uncontrollable aggressive outbursts further increases, as seen, for instance, in intermittent explosive disorder[2] (Coccaro, McCloskey, Fitzgerald, & Phan, 2007). Increased *instrumental aggression* has been primarily associated with a hypofunction of the amygdala (Blair, 2001).

The OFC, the amygdala, the rostral ACC as well as the ventral striatum have also been found to be involved in the processing of angry faces (Blair, Morris, Frith, Perrett, & Dolan, 1999; Phillips et al., 1999; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998). Facial expressions are among the most immediate and significant social signals in nonverbal communication (LeDoux, 1998) and social information processing has been repeatedly shown to be an important construct in the explanation of human aggression (Crick & Dodge, 1994). The link between processing angry facial expressions and aggression has been underlined by work demonstrating increased attention to angry faces in aggression-prone individuals (e.g., van Honk et al., 2001b) as well as an impaired recognition of facial emotions and abnormal neural responses (i.e., exaggerated amygdala

[2] Intermittent explosive disorder is characterized by impulsive acts of aggression and is classified in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) as a impulse-control disorder (Coccaro, Posternak, & Zimmerman, 2005).

reactivity and diminished OFC activation) to angry faces in pathologically aggressive patients (Blair & Cippolotti, 2000; Coccaro et al., 2007). This suggests that aggression-prone individuals focus more on aggression-related social information when encoding situational cues (Crick & Dodge, 1994). Even in samples of healthy individuals, activations in the OFC, the amygdala, the ACC, and the ventral striatum as well as the connectivity between the amygdala and the ACC during the processing of angry faces were related to individual differences in trait impulsivity (Brown, Manuck, Flory, & Hariri, 2006) and appetitive motivation (Beaver, Lawrence, Passamonti, & Calder, 2008; Passamonti et al., 2008). Nevertheless, it has not been studied so far if acute experiences of aggression can alter the processing of angry facial expressions in healthy individuals as well. Investigating this could offer insights into intact emotion regulation mechanisms in challenging situations.

The few studies that have already investigated the online-processing during an aggressive encounter in healthy participants confirmed that frontal and subcortico-limbic structures are involved in reactive (impulsive) aggression. These studies used modified versions of the Taylor Aggression Paradigm (TAP; Taylor, 1967), which is disguised as a reaction time competition and seeks to elicit aggression through provocation, in combination with functional magnetic resonance imaging (fMRI) or electroencephalography (EEG) / event-related potentials (ERP). Lotze and colleagues found that activity in the medial prefrontal cortex (mPFC) increased during retaliation, whereas it increased in the dorsal mPFC, when participants had to select the intensity of revenge (Lotze, Veit, Anders, & Birbaumer, 2007). Moreover, OFC and VMPFC were active when participants watched their opponents suffer and this activation was stronger in more empathetic participants. Krämer et al. (2007) observed higher activations in the anterior insula and the rostral and dorsal ACC following high provocation when participants had to select the intensity of the punishment. In two similar EEG/ERP experiments, Krämer and colleagues reported alterations in frontolateral negativities and theta power related to the participants' aggressive behavior (Krämer, Büttner, Roth, & Münte, 2008; Krämer, Kopyciok, Richter, & Münte, 2009). High provocation was related to an increased frontolateral ERP component as well as increased frontal theta power in participants who refrained from retaliation, but to a decreased theta power in those who got back at the opponent. This may reflect increased dorsal ACC activity and thus indicates increased cognitive effort in order to control the behavioral response to high provocation.

In summary, the results of the studies on impulsivity, anger processing, and impulsive aggression consistently demonstrate the involvement of brain structures implicated in the neural circuitry of emotion processing and regulation, including prefrontal areas (primarily OFC/VMPFC), medial frontal cortex (rostral and dorsal ACC), and limbic structures (amygdala). Studies examining OFC-amygdala interaction in response to anger report an inverse relationship in normal controls, which is disturbed in patients with anger attacks and impulsive aggression. While the hypothalamus and the PAG seem to mediate aggression (Gregg, 2003; Panksepp, 2005), the amygdala, the ACC, and prefrontal regions modulate this circuitry (Blair, 2004).

1.2. Stress and Aggression: the Role of the HPA axis and Cortisol

Stress is one of the most important factors promoting aggression and violence. The General Aggression Model (Anderson & Bushman, 2002) and other prominent aggression models, for instance, Berkowitz's associative aggression model (Berkowitz, 1990), see stressful events of all kinds (including interpersonal provocation, frustration, and physical discomfort; see above) as situation factors that can prime the initiation of aggression. Evidence from both animal and clinical studies indicates tight connections between the stress system and aggression (e.g., Haller, Millar, van de Schraaf, de Kloet, & Kruk, 2000; Kruk et al., 2004; Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; van Goozen & Fairchild, 2006). However, the interaction of stress and aggression in healthy humans has been hardly researched yet.

1.2.1. The Role of the HPA axis in Response to Stress

The HPA axis consists of the hypothalamus, the pituitary gland, and the adrenal gland. Along with the sympathetic nervous system, the HPA axis plays a primary role in the stress response (de Kloet, Joëls, & Holsboer, 2005; Sapolsky, Romero, & Munck, 2000). Psychological stress triggers the activation of the HPA axis, leading to the secretion of corticotropin-releasing hormone (CRF) from the paraventricular nucleus (PVN) of the hypothalamus, which in turn activates the anterior pituitary gland to release adrenocorticotrophic hormone (ACTH) into the blood stream (Vazquez, 1998). ACTH eventually reaches the adrenal cortex, where it stimulates the synthesis and secretion of glucocorticoids (primarily cortisol in humans and corticosterone in rodents).

The biological effects of glucocorticoids are usually adaptive (de Kloet, Oitzl, & Joëls, 1999). For instance, they terminate digestive activity, increase sympathetic nervous system activity, and mobilize energy resources in response to stressful stimuli (de Kloet, 1991; de Kloet et al., 1999; Johnson, Kamilaris, Chrousos, & Gold, 1992). Additionally, basal and stress-induced glucocorticoids prime defense mechanisms and thus prepare responses to future stressors (Sapolsky et al., 2000). Cortisol regulates its own release via a negative feedback loop in the central nervous system, where it binds to glucocorticoid receptors (GRs) and mineralocorticoid receptors (MRs) throughout the limbic system (including hippocampus and amygdala), the prefrontal cortex (PFC), and the ACC (de Kloet et al., 2005; Herman, Ostrander, Mueller, & Figueiredo, 2005; Pruessner et al., 2009). The hippocampus and the prefrontal cortex (in particular the VMPFC and the OFC) are largely inhibitory to HPA axis secretion, whereas the amygdala is implicated in activation of glucocorticoid secretion[3] (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Herman et al., 2005). Findings that the amygdala has positive effects on the HPA axis are consistent with other known functions of this brain region, including activation of autonomic responses (Gray, 1993), and its involvement in fear, threat, and anxiety (Davis, 1992). The hippocampus, the PFC, and the amygdala also possess a high density of MRs and GRs and may thus play a critical role in the HPA feedback mechanism (Herman et al., 2005).

Structural and functional magnetic resonance imaging studies revealed that the exposure to acute or chronic stress may alter hippocampal, PFC, and amygdaloid activity and volume (for a review, see Pruessner et al., 2009). For instance, being exposed to a psychosocial stressor (Montreal Imaging Stress Test; Dedovic et al., 2005) was associated with a hypothalamic deactivation (Pruessner et al., 2008). This was limited to individuals who showed a stress-induced increase in cortisol. As its degree was linked to the amount of cortisol released, this suggests a linear relationship between the hippocampal deactivation and HPA activation. Furthermore, Eisenberger, Lieberman, and Williams (2003) reported enhanced activations in the ACC and regions of the right PFC during social exclusion compared to social inclusion. The experience of social exclusion is a very strong social stressor, which has been shown to increase cortisol levels, autonomic activity, and self-reported distress (Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003; Stroud, Salovey,

[3] Increased hippocampal and PFC activation has inhibitory effects on the PVN, while increased amygdaloid activation has exciatatory effects on the PVN.

& Epel, 2002; Stroud, Tanofsky-Kraff, Wilfey, & Salovey, 2000).[4] In the study of Eisenberger et al. (2003), self-reported distress was positively associated with ACC activation but negatively with right PFC activation during social exclusion relative to inclusion. As the activity in the right PFC was also negatively correlated with ACC activity, this suggests that the right PFC might play a self-regulatory role in mitigating the distressing effects of social exclusion.

Taken together, converging evidence indicates that prefrontal and limbic brain structures are crucially involved in the generation of appropriate responses to stress. These brain structures are part of a fronto-limbic network engaged in emotion regulation – and aggression.

1.2.2. The HPA axis and Aggression

Many studies found a *negative* association between basal cortisol levels and the magnitude of behavioral deviation in antisocial and pathologically aggressive children and adults (for reviews, see Cappadocia, Dosrocher, Pepler, & Schroeder, 2009; van Goozen et al., 2007). The majority of these studies, however, has been correlational in nature, which makes causal inferences impossible. Moreover, the results have been relatively mixed regarding the effect sizes and even the direction of the relationship, maybe due to the use of varying methods for cortisol collection and differing definitions or diagnoses of aggression or antisocial behavior. Despite this, van Goozen et al. (2007) reported a small to moderate effect across studies in the direction of an inverse relationship (d = -0.40) between basal cortisol levels and disruptive behavior disorder symptoms. Other studies found reduced cortisol reactivity to frustration or provocation in pathologically aggressive and impulsive individuals compared to healthy controls (van Goozen, Matthys, Cohen-Kettenis, Buitellar, & Van Engeland, 2000; van Goozen et al., 1998). In addition, attenuated resting autonomic arousal (heart rates, skin conductance, and electrodermal activity) was observed in pathologically aggressive children and adolescents (for reviews, see Patrick, 2008; Patrick & Verona, 2007). Cappadocia et al. (2009) recently suggested that lower cortisol levels and autonomic functioning may reflect a lack of self-control and emotion regulation and thus increase the likelihood for impulsive and aggressive behavior.

[4] In fact, most laboratory paradigms which reliably increase cortisol levels comprise a social-evaluative component, e.g., the Trier social stress test (Kirschbaum, Pirke, & Hellhammer, 1993) or the socially evaluated cold-pressor test (Schwabe, Haddad, & Schachinger, 2008).

In healthy individuals, increased cortisol stress reactivity was *positively* related to self-reported aggression (Lopez-Duran et al., 2009; Scarpa, Fikretoglu, & Luscher, 2000). Gerra and colleagues (Gerra et al., 2001a; 2001b; 2004; 2007) also reported a positive relationship between aggressive behavior in a laboratory aggression paradigm (PSAP; Cherek, 1981) and acute HPA axis activity prior to the aggression induction in healthy male participants. However, not all studies were able to replicate this effect (Berman, Gladue, & Taylor, 1993; Salvador, Suay, Martinez-Sanchis, Simon, & Brain, 1999). The above-mentioned evidence that hot rooms (Anderson et al., 1996), painful cold water immersion (Berkowitz et al., 1981), or the exposure to an air blast stressor (Verona et al., 2002) increase the likelihood of aggression indirectly supports a positive association between cortisol levels and aggression in healthy individuals. In addition, social exclusion has also been reported to increase feelings of anger (e.g., Williams, Cheung, & Choi, 2000), hostile cognitions (DeWall, Twenge, Gitter, & Baumeister, 2009), the urge to aggress (Buckley, Winkel, & Leary, 2004), and even aggressive behavior (e.g., Twenge, Stucke, Baumeister, & Tice, 2001).

Furthermore, the findings of several studies indicate that acutely elevated cortisol levels affect the processing of angry facial expressions. However, the results are inconsistent in regard to the direction of the effect: Some studies reported increased attention for angry faces in participants who were exogenously administered high dosages of cortisol (Putman, Hermans, Koppeschaar, van Schijndel, & van Honk, 2007a; van Peer et al., 2007) or exposed to social stress (Roelofs, Bakvis, Hermans, van Pelt, & van Honk, 2007) compared to a placebo or non-stress condition. Other studies found reduced anger biases in participants with acutely elevated cortisol levels (Oei, Tollenaar, Spinhoven, & Elzinga, 2009; van Honk et al., 1998; van Peer, Spinhoven, & Roelofs, 2009a). As mentioned above, angry faces are threat-related social signals which attract enhanced attention by aggression-prone individuals (van Honk et al., 2001b). Abnormal neural activations as well as an impaired recognition of angry facial expressions in pathologically aggressive individuals imply the relevance of social information processing for the explanation of human aggression (Blair & Cippolotti, 2000; Coccaro et al., 2007; Crick & Dodge, 1994). Brain structures that have been implicated in emotion processing and regulation, in particular the amygdala and OFC, also possess a high density of glucocorticoid receptors that are crucially involved the regulation of HPA axis activity.

1.2.3. 'The Vicious Circle of Aggression': Animal Studies on Stress and Aggression

So far, knowledge about how stress mechanisms and mechanisms involved in aggression interact on a neurobiological level mostly relies on animal research. Kruk et al. (2004) established a fast positive feedback loop – 'the vicious circle of aggression' – between the adrenocortical stress response and the hypothalamic attack area (see Figure 2). In a series of rodent studies, the stimulation of the hypothalamic attack area rapidly activated the HPA axis even in the absence of an opponent. In addition, an injection of corticosterone quickly facilitated hypothalamic aggression. Thus, this fast, mutual, and positive feedback mechanism might contribute to the precipitation and escalation of aggression and violent behavior under stressful conditions (Kruk et al., 2004). This may reflect the aggression-potentiating effects of high levels of arousal, maybe mediated by elevated glucocorticoid levels, in healthy humans.

In addition, a hypoarousal-driven rodent model mimics human pathological aggression (Halász, Liposits, Kruk, & Haller, 2002; Haller, Halász, Mikics, & Kruk, 2004; Haller & Kruk, 2006; Haller, van de Schraaf, & Kruk, 2001), in particular the consequences of low basal levels of cortisol seen, for instance, in antisocial children and adults. When confronted with an intruder in their home-cage, rats with low concentrations of circulating corticosterones[5] showed abnormal patterns of aggression including an inappropriate injuring of vulnerable body parts of the opponent and a decreased threatening behavior (Haller et al., 2001). Acute corticosterone injections prior to exposure to the fighting situation prevented these abnormal aggression patterns (Haller et al., 2001). Interestingly, abnormal patterns of aggression observed in rats with glucocorticoid deficiencies did not affect the function of brain regions involved in the control of aggression (e.g., medial amygdala, hypothalamic attack area, and PAG). Instead, brain regions involved in mediating the stress response (the parvocellular part of the PVN) and fear reactions (the central nucleus of the amygdala) were activated (Halász et al., 2002). This suggests that glucocorticoid hypofunction increases the sensitivity to stressors and fear reactions, possibly involving a misinterpretation of social signals sent by the opponent. The resulting overreaction may then contribute to abnormal aggressiveness observed in these animals.

[5] These rats were adrenolectomized and implanted a slow release pellets.

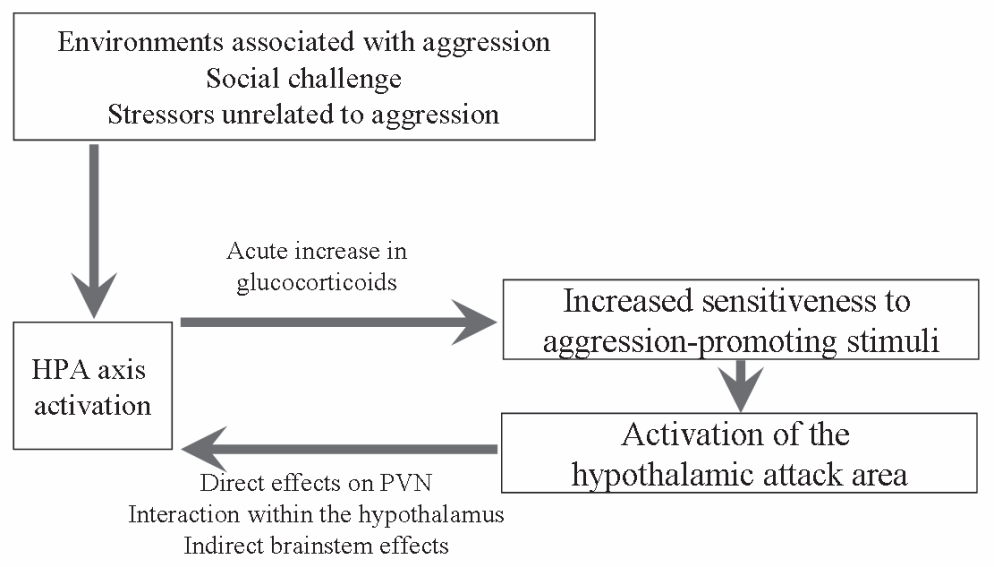

Figure 2. 'The vicious circle of aggression': a fast positive feedback loop between the adrenocortical stress response and the hypothalamic attack area (Kruk et al., 2004, p. 1067). HPA: hypothalamic-pituitary-adrenal; PVN: paraventricular nucleus of the hypothalamus.

The findings of these studies are in agreement with (1) aggression-enhancing effects of elevated cortisol levels in healthy individuals and (2) hypoarousal, in the sense of lower basal cortisol levels and autonomic functioning, (3) blunted cortisol and autonomic reactivity to frustration and provocation as well as (4) deficiencies in emotion recognition and dysfunctions of emotion regulation observed in pathologically aggressive individuals. In their 'vicious circle of aggression', Kruk et al. (2004), therefore hypothesized that the rapid processing and appraisal of aggression-promoting social signals may be an important mediator in the association between stress and aggression (see Figure 2). The fronto-limbic emotion regulation network, and particularly the amygdala, parts of the frontal cortex, and the ACC, could be of particular importance for the regulation of aggressive behavior *and* the generation of appropriate stress responses. Deficiencies in this network are associated with deficits in emotion recognition and regulation, abnormal aggressiveness, and problems with stress response systems, as observed in pathologically aggressive individuals. So far, less is known about the association of stress and aggression and the role of social information processing / emotion regulation in healthy humans. There is, however, some evidence for effects of acutely elevated cortisol levels as well as trait aggression on the processing of angry facial expressions in healthy individuals.

1.3. Summary and Aims of the Thesis

Aggression is a common social behavior in humans and animals. Current psychosocial and (neuro)biological models propose that stressful situations may be an important promoting factor for aggression and are probably mediated by alterations in social information processing and emotion regulation. Evidence has primarily been established in animal and clinical studies. Most of the findings in humans have been correlative in nature, which does not allow for causal inferences. Although effects of trait aggression as well as acute levels of cortisol on social information processing / emotion regulation have been reported, the influence of both – acutely elevated cortisol and experimentally induced aggression – on social information processing in healthy individuals remain unclear. This is necessary to gain more insights into intact emotion regulation processes and their involvement in the relationship of stress and aggression.

Therefore, the aims of the present work are: (1) to investigate the relationship between stress and aggression, (2) to investigate the influence of both stress and aggression on social information processing, i.e., the processing of emotional facial expressions, and (3) to gain more insights into the temporal dynamics of stress and aggression effects on information processing in healthy individuals. This was investigated in three consecutive studies.

In all three studies, healthy participants performed the Taylor Aggression Paradigm (TAP; Taylor, 1967), a well-validated and often used laboratory paradigm to provoke and measure aggression (for a review, see Giancola & Chermack, 1998). This was followed by an emotional Stroop task (Williams, Mathews, & MacLeod, 1996) with emotional (angry, fearful, and happy) and neutral facial expressions. In this task, the emotional valence of the faces is irrelevant to the task itself, which, for instance, requires the participants to name as quickly as possible the color in which the face is displayed. Individuals with high levels of trait anger or aggression as well as violent criminals have been found to allocate more attention to angry facial expressions, which has been interpreted as an implicit bias effect for aggression-related social information (e.g., Smith & Waterman, 2003, 2004; van Honk et al., 2001b). As acute cortisol levels have also been reported to influence bias effects in emotional Stroop tasks (e.g., Roelofs et al., 2007; van Peer et al., 2009a), this task appears suitable for the purposes of the present work.

The first study (Chapter II) examines the effects of experimentally provoked aggression on the processing of emotional facial expressions. The second study (Chapter III) extends this by including a pharmacological manipulation of acute cortisol levels prior

to the experiment. Thus, this study investigates the effects of exogenously elevated cortisol levels on aggressive behavior in the TAP and the effect of both elevated cortisol and aggression on the processing of emotional facial expressions. Both studies comprised the measurement of aggression in the TAP as well as the measurement of stimulus-locked reaction times and ERPs in the emotional Stroop task. As ERPs allow a more direct and sensitive examination of differences in the time course of information processing and the cortical resources used herein (Hillyard & Kutas, 1983), they can provide valuable insights into early stages of information processing. In addition, salivary cortisol was obtained in the second study to gain information about acute cortisol levels. The third study (Chapter IV) examines the effects of ostracism, a very strong social stressor including the experience of being ignored and excluded (Williams, 2001), on aggressive behavior in the TAP as well as the combined effects of ostracism and aggression on emotional face processing. In addition, two different versions of the TAP were applied in order to see if aggressive behavior depends on the type of aggression allowed by the paradigm.

Chapter II

Influence of Aggression on Information Processing in the Emotional Stroop Task – an Event-Related Potential Study

Abstract

Aggression is a common behavior which has frequently been explained as involving changes in higher level information processing patterns. Although researchers have started only recently to investigate information processing in healthy individuals while engaged in aggressive behavior, the impact of aggression on information processing beyond an aggressive encounter remains unclear. In an ERP study, we investigated the processing of facial expressions (happy, angry, fearful, and neutral) in an emotional Stroop task after experimentally provoking aggressive behavior in healthy participants. Compared to a non-provoked group, these individuals showed increased early (P2) and late (P3) positive amplitudes for all facial expressions. For the P2 amplitude, the effect of provocation was greatest for threat-related expressions. Beyond this, a bias for emotional expressions, i.e., slower reaction times to all emotional expressions, was found in provoked participants with a high level of trait anger. These results indicate significant effects of aggression on information processing, which last beyond the aggressive encounter even in healthy participants.

2.1. Introduction

Aggression is a common social behavior in both humans and animals. Not surprisingly, aggression and violence are among the leading causes of death worldwide (e.g., more than 1.6 million lives in 2000) and exert enormous economic costs (Krug et al., 2002). Neuroscientific research has mainly focused on pathologic aggression (e.g., Blair, 2004; Raine, 1989; Raine & Venables, 1988). However, aggression is also common in psychologically and neurologically healthy individuals. Aggression is necessary for human survival as it serves important purposes of allowing an individual to compete effectively for limited resources and to establish and maintain his/her position in society. The omnipresence of aggression and its impact on our everyday lives highlights the importance of finding an explanation of its causes and underlying mechanisms.

Information processing patterns at "higher levels" (e.g., scripts and schemata) have frequently been proposed as a possible explanation for aggressive behavior (e.g., Anderson & Bushman, 2002; Bushman & Anderson, 2001; Dodge & Crick, 1990; Huesmann, 1988). Even so, researchers have started to investigate the influence of aggression on basic information processing operations in healthy individuals only in the last decade. These studies show alterations in the cortical activity of healthy participants *while* they were engaged in a reactive aggression paradigm (Krämer et al., 2007; 2008; Lotze et al., 2007). For instance, participants with a high level of trait aggression displayed an enhanced early frontal negative event-related potential (ERP) in trials with high provocation while deciding about punishing an opponent (Krämer et al., 2008). A similar study with functional magnetic resonance imaging (fMRI) (Krämer et al., 2007) revealed that, in this decision phase, the activity in the rostral and dorsal parts of the anterior cingulate cortex and the anterior insula was greater in highly provocative than less provocative trials. Activity in these brain areas has been associated with emotional processing and this might reflect heightened emotional involvement of the participants under high provocation. Furthermore, an increase in activity in the medial prefrontal cortex (mPFC) has been reported during retaliation in a similar paradigm (Lotze et al., 2007). Enhanced activity in the dorsal mPFC might represent a stronger need for conflict management and response selection in the provoking situations. Increased activity in the ventral mPFC might indicate affective processes, such as compassion with the opponent. Hence, these findings show changes in the processing of information *during* an aggressive encounter in a laboratory setting.

From a therapeutic point of view, it may be even more interesting to investigate why aggressive behavior is often hard to stop and why it is easily transferred from one setting to another. Thus, the aim of the current study is to discover possible alterations in psychophysiological indicators of information processing *after* an aggressive encounter.

So far, this has only been addressed by a few behavioral studies, which have mainly focused on the influence of trait anger on reaction times for responding to threat-related stimuli. These studies have revealed that trait anger (Cohen et al., 1998; Eckhardt & Cohen, 1997; van Honk et al., 2001a; 2001b), previous self-reported aggressive experience (Smith & Waterman, 2004), and criminal convictions for violent offending (Smith & Waterman, 2003) predict an information processing bias for threat- or aggression-related material in various cognitive tasks. Thus, individuals with a high level of trait anger, who have experienced many incidences of aggression or violence seem to spend more attention on threat- or aggression-related information than less angry, aggressive, or violent individuals. This results in slower reactions to stimuli which are threat-related (emotional Stroop task) or surrounded by threat-related stimuli (visual search task) compared to neutral stimuli. In addition, there is evidence that participants with a high level of trait anger, who are experimentally induced to experience anger, process task irrelevant anger-related material in an emotional Stroop task (Cohen et al., 1998) and a visual search task (Eckhardt & Cohen, 1997). Nevertheless, the underlying mechanisms of these changes in information processing associated with anger and/or aggression remain unclear. Research has only investigated differences in behavioral responses (i.e., reaction times) between individuals with high and low levels of trait anger or self-reported aggression. However, impaired reaction times are only an indirect measure of attention towards threat-related material or of an information processing bias.

Recent neuroimaging studies have proposed the involvement of a neural network consisting of the amygdala, the ventral anterior cingulate, and the ventral striatum in the processing of facial signals of aggression (Beaver et al., 2008; Passamonti et al., 2008). In particular, the ventral striatum and its associated dopaminergic system seem to play a specific role in the recognition of angry facial expressions. Selectively impaired recognition of angry expressions has been reported in patients with lesions in the ventral striatum (Calder, Keane, Lawrence, & Manes, 2004) as well as after the administration of the dopamine antagonist sulpiride to healthy participants (Lawrence, Calder, McGowan, & Grasby, 2002). Thus, biased responses for angry or threat-related material in participants

with high state and/or trait anger or aggression might steam from an increased striatal activity.

In addition to functional neuroimaging, event-related potentials (ERP) studies might help to shed light at attention-related cortical processes related to anger and aggression. Because of their excellent temporal resolution, ERPs allow a finer, more sensitive, and more direct examination of differences in the time course and cortical resources of information processing (Hillyard & Kutas, 1983). This is especially important, as numerous behavioral studies have reported no differences in the reaction times of healthy participants towards threat-related and neutral stimuli in cognitive tasks like the emotional Stroop task or the visual search task (for a meta-analytic review, see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007). However, even in the absence of behavioral effects, significant differences could be found in the ERPs in some of the studies (Bar-Haim, Lamy, & Glickman, 2005; Bernat, Bunce, & Shevrin, 2001; Carretié, Martín-Loeches, Hinojosa, & Mercado, 2001a; Thomas, Johnstone, & Gonsalvez, 2007; Weinstein, 1995).

For instance, Thomas et al. (2007) reported greater parietal positivities to threat-related compared to neutral words in an emotional Stroop task with healthy individuals. Although there were no differences in the reaction times, increased P2 (150 to 210 ms) and P3 (340 to 600 ms) amplitudes indicate an enhanced processing of threat-related compared to neutral words. Similar ERP responses have been found during the processing of emotional material when pictures of facial expressions were used. These studies also found rapid effects (< 250 ms post-stimulus), indicating a very early preferential processing or categorization of emotional – especially threat-related – facial expressions (Ashley, Vuilleumier, & Swick, 2004; Bar-Haim et al., 2005; Bediou, Eimer, d'Aato, Hauk, & Calder, 2009; Eimer & Holmes, 2002; Williams, Palmer, Liddell, Song, & Gordon, 2006), followed by an alteration of later stages of ERP responses (Eimer & Holmes, 2002; Schupp et al., 2004; Williams et al., 2006). Alterations in the P2 and P3 amplitudes are thus sensitive indicators for early and later processing stages of emotional information. However, the levels of trait or state anger or aggression were not measured in these studies.

In summary, research indicates strong influences of anger and/or aggression on information processing. Trait anger and trait aggression are associated with reaction time biases towards threat- and aggression-related stimuli. Furthermore, healthy individuals show altered information processing *while* involved in aggressive behavior. However, the influence of experimentally induced aggression on information processing *beyond* an

aggressive encounter and its underlying neural mechanisms has not been reported. Therefore, the present paper reports on an ERP study that investigated this research question.

Like previous studies, our present ERP study used an emotional Stroop task (Williams et al., 1996) to investigate information processing biases. This task requires the participant to identify the color of an emotional word, picture, or facial expression as fast as possible while ignoring its emotional content. An information processing bias (or interference) is inferred when the color naming takes longer with a threat-related stimulus than with a neutral stimulus. This has been frequently reported for clinically and subclinically anxious individuals (see Bar-Haim et al., 2007), but also for individuals with high levels of trait anger (see above). Before performing an emotional Stroop task with happy, neutral, angry, and fearful facial expressions, the participants took part in a competitive reaction time task. This task was a modified Taylor Aggression Paradigm (TAP; Taylor, 1967), which was used to induce aggression in half of our group of healthy participants.

We expected differences in the reaction times of the provoked and non-provoked participants in the emotional Stroop task. In particular, we hypothesized an information processing bias that is longer reaction times for angry and fearful facial expressions in the provoked participants. Furthermore, we anticipated an increase in both early (P2) and late (P3) positive amplitudes in the provoked participants compared to the non-provoked participants for angry and fearful facial expressions.

2.2. Material and Methods

Participants

Twenty students of the University of Trier (10 female and 10 male, mean age = 23 years, *SE* = 0.6, range = 20–29 years) took part in the study. Exclusion criteria were left-handedness, color blindness, psychiatric disorders, regular medication (besides contraceptives), or any acute or chronic medical disease. The study had been approved by the local ethics committee. Participation was compensated with €30 (approximately US-$40). The participants were randomly assigned to either an experimental (provoked participants) or a control (non-provoked participants) group, but sex was balanced across groups (five male and female participants in each group).

Materials

The Taylor Aggression Paradigm

Aggression was elicited by and assessed with a modified version of the Taylor Aggression Paradigm (TAP; Taylor, 1967) . The TAP has shown good construct, external, discriminant, and convergent validity (Anderson, Lindsay, & Bushman, 1999; Bernstein, Richardson, & Hammock, 1987; Giancola & Chermack, 1998; Giancola & Zeichner, 1995).

The participants were led to believe that they were playing a competitive reaction time game with another participant who they met before the experiment started. The TAP consisted of 30 trials, which were divided into three blocks of ten trials. The participants' task was to react as fast as possible to a green square shown on the screen by pressing a key. They were told that whoever reacted slower would receive a blast of aversive noise. Prior to each trial, the participants had to set the volume and the duration of the noise for the opponent on two separate scales each ranging from 0 to 10. Corresponding to the 11 levels, the duration could be varied between 0 (level 0) and 5 seconds (level 10) in 0.5 second increments. The volume varied between 60 (level 1) and 105 dB (level 10) in 5 dB increments. The level 0 on the volume scale corresponded to 0 dB. After each trial, the participants received feedback about the outcome of the trial, i.e., whether they won or lost, as well as about the opponent's settings. In fact, there was no opponent and the outcome of the trials was held constant for all participants – each of them won and lost half of the trials. The experimenter also set in advance the "opponent's" volume and duration settings according to the block and experimental condition of the participant. During the first block, all participants received short and gentle noises when they lost a trial (volume: M = 62.5 dB, range 0–70 dB; duration: M = .075 s, range 0–1.5 s). Participants of the non-provoked group received noises of the same volume and duration during the second and third block as well. Participants of the provoked group were exposed to noises of intermediate volume and duration in the second block (volume: M = 82.5 dB, range 75–90 dB; duration: M = 2.75 s, range 2–3.5 s) and of high volume and duration in the third block (volume: M = 99 dB, range 90–105 dB; duration: M = 4.4 s, range 3.5–5 s) when they lost a trial. The volume and duration settings of the participants were recorded in each trial from 0 to 10. For each participant and each trial, an average of the volume and duration setting was computed, except for those trials in which one of the settings was 0. In that case, the total score was set to 0, since no noise would have been presented to the opponent and this trial would not have constituted an aggressive act. Finally, the 10 trials which belonged to one

block of TAP were averaged for each participant. These values were then used as the dependent variable of aggressive behavior in the statistical analysis.

The emotional Stroop task

Stimuli were taken from Ekman and Matsumoto's *Japanese and Caucasian Facial Expressions of Emotion* (JACFEE) and *Japanese and Caucasian Neutral Faces* (JACNeuF) (Matsumoto & Ekman, 1988). We used pictures of four male and four female faces, displaying happy, angry, fearful, and neutral expressions. Duplications of each picture were colored in transparent red, blue, yellow, and green, resulting in 32 different stimuli. In total, the emotional Stroop task consisted of eight practice trials and 256 experimental trials. Each trial comprised the presentation of a colored facial expression, which was backwardly masked after 26.7 ms (2 frames at 75 Hz), since backward masking after 25 to 30 ms has been reported to produce large effects in regard to anger (e.g., Putman et al., 2004; van Honk et al., 2001a). The masks were individually constructed for each facial expression and represented a distorted version of the picture, keeping hue and saturation constant. The mask remained on the screen until the participant responded by orally naming the color of the picture. The participants were instructed to respond as fast as possible, whilst making as few errors as possible. The responses were recorded via microphone, and reaction times (i.e., voice onset times) were measured for each trial. The voice onset times were measured online with a microphone and serial voice response box (both provided by Psychology Software Tools, Inc.). Prior to each facial presentation, a fixation cross appeared at the center of the screen for 1990 ms (the timing of a single trial is displayed in Figure 3).

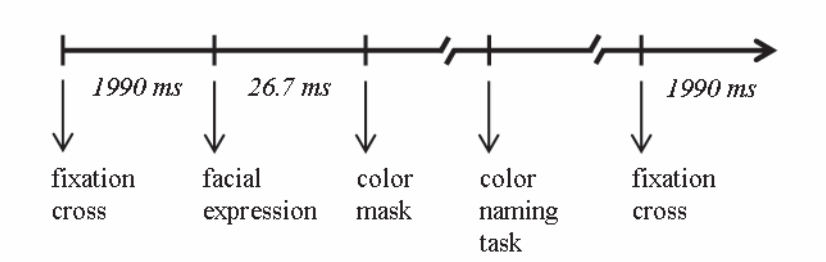

Figure 3. Time line for a single trial of the emotional Stroop task

All stimuli were presented in the center of the screen on a black background. The image sizes were 5.55" x 5.20" and the vertical and horizontal visual angles were 0.28° and 0.26°, respectively. The stimuli were presented in a pseudorandomized fashion, which allowed a presentation of no more than three pictures of the same color or facial expression in a row. The task was divided into two random blocks of 128 pictures by a 2 min break.

Trait measures

Trait anger was measured prior to the experiment with the subscale anger of the *Buss and Perry Aggression Questionnaire* (BPAQ; Buss & Perry, 1992; German version: Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2004). The BPAQ is a 29-item questionnaire, which consists of four subscales: anger, physical aggression, verbal aggression, and hostility. The four subscales have shown high internal and construct validity as well as high test-retest reliability (Buss & Perry, 1992; Harris, 1997). All items are scored using a 5-point Likert scale (1 = never or hardly ever applies to me, and 5 = very often applies to me). Two items on the scale are reverse scored. The BPAQ anger subscale is a good predictors for information processing biases in the emotional Stroop task and other cognitive tasks (Smith & Waterman, 2003, 2005).

Procedure

All participants were tested individually. We invited them to a preliminary interview, at which we checked for exclusion criteria and informed them about the aim of the study and the experimental procedure. They were told that the study would concern the investigation of the relationship between the steroid hormone cortisol, personality, and the perception of and reaction to visual stimuli. After a description of the experiment, the EEG and salivary cortisol measurement, written informed consent was obtained form all participants. Finally, the participants also received a battery of personality questionnaires to fill out at home as well as home sampling devices for salivary cortisol.

The experimental procedure was kept constant for all participants. On arrival, the participant was acquainted to another participant of the same sex, who was in fact a confederate of the experimenter. The participant and the confederate both handed over the filled-out questionnaires and the salivary cortisol samples. The participants were then led to the EEG laboratory, where they were comfortably seated in a dimly lit sound-attenuated room one meter away from the 19" computer screen with a computer keyboard on a table in front of them. After the EEG electrodes were applied, the participants were left alone in the room for the remainder of the experiment and received all instructions via the computer screen. All participants first played the Taylor Aggression Paradigm, which lasted for about ten minutes, and then performed the emotional Stroop task for about 15 minutes. Before and after the TAP, as well as after the emotional Stroop task, the participants gave salivary cortisol samples, filled out a short mood questionnaire, and relaxed while the baseline resting EEG was measured during a 2 min period. A forth salivary cortisol sample was collected shortly before the participants left the laboratory. The results of these cortisol

data are reported elsewhere (Böhnke, Bertsch, Kruk, & Naumann, under review). Finally, the participants were debriefed about the true aim of the study as well as the TAP and the confederate. We thanked and compensated them for their participation.

Stimulus presentation and response logging were controlled for using E-Prime software (Version 1.1, Psychology Software Tools, Inc.) and a serial voice response box and microphone. The experiment, including preparation and debriefing had a duration of 90 minutes.

EEG recording and quantification

The EEG was recorded from 32 electrode sites according to the 10–10 electrode reference system (Chatrian, Lettich, & Nelson, 1988) including the mastoids with the Easy-Cap electrode system (Falk Minow Services). All sites were referenced to vertex (Cz). A bipolar horizontal electrooculogram (EOG) was recorded from the epicanthus of each eye, and a bipolar vertical EOG was recorded from supra- and infra-orbital positions of the left eye. The EEG and the EOG were recorded with Ag/AgCl electrodes. Prior to the electrode placement, the electrode sites on the participant's scalp and face were cleaned with alcohol and gently abraded. All impedances of the EEG electrodes were below 5 kΩ. EEG and EOG were amplified with a 32-channel SynAmps Model 5083 amplifier (input impedance: 10 MΩ; Neuroscan, Inc.) in AC mode. The pass-band was set to 0.05 to 40 Hz (-12 dB/octave rolloff); the signals were digitalized at 500 Hz and stored to hard disk for later analysis.

The EEG was re-referenced to linked mastoids. Artifacts due to eye movements were corrected via the algorithm developed by Gratton, Coles, and Donchin (1983). Trials with non-physiological artifacts were excluded from analysis via semiautomatic artifact rejection. EEG and EOG were epoched off-line into 1400-ms periods, starting 200 ms prior to stimulus onset and ending 1200 ms after stimulus onset. A baseline correction was performed using the first 200 ms as a reference. Separate averages were computed for each electrode, individual, and facial expression condition (happy, angry, fearful, and neutral).

Based on visual inspection of grand average ERPs, averaged across all participants and emotional facial expressions and a point-by-point inspection of effect sizes (Strelzyk, Britz, & Naumann, under review) performed on all channels and time-frames, the following two stimulus-locked ERP components (peak amplitude relative to baseline) were identified and used for further analysis: P2 (the first major positive wave occurring 160–200 ms post-stimulus) and P3 (300–400 ms). The P2 and P3 waveforms had a

centroparietal to parietal maximum. Therefore, we used the following nine central to parietal positions for further analyses: C3, Cz, C4, CP3, CPz, CP4, P3, Pz, and P4.

Statistical analyses

Aggressive behavior in the TAP. To check whether the induction of aggressive behavior in the provoked group was successful, we performed a 2 × 3 analysis of variance (ANOVA) including the factors Provocation (provoked, non-provoked participants; between-subject) and TAP Block (Blocks 1, 2, 3; repeated measure).

Behavioral data in the emotional Stroop task. Outliers (± 2 SD) and trials with incorrect responses were individually rejected for each participant. We calculated the mean reaction time for each of the four emotional facial expression conditions. Bias scores were computed by subtracting the mean reaction time for neutral pictures from each of the three emotional categories (e.g., the individual mean response latencies for angry faces minus the individual mean response latencies for neutral faces; see, for instance, Smith & Waterman, 2003; 2005; van Honk et al., 2001a). Note that positive bias scores are referred to as *interference* and negative scores as *facilitation*. To examine differences in reaction times towards facial expressions, we submitted the mean correct responses to a 2 × 3-mixed-design ANOVA examining the factors Provocation (provoked, non-provoked participants; between-subject) and Facial Expression Conditions (happy, angry, fearful; within-subject).

Electrophysiological data in the emotional Stroop task. For the ERP average amplitudes, we calculated separate 2 × 4 × 3 × 3-mixed-design ANOVAs including the factors Provocation (provoked participants, non-provoked participants; between-subject), Facial Expression Conditions (happy, angry, fearful, neutral; repeated measure), Caudality (central, centroparietal, parietal; repeated measures), and Lateralization (left, middle, right; repeated measures) for each component (P2, P3).

Additional analyses. As trait anger has been previously found to be associated with an information processing bias (i.e., more interference) for threat-related stimuli (e.g., Eckhardt and Cohen, 1997; van Honk et al., 2001a; Smith and Waterman, 2003; 2005), we recalculated the statistical analyses for the behavioral and the electrophysiological data including the continuous between-subject factor of trait anger. For the *behavioral data*, we performed a 2 × 3-mixed-design ANOVA including the factors Provocation (provoked participants, non-provoked participants; between-subject), Facial Expression Conditions (happy, angry, fearful; repeated measure), and Trait Anger (continuous between subject). Prior to this analysis, the mean trait anger scores were z-standardized (Aiken & West,

1991). For the *electrophysiological data*, separate 2 × 4 × 3 × 3-mixed-design ANOVAs were calculated for the P2 and P3 components including the factors Provocation (provoked participants, non-provoked participants; between-subject), Facial Expression Conditions (happy, angry, fearful, neutral; repeated measure), Caudality (central, centroparietal, parietal; repeated measure), Lateralization (left, middle, right; repeated measure), and Trait Anger (continuous between subject; again, the z-standardized mean trait anger values were used for this analysis; Aiken and West, 1991).

For all ANOVAs, the degrees of freedom were Huynh-Feldt corrected if the assumption of sphericity was violated (Huynh & Feldt, 1976). We calculated Hays' ω^2 (Hays, 1974) as an effect size measure, with 1% considered a small effect, 5% considered a medium effect, and 14% considered a large effect (Cohen, 1988). A power analysis performed with GPOWER 2.0 (Buchner, Erdfelder, & Faul, 1996; Erdfelder, Faul, & Buchner, 1996) revealed a statistical power of 1-ß ≥ .90 for medium sized interaction effects of $\omega^2 = .05$ for the ERP data. According to Cohen (1962, 1988, 1992), values of 1-ß ≥ .80 can be regarded as adequate statistical power for the interpretation of non-significant effects.

In case of significant effects, we used Dunn's Multiple Comparison Tests as well as Pearson product moment correlations as post-hoc tests. All statistical analyses were conducted with SPSS for Windows (Version 14.0, SPSS Inc.).

2.3. Results

Aggressive behavior in the TAP

The experimental manipulation of aggressive behavior was successful. The provoked group showed generally more aggressive behavior ($M = 3.3$, $SE = 0.4$) than the non-provoked group ($M = 2.1$, $SE = 0.4$), $F(1,18) = 4.59$, $p = .046$, $\omega^2 = .15$. Aggressive behavior increased from the first to the third block of the TAP, $F(2,36) = 12.92$, $p = .001$, $\omega^2 = .28$. Post-hoc tests showed significant differences between Block 1 ($M = 2.1$, $SE = 0.3$) and Block 3 ($M = 3.4$, $SE = 0.4$, $p < .010$) as well as between Block 2 ($M = 2.6$, $SE = 0.3$) and Block 3 ($M = 3.4$, $SE = 0.4$, $p < .010$) of the TAP. There was also a significant interaction between Provocation and TAP Block, $F(2,36) = 9.03$, $p = .003$, $\omega^2 = .21$. According to post-hoc tests, aggressive behavior increased only in the provoked group ($p < .010$) but not in the non-provoked group. Moreover, the mean aggressive behavior of the groups differed in TAP Block 2 ($p < .050$) and Block 3 ($p < .010$), but not in Block 1,

where no provocation had taken place. Means and standard errors of each group and TAP block are presented in Figure 4.

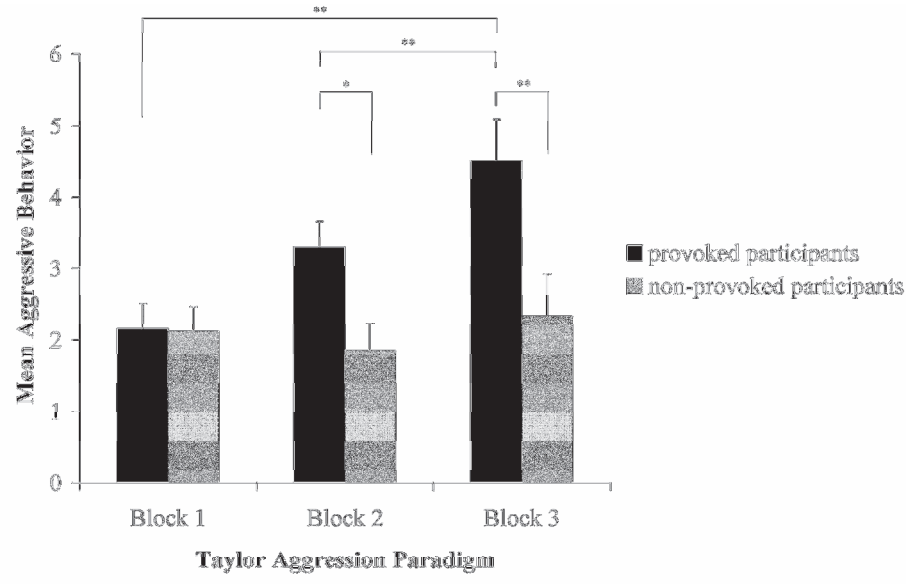

Figure 4. Mean aggressive behavior of the provoked and the non-provoked group in the three blocks of the Taylor Aggression Paradigm. Mean aggressive behavior represents the average of the loudness and duration setting. Each block consists of ten trials (Block 1: trial 1 to 10, Block 2: trial 11 to 20, Block 3: trial 21 to 30). The error bars represent ± one standard error.

Behavioral data in the emotional Stroop task

The error rate in this task was 2.2% (M = 5.6, SE = 0.8) and the provoked and non-provoked participants did not differ in their error rates, $t < 1.00$, $p > .050$. The behavioral performance in the emotional Stroop task of the provoked and non-provoked participants is summarized in Table 1.

Analysis of the bias scores revealed a marginally significant main effect of Provocation, $F(1,18) = 3.08$, $p = .096$, $\omega^2 = .09$, with provoked participants showing more interference (M = 11.7, SE = 6.5) for all emotional expressions (i.e., they were slower to name the color of emotional compared to neutral expressions) than the non-provoked participants (M = -4.4, SE = 6.5). Beyond this, no further significant effects were found (all Fs < 1.0, ps > .050).

Table 1. Reaction times and bias scores in the emotional Stroop task (means and standard errors).

	all participants		provoked participants		non-provoked participants	
	M	*SE*	*M*	*SE*	*M*	*SE*
RT neutral	527.9	12.8	526.8	23.7	529.1	11.5
RT happy	533.6	15.1	540.7	26.5	526.5	15.6
RT angry	530.8	15.6	537.4	27.6	524.1	15.8
RT fearful	530.4	14.5	537.5	26.6	523.3	13.1
Bias happy	5.6	4.2	13.9	6.0	-2.6	6.0
Bias angry	2.8	5.6	10.6	7.9	-4.9	7.9
Bias fearful	2.5	5.4	10.7	7.6	-5.7	7.6

M = means (estimated marginals), *SE* = standard error. *Note.* Reaction time was measured in units of ms. Bias scores represent difference values (mean reaction times for emotional minus neutral expressions) in ms.

Electrophysiological data in the emotional Stroop task

Figure 5 shows grand average ERP responses to the facial expressions for the provoked and the non-provoked participants, averaged over the four facial expression conditions for all electrode positions. The general morphology of the waveform included a prominent, early negative peak at 135 ms (N1), followed by a positive wave at 180 ms (P2), a second negative wave at 250 ms (N2), and a final positive wave at 350 ms (P3). Descriptively large differences between the provoked and non-provoked participants can be observed in the time window of the P2 (160–220 ms) and P3 (300–400 ms) (see Figure 5).

P2 (160–200 ms). The P2 amplitude was greater in the provoked than in the non-provoked group, $F(1,18) = 5.77$, $p = .027$, $\omega^2 = .19$. This main effect of Provocation was qualified by a significant interaction between Provocation, Caudality, and Lateralization, $F(3,72) = 2.93$, $p = .031$, $\omega^2 = .04$. According to post-hoc tests, the group difference was greatest at P3 and Pz ($p < .010$). Moreover, there was a significant interaction between Provocation and Facial Expression, $F(3,54) = 3.78$, $p = .016$, $\omega^2 = .09$ (see Figure 6). Post-hoc tests indicated that the difference between the provoked and non-provoked participants was greatest for fearful and angry expressions (both ps $< .001$), although significant group differences were also found for happy and neutral expressions (both ps $< .010$).

P3 (300–400 ms). We found a large main effect of Provocation, $F(1,18) = 4.70$, $p = .004$, $\omega^2 = .16$, with a greater positivity in the provoked compared to the non-provoked participants. In addition, there was a significant interaction between Facial Expression and

Lateralization, $F(6,108) = 2.82$, $p = .014$, $\omega^2 = .04$. Post-hoc tests showed that the P3 amplitude was greater for happy than for neutral expressions at all electrode positions ($p < .050$), and greater for neutral than for angry expressions at right hemispheric electrode positions (C4, CP4, and P4; $p < .050$).

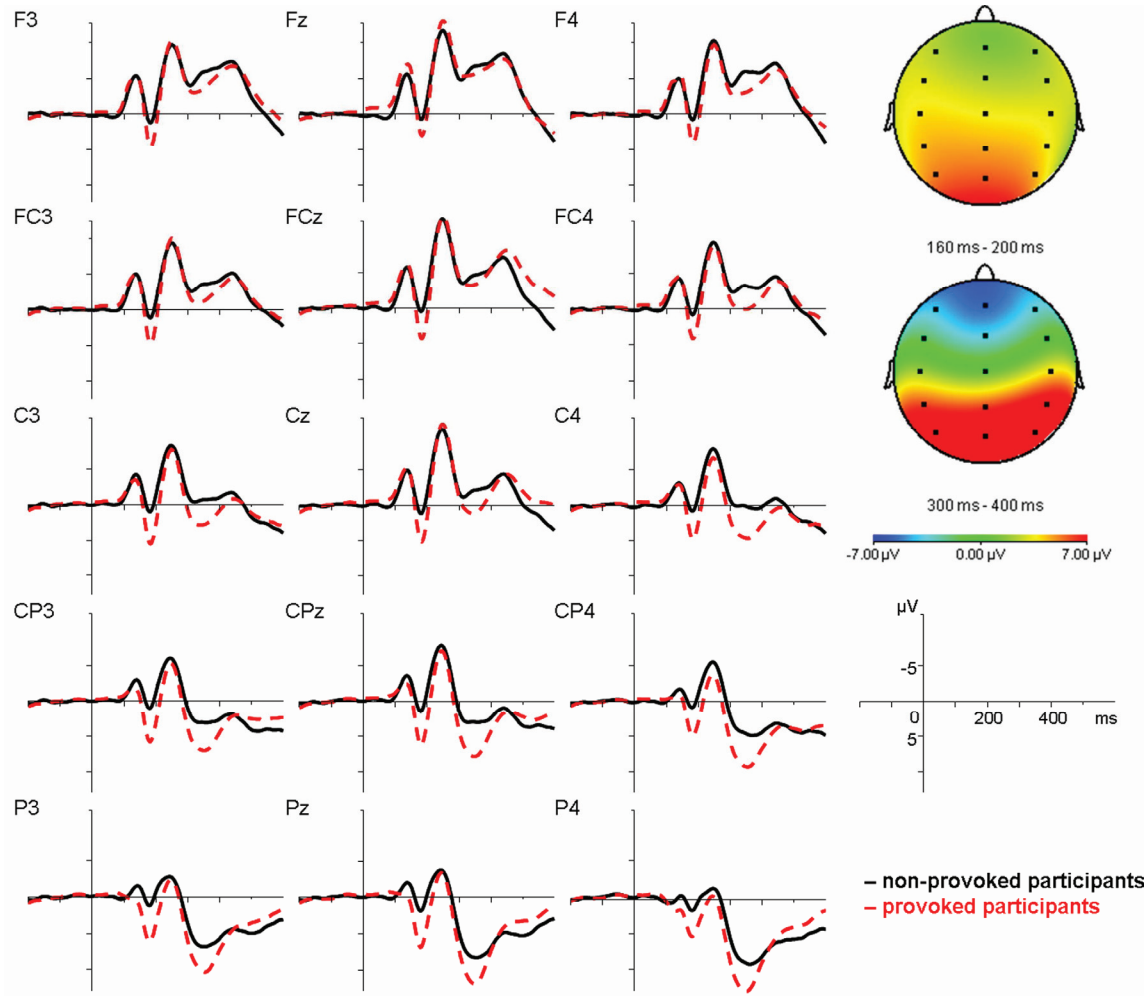

Figure 5. Grand average ERP waveforms for the provoked (--) and the non-provoked (–) group averaged over the four facial expressions (happy, neutral, fearful, and angry) and difference maps averaged over the four facial expressions (happy, neutral, fearful, and angry) for the time domains of the P2 (160–200 ms) and P3 (300–400 ms). In the difference maps, red indicates a greater positivity in the provoked than in the non-provoked group and blue refers to a greater positivity in the non-provoked compared to the provoked group.

Additional analyses

As in previous studies a greater information processing bias (i.e., more interference) was found in the emotional Stroop task for individuals with higher levels of trait anger, we recalculated the repeated measure ANOVAs and included the continuous between-subject factor of trait anger.

For the *behavioral data*, this analysis revealed a significant main effect of Provocation, $F(1,18) = 7.10$, $p = .017$, $\omega^2 = .23$, a significant main effect of Trait Anger, $F(1,18) = 5.06$, $p = .039$, $\omega^2 = .17$, as well as a significant interaction between Provocation and Trait Anger, $F(1,18) = 6.07$, $p = .025$, $\omega^2 = .20$. Again, the provoked participants showed more interference for all emotional expressions than the non-provoked participants. Bivariate correlations revealed a positive association between trait anger and the bias scores for all emotional expressions only in the provoked group, $.81 \leq r \leq .86$, $p \leq .005$. Thus, participants with a high level of trait anger who were experimentally provoked showed more interference to all emotional expressions (i.e., they were slower to name the color of emotional compared to neutral expressions) than participants with lower levels of trait anger and non-provoked participants.

For the *electrophysiological data*, the additional analyses did not reveal any further effects.

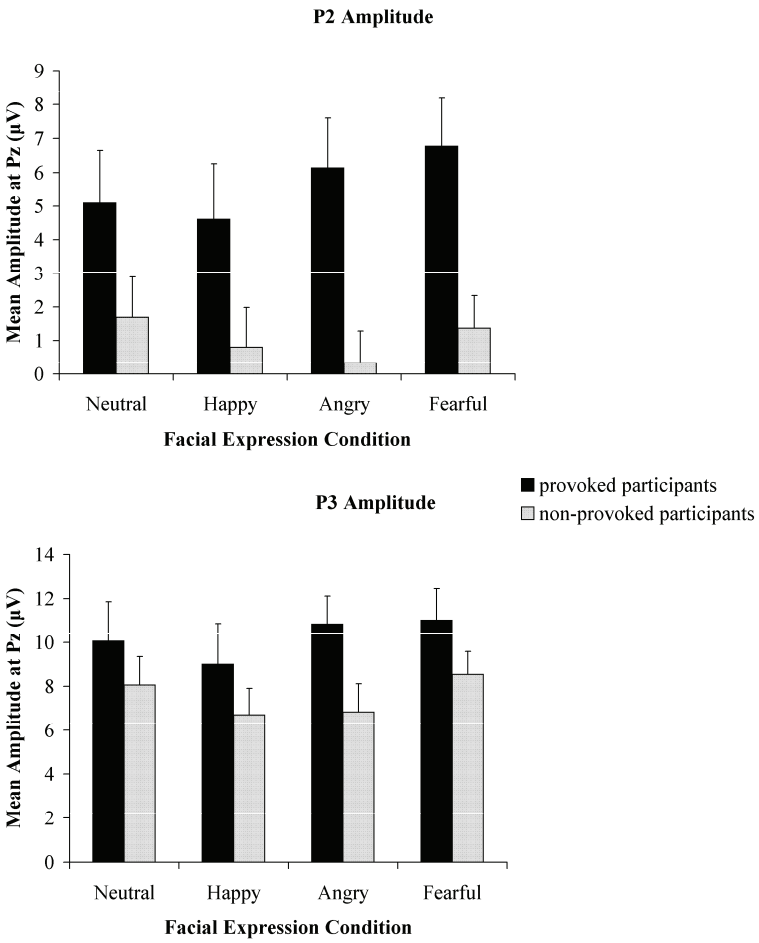

Figure 6. Mean P2 and P3 amplitudes (µV) for the provoked and non-provoked participants at Pz electrode site separately for the neutral, happy, angry, and fearful expressions.

2.4. Discussion

Changes in information processing have been discussed in the context of aggression and higher level information-processing patterns (i.e., scripts or schemata), and have frequently been used to explain the occurrence of aggressive behavior (Anderson & Bushman, 2002; Dodge & Crick, 1990; Huesmann, 1988). There are also indications from recent ERP and fMRI studies that, even in healthy individuals, information processing is changed *while* they are involved in aggressive encounters (Krämer et al., 2008; Krämer et al., 2007; Lotze et al., 2007). Moreover, anger, self-reported aggression, and violent convictions have been associated with information processing biases for threat- and aggression-related material in several behavioral studies (Cohen et al., 1998; Eckhardt & Cohen, 1997; Smith & Waterman, 2003, 2004; van Honk et al., 2001a; 2001b). However, the influence of experimentally induced aggression on information processing and its underlying neural mechanisms has not been reported. Thus, in this ERP study, we measured reaction times as well as ERPs during the presentation of facial expressions in an emotional Stroop task (Williams et al., 1996) after provoking aggressive behavior in half of our healthy participants with the Taylor Aggression Paradigm (Taylor, 1967).

The provocation of aggressive behavior was successful. On average, the participants in the provoked group set significantly louder and longer noises for their opponents when provoked (TAP Blocks 2 and 3) compared to the non-provoked group and compared to TAP Block 1, where no provocation had taken place. This experimental provocation of aggression led to a changed processing of facial expressions in both early and later stages of information processing, and on the behavioral level to more interference for emotional facial expressions.

Electrophysiological data in the emotional Stroop task

One aim of the study was to investigate differences in the ERPs directly after experimentally induced aggression as indicators for the processing of facial expressions in the emotional Stroop task. The principle advantage of ERPs is their excellent temporal resolution, which allows for the direct examination of differences in information processing and its time course (Hillyard & Kutas, 1983). The ERP results showed large differences between the provoked and non-provoked participants in two positive ERP components: the P2 and P3. This large main effect of provocation indicates that the experimental provocation had an impact on information processing in early as well as later stages of information processing, partly independent of the emotional content of the facial expressions.

First, the provoked participants showed an enhanced P2 amplitude compared to the non-provoked participants at posterior electrode positions. This very early component has been associated with bottom up or low level processing of information, such as stimulus classification and categorization (Crowley & Colrain, 2004). In this time window, we also found a significant interaction between provocation and facial expression condition, due to a greater positivity for threat-related (angry and fearful) expressions in the provoked participants. Similarly, Carretié et al. (2001b) reported a greater posterior P2 amplitude for negative compared to neutral and positive emotional pictures. This was interpreted in terms of a greater mobilization of attentional resources. Recently, Thomas et al. (2007) also found a greater P2 amplitude for threat-related compared to neutral words in an emotional Stroop task.

Schapkin, Gusev, and Kuhl (2000) understand the underlying processes of enhanced centro-parietal P2 amplitudes for emotional relative to neutral stimuli as an early global affective evaluation, which appears to be critical for further approach or withdrawal behavior. According to studies by Calder and colleagues (Beaver et al., 2008; Calder et al., 2004; Lawrence et al., 2002; Passamonti et al., 2008), a neural network consisting of amygdala, anterior cingulate, and ventral striatum is involved in the processing of facial signals of aggression, i.e., angry facial expressions. One might speculate that the induction of aggression in the present study might have altered early global affective evaluation or categorization processes of all, and particularly threat-related facial expressions, potentially involving an altered striatal activity. However, this can only be resolved with studies using simultaneous measurements of EEG and fMRI, a now evolving technique (see, for instance, Debener, Ullsperger, Siegel, & Engel, 2006).

Second, we found a greater P3 amplitude in the provoked compared to the non-provoked participants. This component, which had a definite parietal localization, was independent of the emotional content of the facial expressions. An enhanced P3 with a centroparietal distribution has been previously found for less frequent, more salient and meaningful stimuli (Johnson, 1993; Naumann et al., 1992b; Picton, 1992). Unlike preceding ERP studies, we did not find a greater P3 amplitude for threat-related (Thomas et al., 2007) or emotional (Carretié et al., 2001a; Herbert, Kissler, Junghöfer, Peyk, & Rockstroh, 2006; Naumann, Bartussek, Diedrich, & Laufer, 1992a) compared to neutral stimuli. Contrary to Thomas et al. (2007), we found significantly greater P3 amplitudes for neutral than for angry expressions, at least at right hemispheric electrode sites. However, Thomas et al. (2007) reported that P3 amplitude differences between threat and neutral

words were considerably smaller when word meaning was not relevant for the task performance. It should be noted that there are several differences concerning the experimental design and material between the study of Thomas at al. (2007) and the present study (i.e., the use of angry and neutral words versus happy, angry, fearful, and neutral facial expression as well as a stimulus presentation time of 200 ms versus 26.7 ms). In particular, the different presentation times might at least partly account for the dissimilar results (see, for instance, Kiss & Eimer, 2008). Moreover, none of those earlier studies included an experimental induction of aggression prior to the processing of emotional and neutral stimuli.

Recently, the P3 has been discussed with regard to the locus coeruleus norepinephrine (LC-NE) system (Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005). According to this theory, motivationally significant stimuli elicit a greater P3 amplitude due to a norepinephrine induced phasic enhancement of neural responsivity in the neocortex (especially the temporal-parietal junction). This enhancement is triggered by the outcome of task-relevant decision processes (e.g., stimulus categorization). It is supposed to optimize information processing by modulating post-decision response processes. According to Nieuwenhuis et al. (2005), the LC-NE system might be a generator of the P3.

When provoked, all facial expressions are motivationally significant, because they might contain important and lifesaving information about the opponent. Thus, provocation affects the processing of facial expressions at different levels. First, it alters early global affective evaluation processes. At this early stage of information processing, all facial expressions and particularly threat-related expressions are classified as motivationally significant (indicated by increased P2 amplitudes). Following this, an enhancement in the phasic LC-NE activity to *all* (motivationally significant) facial expressions results in an enhanced neural responsivity in the neocortex (indicated by increased P3 amplitudes). As mentioned above, such an interpretation has to be validated by joined fMRI and EEG measurements.

Behavioral data in the emotional Stroop task

Beyond the large group differences between provoked and non-provoked participants in the positive components of the ERPs, we also found behavioral effects related to the experimental induction of aggression. The provoked participants displayed more interference for all emotional (happy, angry, and fearful) facial expressions (i.e., they were slower to name the color of emotional compared to neutral facial expressions) than

the non-provoked participants. This was especially the case when trait anger was included as a continuous between-subject factor. This additional analysis revealed positive correlations between trait anger and the bias scores for all emotional expressions within the provoked group, indicating slower reaction times in participants with high levels of trait anger after provocation.

This is partly in line with the results of previous behavioral studies, which found an information processing bias for threat-related material associated with anger and aggression (e.g., Eckhardt and Cohen, 1997; Cohen et al., 1998; van Honk et al., 2001a; 2001b; Smith and Waterman, 2003; 2004b). However, the induction of aggression in the present study resulted in a rather broad, less specific change of information processing and a processing bias (i.e., more interference) for all emotional facial expressions. In contrast, the information processing bias of participants with high levels of trait anger, self-reported experiences of aggression, or violent incidences, which has been reported by other studies, was specific for threat- or aggression-related material. So far, it remains unclear why induced aggression should lead to a broader change in information processing. One could speculate that all emotional facial expressions gain relevance after being involved in an aggressive encounter. Facial expressions inform us more rapidly than language about the state of mind of other individuals and are, thus, biologically and socially salient stimuli in human nonverbal communication (LeDoux, 1998). Even a laughing face might be provoking in such a situation, as this person might be laughing at you. Moreover, the immediate and reliable awareness about potential friends and enemies might be more important and even lifesaving in the context of an acute aggressive encounter. This is supported by the ERP data of the present study. An increase in the P2 and P3 amplitudes in the provoked participants indicates a greater relevance and salience of all facial stimuli after an aggressive encounter independent of the individual's level of trait anger. In other words, the induction of aggression seems to produce a general gating effect of the neural response at the level of both early and later ERP components. Only in participants with high levels of trait anger, the provocation also resulted in behavioral differences, i.e., more interference for emotional expressions.

Limitations

Before strong conclusions can be drawn, three limitations of the present study should be noted. First, we did not include a control condition with non-facial stimuli in the present study. Hence, it remains unclear whether the changes in information processing

due to aggression are specific for facial expressions or are more general, going beyond (or not depending upon) facial expressions.

Second, like previous studies using the emotional Stroop task (e.g., Putman et al., 2004, Smith and Waterman, 2003; 2004, 2005; van Honk et al., 2001a; 2001b), we requested the participants to orally name the color of the presented stimuli. This might have introduced artifacts in the EEG. However, it should be noted, that the mean response latencies (around 530 ms) did not overlap with the time domains of investigated components (160–200 ms and 300–400 ms).

Third, we only found a marginally significant and emotion-unspecific effect of aggression in the behavioral data. Greater behavioral effects were found, when trait anger was included as a continuous between-subject factor. However, the study was not designed to investigate interaction effects of trait anger. As the statistical power for the behavioral data is not adequate to interpret non-significant effects, these behavioral results need to be replicated in a larger sample. Nevertheless, for the statistical analyses of the ERP data the statistical power was sufficient to interpret non-significant interaction effects (see Method and Material section). To overcome these limitations, a second study with more participants, which also includes a non-facial control condition, is in preparation.

Conclusion

In summary, this study showed that experimentally induced aggression has a strong impact on early as well as later stages of information processing. The ERPs revealed large differences between provoked and non-provoked participants during the processing of facial expressions in an emotional Stroop task, largely independent of the emotional content of the facial expressions and the individual level of trait anger. Moreover, aggression led to slower reaction times and therefore an information processing bias for emotional facial expressions, especially in participants with a high level of trait anger. Together with the findings from previous studies, our results demonstrate pronounced effects of aggression on information processing during and after an aggressive encounter. It is intriguing that even a mild provocation in a laboratory setting affects several stages of information processing and results in behavioral differences even up to 15 min after the aggressive encounter took place. This suggests profound effects from real-life conflicts and aggressive encounters on information processing and consequent behavior.

Chapter III

Exogenous Cortisol and Aggression Have Independent Effects on Information Processing – an ERP Study

Abstract

Stress is one of the major factors promoting aggression. Previous findings indicate that there is a relationship between stress and aggression and that both factors influence the processing of threat-related information. In an ERP study, we investigated the effects of the stress hormone cortisol and aggression on the processing of emotional faces. Healthy participants were exposed to high or low provocation of aggression after receiving either 20 mg hydrocortisone or a placebo. Thereafter, all participants performed an emotional Stroop task with angry, fearful, happy, and neutral facial expressions. Angry faces elicited enhanced fronto-central P2 amplitudes. After cortisol administration, this early processing bias for angry faces was diminished. Aggression led to increased posterior P1 and late positive potentials for all faces. These results indicate different effects of cortisol and aggression on information processing. While cortisol reduced the discriminating sensitivity for threat-related signals, aggression rather broadly changed early and later processing of all facial expressions.

3.1. Introduction

Stress has been identified as one of the major factors promoting aggression. There are indications that the stress system plays an important role in explaining individual differences in aggressive and violent behavior (van Goozen & Fairchild, 2006; Verona & Kilmer, 2007; Wommack & Deville, 2007). Acutely elevated levels of the stress hormone cortisol (corticosterone in rodents) have been related to enhanced aggressive behavior in humans and animals (e.g., Gerra et al., 2001a, 2001b; 2004; 2007; Kruk et al., 2004; Lopez-Duran et al., 2009).

Based on a series of experiments in rodents, Kruk et al. (2004) have suggested that the processing of social signals might play an important role in the association of stress (hormones) and aggression. In fact, pathologically aggressive individuals not only show an altered activity of the stress system (for reviews, see Cappadocia et al., 2009; Patrick & Verona, 2007; van Goozen et al., 2007), but also severe deficits in recognizing emotional facial expressions (Davidson et al., 2000; Herpertz & Sass, 2000). Emotional facial expressions are among the most significant and immediate social signals in personal communication (LeDoux, 1998). Thus, the inherent ability in rapidly recognizing and distinguishing different emotional expressions is necessary to read the emotional states of others and hence for effective social interaction (Dolan, 2002).

But even in healthy individuals cortisol and aggression seem to influence the processing of emotional facial expressions. There is some evidence for a preferential processing of angry facial expressions when cortisol levels are experimentally or pharmacologically increased in healthy participants (e.g., Putman, Hermans, & van Honk, 2007b; Roelofs et al., 2007). For instance, healthy young men who were exogenously administered cortisol showed an immediate memory bias for angry faces in a spatial working memory task (Putman et al., 2007b). In addition, a response bias for angry faces in an emotional Stroop task (i.e., slower reactions to angry relative to neutral faces) was reported in participants with high increases in cortisol levels to a social stressor (Roelofs et al., 2007). However, there are also indications of reduced anger biases in association with acutely elevated cortisol levels (e.g., Oei et al., 2009; Roelofs, Elzinga, & Rotteveel, 2005). Oei et al. (2009), for instance, recently reported a reduced interference by task-irrelevant negative pictures in a modified Sternberg working memory task after cortisol administration compared to a placebo.

Similar to cortisol, aggression has been found to influence the processing of threat-related material, such as angry facial expressions. Individuals with high levels of trait anger, previous self-reported aggressive experience, and criminal convictions for violent offending showed biased responses, i.e., slower reaction times for angry faces or other aggression-related stimuli in various cognitive tasks (Smith & Waterman, 2003, 2004; van Honk et al., 2001a; van Honk et al., 2001b). Moreover, when participants are induced to experience anger, they tend to process task irrelevant anger information in an emotional Stroop task (Cohen et al., 1998) and a visual search task (Eckhardt & Cohen, 1997). These findings could be seen as indications for the potential presence of information processing patterns that may be implicated in aggressive behavior (Todorov & Bargh, 2002) in pathologically aggressive as well as in "normal" individuals. Deficiencies in the processing of and responding to social signals, such as emotional facial expressions, might further increase the likelihood for maladaptive or inappropriate behavior (Geen, 2001), and hostile attribution bias could have negative effects on the cognitive construction of future social interactions.

Taken together, there is evidence for both a strong correlation between stress and aggression and for effects of the stress hormone cortisol and aggression on the processing of threat-related social signals, such as angry facial expressions. However, this evidence is primarily based on behavioral studies, which, for instance revealed slower reactions for angry faces in emotional Stroop tasks. Reaction times are only an indirect measure of information processing, though. Another useful method is the recording of event-related potentials (ERPs). Because of their excellent temporal resolution, ERPs allow a finer, more sensitive and more direct examination of differences in the time course of information processing and the cortical resources used therein (Hillyard & Kutas, 1983). Therefore, they can provide valuable insights into early stages of information processing, which are not reflected in behavioral measures (e.g., Bar-Haim et al., 2005; Thomas et al., 2007).

Evidence for the suitability of ERPs for the investigation of emotional face processing comes from a broad number of studies (for reviews, see Eimer & Holmes, 2007; Vuilleumier & Driver, 2007). These studies reported an increased *exogenous visual P1* component for negative faces around 130 ms post-stimulus over the posterior cortex (Eger, Jedynak, Iwaki, & Skrandies, 2003; Holmes, Kragh Nielsen, & Green, 2008; Pizzagalli, Regard, & Lehmann, 1999). This early enhanced positivity might reflect selective attentional processing of negative facial emotions in the extrastriate visual cortex, which may be modulated by re-entrant processes from more anterior structures, such as the

amygdala (Lang et al., 1998; Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). In addition, unlike neutral faces, emotional faces elicit an increased positivity over prefrontal (fronto-central) areas (Ashley et al., 2004; Bar-Haim et al., 2005; Eimer & Holmes, 2002; Williams et al., 2006). This effect – also described as *fronto-central P2* – usually begins within 250 ms post-stimulus. The fronto-central P2 may be generated by prefrontal or orbitofrontal mechanisms which are involved in the rapid detection and categorization of facial expressions (Eimer & Holmes, 2007). Following this increased early fronto-central P2, a sustained positivity (*Late Positive Potential*, LPP) broadly distributed over the parietal cortex has been observed beyond 300 ms after the presentation of emotional faces (Ashley et al., 2004; Eimer & Holmes, 2002; Krolak-Salmon, Fischer, Vighetto, & Mauguière, 2001). The LPP therefore may reflect subsequent higher level stages of emotional face processing, such as conscious evaluation of emotional content (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Eimer & Holmes, 2007). In summary, emotional and in particular threat-related facial expressions have been found to elicit distinct ERP responses compared to neutral faces, supposedly reflecting different stages of facial emotion processing (Eimer & Holmes, 2007).

Considering the suitability of the ERP technique to study the processing of emotional facial expressions, studies using ERPs to investigate the effects of acutely elevated cortisol levels or aggression on facial emotion processing are scarce. In two studies van Peer et al. (2007; 2009b) reported increased P2 and LPP amplitudes to angry faces after cortisol administration in high trait avoidant and social phobic individuals during an explicit emotion evaluation task. This indicates an enhanced allocation of processing resources to motivationally salient stimuli after cortisol administration. However, when the same social phobic patients were performing an emotional Stroop task, in which the emotionality of the facial expressions was irrelevant for the task performance, cortisol administration decreased P2 amplitudes for angry faces (van Peer et al., 2009a). Moreover, we recently found significantly increased P2 and LPP amplitudes to emotional facial expressions after experimentally provoking aggression in healthy participants (Bertsch, Böhnke, Kruk, & Naumann, 2009). In aggressive participants, P2 amplitudes were greatest for threat-related (angry and fearful) expressions.

Hence, the findings of ERP studies indicate that cortisol and aggression already affect very early but also later stages of social information processing, which confirms and extends the findings of previous behavioral studies. However, evidence in healthy individuals is scarce, and so far, effects of both cortisol *and* aggression on the processing

of emotional faces have not yet been studied. This was the aim of the present study. In addition, we wanted to gain more insights into the temporal dynamic of cortisol and aggression effects on information processing.

In an ERP study, we therefore orally administered either 20 mg hydrocortisone (cortisol group) or a placebo (placebo group) to a group of 56 healthy participants. One hour after they took the capsule, aggressive behavior was provoked in half of the participants of each group with the Taylor Aggression Paradigm (high provocation group). The other half played a non-provocative version of the paradigm (low provocation group). Thereafter, all participants performed a masked emotional Stroop task with emotional (angry, fearful, and happy) and neutral facial expressions. To investigate if the effects of cortisol and provocation are specific to social stimuli (i.e., facial expressions), we included a non-facial control task, in which the facial expressions were omitted and only color masks were presented.

We expected that cortisol as well as aggression would have an impact on the processing of emotional faces. In particular, we expected a preferential processing of angry faces after cortisol administration and induced aggression. Based on previous studies (Bertsch et al., 2009; van Peer et al., 2007; 2009b), we expected alterations in the ERPs, i.e., increased amplitudes in the exogenous visual P1 component, the early fronto-central positivity (P2), and the later posterior positivity (LPP) due to cortisol and aggression, particularly for angry facial expressions. This effect may be followed by biased, i.e., longer response latencies, for angry faces.

3.2. Materials and Methods

Participants

56 students of the University of Trier (28 female and 28 male, mean age = 23 years, *SE* = 0.3, range = 19–32 years) took part in the study. However, the data of four participants had to be discarded because of excessive eye blinks. Moreover, the data of one participant were removed because of outlier values (± 2 SD) in all three blocks of the Taylor Aggression Paradigm. The remaining 51 participants (28 females and 23 males, mean age = 22.9 years, *SE* = 0.4, range 19–32) were native speakers of German. Exclusion criteria were as follows: any acute or chronic disease, a presence or history of mental illness, use of medication. In addition, only non-smoking participants with a BMI between 19 and 25 were included, because cigarette smoking and weight are associated with changes in HPA axis activity. Furthermore, left-handed and color-blind individual were

excluded from the study, since handedness is related to brain activity and the emotional Stroop task requires the discrimination between different colors. Only non-pregnant women who used hormonal contraceptives were included in the study to control for hormonal status. However, women using the oral contraceptives Yasmine, Yasminelle, and Petibelle were excluded, since these medications comprise the component Drospirenone, which is an antagonist for the mineralocorticoid receptor and therefore might have interfered with the effect of cortisol administration (Genazzani, Mannella, & Simoncini, 2007). Medical exclusion criteria were assessed by a physician. The experiment was conducted in accordance with the Declaration of Helsinki and had been approved by the Ethical Committee of the State's Medical Association (Landesärztekammer Rheinland-Pfalz). All participants gave written informed consent and were compensated with €40 (approximately US-$55). The participants were randomly assigned to either a cortisol or a placebo group as well as to a high provocation or a low provocation group, but sex was balanced across groups. Thus, each of the groups consisted of six male and seven female participants, except for the high provocation-placebo group, which only comprised five male and seven female participants.

Materials

Taylor Aggression Paradigm

Aggression was elicited by and assessed with a modified version of the Taylor Aggression Paradigm (TAP; Taylor, 1967). The TAP has shown good construct, external, discriminant, and convergent validity (Anderson et al., 1999; Bernstein et al., 1987; Giancola & Chermack, 1998; Giancola & Zeichner, 1995).

The participants were led to believe that they were playing a competitive reaction time game with another participant whom they had met prior to the experiment. The TAP consisted of 30 trials, which were divided into three blocks of ten trials. The participants' task was to react as fast as possible to a green square shown on the screen by pressing a key. They were told that whoever reacted slower would receive a blast of aversive noise. Prior to each trial, the participants had to set the volume and the duration of the noise blast for the opponent on two separate scales, each ranging from 0 to 10. Corresponding to the 11 levels, the duration could be varied between 0 (level 0) and 5 seconds (level 10) in 0.5 second increments. The volume could be varied between 60 (level 1) and 105 dB (level 10) in 5 dB increments. Level 0 on the volume scale corresponded to 0 dB. After each trial, the participants received feedback about the outcome of the trial, i.e., whether they had won or lost, as well as about the opponent's settings. In fact, there was no opponent and the

outcome of the trials was held constant for all participants – each of them won and lost half of the trials. The experimenter also set the "opponent's" volume and duration settings in advance, according to the block and experimental condition of the participant. During the first block, all participants received short and gentle noises when they lost a trial (volume: *M* = 62.5 dB, range 0–70 dB; duration: *M* = .075 s, range 0–1.5 s). Participants of the low provocation group received noises of the same volume and duration during the second and third block as well. Participants of the high provocation group were exposed to noises of intermediate volume and duration during the second block (volume: *M* = 82.5 dB, range 75–90 dB; duration: *M* = 2.75 s, range 2–3.5 s) and of high volume and duration during the third block (volume: *M* = 99 dB, range 90–105 dB; duration: *M* = 4.4 s, range 3.5–5 s) when they lost a trial. The volume and duration settings of the participants (from 0 to 10) were recorded for each trial. For each participant and each trial, an average of the volume and duration setting was computed, except for those trials in which one of the settings had been 0. In that case, the total score was set to 0, since no noise would have been presented to the opponent and this trial would not have constituted an aggressive act. Finally, the 10 trials which belonged to one block of TAP were averaged for each participant. These values were then used as the dependent variable (aggressive behavior) in the statistical analysis.

Modified emotional Stroop task

Emotional Stroop task. Facial stimuli were taken from Ekman and Matsumoto's *Japanese and Caucasian Facial Expressions of Emotion* (JACFEE) and *Japanese and Caucasian Neutral Faces* (JACNeuF) (Matsumoto & Ekman, 1988). We used pictures of three male and three female faces, displaying happy, angry, fearful, and neutral expressions. Duplicates of each picture were colored in transparent red, blue, yellow, and green, resulting in 96 different stimuli. Each picture was presented twice. In total, the emotional Stroop task comprised 192 experimental trials. Each trial consisted of the presentation of a colored facial expression, which was backwardly masked after 16.6 ms (1 frame at 60 Hz). The masks were individually constructed for each facial expression and represented a distorted version of the picture, keeping hue and saturation constant. The mask remained on the screen until the participant responded by pressing a key. Prior to each facial presentation, a fixation cross appeared at the center of the screen for 1990 ms.

Non-facial control task. In order to check whether the effects of cortisol and aggression are face-specific, the participants also performed a non-facial control task. This task comprised 128 trials, in which identical blue, green, yellow, and red color masks were

presented, but the facial expressions were omitted. Thus, each of these 128 trials was composed of a presentation of a fixation cross at the center of the screen for 1990 ms, which was followed by a colored mask. The masks were presented until the participants responded by pressing a key.

Half of the participants in each experimental group started with the non-facial control task and the other half with the emotional Stroop task. All participants began with 8 practice trials. There were four colored response keys. The participants were instructed to identify the color of the picture by pressing the appropriate key as fast as possible, whilst making as few errors as possible. The responses and reaction times were recorded for each trial. There was a 2 min break between the non-facial control task and the emotional Stroop task as well as after 96 trials of the emotional Stroop task. Thus, in total, this modified emotional Stroop task consisted of three blocks, two comprising 96 facial expressions and masks and one comprising 128 colored masks only. All stimuli were presented at the center of the screen on a black background. The image sizes were 5.55" x 5.20" and the vertical and horizontal visual angles were 0.28° and 0.26°, respectively. The stimuli were presented in a pseudo-randomized fashion, which allowed a presentation of no more than three pictures of the same color or facial expression in a row.

Salivary cortisol measurement

Salivary cortisol samples were collected at five assessment points over a 115 min period: before capsule ingestion (T0; -5 min with reference to capsule ingestion), before the induction of aggression (T1; +60 min), after the induction of aggression (T2; +75 min), after the emotional Stroop task (T3; +100 min), and before the debriefing (T4; +110 min). Native saliva samples were obtained in 2 ml reaction tubes (Sarstedt, Nümbrecht, Germany) since typically-used cotton-based devices artificially reduce free cortisol concentrations (Gröschl & Rauh, 2006). Directly after the experiment, the samples were frozen for biochemical analysis. Salivary cortisol was analyzed with a time-resolved immunoassay with fluorescence detection as has been described in detail in Dressendörfer, Kirschbaum, Rohde, Stahl, and Strasburger (1992). Intra- and interassay variability was less than 10 and 12%, respectively. For the participants who were administered hydrocortisone, a cut-off was set at 100 nmol/l for the samples taken after hydrocortisone administration (samples T1–T4). In addition, all participants were asked to guess whether they received cortisol or a placebo at T3.

Subjective measures

Self-reported mood was assessed with the German version of the *Positive and Negative Affect Schedule* (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996) at T1, T2, and T3.

Procedure

All participants were tested individually. We invited them to a preliminary interview in order to check for exclusion criteria and to inform them about the aim of the study and the experimental procedure. They were told that the study would concern the investigation of the relationship between the steroid hormone cortisol, personality, and the perception of and reaction to visual stimuli. After a description of the experiment, the EEG and salivary cortisol measurement, written informed consent was obtained from all participants. Finally, the participants also received a battery of personality questionnaires to fill out at home as well as home sampling devices for salivary cortisol. In a separate meeting, a physician conducted a clinical interview with the participants and checked for health status and medical exclusion criteria.

The experiment was conducted between 1200h and approximately 1930h, beginning at 1200h, 1430h, and 1700h when endogenous cortisol levels are low (Schreiber et al., 2006). The experimental procedure was kept constant for all participants. On arrival, the participant was acquainted with another participant of the same sex, who was in fact a confederate of the experimenter. The participant and the confederate both handed over the filled-out questionnaires and the salivary cortisol samples. The participants were then seated in a waiting room, where they were administered 20 mg of hydrocortisone or a placebo. The confederate was ostensibly seated in a different room, but in fact was free to go. Then, the participants were left alone in the room to fill out questionnaires. After 30 min, the participants were led to the EEG laboratory, where they were comfortably seated in a dimly lit, sound-attenuated room one meter away from a 19" computer screen with a computer keyboard on a table in front of them. After the EEG electrodes were applied, the participants were left alone in the room for the rest of the experiment and received all instructions via computer screen. The computer program was started exactly 1 h after the administration of hydrocortisone / placebo. All participants first played the Taylor Aggression Paradigm, which took about ten minutes, and then performed the modified emotional Stroop task (including the non-facial control task) for about 20 minutes. Prior to the administration of the capsule, before and after the TAP as well as after the emotional Stroop task, the participants gave salivary cortisol samples, and filled out a short mood

questionnaire. A fifth salivary cortisol sample was collected shortly before the participants left the laboratory. Finally, the participants were debriefed about the true aim of the study as well as the TAP and the confederate. We thanked and compensated them for their participation.

Stimulus presentation and response logging were controlled for using E-Prime software (Version 2.0, Psychology Software Tools, Inc.). The entire experiment, including preparation and debriefing, took 130 minutes.

EEG recording and quantification

The EEG was recorded from 32 electrode sites according to the 10–10 electrode reference system (Chatrian et al., 1988) including the mastoids with the Easy-Cap electrode system (Falk Minow Services). All sites were referenced to FCz. A bipolar horizontal electrooculogram (EOG) was recorded from the epicanthus of each eye, and a bipolar vertical EOG was recorded from supra- and infra-orbital positions of the left eye. The EEG and the EOG were recorded with Ag/AgCl electrodes. Prior to electrode placement, the electrode sites on the participant's scalp and face were cleaned with alcohol and gently abraded. All impedances of the EEG electrodes were below 5 kΩ. EEG and EOG were amplified with a 32-channel BrainAmp amplifier (input impedance: 10 MΩ; Brain Products, GmbH) in AC mode. The pass-band was set to 0.05 to 35 Hz (-12 dB/octave rolloff); the signals were digitalized at 1000 Hz and stored to hard disk for later analysis.

The EEG was re-referenced to linked mastoids. Artifacts due to eye movements were corrected via the algorithm developed by Gratton, Coles, and Donchin (1983). Trials with non-physiological artifacts were excluded from analysis via semiautomatic artifact rejection. EEG and EOG were epoched off-line into 1400-ms periods, starting 200 ms prior to stimulus onset and ending 1200 ms after stimulus onset. A baseline correction was performed using the first 200 ms as a reference.

Separate averages were computed for each electrode, individual, and emotion (happy, angry, fearful, and neutral) as well as color condition (red, blue, yellow, and green).[6] Analyses of variance were conducted on ERP mean amplitudes obtained for specific sets of electrodes within predefined measurement windows based on previous studies (Bertsch et al., 2009; Holmes et al., 2008; van Peer et al., 2007; 2009b). Analyses focused on frontal (F3/Fz/F4), frontocentral (FC3/FCz/FC4), centroparietal

[6] For the non-facial control condition, separate averages were computed for each electrode, individual, and color condition (red, blue, yellow, green).

(CP3/CPz/CP4), parietal (P3/Pz/P4), and occipital (O1/Oz/O2) electrodes. Analyses were conducted at frontal and frontocentral electrode sites for the early fronto-central positivity (P2) within a stimulus-locked time interval of 180–220 ms. For the posterior late positive potential (LPP), analyses focused on centroparietal and parietal electrode sites within three successive stimulus-locked time intervals (300–450 ms, 450–600 ms, and 600–800 ms post-stimulus, respectively). In addition, analyses were performed at occipital electrodes within the 100–170 ms time window to examine P1 component effects (see, for instance, Holmes et al., 2008). Before calculating average amplitudes, the EEG data were filtered using a 12-Hz (-12dB/octave rolloff) digital low pass filter.

Statistical analyses

Basic design. The influence of cortisol administration and aggression induction on aggressive behavior in the TAP, salivary cortisol, subjective measures, emotional Stroop task performance, and ERP amplitudes were tested using repeated measures analyses of variance (ANOVAs) using SPSS for Windows (Version 17.1, SPSS Inc.). All ANOVAs included the between-subject factors Cortisol Group (cortisol vs. placebo) as well as Provocation (high vs. low provocation).

Manipulation check

Salivary cortisol. An ANOVA including the factor Time (T0, T1, T2, T3, T4; repeated measure) was performed to check if the cortisol administration led to elevated salivary cortisol levels. Since the salivary cortisol measures (nmol/l) were skewed, they were log transformed before statistical analysis. Awareness of cortisol/placebo administration was tested with a Chi-squared test for independent groups.

Aggressive behavior during the TAP. An ANOVA with the additional factor TAP Block (Blocks 1, 2, 3; repeated measure) was performed to check if the induction of aggressive behavior had been successful.

Subjective measures. Effects of the experimental treatment on positive and negative mood as well as state anger were analyzed with ANOVAs including the factor Time (T1, T2, T3; repeated measure).

Emotional Stroop task

Behavioral data. Outliers (± 2 SD) and trials with incorrect responses were individually rejected for each participant. We calculated the mean reaction times for each of the four facial expressions. Bias scores were computed by subtracting the mean reaction time for neutral pictures from each of the three emotional categories (e.g., the individual mean response latencies for angry faces minus the individual mean response latencies for

neutral faces, see, for instance, van Honk et al., 2001a). Note that positive bias scores are referred to as *interference* and negative scores as *facilitation*. Mean bias scores were analyzed using an ANOVA with the additional factor Emotion (happy, angry, fearful; within-subject).

Electrophysiological data. For each component, average ERP amplitudes were submitted to separate ANOVAs with the additional factors Emotion (happy, angry, fearful, neutral; repeated measure), Caudality (repeated measure), and Lateralization (repeated measure).

Comparison of facial and non-facial control task. (1) Behavioral data: to check whether the effects of cortisol and aggression were specific to faces or independent of the presented stimuli, we calculated the mean reaction times for each of the four color conditions separately for the emotional Stroop task and the non-facial control task.[7] The mean reaction times were then submitted to an ANOVA with the additional factors Task (facial and non-facial task; repeated measure), and Color (red, blue, yellow, green; repeated measure). *(2) ERP data:* for the ERP data, ANOVAs including the factors Task (facial and non-facial task; repeated measure), Color (red, blue, yellow, green; repeated measure), Caudality (repeated measure), and Lateralization (repeated measure) were performed separately for each component.

For all ANOVAs, the degrees of freedom were Huynh-Feldt corrected if the assumption of sphericity was violated (Huynh & Feldt, 1976). All statistical analyses employed a two-tailed alpha of .05. Effect sizes of significant results are reported as proportion of explained variance (partial eta squared [η^2]). In case of significant effects, we used Dunn's Multiple Comparison Tests as post-hoc tests.

3.3. Results

Manipulation check

Salivary cortisol. Mean free salivary cortisol levels of the cortisol and placebo group are shown in Table 2. The ANOVA yielded a significant main effect of Cortisol Group, $F(1,47) = 815.81$, $p < .001$, $\eta^2 = .95$, and Time, $F(4,188) = 251.33$, $p < .001$, $\eta^2 = .84$, as well as a significant interaction between Cortisol Group and Time, $F(4,188) = 334.84$, $p < .001$, $\eta^2 = .88$. Post-hoc tests showed that, as expected, salivary cortisol levels did not differ between the placebo and cortisol group before capsule intake (T0; $p > .050$)

[7] Again, outliers (± 2 SD) and trials with incorrect responses were individually rejected for each participant.

but were significantly increased in the cortisol group compared to the placebo group from one hour after capsule intake until the end of the experiment (T1-T5; $p < .010$). Provocation had no significant effect on cortisol levels (all Fs < 1.90, $p > .050$). The participants were not aware whether they received cortisol or a placebo, $\chi^2(1) = 0.16$, $p = .689$.

Table 2. Mean free salivary cortisol levels (nmol/L) before (T0) and after (T1-T4) cortisol or placebo administration

	Cortisol Group		Placebo Group	
Time	*M*	*SE*	*M*	*SE*
T1	5.2	0.5	5.0	0.5
T2***	97.2	1.9	4.0	0.4
T3***	90.6	3.3	3.8	0.3
T4***	84.4	4.6	4.1	0.4
T5***	84.8	4.9	4.3	0.4

Note. *** $p < .001$ placebo vs. cortisol group. T0: before capsule ingestion (-5 min), T1: before aggression induction (+60 min), T2: after aggression induction (+75 min), T3: after emotional Stroop task (+100 min), T4: before depriving (+110 min).

Aggressive behavior during the TAP. The experimental manipulation of aggressive behavior was successful. The ANOVA revealed a main effect of Provocation, $F(1,47) = 16.28$, $p < .001$, $\eta^2 = .26$, and TAP Block, $F(2,94) = 19.54$, $p < .001$, $\eta^2 = .29$, as well as a significant interaction between Provocation and TAP Block, $F(2,94) = 34.09$, $p < .001$, $\eta^2 = .34$. Aggressive behavior increased only in the high ($p < .010$) but not in the low provocation group ($p > .050$). The high provocation group showed more aggressive behavior than the low provocation group in TAP Blocks 2 ($p < .010$) and 3 ($p < .010$), but not in Block 1 ($p > .050$), where no provocation had taken place (see Figure 7).

Moreover, a marginally significant interaction between Cortisol Group and Provocation, $F(1,47) = 3.99$, $p = .052$, $\eta^2 = .08$, indicated that the effect of provocation was greater in the placebo group than in the cortisol group (see Figure 8). In the placebo group, strongly provoked participants showed significantly more aggressive behavior than little provoked participants ($p < .010$), but no such difference was found in the cortisol group ($p > .050$).

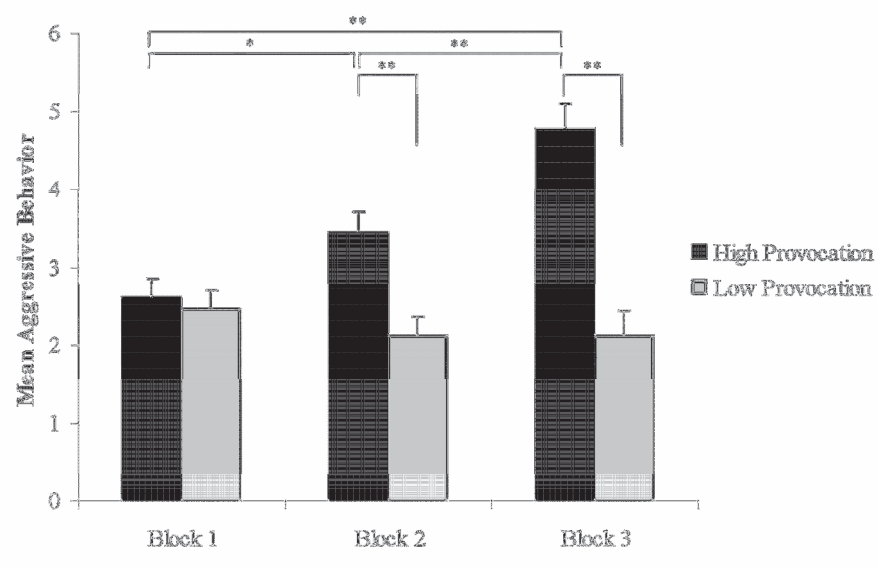

Figure 7. Mean aggressive behavior of the high and low provocation group in the three blocks of the Taylor Aggression Paradigm. Mean aggressive behavior represents the average of the loudness and duration setting. Each block consists of ten trials (block 1: trial 1 to 10, block 2: trial 11 to 20, block 3: trial 21 to 30). The error bars represent ± one standard error.

Subjective measures. Participants in the high provocation group reported significantly higher levels of negative affect, $F(2,94) = 8.69$, $p < .001$, $\eta^2 = .16$, and anger, $F(2,94) = 16.60$, $p < .001$, $\eta^2 = .26$, after the TAP than participants in the low provocation group. No significant effects on positive mood (all Fs ≤ 1.02, ps $\geq .365$) and no effects of cortisol on mood ratings (all Fs ≤ 1.70, ps $\geq .189$) were found.

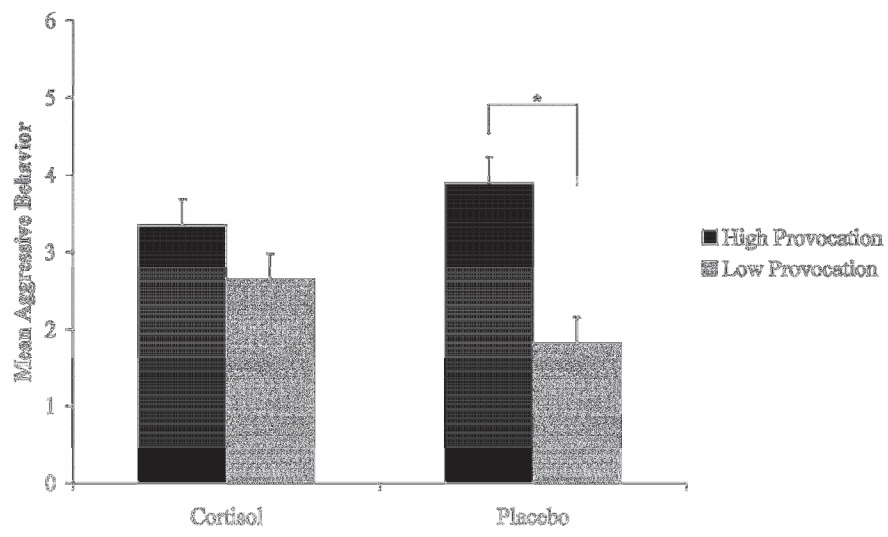

Figure 8. Mean aggressive behavior of the four experimental groups averaged over all trials of the Taylor Aggression Paradigm. Mean aggressive behavior represents the average of the loudness and duration settings.

Emotional Stroop task

Behavioral results. The error rates in the emotional Stroop task (facial task) were 4.0% (M = 5.1, SE = 0.5) and 4.5% (M = 8.7, SE = 0.8) in the non-facial control task. The four experimental groups did not differ with respect to their error rates (all Fs < 1.00, ps > .050). The behavioral performance of all groups in both tasks is summarized in Table 3. Statistical analysis of the bias scores revealed a significant effect of Emotion, $F(2,94)$ = 3.35, p = .040, η^2 = .07. Post-hoc tests revealed that, relative to neutral faces, all participants reacted faster to angry faces (M = -8.1, SE = 5.5) and slightly slower to happy faces (M = 4.3, SE = 4.0; p < .050).

Table 3. Reaction times and bias scores in the modified emotional Stroop task (means ± one standard error).

	All Participants	Cortisol Group		Placebo Group	
		High Provocation	Low Provocation	High Provocation	Low Provocation
RT Neutral	645.8 ± 10.8	619.1 ± 25.2	638.9 ± 23.5	681.5 ± 16.5	646.4 ± 18.0
RT Happy	649.9 ± 10.8	625.2 ± 21.1	638.2 ± 24.1	697.5 ± 16.1	642.1 ± 20.4
RT Angry	637.6 ± 11.9	604.2 ± 23.1	633.5 ± 29.7	677.3 ± 19.9	638.6 ± 18.3
RT Fearful	641.5 ± 10.8	617.6 ± 19.8	629.9 ± 26.1	675.5 ± 15.2	645.5 ± 21.7
Bias Happy	4.3 ± 4.0	6.2 ± 7.9	-0.6 ± 7.9	16.0 ± 8.2	-4.2 ± 7.9
Bias Angry	-8.1 ± 5.5	-14.9 ± 10.8	-5.4 ± 10.8	-4.2 ± 11.2	-7.8 ± 10.8
Bias Fearful	-4.3 ± 4.3	-1.4 ± 8.8	-9.0 ± 8.8	-6.0 ± 9.1	-0.9 ± 8.8

Note. Reaction time (RT) was measured in units of ms. Bias scores (Bias) represent difference values (mean reaction times for emotional minus neutral expressions) in ms.

Electrophysiological results.

P1 (100–170 ms at occipital electrodes). For the occipital P1 amplitudes, results showed a significant main effect of Provocation, $F(1,47)$ = 7.53, p = .009, η^2 = .14, with greater (more positive) P1 amplitudes for participants of the high provocation group compared to those of the low provocation group (see Figure 9). In addition, there was a significant main effect of Emotion, $F(3,141)$ = 5.79, p = .001, η^2 = .11. Post-hoc tests indicated larger P1 amplitudes for fearful faces compared to neutral faces (p < .050). No further effects including Cortisol Group, Provocation, or Emotion reached significance (all Fs ≤ 2.29, ps ≥ .137).

P2 (180–220 ms at fronto-central electrodes). The ANOVA showed a significant interaction between Cortisol Group and Emotion on P2 amplitudes, $F(3,141)$ = 4.17, p = .007, η^2 = .08. Post-hoc tests revealed that in the placebo group, the P2 amplitudes were

significantly larger (more positive) for angry compared to neutral faces ($p < .050$) but not in the cortisol group ($p > .050$). No such difference was found between fearful and neutral or happy and neutral faces, respectively ($p > .050$). Relative to placebo administration, the administration of cortisol resulted in decreased P2 amplitudes for happy, fearful ($p < .050$), and particularly angry ($p < .010$) faces, but did not significantly affect P2 amplitudes for neutral faces ($p > .050$; see Figure 10). No other effects including Cortisol Group, Provocation, or Emotion reached significance (all Fs $\leq .2.19$, ps $\geq .146$).

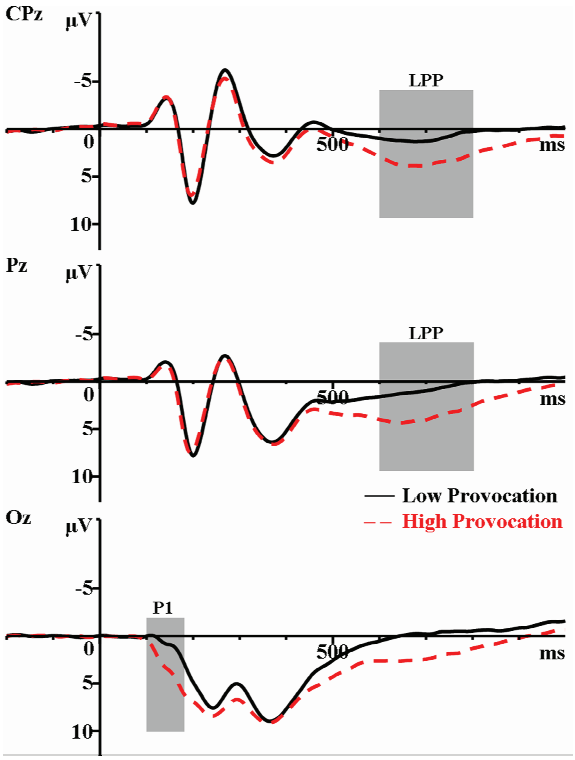

Figure 9. Grand average ERP waveforms at Pz and Oz, averaged over the four facial expressions for the high and low provocation group.

LPP (300–450 ms, 450–600 ms, 600–800 ms at centro-parietal electrodes). The posterior LPP was larger (more positive) for all facial expressions in the high provocation than in the low provocation group (see Figure 9). This effect of provocation was largest between 600 and 800 ms post-stimulus (Provocation: $F(1,47) = 10.32$, $p = .002$, $\eta^2 = .18$), but already started around 300 ms post-stimulus at the CP3 electrode (Provocation × Caudality × Hemisphere $F(2,94) = 2.80$, $p = .066$, $\eta^2 = .06$ between 300-450 ms and $F(2,94) = 2.75$, $p = .069$, $\eta^2 = .06$ between 450-600 ms, respectively). No further effects including Cortisol Group, Provocation, or Emotion reached significance (all Fs ≤ 2.06, ps $\geq .135$).

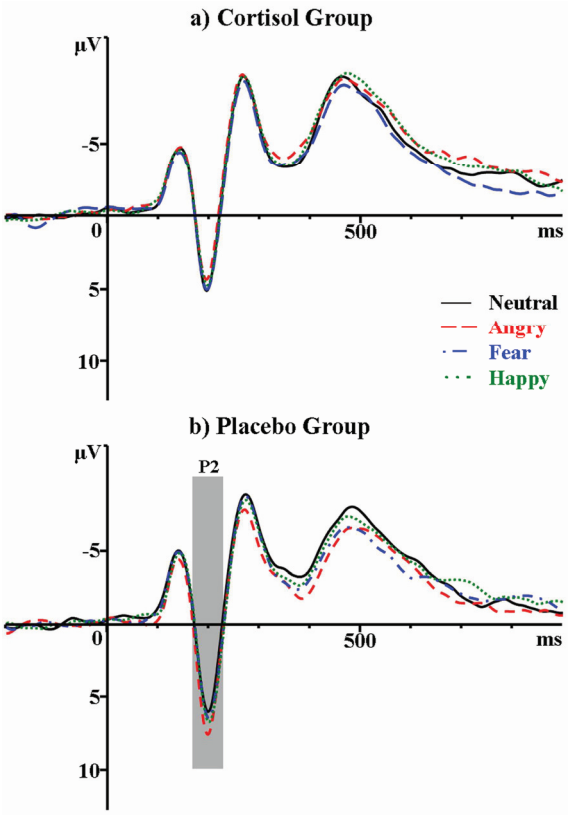

Figure 10. Grand average ERP waveforms at Fz elicited in response to happy, neutral, fearful, and angry faces for the cortisol and the placebo group.

Comparison of facial and non-facial control task. (1) Behavioral results. Concerning the reaction times in the two task conditions of the modified emotional Stroop task, the results showed no significant main effect Task, $F(1,47) = .180$, $p = .674$. Thus, the reaction times did not differ significantly between the facial and the non-facial control task. However, there was a significant interaction between Cortisol Group and Task, $F(1,47) = 4.36$, $p = .042$, $\eta^2 = .09$. Post-hoc tests revealed that, relative to placebo administration, cortisol administration resulted in faster reactions in the facial task ($p < .010$) but not in the non-facial control task ($p > .050$).

(2) Electrophysiological results. Figure 11 shows grand average ERP responses to the two task conditions: the facial – emotional Stroop – task as well as the non-facial control task for Fz, Cz, Pz, and Oz, averaged over all participants and the four color conditions, respectively. The results of the statistical analyses, which included the data of the facial as well as the non-facial control task, showed significant differences in amplitudes between the two tasks. The short presentation of the emotional facial expressions resulted in enhanced amplitudes in the time window of occipital P1 amplitudes, $F(1,47) = 20.53$, $p < .001$, $\eta^2 = .30$ and fronto-central P2 amplitudes, $F(1,47) = 57.60$, p

<.001, η^2 = .55 (see Figure 11). However, no significant main effect of Task could be found for the posterior LPP (*F*s < 1.00, *p*s > .400).

In addition, for the *occipital P1* amplitudes, there was a significant main effect of Provocation, $F(1,47) = 8.09$, $p = .007$, $\eta^2 = .15$. Thus, provocation resulted not only in larger (more positive) P1 amplitudes for the facial expressions (see above), but also for the non-facial stimuli. Furthermore, for the *posterior LPP*, we found a significant main effect Provocation, $F(1,47) = 6.23$, $p = .016$, $\eta^2 = .12$, and a significant interaction between Provocation and Task, $F(1,47) = 4.95$, $p = .031$, $\eta^2 = .10$, within the time interval of 600-800 ms. Post-hoc tests revealed that high provocation only resulted in increased (more positive) LPP amplitudes in the facial task ($p < .010$; see also above) but not in the non-facial control task ($p > .050$).

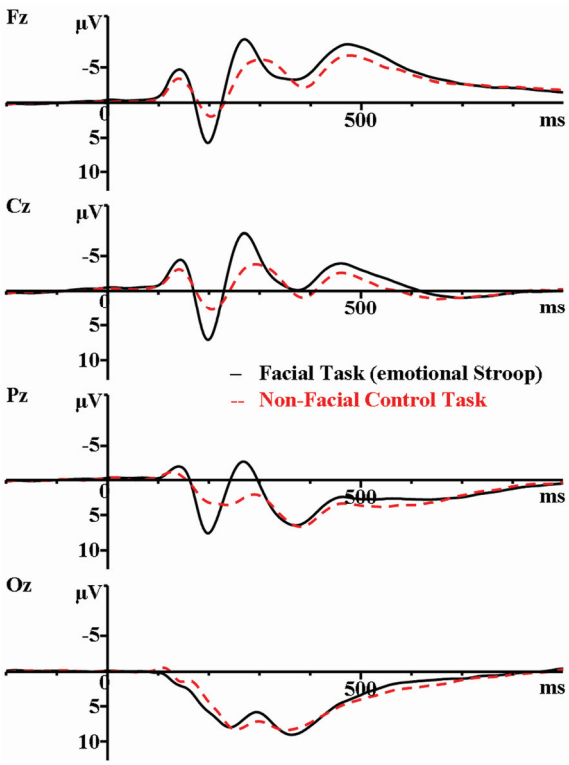

Figure 11. Grand average ERP waveforms at Fz, Cz, Pz, and Oz for the facial task (emotional Stroop task) and the non-facial control task, averaged over all colors and participants.

3.4. Discussion

The present study established different effects of exogenously elevated levels of the stress hormone cortisol and experimentally provoked aggression on social information processing. Cortisol and aggression affected temporally and spatially diverse stages of

emotional face processing during an emotional Stroop task. In addition, cortisol and aggression effects differed with respect to their specificity for threat-related facial expressions.

Effects of cortisol

Participants who were administered cortisol prior to the experiment tended to show intermediate levels of aggression in the TAP independent of their counterparts' behavior. This might indicate a diminished discriminating sensitivity for real threats and lower thresholds for aggressive behavior in individuals with exogenously elevated cortisol levels. Lower thresholds for aggressive behavior have been reported in rodents with elevated corticosterone levels (Kruk et al., 2004). Animals in this state also attack almost anything and do not discriminate between potentially threatening perpetrators and innocent (female or frozen) animals (Kruk et al., 2004).

In the emotional Stroop task, participants with exogenously elevated cortisol levels showed a diminished early processing bias for angry faces. Similar to the previous studies without any drug treatment (Ashley et al., 2004; Bediou et al., 2009; Williams et al., 2006), the placebo group displayed increased (more positive) fronto-central P2 amplitudes for angry compared to neutral faces. Participants in the cortisol group had diminished P2 amplitudes for all emotional faces, and thus showed no significant amplitude differences between angry and neutral faces. This replicates the findings of van Peer et al. (2009a) and indicates that a cortisol-induced reduction of early processing biases for threat information in an emotional Stroop task is not specific for social phobic patients but can also be found in healthy participants.

Increased early fronto-central positivities have been linked to an initial, rapid detection and attentional prioritization of salient facial emotions (Eimer & Holmes, 2007). A rapid allocation of attention to threat-related social signals is necessary to facilitate appropriate perceptual and behavioral responses to potential sources of danger in the environment, especially in highly competitive situations such as the Taylor Aggression Paradigm (e.g., LeDoux, 1998; Öhman, Flykt, & Lundqvist, 2000). In line with the behavioral data obtained during the TAP, participants with exogenously elevated cortisol levels showed signs of a reduced sensitivity for social threat signals in the emotional Stroop task.

According to intracranial recordings and lesion studies, early fronto-central positive ERP components may reflect activity in the orbitofrontal cortex (OFC; Ashley, Vuilleumier, & Swick, 2002; Kawasaki et al., 2001). The OFC receives sensory

information from primary and secondary association cortices (Rolls, 2004) and is part of an interconnected network (also including the anterior cingulate cortex and the amygdala) that is involved in the processing of emotions, such as anger (Davidson et al., 2000). Thus, the OFC plays a key role in the processing of anger and dysregulations contribute to impulsive aggression (Lee & Coccaro, 2007). In addition, the OFC is also known as one of the target structures of cortisol (Pruessner et al., 2009). One might therefore speculate that the present results indicate a cortisol-induced attenuation of OFC activity to social threat information, reflected by decreased early fronto-central processing bias for angry faces. A possible consequence may be an increase in automatic, impulsive behavioral tendencies such as fight and flight. In the present study, we found indications for both: reduced thresholds for aggressive behavior in the TAP (fight) and faster reactions to facial signals in the emotional Stroop task (flight).

Previous studies have also reported changes in the processing of angry faces in individuals with acutely elevated cortisol levels (Oei et al., 2009; Putman et al., 2007b; Roelofs et al., 2007; 2005; van Peer et al., 2007; 2009b). However, the results are inconsistent with regard to the direction of the effect. A possible explanation of these contrasting findings can be seen task-related differences (van Peer et al., 2009a). The effects of cortisol on the processing of threat-related information may depend on the task-relevance of emotional material as well as on the preclusion of cognitive control processes (e.g., by brief stimulus presentation). This is in line with a notion by de Kloet, Oitzel, and Joël (1999) according to which glucocorticoids such as cortisol influence information processing systems conditionally, so that adaptive behavior that is most relevant to the situation is facilitated.

Effects of provocation

The experimental provocation of aggression resulted in significantly increased aggressive behavior and anger in strongly provoked participants. In the emotional Stroop task, the strongly provoked participants showed increased (more positive) early (P1) and late (LPP) posterior ERP amplitudes. These effects were rather independent of the emotional expression and, in case of the occipital P1 amplitudes, also affected the processing of non-facial stimuli.

Similar to previous studies, we also found increased occipital P1 amplitudes for negative (fearful) relative to neutral faces in all participants (e.g., Eger et al., 2003; Holmes et al., 2008; Pizzagalli et al., 1999). This very early effect may reflect a coarse perceptual discrimination of visual signals of emotion in the inferior occipital areas (Adolphs, 2002),

which is possibly modulated by the amygdala (Vuilleumier et al., 2004). After a highly aggressive encounter, early discrimination processes may go on alert in order to rapidly distinguish threat-related signal within a continuous stream of information. A rapid extraction of potential threat is advantageous in the context of an aggressive encounter and may enable rapid responses to further sources of danger.

High provocation also increased posterior late positive potential (LPP) amplitudes. Starting around 300 ms post-stimulus, this effect was maximal between 600 and 800 ms after stimulus onset. This replicates the results of our previous study (Bertsch et al., 2009), in which we also found enhanced LPPs to emotional facial expressions in strongly provoked participants. It is generally assumed that the LPP reflects attentional processes in terms of task-induced and motivated attention, since it is evoked by stimuli that trigger motivational processes, i.e., approach or avoidance (Schupp et al., 2004). Indeed, previous studies reported increased posterior LPP amplitudes for personally significant, intrinsically relevant, or meaningful stimuli (e.g., Franken, Stam, Hendriks, & Van den Brink, 2003; Namkoong, Lee, Lee, Lee, & An, 2004).

A potential source of the modulations in LPP amplitudes can be seen in a composite of activity of the extrastriate occipital, inferior temporal, and the medial parietal cortex (Sabatinelli, Lang, Keil, & Bradley, 2007), maybe involving influences of re-entrant projections from the amygdala to the visual system (Sabatinelli, Bradley, Fitzsimmons, & Lang, 2005). Thus, aggression might lead to an enhanced allocation of attention to facial expressions, as these could contain important, lifesaving, and conflict-relevant information. Unlike preceding ERP studies, we did not find greater LPP amplitudes for emotional compared to neutral faces (e.g., Ashley et al., 2004; Eimer & Holmes, 2002; Krolak-Salmon et al., 2001). However, our present findings replicate the results of our previous study (Bertsch et al., 2009) in which the LPP was also not modulated by the emotion of the facial expressions. It is plausible that the short stimulus presentation time of 16.6 ms as well as the irrelevance of the facial expression in this task might at least partly account for this (see e.g., Kiss & Eimer, 2008; Thomas et al., 2007).

Despite the large effects of aggression on ERPs, we did not find a response bias in the reaction times for angry faces due to aggression. This further illustrates the greater sensitivity of ERP data in information processing research (Hillyard & Kutas, 1983). However, this contradicts previously reported aggression- and anger-related bias effects for threat-related information (e.g., Cohen et al., 1998; Eckhardt & Cohen, 1997; van Honk et al., 2001a; 2001b). It should be noted that previous behavioral studies have included trait

measures of anger or aggression (Cohen et al., 1998) and mostly selected participants with particularly high and low trait anger or aggression (Eckhardt & Cohen, 1997; van Honk et al., 2001a; van Honk et al., 2001b) or violent offenders (Smith & Waterman, 2003, 2004). The aggression-induced ERP modulation may therefore reflect intact processes of emotion regulation in healthy individuals, which suppress impulsive behaviors. Dysregulations in these mechanisms could be a reason for behavioral response biases for threat-related stimuli in high trait aggressive or angry individuals (Davidson et al., 2000). Indications for this have been found in highly aggressive and antisocial individuals, who show diminished LPP amplitudes to emotional stimuli (see, for instance, Patrick, 2008, for a review) and deficits in facial emotion recognition (Herpertz & Sass, 2000). Future studies are necessary to compare the processing of emotional faces of highly aggressive and "normal" individuals with ERPs in emotional Stroop tasks.

Summary and conclusion

There is evidence for (1) an association between enhanced acute cortisol levels and aggression as well as for (2) cortisol- and aggression-induced effects on the processing of (threat-related) information. The results of the present study indicate that, in healthy participants, cortisol and aggression have distinct temporal and spatial effects on social information processing.

As this is the first study investigating the effects of both cortisol and aggression on information processing, its results raise questions which should be addressed by future studies. First, a replication of these effects in other cognitive tasks, for instance, visual search or memory tasks would be interesting. Additionally, a within-subject design including cognitive task performance prior to the experimental treatment might help to further investigate cortisol- and aggression-induced changes in information processing. Second, it might be interesting to see how dynamic changes in cortisol (e.g., due to social stress) affect aggression and how both affect information processing. Furthermore, the investigation of samples consisting of participants with particularly high or low levels of (basal or acute) cortisol or aggression as well as pathologically aggressive individuals might be helpful to gain more insights into the effects of stress and aggression on information processing. Third, future studies should include non-facial, but nevertheless socially relevant stimuli in order to further disentangle the specificity of the effect for facial expressions. Fourth, future studies using a combined measurement of ERPs and fMRI are necessary to gain more information about underlying mechanisms and brain structures.

Taken together, in a highly competitive context, the administration of cortisol abolished an early processing bias for threat-related social information, i.e., angry faces, resulting in enhanced impulsive reactions such as fight and flight tendencies. Indications for this can be seen in reduced thresholds for aggressive behavior in the TAP (fight) and generally facilitated reactions for social information in the emotional Stroop task (flight). In addition, experiencing a highly aggressive encounter increased the initial visual processing of all stimuli and allocated more attention to all facial expressions in later processing stages. These may reflect emotion regulation processes which suppress impulsive behavior in healthy individuals.

Chapter IV

Acute Experiences of Ostracism and Provocation Increase the Implicit Bias for Social Threat Information

Abstract

Dramatic incidences of violence such as school shootings have raised new questions about reasons and prevention strategies. Social rejection such as ostracism, bullying, or social harassment are among the most important factors that promote aggression and violence. The present study investigates the effects of ostracism and constantly increasing levels of provocation on two different types of aggressive behavior and on implicit social information processing. In line with previous studies, we found increased anger and aggression after provocation. However, effects of ostracism on aggression depended on (1) the type of aggressive behavior, (2) the level of provocation, and (3) the participants' sex. Interestingly, ostracism alone resulted in an implicit processing bias for fearful facial expressions while ostracism and provocation shifted this bias towards angry expressions. Thus, although ostracism and high levels of provocation do not necessarily result in immediate aggressive outbursts, they increase feelings of anger and lead to changes in the processing of social information even in healthy student participants. Prevention programs which take implicit cognitive biases into account may therefore be a promising strategy to prevent aggression and violence.

4.1. Introduction

A new series of school shootings in Germany (e.g., Erfurt 2002, Emsdetten 2006, Winnenden 2007, and Ansbach 2009) but also in other countries all over the world has evoked nationwide shocks and raised questions about possible causes and prevention strategies. Social rejection has been discussed as one of the motivators for these seemingly random acts of outrageous violence. In fact, a careful case study revealed that almost all of the perpetrators of 15 US school shooting incidences between 1995 and 2001 had previously suffered both acute and chronic social rejection, such as ostracism, bullying, or social harassment by peers or relationship partners (Leary, Kowalski, Smith, & Phillips, 2003). An infamous example can be found in the farewell letter of the 19-year-old Sebastian B., who severely injured 17 pupils in Erfurt in 2002 before killing himself. In this letter, he claimed that he had repeatedly been bullied and ostracized by his peers. This confirms the US General's Report on Youth Violence (2001), which found social exclusion to be the strongest risk factor for adolescent violence and an even better predictor than gang membership, poverty, or drug use. According to correlational studies, ostracism, which is defined as being ignored and excluded (Williams, 2001), and constant provocation of relevant peers may thus be important factors with respect to promoting aggression and violence. In fact, interpersonal provocation probably constitutes the most important single cause of aggression (Anderson & Bushman, 2002; Berkowitz, 1993) and is used to reliably induce aggression (e.g., Bertsch et al., 2009; Chermack, Berman, & Taylor, 1997; Epstein & Taylor, 1967) in several laboratory paradigms, such as the Taylor Aggression Paradigm (TAP; Taylor, 1967).

There is also evidence for a causal connection between social exclusion and aggressive behavior against other individuals (Buckley et al., 2004; DeWall et al., 2009; Kirkpatrick et al., 2002; Twenge et al., 2001; Warburton, Williams, & Cairns, 2006). For instance, participants who were told that no one had chosen to work with them after a get-acquainted task applied higher levels of physical pain to individuals who provoked them (Twenge et al., 2001). Similarly, Warburton et al. (2006) showed a link between ostracism and aggression. When additionally exposed to an uncontrollable aversive situation, participants who were ostracized in a virtual ball-tossing game were more aggressive towards an innocent target. Self-reports of perceived social exclusion also predicted a greater allocation of hot sauce towards a person who disliked hot food in a quasi-experimental study (Kirkpatrick et al., 2002). Moreover, excluded participants reported

more feelings of anger (Buckley et al., 2004; Williams et al., 2000; 2002; Zadro, Williams, & Richardson, 2004) and a greater urge for aggressive behavior (Buckley et al., 2004) than included participants. In summary, several experimental studies have demonstrated that individuals who think that other people do not value them report more anger as well as a stronger urge to aggress and even do behave more aggressively against a provoker or an innocent bystander than people who are socially valued. These effects were obtained using various manipulations of social exclusion (e.g., being voted out of a group, being ignored, receiving explicit feedback that others are disinterested) as well as different outcome measures (e.g., self-reported feelings of anger or the urge to aggress, allocation of hot sauce, application of loud noises). Previous studies have concentrated on a single (e.g., allocation of hot sauce) or the first (e.g., application of loud noise) outburst of aggression because this was regarded as a measure of unprovoked aggression (Bushman & Baumeister, 1998).

But what happens when people are ostracized *and* simultaneously exposed to constantly increasing levels of provocation by their peers? Furthermore, does ostracism increase the likelihood of aggressive behavior even in face of retaliation for aggressing? The present study addresses these questions by combining two well established and validated laboratory paradigms, namely, a virtual ball-tossing game (Cyberball; Williams & Jarvis, 2006) to ostracize participants, and the Taylor Aggression Paradigm (TAP; Taylor, 1967) to provoke and measure aggression. In addition, two versions of the TAP were used to investigate whether the effects of ostracism and provocation differ when the aggressive behavior causes either physical pain or monetary setback to another person. Aggressors such as school shooters often focus their violence on persons they know and by whom they have been previously ostracized or rejected (see, e.g., Leary et al., 2003). We therefore investigated effects of ostracism and increasing levels of provocation by relevant peers on aggressive behavior against these peers.

Research has recently started to investigate why social rejection increases the likelihood for aggressive and violent behavior. Although people frequently report greater negative emotions, particularly anger after experiencing social rejection (e.g., Williams et al., 2000; 2002; Zadro et al., 2004), several studies have shown that self-reported mood does not significantly moderate rejection-induced aggression (e.g., DeWall et al., 2009; Twenge et al., 2001). The investigation of social cognition, on the other hand, seems more promising. In a very recent series of experiments, DeWall et al. (2009) found increases in hostility-related cognitive processes (i.e., rating aggressive and ambiguous words as

similar, completing word fragments with aggressive words, and rating the ambiguous actions of another person as hostile) in participants who experienced social exclusion compared to socially accepted and control participants. Interestingly, hostile cognitions mediated the relationship between social exclusion and aggression substantially. Similar cognitive biases for aggression- or anger-related material have been previously reported in individuals with high levels of trait anger, previous self-reported aggressive experience, and criminal convictions for violent offending (Smith & Waterman, 2003, 2004; van Honk et al., 2001a; 2001b). In various cognitive tasks, such as the emotional Stroop task, aggressive and violent individuals showed biased responses, i.e., slower reaction times for angry faces or other aggression-related stimuli. Moreover, when high trait angry participants were induced to experience anger, they tended to process task-irrelevant anger information in an emotional Stroop task (Bertsch et al., 2009; Cohen et al., 1998) and a visual search task (Eckhardt & Cohen, 1997).

These findings indicate changes in information processing patterns in pathologically aggressive as well as in healthy individuals who were socially rejected, insulted, or provoked (Todorov & Bargh, 2002). A biased processing and responding to aggression-related social information might increase the likelihood for maladaptive or inappropriate behavior (Crick & Dodge, 1994; Geen, 2001), and hostile attribution bias could have negative effects on the cognitive construction of future social interactions (Anderson & Bushman, 2002; Crick & Dodge, 1994). Therefore, another aim of the present study was to investigate the influence of both ostracism and provocation on the implicit processing of social information.

In summary, the results of previous studies indicate that ostracism and provocation increase negative emotions, in particular feelings of anger, and the likelihood for aggressive behavior. Moreover, social cognitions seem to play an important role in the context of social rejection and aggression. The emotional Stroop task (Williams et al., 1996) has been reported a suitable measure to assess implicit social information processing biases in the context of aggression and anger. Therefore, an emotional Stroop task with emotional facial expressions was used to assess the effects of ostracism and provocation on social information processing in the present study.

We first manipulated the inclusionary status (ostracism vs. inclusion) of 64 male and female students with the Cyberball game (Williams & Jarvis, 2006). Following this, all participants performed the Taylor Aggression Paradigm (Taylor, 1967). Half of the participants of each inclusionary status group were exposed to continuously increasing

levels of provocation (high provocation group), while the others received constantly low levels of provocation (low provocation group). There were two versions of the TAP, which differed in the type of provocation and aggressive behavior: in the *noise version*, high levels of aggression resulted in the exposure to (loud and aversive) noise, while in the *money version*, high levels of aggression resulted in a monetary setback for another participant. Finally, all participants performed an emotional Stroop task with angry, fearful, happy, and neutral facial expressions.

We expected that ostracism and high levels of provocation would lead to enhanced aggressive behavior. We also expected that both ostracism and high levels of provocation would affect the processing of emotional facial expressions. In particular, we hypothesized a bias for threat-related (angry) facial expressions in ostracized and highly provoked participants.

4.2. Materials and Methods

Participants

Sixty-four first-year psychology undergraduates enrolled at the University of Trier (32 female and 32 male, mean age = 21.5 years, *SE* = 0.3, range = 19–29 years) took part in the study. The study had been approved by the local ethics committee. Participants volunteered to take part in the experiment in return for course credit. In addition, participation was compensated with €7 (approximately US-$10). The participants were randomly assigned to a 2 (Inclusionary Status: inclusion vs. ostracism) × 2 (TAP-version: money vs. noise) × 2 (Provocation: high vs. low provocation) between-subject design, but sex was balanced across groups (four male and female participants in each group).

Materials

Cyberball

Cyberball is a virtual ball-tossing game developed by Williams and Jarvis (2006). Cyberball and its preliminary versions have been found to reliably induce ostracism in several studies (e.g., Warburton et al., 2006; Williams et al., 2000; 2002; Zadro, Boland, & Richardson, 2006; Zadro et al., 2004).

Participants were told that the study involved the effects of mental visualization on reaction time tasks. To assist them in practicing their mental visualization skills, they were going to play a ball-tossing game on the computer with the two other participants. They were told that the game was merely a means for them to train their mental visualization

skills and that therefore performance in the game was of no importance. The participants were asked to visualize the situation, themselves, and the other players. The game depicts three ball-tossers, the middle one representing the participant. The game is animated and shows one icon throwing a ball to one of the other two. When the ball was tossed at the participants, they were instructed to click on '1' in order to throw the ball to the recipient on the left side or on '2' in order to throw the ball to the recipient on the right side of the screen, and the ball would move towards that recipient. The game was set for 30 total throws and lasted approximately 5 minutes. Once the instructions had been read, the participant clicked the 'Next' link to start the game.

If randomly assigned to the *inclusion* condition, participants received the ball for roughly one-third of the total throws. If assigned to the *ostracism* condition, participants received the ball twice at the beginning of the game, but never received the ball again for the remainder of the game.

Taylor Aggression Paradigm

Aggression was elicited by and assessed with two modified versions of the Taylor Aggression Paradigm (TAP; Taylor, 1967) . The TAP has shown good construct, external, discriminant, and convergent validity (Anderson et al., 1999; Bernstein et al., 1987; Giancola & Chermack, 1998; Giancola & Zeichner, 1995).

The participants were led to believe that they were playing a competitive reaction time game with one of the other participants whom they had met prior to the experiment. The TAP consisted of 30 trials, which were divided into three blocks of ten trials each. The participants' task was to react as fast as possible to a green square shown on the screen by pressing a key. They were told that whoever reacted slower would receive a punishment. Prior to each trial, the participants set the punishment for the opponent (see below). After each trial, the participants received feedback about the outcome of the trial, i.e., whether they had won or lost, as well as about the opponent's settings. In fact, the participants did not play against each other and the outcome of the trials was held constant for all participants – each of them won and lost half of the trials. The experimenter also set the "opponent's" punishment settings in advance, according to the block and experimental condition of the participant. There were two versions of the TAP – a *noise* and a *money version* – and each participant was randomly assigned to one of the versions.

Noise Version. In the noise version, the punishment consisted of a blast of aversive noise, which was applied over headphones. Prior to each trial, the participants had to set the volume and the duration of the noise for the opponent on two separate scales, each

ranging from 0 to 10. Corresponding to the 11 levels, the duration could be varied between 0 (level 0) and 5 seconds (level 10) in 0.5 second increments. The volume varied between 60 (level 1) and 105 dB (level 10) in 5 dB increments. Level 0 on the volume scale corresponded to 0 dB. During the first block, all participants received short and gentle noises when they lost a trial (volume: M = 62.5 dB, range 0–70 dB; duration: M = .075 s, range 0–1.5 s). Participants of the low provocation group received noises of the same volume and duration during the second and third block as well. Participants of the high provocation group were exposed to noises of intermediate volume and duration in the second block (volume: M = 82.5 dB, range 75–90 dB; duration: M = 2.75 s, range 2–3.5 s) and of high volume and duration in the third block (volume: M = 99 dB, range 90–105 dB; duration: M = 4.4 s, range 3.5–5 s) when they lost a trial. The volume and duration settings of the participants (from 0 to 10) were recorded for each trial. For each participant and each trial, an average of the volume and duration setting was computed, except for those trials in which one of the settings was 0. In that case, the total score was set to 0, since no noise would have been presented to the opponent and this trial would not have constituted an aggressive act. Finally, the 10 trials which belonged to one block of the TAP were averaged for each participant. These values were then used as the dependent variable (aggressive behavior) in the statistical analysis.

Money Version. In the money version, participants were told that they could gain up to €7 (approximately US-$10) in the TAP. All participants started with a deposit of €4 (approximately US-$6). In each trial, they could chose whether they would like to gain 30 cents without taking money from their opponent's account, gain 20 cents and take 20 cents from the opponent, gain 10 cents and take 40 cents from the opponent, or gain 0 cents and take 60 cents from their opponent's account. During the first block, all participants of the money version lost no or only very small amounts of money (M = -10 cents, range 0 to -20 cents). Participants of the low provocation group were exposed to the same settings during the second and third block as well. Participants of the high provocation group were exposed to intermediate losses of money in the second (M = -30 cents, range 0 to -60 cents) and high losses in the third block (M = -48 cents, range -20 to -60 cents). For each participant, we computed the average money they chose to subtract from their opponent across the 10 trials which belonged to one block of the TAP. Again, these values were used as the dependent variable (aggressive behavior) in the statistical analysis.

Emotional Stroop task

Stimuli were taken from Ekman and Matsumoto's *Japanese and Caucasian Facial Expressions of Emotion* (JACFEE) and *Japanese and Caucasian Neutral Faces* (JACNeuF) (Matsumoto & Ekman, 1988). We used black and white pictures of four male and four female faces, displaying happy, angry, fearful, and neutral expressions. In total, the emotional Stroop task consisted of eight practice trials and 128 experimental trials. The participants' task was to name the person's sex as fast as possible by pressing a key, whilst making as few errors as possible. In each trial, the facial expression was presented until the participant responded. Thereafter, a fixation cross appeared at the center of the screen for 1.9 s.

All stimuli were presented at the center of a 19" screen on a black background. The image size was 5.55" x 5.20" and the vertical and horizontal visual angles were 0.28° and 0.26°, respectively. The stimuli were presented in a pseudo-randomized fashion, which allowed a presentation of no more than three pictures of the same color or facial expression in a row. The task was divided into two random blocks of 64 pictures with a 2 min break in between.

The responses and reaction times were recorded for each trial. Stimulus presentation and response logging were controlled for using E-Prime software (Version 1.1, Psychology Software Tools, Inc.). Outliers (± 2 SD) and trials with incorrect responses were individually rejected for each participant. We calculated the mean reaction times for each of the four facial expressions. Bias scores were computed by subtracting the mean reaction time for neutral pictures from each of the three emotional categories (e.g., the individual mean response latencies for angry faces minus the individual mean response latencies for neutral faces; see for instance, van Honk et al., 2001b).

Subjective measures

Self-reported mood was assessed three times with the German version of the *Positive and Negative Affect Schedule* (PANAS; Krohne et al., 1996): at the beginning of the experiment (T0), after the Cyberball game (T1), and after the Taylor Aggression Paradigm (T2).

At the end of the experiment (after finishing the emotional Stroop task), participants completed a post-experimental questionnaire. Following previous experiments (Williams et al., 2002; Zadro et al., 2006; 2004), this questionnaire contained 12 items assessing the effect of the Cyberball game on the fulfillment of the four needs: *belonging* (e.g., "I felt like and outsider"), *self-esteem* (e.g., "I felt good about myself"), *control* (e.g.,

"I felt like I had control over the course of the interaction"), and *meaningful existence* (e.g., "I felt non-existent") rated on a 5-point scale, ranging from 1 = not at all to 5 = very much.

The post-experimental questionnaire also contained three *manipulation checks* to confirm participants' perception of their inclusionary status during the Cyberball game (i.e., "I was well integrated within the ball-tossing game" and "I felt rejected during the ball-tossing game", both answered on the same 5-point scale described above, and an open question: "What percentage of throws do you think you received during the ball-tossing game?").

Additionally, we assessed the participants' *feelings of anger* during the Cyberball game and the Taylor Aggression Paradigm with 7 items, respectively (e.g., "I felt very angry during the ball-tossing game [reaction time game]"). The participants were asked to answer the questions according to how they felt, "while playing the ball-tossing game [reaction time game]" (rated on a 5-point scale as described above).

Procedure

Three participants of the same sex arrived at the laboratory and were introduced to each other. Participants were told that the study involved the effects of mental visualization on reaction time tasks. To assist them in practicing their mental visualization skills they would be presented with a computer-based ball-tossing game with the two other participants. Therefore, each participant was led to a separate room, where he or she had to fill out several trait and mood questionnaires. After all participants were finished filling out the questionnaires, the experimenter signaled the participants that they could now start the Cyberball game. After the Cyberball game, they had to fill out a second mood questionnaire, followed by instructions for the Taylor Aggression Paradigm. After the TAP, they filled out a third mood questionnaire and performed the emotional Stroop task. Finally, the participants filled out a post-experiment questionnaire and were fully debriefed and thanked for their participation. The entire experiment, including debriefing, took about 90 minutes.

Statistical analyses

Manipulation checks and self-reported levels of need fulfillment and mood. Univariate analyses of variance (ANOVAs), which included the factors Inclusionary Status (inclusion vs. ostracism), TAP Version (noise vs. money), Provocation (high vs. low provocation), and Sex (male vs. female) were performed for the *manipulation checks* (perceived integration, rejection, and perceived percentage of received ball-tosses), *self-reported levels of need fulfillment* (belonging, control, self-esteem, and meaningful

existence), and *feelings of anger* during the Cyberball game and Taylor Aggression Paradigm. The repeated self-report measures of *positive and negative affect* were submitted to mixed-design ANOVAs with the factors Inclusionary Status, Provocation Group, TAP Version, Sex, and Measurement Time (T1, T2; repeated measure). Self-reported positive and negative affect prior to the experiment (T0) was added as a covariate in order to control for baseline mood differences.

Ostracism and Provocation effects on aggressive behavior. A mixed-design ANOVA with the factors Inclusionary Status, TAP Version, Provocation, Sex, and TAP Block (Blocks 1, 2, 3; repeated measures) was performed to analyze effects of ostracism and provocation on aggressive behavior in the TAP. As the measures of aggressive behavior were skewed, they were log transformed prior to statistical analysis.

Ostracism and Provocation effects on social information processing. Effects of ostracism and provocation on social information processing in the emotional Stroop task were analyzed using a mixed-design ANOVA with the factors Inclusionary Status, TAP Version, Provocation, Sex, and Emotion (angry, fearful, happy; repeated measures).

For all ANOVAs, the degrees of freedom were Huynh-Feldt corrected if the assumption of sphericity was violated (Huynh & Feldt, 1976). All statistical analyses employed a two-tailed alpha of .05. Effect sizes of significant results are reported as the proportion of explained variance (partial eta squared [η^2]). In case of significant effects, we used Dunn's Multiple Comparison Tests as post-hoc tests. All statistical analyses were conducted with SPSS for Windows (Version 17.1, SPSS Inc.).

4.3. Results

Manipulation checks. Three manipulation checks assessed the inclusionary status. Participants in the ostracism condition reported that they felt significantly less integrated and more rejected than participants in the inclusion condition (integration: $F(1,48) = 162.91$, $p < .001$, $\eta^2 = .77$; rejection: $F(1,48) = 24.90$, $p < .001$, $\eta^2 = .34$). Participants in the ostracism condition also reported that they received the ball less often than participants in the inclusion condition, $F(1,48) = 65.39$, $p < .001$, $\eta^2 = .59$. This suggests that participants correctly perceived whether they were included or ostracized during the ball-tossing game. For means and standard errors of self-report measurements see Table 4.

Self-reported levels of need fulfillment. Ostracism significantly threatened the fulfillment of the four needs belonging, control, self-esteem, and meaningful existence (belonging: $F(1,48) = 66.14$, $p < .001$, $\eta^2 = .57$; control: $F(1,48) = 88.13$, $p < .001$, $\eta^2 = .64$;

self-esteem: $F(1,48) = 13.21$, $p = .001$, $\eta^2 = .21$; meaningful existence: $F(1,48) = 44.83$, $p < .001$, $\eta^2 = .48$). There were no significant main effects of provocation, nor did the inclusionary status interact with provocation condition (all Fs ≤ 1.27, ps $\geq .265$).

Table 4. Mean and standard errors (in parenthesis) of self-report measurements (all scales 1 = not at all to 5 = very much).

	Inclusion		Ostracism	
	High Provocation	Low Provocation	High Provocation	Low Provocation
Fundamental needs [a]				
Belonging	3.9 (0.2)	3.9 (0.2)	2.4 (0.2)	2.2 (0.2)
Control	3.5 (0.2)	3.4 (0.2)	2.0 (0.2)	2.0 (0.1)
Self-esteem	4.0 (0.2)	4.3 (0.2)	3.5 (0.2)	3.4 (0.2)
Meaningful existence	3.4 (0.2)	3.4 (0.2)	2.0 (0.2)	2.1 (0.2)
Feelings of angers [b]				
– during Cyberball	1.2 (0.1)	1.1 (0.1)	2.0 (0.1)	1.8 (0.1)
– during TAP	2.2 (0.1)	1.2 (0.1)	1.7 (0.1)	1.3 (0.1)
Manipulation checks				
Feelings of integration	3.6 (0.2)	3.9 (0.2)	1.3 (0.2)	1.2 (0.2)
Feelings of rejection	1.4 (0.3)	1.4 (0.3)	3.1 (0.3)	2.5 (0.3)
% of received ball-tosses	35.8 (1.2)	36.5 (1.2)	11.0 (1.2)	13.2% (1.1)

[a] Each fundamental need score represents an average of three questions.
[b] Feelings of anger during the Cyberball game and the TAP each represent an average of seven questions.

Mood. Both ostracism and high provocation resulted in lower levels of self-reported *positive affect* directly after the manipulation. Thus, ostracized participants reported significantly lower positive affect than included participants directly after the Cyberball game (Inclusionary Status × Measurement Time: $F(1,47) = 8.6$, $p = .005$, $\eta^2 = .16$), and strongly provoked participants reported significantly lower positive affect directly after the Taylor Aggression Paradigm (Provocation × Measurement Time: $F(1,47) = 7.68$, $p = .008$, $\eta^2 = .15$). For *negative affect*, we found a significant interaction between Inclusionary Status and Measurement Time, $F(1,47) = 4.36$, $p = .043$, $\eta^2 = .09$. Included participants reported lower negative affect after Cyberball than after the TAP ($p < .050$) and than ostracized participants after Cyberball ($p < .100$). Moreover, ostracized participants reported feeling *angrier* than included participants during Cyberball, $F(1,48) = 31.01$, $p < .001$, $\eta^2 = .39$, and strongly provoked participants reported feeling *angrier* than little

provoked participants during the Taylor Aggression Paradigm, $F(1,48) = 25.54$, $p < .001$, $\eta^2 = .35$.

Aggressive behavior during the TAP

Aggressive behavior during the TAP depended on the level of provocation as well as on inclusionary status and sex of the participants. These effects were different in the two versions of the TAP (see Figures 13 and 14), which was confirmed by the omnibus ANOVA (Inclusionary Status × TAP Version × Sex: $F(1,48) = 4.67$, $p = .036$, $\eta^2 = .09$; Inclusionary Status × TAP Version × Provocation × TAP Block: $F(1,96) = 2.84$, $p = .064$, $\eta^2 = .06$). For reasons of interpretability, separate ANOVAs were performed for the two TAP versions, which included the factors Inclusionary Status, Provocation, Sex, and TAP Block. The results of these analyses will be reported in the following sections.

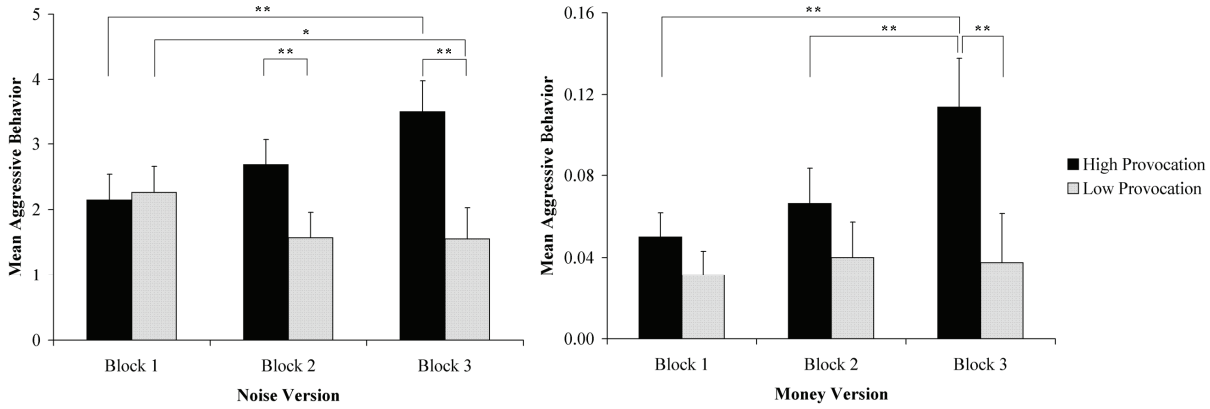

Figure 12. Mean aggressive behavior of the high and low provocation group in the three blocks of the Taylor Aggression Paradigm separately for the *Noise* and *Money Version* of the paradigm. In the *Noise Version*, mean aggressive behavior represents the average loudness and duration setting and in the *Money Version*, the average money subtraction. Each block consists of ten trials (Block 1: trial 1 to 10, Block 2: trial 11 to 20, Block 3: trial 21 to 30). The error bars represent ± one standard error. *$p < .050$; **$p < .010$.

(1) Effects of Provocation. The provocation of aggression was successful in both versions of the TAP (see Figure 12), although the effect was larger in the noise version than in the money version. In both versions, there was a significant interaction between Provocation and TAP Block (Noise Version: $F(2,48) = 12.20$, $p < .001$, $\eta^2 = .34$; Money Version: $F(2,48) = 4.21$, $p = .021$, $\eta^2 = .15$). In the *Noise Version*, aggressive behavior significantly increased from Block 1 to Block 3 in the high provocation group ($p < .010$) while decreasing from Block 1 to Block 3 in the low provocation group ($p < .050$). Thus, the strongly provoked participants of the noise version showed significantly more aggressive behavior than the little provoked participants in Block 2 ($p < .010$) and Block 3

($p < .010$) of the TAP. In the *Money Version*, there was a significant increase in aggression in the high provocation group from Block 1 to Block 3 ($p < .010$) and from Block 2 to Block 3 ($p < .010$). The strongly provoked participants of the money version showed significantly more aggression than the little provoked participants in Block 3 of the TAP ($p < .010$).

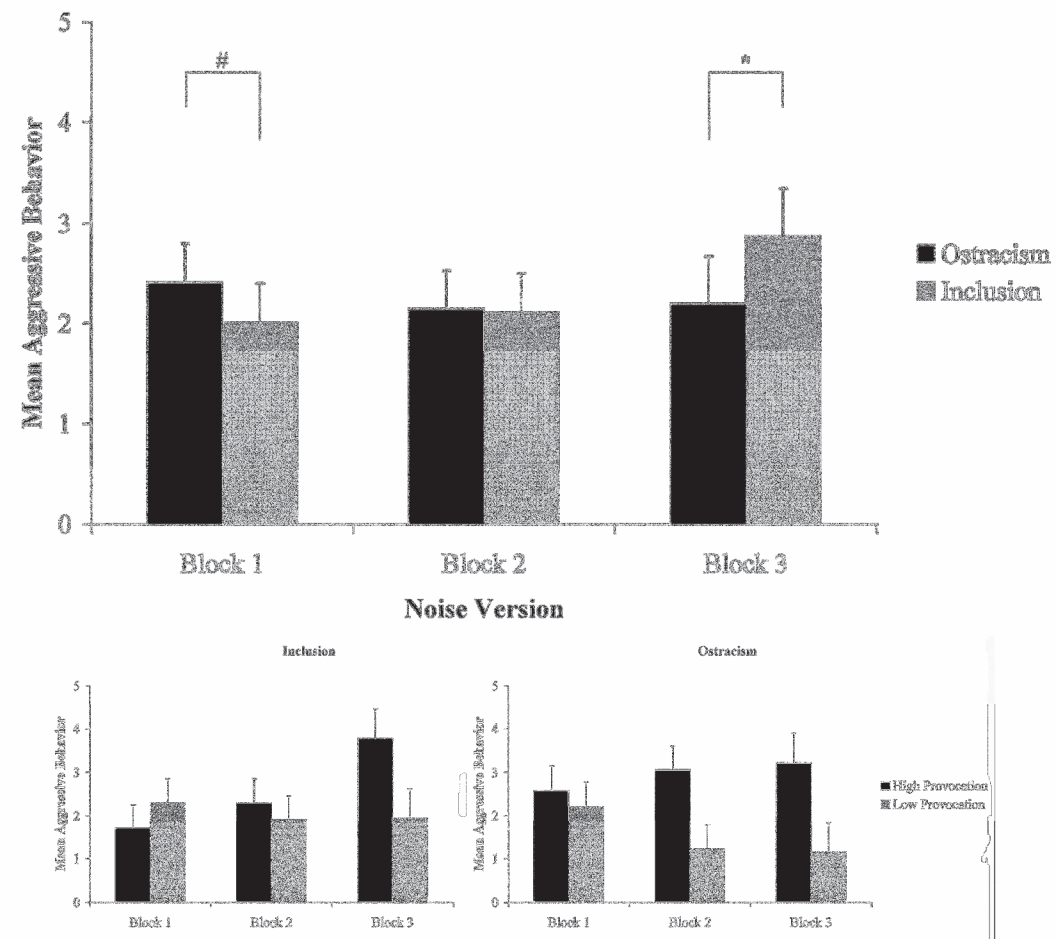

Figure 13. Top: mean aggressive behavior of included and ostracized participants in the three blocks of the TAP *Noise Version*. *Bottom:* mean aggressive behavior of the high and low provocation groups in the three blocks of the TAP *Noise Version* separately for the inclusion and ostracism conditions. Mean aggressive behavior represents the average of loudness and duration settings. Each block consists of ten trials (Block 1: trial 1 to 10, Block 2: trial 11 to 20, Block 3: trial 21 to 30). The error bars represent ± one standard error. $^{\#}p < .100$; $^{*}p < .050$.

(2) Effect of Ostracism. In the *Noise Version*, ostracism seemed to increase aggression in the first TAP Block but to decrease aggression in the third TAP Block (see Figure 13). In the high provocation condition, ostracized participants showed less retaliation to increasing levels of provocation than included participants as can be seen in the lower increase of aggressive behavior from Block 1 to Block 3 in ostracized compared

to included participants (see Figure 13). Ostracized participants in the low provocation group appeared to continually decrease their aggressive behavior. A significant interaction between Inclusionary Status and TAP Block confirmed this, $F(2,48) = 3.39$, $p = .042$, $\eta^2 = .12$. According to post-hoc tests, ostracized participants tended to show more aggressive behavior in Block 1 ($p < .10$) but less aggressive behavior in Block 3 of the TAP ($p < .050$).

In the *Money Version*, participants' sex appeared to be an important predictor of aggressive behavior in the TAP (see Figure 12). There was a significant interaction between Sex and Inclusionary Status, $F(1,24) = 5.09$, $p = .033$, $\eta^2 = .18$. Post-hoc tests revealed that in the inclusion condition, women were significantly more aggressive than men ($p < .050$). Having experienced ostracism resulted in an increase of aggressive behavior in men. Ostracized men tended to subtract more money from their opponent's account than included men ($p < .100$; see Figure 14). In addition, we found a significant interaction between Sex and TAP Block, $F(2,48) = 3.30$, $p = .045$, $\eta^2 = .12$. Post-hoc tests indicated that the women's aggressive behavior significantly increased from Block 1 to Block 3 ($p < .010$) and from Block 2 to Block 3 ($p < .050$), while the men's levels of aggression remained constant throughout the experiment. This resulted in a significant difference between male and female participants in TAP Block 3 ($p < .010$), with female participants showing higher levels of aggressive behavior than males. There were no significant interactions of Inclusionary Status and Provocation in the Noise or in the Money Version (all Fs ≤ 2.25, ps $\geq .117$).

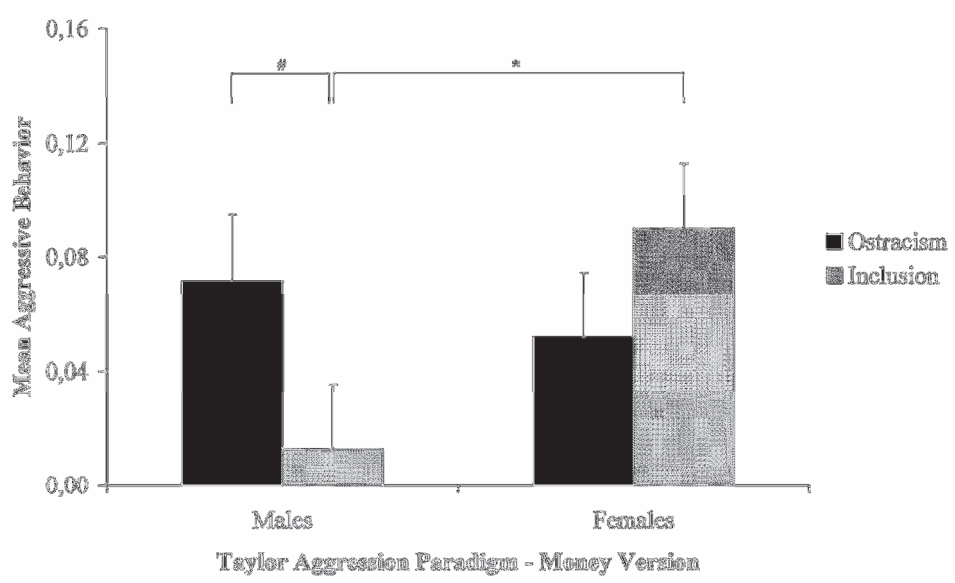

Figure 14. Mean aggressive behavior of included and ostracized males and females in the TAP *Money Version*. Mean aggressive behavior represents the average money subtractions. The error bars represent ± one standard error. $^{\#}p < .100$; $^{*}p < .050$.

Emotional Stroop task

The error rate in the emotional Stroop task was 6.4% (M = 5.0, SE = 0.4). The experimental groups did not differ in their error rates (all Fs < 2.30, ps > .100). Statistical analysis of the bias scores revealed a significant main effect of Emotion, $F(2,96) = 3.29$, $p = .041$, $\eta^2 = .06$. Post-hoc tests revealed that all participants responded slower to angry than to happy faces ($p < .050$). Interestingly, there was also a significant interaction between Inclusionary Status, Provocation, and Emotion, $F(2, 96) = 3.41$, $p = .037$, $\eta^2 = .07$. Relative to included participants, ostracized participants who were exposed to low provocation showed a greater response bias for fearful faces ($p < .050$). After high provocation, however, ostracized participants tended to show a response bias for angry faces ($p < .100$; see Figure 15). While the little provoked ostracism group reacted slower to fearful than to happy faces ($p < .050$), the strongly provoked ostracism group showed slower reactions for angry than for happy faces ($p < .050$). Thus, provocation seemed to shift the implicit cognitive bias in ostracized participants from fearful to angry faces.

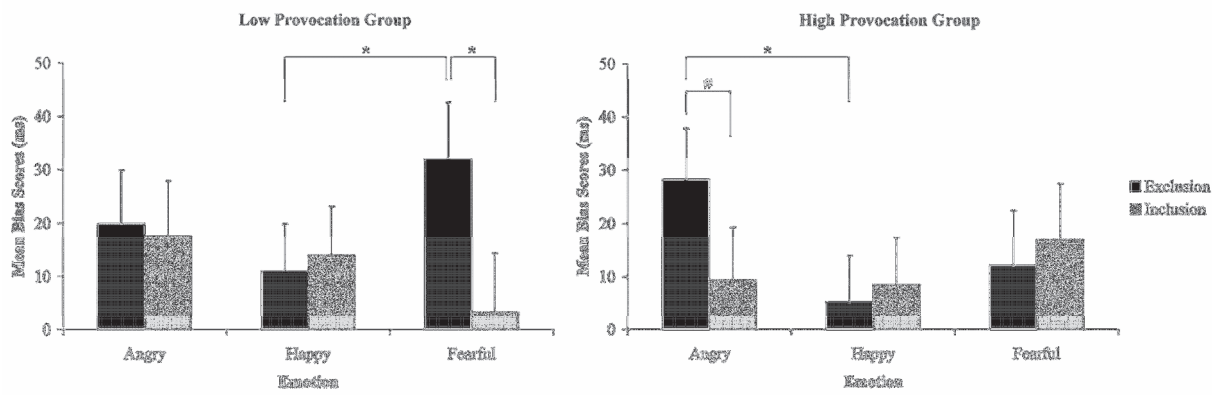

Figure 15. Mean bias scores of the four experimental groups for angry, happy, and fearful faces. Bias scores represent difference values (mean reaction times for emotional minus neutral facial expressions) in ms. The error bars represent ± one standard error. $^{\#}p < .100$; $^{*}p < .050$.

4.4. Discussion

The present study established effects of ostracism and continually increasing levels of provocation by peers on aggressive behavior against those peers in two different versions of the Taylor Aggression Paradigm. In addition, we revealed effects of both ostracism and provocation on the implicit processing of threat-related social information in an emotional Stroop task.

In both versions of the TAP, we found an increase in aggression with increasing levels of provocation replicating previous studies (Bertsch et al., 2009; Chermack et al., 1997; Epstein & Taylor, 1967). Strongly provoked participants also reported more feelings of anger than little provoked participants after the TAP. In line with previous studies, experienced social rejection acutely threatened interpersonal bonds and resulted in severe intrapersonal consequences, such as a low sense of belonging, control, self-esteem, and meaningful existence for the ostracized person as well as negative emotions, in particular anger (Buckley et al., 2004; Williams et al., 2000; Williams et al., 2002; Zadro et al., 2004). However, the effects of ostracism on aggressive behavior in the TAP depended on (1) the type of provocation and aggressive behavior, (2) the level of provocation, and (3) the participants' sex.

In the *noise version*, ostracized participants exposed their game partners to more painful noises than included participants directly after experiencing ostracism (TAP Block 1). This is in line with other studies using noise blasts in similar competitive reaction time games as a measure for rejection-induced aggression (DeWall et al., 2009; Twenge et al., 2001). However, the present study also revealed that ostracized participants did not respond to their partner's increasing provocation by showing similar strong retaliations as observed in the highly provoked included participants (TAP Blocks 2 and 3). When exposed to very high levels of provocations (TAP Block 3), ostracized participants were even less aggressive than included participants.

Previous studies have concentrated on the noise blast settings in the first trial or block of similar competitive reaction time games, as these were regarded as a measure of rejection-induced unprovoked aggression (DeWall et al., 2009; Twenge et al., 2001). As most real-life interpersonal aggressive encounters are characterized by a reciprocal escalatory interchange of provocations (Taylor & Chermack, 1993), we were interested in studying the effects of ostracism *and* continually increasing levels of provocation on aggression. Moreover, acts of extreme violence such as school shootings are often preceded by episodes of ostracism and/or social harassment (Leary et al., 2003). Interestingly, the present results indicate that when provocation increases, ostracized participants do not directly retaliate with likewise increasing levels of aggression. Their behavior to increasing peer provocation may rather reflect learned helplessness. When individuals expect that whatever they do does not matter, they will become helpless, fail to initiate any action, and consequently show reduced aggressive behavior (Peterson, Maier, & Seligman, 1993). In the present study, ostracized participants were exposed to a highly

aversive, uncontrollable social situation during the Cyberball game. At the beginning of the reaction time game (TAP), these participants took the opportunity to retaliate for being ostracized by exposing their excluders to more painful noises. When the game went on and ostracized participants realized that they were again in an uncontrollably aversive situation, in which their partners exposed them to more and more painful noises no matter how they behaved (in the high provocation group), this might have resulted in a state of learned helplessness and they may just have waited for the game to end.

In the low provocation condition of the noise version, ostracized participants were also more aggressive than included participants directly after being socially excluded (TAP Block 1). However, when the game went on (TAP Blocks 2 and 3), these participants showed the least aggressive behavior of all groups. After the aversive experience during the Cyberball game, they may have expected a similar treatment by their partners in the Taylor Aggression Paradigm, i.e., being exposed to uncontrollably painful noises. Their partner's friendly and less provocative behavior may have been a positive surprise to the ostracized participants, which they could have seen as an opportunity to reestablish the thwarted need of belonging. The best way to achieve this was by showing friendly and socially desirable, less aggressive behavior themselves. Similar to this, previous studies found that socially excluded participants were not aggressive towards individuals who praised them (Twenge et al., 2001) or with whom they expected to interact later on in the experiment (for a review, see Twenge, 2005). In fact, these participants were even less aggressive then included participants who expected a future interaction with their game partner. Also, a friendly social interaction or writing about a friend, a family member, or a famous celebrity reduced aggression after social rejection (Twenge et al., 2007). All participants of the present experiment were freshmen in psychology – the experiment took place during the first weeks of the semester. Thus, future interactions with the other participants after the experiment were to be expected.

In the *money version*, participants' sex played a major role with regard to aggression. In the inclusion group, females were more aggressive than males. In the ostracism group, males tended to be more aggressive than females. Female participants were generally more responsive to increasing levels of provocation than male participants. Thus, ostracism increased aggression in males but rather decreased aggression in females. Moreover, the effects of increasing levels of provocation on aggression were stronger in female than in male participants.

Sex differences in social behavior after ostracism have also been found in a study by Williams and Sommer (1997). After a ball-tossing game with two other individuals, participants had to work on an idea-generation task either coactively, with the individual effort being evaluated, or collectively, so that the group's effort would be assessed. Males were socially loafing, i.e., they worked less hard collectively than coactively, whether they had been ostracized or included in the ball-tossing game. However, ostracized females socially compensated, i.e., they worked harder in the collective than in the coactive condition. In line with this, diminished aggression in ostracized women in the present study may be interpreted as an enhanced effort towards a pro-social goal, namely, being accepted by the group by showing socially desirable behavior.

In the money version, the present results replicated previously reported exclusion-induced aggression effects (e.g., DeWall et al., 2009; Twenge et al., 2001), but only for male participants. Sex differences in regard to the effects of ostracism on aggression have not been reported so far. A plausible reason for this can be seen at least partly in task-related differences. Although the TAP money version successfully induced aggression, as can be seen in the increased aggressive behavior and feelings of anger in the high provocation group, this task may have resembled a gambling task. The subtraction of money did not cause direct physical harm but a delayed monetary setback. Subtracting some money from the partner's account may have made the game more interesting and exciting by increasing one's own risk to gain less money at the same time. On average, females have been found to be more risk averse than males in financial decision-making (Byrnes, Miller, & Schafer, 1999). The present results indicate that these sex differences could be modulated by interpersonal devaluation. Thwarted feelings of belonging rather seem to increase risk aversion in females while they may increase risk taking in males.

Taken together, the present results indicate that being ostracized by socially significant peers enhances physical aggression directly after the experience of social exclusion. But if ostracized participants are additionally exposed to ongoing and increasing physical provocation, this rather leads to learned helplessness. Moreover, peer ostracism enhances risk taking behavior in males causing monetary harm to the ostracizing peers, but decreases risk taking in females. The present results also confirm the importance of an anticipation of future interactions with regard to the effects of ostracism and aggression (Twenge, 2005). Thus, the extent to which we were able to experimentally provoke aggression towards socially significant peers, particularly in face of a highly likelihood of

future (real word) interactions, is astonishing and points out the enormous power of social exclusion and peer harassment.

This was even more obvious with regard to effects of ostracism and provocation on social information processing. Although ostracism and high provocation did not directly lead to aggressive outbursts in all participants, it resulted in a lasting implicit cognitive bias for angry facial expressions. Compared to included participants, ostracized participants in the low provocation group showed a bias for or interference with fearful faces, i.e., longer reaction times for fearful than for neutral faces. Bias effects are seen as reflecting the salience of the stimulus to an individual (Riemann & McNally, 1995). Ostracism is a painful experience (Eisenberger et al., 2003) and seems to enhance the sensitivity to and salience of social signals related to rejection and fear even after having experienced a positive peer interaction in the TAP. This bias shifted towards angry faces in ostracized participants who were also exposed to high levels of provocation. Similar cognitive biases have previously been reported in highly aggressive and violent individuals (Smith & Waterman, 2003, 2004; van Honk et al., 2001a; 2001b), particularly after they had been insulted (Cohen et al., 1998; Eckhardt & Cohen, 1997). Moreover, the presence of biases for anger and threat-related material may indicate a potential to perceive situations as threatening and/or hostile (Cohen et al., 1998; Smith & Waterman, 2004). The biased response to threat-related social information of highly provoked, ostracized participants may thus indicate an increased likelihood for future maladaptive or inappropriate behavior (Crick & Dodge, 1994; Geen, 2001). Additionally, this hostile attribution bias could have negative effects on the cognitive construction of upcoming social interactions (Anderson & Bushman, 2002; Crick & Dodge, 1994). The present results confirm the findings of DeWall et al. (2009) in regard to the relevance of social cognitions in the relationship of social exclusion and aggression. They indicate that already a single (mild) experience of social exclusion and provoked aggression can result in implicit cognitive biases for aggression-related social information similar to highly trait or pathologically aggressive individuals.

Thus, experiences of ostracism and continually increasing provocation may not lead to an immediate outburst of aggression but to a lasting cognitive bias for threat-related social information. As similar implicit cognitive biases for aggression-related social information have been found in highly aggressive individuals, they may indicate an enhanced likelihood of future aggressive and violent behavior. Teachers, parents, and social workers should aim at identifying not only highly aggressive youngster but also the

socially excluded and harassed ones. Intervention programs which take implicit cognitive biases into account may therefore be a promising strategy to prevent aggression and violence.

Chapter V

General Discussion

In the first chapter of the present thesis, previous research on stress and aggression has been outlined. The chapter also introduced a psychosocial framework of aggression, the General Aggression Model (GAM; Anderson & Bushman, 2002), and gave an overview of neuroscientific findings on the neural correlates of aggression. Taken together, these – psychosocial, neuroscientific, and neuroendocrine – approaches indicate that (1) stress may be an important promoting factor for aggression, (2) there is considerable overlap in brain structures involved in the regulation of aggression and the generation of appropriate stress responses, (3) these brain structures are part of a fronto-limbic emotion regulation network, and (4) the processing of social signals and regulation of emotions might therefore play a crucial role in the relationship of stress and aggression.

Based on this, the aims of this thesis comprised the investigation of (1) the relationship of stress and aggression, (2) effects of stress and aggression on social information processing, i.e., the processing of emotional facial expressions, and (3) the temporal dynamics of these effects in healthy individuals. Therefore, three consecutive studies were performed which included an experimental manipulation of aggression (Studies 1, 2, 3) and social exclusion (Study 3), a pharmacological manipulation of acute cortisol levels (Study 2) as well as the measurement of aggressive behavior (Studies 1, 2, 3), reaction times (Studies 1, 2, 3) and ERPs (Studies 1, 2) in response to emotional facial expressions.

This final chapter will summarize and discuss the results of the three studies. After outlining the strengths and limitations of these studies, the chapter will conclude with suggestions on how the present work can be continued.

5.1. Summary of the results

In all three studies of the present work, aggression was provoked and measured with the Taylor Aggression Paradigm (TAP; Taylor, 1967). As high provocation significantly increased aggressive behavior in all studies, this further confirms the TAP as a valid measure to induce aggression in healthy individuals in laboratory settings.

The first study (Chapter II) examined the effects of aggression (high vs. low provocation in the TAP) on the processing of emotional faces in an emotional Stroop task using behavioral (reaction times) as well as electrophysiological (ERPs) data. Results revealed that experimentally provoked aggression influences social information processing beyond the actual aggressive encounter. Differences between strongly and little provoked participants were found at very early (P2) as well as later (P3) stages of emotional face

processing with greater centroparietal positivities due to high provocation. For P2 amplitudes, the effect of provocation was greatest for threat-related (angry and fearful) facial expressions, whereas this effect was independent of the emotional valence of the faces regarding P3 amplitudes. In addition, participants with high levels of trait anger showed slower reactions to all emotional faces when exposed to high provocation in the TAP. These findings indicate that after an aggressive encounter, people allocate more attention towards all kinds of socially relevant information.

The second study (Chapter III) investigated effects of exogenously elevated cortisol levels on aggressive behavior and the effects of both cortisol and aggression on social information processing. As in the first study, highly provoked participants showed enhanced early and late parieto-occipitally distributed positivities to all facial expressions compared to less provoked participants. However, the effects of provocation started somewhat earlier (P1) and were located more posterior than in the first study, maybe reflecting enhanced early visual processes in inferior occipital areas. Although the effect of provocation on the late positive potential started as early as 300 ms post-stimulus, its maximum was somewhat later (600–800 ms) than in the first study (P3: 300–400 ms). Interestingly, exogenously administered cortisol had temporally and spatially different effects on social information processing. Elevated cortisol levels decreased the sensitivity for (real) threats, as could be seen in rather intermediate and provocation-independent aggressive behavior as well as in a reduced early processing bias (reduced P2 amplitudes) for angry faces in the cortisol compared to the placebo group. In addition, elevated cortisol levels decreased reaction times to all facial expressions, indicating increased avoidance behavior for all kinds of social information.

The third study (Chapter IV) comprised a behavioral experiment in which participants were either exposed to ostracism or inclusion in a virtual ball-tossing game (Cyberball; Williams & Jarvis, 2006) and were then either strongly or little provoked in the TAP. Again, the effects of aggression, and this time social exclusion, on social information processing were investigated with an emotional Stroop task. Two different kinds of provocation (and aggressive behavior) were applied, namely, loud noise and monetary setback. Effects of ostracism on aggression depended on the level and type of provocation as well as the participants' sex. Although ostracism did not directly increase aggression in all participants, it enhanced the sensitivity for and attention to angry faces when combined with high levels of provocation.

5.2. Discussion and Integration of the Results

5.2.1. Stress and Aggression

The administration of cortisol and the exposure to social exclusion were both able to alter aggressive behavior in healthy individuals. However, the effects were more complex than one might expect based on the findings of previous studies.

Concerning the effects of cortisol on aggression, most previous studies have been correlational in nature, assessing associations between basal or acute levels of cortisol and the severity of symptoms in pathologically aggressive individuals (see, for instance, Cappadocia et al., 2009; van Goozen et al., 2007, for reviews). Contrary to their findings, the few studies with healthy volunteers reported positive correlations between acute cortisol levels and aggressive behavior in laboratory settings (Gerra et al., 2001a; 2001b; 2004; 2007). However, these studies only included male participants and did not manipulate cortisol levels pharmacologically or experimentally. The second study of the present work was the first to do this. It can thus be regarded as a continuation of the animal studies of Kruk and colleagues (2004), who found lowered thresholds for (hypothalamic) aggression in rodents with pharmacologically elevated corticosterone levels. Contrary to this, the exogenous administration of cortisol in the second study did not result in a prominent increase of aggression in healthy humans. However, it did reduce the discriminative sensitivity for real threats and led to rather intermediate levels of aggression that were independent of provocation. In line with the rodent findings, this may indicate a cortisol-induced decrease in thresholds for aggression. Of course, more research is needed to systematically disentangle the effects of cortisol on aggression in humans (see also Chapter 5.3.3). This becomes even more obvious when one takes into account the differing effects of pharmacologically elevated levels of the stress hormone cortisol and those of experimentally induced social exclusion, a strong social stressor. Although the third study did not include any kind of physiological (endocrine) measure, it seems plausible that the dynamic effects of social stress, including endogenous increases in cortisol levels, autonomic arousal as well as feelings of distress and negative affect, differ from those associated with the intake of 20 mg hydrocortisone.

Regarding the effects of social exclusion on aggression, the results of the third study partly confirmed previous finding indicating aggression-enhancing effects of social exclusion (e.g., DeWall et al., 2009; Kirkpatrick et al., 2002; Twenge et al., 2001; Warburton et al., 2006). However, this study also showed that social exclusion may not

necessarily and under all circumstances result in immediate aggressive outbursts. Several person and situation factors, such as the level and type of provocation (Chapter IV), the individual's sex (Chapter IV), expectations about future social interactions (Chapter IV; Twenge, 2005; 2007), perceived control (Warburton et al., 2006), and social cognitions (DeWall et al., 2009), seem to mediate the link between social exclusion and aggression. This is in line with Anderson and Bushman's General Aggression Model, which sees aggression as a result of complex interactions of multiple situation and person factors (Anderson & Bushman, 2002).

5.2.2. Stress, Aggression, and Social Information Processing

Exogenously administered cortisol as well as experimentally induced social exclusion and aggression affected the processing of emotional facial expressions. Again, the effects were complex. After a highly aggressive encounter, all kinds of social information gained relevance. Thus, the highly provoked participants of the first study, and in particularly those with high levels of trait anger, showed slower (biased) reactions to all emotional faces (Chapter II). This indicates that aggression influences how a person perceives and behaves in future social encounters (Anderson & Bushman, 2002) and also stresses the high relevance of social information processing in the context of aggression (Crick & Dodge, 1994). Furthermore, experiences of social exclusion *and* aggression enhanced the specific relevance of angry facial expressions in the third study (Chapter IV). Similar implicit biases for threat-related material have been reported in individuals with high levels of trait anger as well as violent criminals (Smith & Waterman, 2003, 2004; van Honk et al., 2001a; 2001b). In line with assumptions by Berkowitz (1990) and Kruk et al. (2004), this may suggest that multiple experiences of social rejection prime information processing towards social threats and thus increase the likelihood for attack in future social encounters.

Contrary to this, exogenously administered cortisol reduced the discriminative sensitivity for threat-related social signals (angry faces). This may have led to faster responses for all kinds of social information and rather intermediate and provocation-independent levels of aggression in the second study (Chapter III). In line with these findings, the lower thresholds for aggression in rodents with acutely elevated cortisol levels may be explained by a reduced ability to discriminate real threats from non-threatening social signals. In the context of aggression or social competition, increased fight/flight tendencies (faster attacks in rodents, general avoidance of social signals and provocation-independent levels of aggression in humans) may be adaptive and even

lifesaving. De Kloet et al. (1999) assume that the effects of glucocorticoids on cognition are mediated by mineraloglucocorticoid receptors (MRs) and glucocorticoid receptors (GRs) in the brain and depend on the relative activation of these receptors at various stages of information processing. The relative activation of MRs and GRs depends on the context, and glucocorticoids influence information processing conditionally so that the adaptive behavior which is most relevant to the situation is increased.

Taken together, the results suggest that, in healthy individuals, stress and aggression affect the processing of social information conditionally by supporting behavioral tendencies that may help protect the organism in future social challenges. The administration of drugs in the second study by itself may have increased the participants' level of arousal and maybe their subjective anxiety. Along with the exposure to a highly competitive social situation, this may be the reason for increased avoidance tendencies to socially threatening information (faster reactions to angry faces) in all participants. Exogenously administered cortisol may have increased and extended this adaptive avoidance behavior to all kinds of social information. Contrary to this, the setting of the third study, including an introduction to socially relevant peers and two interactive games with them, may have increased the relevance of social threat-information overall, which would explain the greater attendance of and biased responses to angry faces in all participants. Being exposed to social rejection and provocation increased this effect. Unfortunately, this study did not include the measurement of acute cortisol levels or underlying cortical processes.

Additionally, differences in the experimental design of the three studies (i.e., drug administration; manipulation of the inclusionary status) and variations in the emotional Stroop task itself (i.e., masking after 26.7 ms vs. 16.6 ms vs. unmasked presentation; color- vs. sex-naming; and vocal vs. manual response mode) may also account for the differing results. In fact, a recent meta-analytic study (Bar-Haim et al., 2007) revealed a strong dependency of threat-related bias effects on experimental paradigms and conditions, including the stimulus material and presentation time. Biased (e.g., Wilson & MacLeod, 2003), but also indifferent (e.g., Thomas et al., 2007) and even facilitated (e.g., Williams, Watts, MacLeod, & Matthews, 1988) responses to threat-related stimuli have previously been reported in healthy individuals. Hence, more studies using other cognitive-emotional tasks are necessary to further investigate the role of social information processing in the context of stress and aggression (see Chapter 5.3.3).

5.2.3. Temporal Dynamics of Cortisol and Aggression Effects on Information Processing

In two studies (Chapters II and III) of the present work, ERPs were measured during the emotional Stroop task to gain more insights into the temporal dynamics of stress and aggression effects on information processing. In both studies, highly provoked participants allocated more attention to any kind of social information, as could be seen in enhanced early and later posterior positivities. In other words, the induction of aggression produced a general gating effect of the neural responses at the level of both early and later ERP components. Along with previous EEG and fMRI studies (Krämer et al., 2007; 2008; 2009; Lotze et al., 2007), this shows that, even in healthy individuals, the exposure to a mild aggressive encounter in a laboratory setting has lasting effects on social information processing. These alterations could thus reflect intact emotion regulation mechanisms that prevent an escalation or transfer of aggression from one situation to another. The fast and reliable knowledge about friends and enemies as well as the discrimination between threatening and non-threatening signals may be lifesaving. It is possible that these mechanisms fail in pathologically aggressive individuals, thus leading to impulsive and socially deviant behaviors. The present work also revealed that not only interindividual differences in trait anger, aggression, or impulsivity (e.g., Beaver et al., 2008; Brown et al., 2006; van Honk et al., 2001b), but also acute experiences of aggression influence the processing of social signals. Confirming Anderson and Bushman's (2002) assumptions, the present findings show that experiences in an aggressive encounter may alter internal (re)appraisal processes, thereby influencing the perception of and behavior in future social encounters.

Because of the relatively low spatial resolution of the EEG, it is difficult to hypothesize about the neural generators of these ERP components. However, evidence from intracranial recordings, lesion and neuroimaging studies indicate that these components may reflect activations in the fronto-limbic emotion regulation network: Occipital P1 amplitudes may reflect amygdaloid influences on early visual processes in the inferior occipital cortex (Adolphs, 2002; Vuilleumier et al., 2004); centroparietal P2 amplitudes may mirror striatal activation to threat-related information (Calder et al., 2004; Passamonti et al., 2008); and the broad, centroparietal distribution of P3/LPP amplitudes could reflect activity of subcortical structures, maybe modulated by re-entrant amygdaloid projections (Nieuwenhuis et al., 2005; Sabatinelli et al., 2005; 2007).

Interestingly, the second study revealed temporally and spatially distinct effects of provoked aggression and exogenously elevated cortisol on information processing. Contrary to the broad, posterior effects of aggression, which affected the processing of all facial expressions, acutely elevated cortisol levels reduced the early processing bias for angry faces, as could be seen in reduced frontocentral P2 amplitudes compared to the placebo group. This may reflect a cortisol-induced attenuation of OFC activation to threat-related information (see, for instance, Ashley et al., 2002; Kawasaki et al., 2001). The OFC plays an important regulatory role in the fronto-limbic emotion regulation network (Davidson et al., 2000). A possible consequence of reduced OFC activity may be decreased inhibitory influences on the amygdala and other subcortico-limbic structures such as the hypothalamus and the PAG, and thus increased fight/flight tendencies.

The results demonstrate that investigating neural mechanisms involved in the regulation of aggression and in the generation of appropriate responses to stress is of crucial importance. Examining biological correlates of aggression is necessary to gain further insights into the internal processes and mechanisms proposed by the GAM (Anderson & Bushman, 2002) and other prominent theories on aggression (Berkowitz, 1990; Crick & Dodge, 1994).

5.3. Strengths, Limitations, and Suggestions for Future Research

5.3.1. Strengths of the Studies

The present work is the first to integrate behavioral, self-report, psychophysiological, and endocrine measures to investigate effects of stress and aggression. The results confirm that various psychological and physiological methods are necessary to adequately examine causes and consequences of complex social behaviors such as aggression.

Second, using similar paradigms in three consecutive studies made a systematic investigation and the comparison of the effects of different experimental manipulations possible.

Third, whereas many previous studies relied on self-reported aggression or anger, aggressive behavior was experimentally provoked and measured in the present work. As self-reports only reflect general behavioral tendencies and are susceptible to social desirability and impression-making, particularly in the context of aggression, this is a noteworthy strength of the three studies.

Fourth, besides investigating effects of a social stressor (ostracism) on aggression, the present work also included a pharmacological manipulation of cortisol levels. The exogenous administration of cortisol allows to investigate the causal role of cortisol in (normal) aggression independently of other factors interacting with endogenous cortisol levels during a stress induction (e.g., autonomic arousal, individual differences in responses to stress). The actual manipulation of the stress system extends findings of previous studies but also shows that much more work is needed to disentangle the effects of specific stressors on aggression.

Fifth, ERPs were used in two studies to investigate the underlying mechanisms of social information processing in the context of stress and aggression. ERPs are highly sensitive measures of temporal dynamics of neural processing and thus highly suitable for the study of early stages of information processing, which are much harder to measure using purely behavioral paradigms (Hillyard & Kutas, 1983).

Finally, the inclusion of fearful and happy facial expressions as negative and positive emotional control stimuli in the emotional Stroop task as well as a non-facial control task (Chapter III) extends previous studies and offers new information about the specificity of the effects of stress and aggression.

5.3.2. Limitations

The limitations of the individual studies are addressed in detail in the respective chapters. This chapter addresses the general limitations of all three studies.

First, the experimental manipulation and measurement of aggression in a laboratory setting could be regarded as unnatural and artificial. However, the external validity of laboratory aggression paradigms has been confirmed, i.e., by considerably high correlations between effects in laboratory and field studies (Anderson et al., 1999) as well as the successful discrimination of violent and non-violent individuals (e.g., Wolfe & Baron, 1971). The Taylor Aggression Paradigm (Taylor, 1967) is a well-established method that has been extensively validated (Bernstein et al., 1987; Giancola & Chermack, 1998; Giancola & Parrott, 2008; Giancola & Zeichner, 1995; Wolfe & Baron, 1971). It can therefore be considered as one of the best methods to induce and measure aggression in the laboratory.

A second limitation is that only one task, namely, the emotional Stroop task, was used to investigate effects of stress and aggression on social information processing. This task was chosen as it had been shown to reveal large effects in the context of aggression /

anger as well as in stress / cortisol (Eckhardt & Cohen, 1997; Roelofs et al., 2007; Smith & Waterman, 2003, 2004; 2005; van Honk et al., 1998; 2001a; 2001b; van Peer et al., 2009a) and for its suitability for ERP measurements. Nevertheless, the above-mentioned inconsistency in the behavioral results has to be clarified with other cognitive-emotional tasks in further studies (see Chapter 5.3.3).

Finally, no physiological and endocrine measures were assessed in the third study. Although ostracized participants reported more distress, the physiological correlates of ostracism remain unclear in the present work. However, it is known from previous work that social exclusion may significantly increases HPA axis activity and autonomic arousal (Gunnar et al., 2003; Stroud et al., 2000; 2002).

5.3.3. Suggestions for Future Research

The present work is the first attempt to investigate the relationship between stress and aggression as well as the role of social information processing in the 'vicious circle of aggression' in healthy humans. As could be expected, these first findings raise a number of further research questions that may be answered by future studies. This last section of the thesis aims at capturing some of these questions and thus generates possible future research directions.

First, studies examining effects of stress and aggression on social information processing with other cognitive-emotional tasks, for instance, approach-avoidance task (Rotteveel & Phaf, 2004) or dot-probe task (MacLeod, Mathews, & Tata, 1986), are necessary to clarify and extend the results of the present work. Also, within-subject designs may be useful as they allow the investigation of intraindividual changes in information processing.

Second, a replication and extension of the third study using electrophysiological and endocrine measures could yield insights into the underlying neural processes and biological correlates of the effects of ostracism on aggression and social information processing.

Third, more research is needed to examine specific effects of different social and non-social stressors on aggression. A consequent next step would be to investigate dynamic effects of endogenously elevated cortisol levels on aggression. In addition, measures of autonomic activation, i.e., heart rate and blood pressure, as well as of basal HPA axis activity may offer further information about associations between the systems regulating stress and aggression. An ultimate goal could be an integration of genetic

analysis and the measurement of other hormones and peptides related to aggression, e.g., testosterone and serotonin.

Fourth, investigating individuals with particularly high or low levels of cortisol or trait aggression in similar psychosocial paradigms with psychophysiological methods could further clarify the role of social information processing in the relationship of stress and aggression.

Finally, studies using neuroimaging techniques such as fMRI should further investigate brain areas that play a crucial role in stress and aggression.

5.4. Conclusion

The presents work constitutes a first attempt to investigate the relationship between stress and aggression in healthy humans. As the processing of social signals is regarded as a potential link between stress and aggression, effects of both variables on the processing of social information, i.e., of emotional facial expressions, were examined. The results of three consecutive studies revealed that (1) aggression lastingly enhances the relevance of all kinds of social information, (2) the experience of social exclusion *and* aggression biases attention towards threat-related social information, (3) exogenously administered cortisol reduces the discriminative sensitivity for (real) social threats, which, in turn, may enhance fight/flight tendencies within a highly competitive social encounter. Confirming previous work, these findings indicate an association between stress and aggression and further underline that the pattern of effects regarding stress and aggression seems to be complex, confirming Anderson's and Bushman's (2002) assumptions that the causes of human aggression are manifold and comprise interacting effects of multiple situation and person variables. The present work also confirmed the relevance of social information processing and emotion regulation in the context of stress and aggression (Crick & Dodge, 1994; Davidson et al., 2000; Kruk et al., 2004). Besides raising a number of new research questions, the findings demonstrate the high potential of investigating complex social behaviors such as aggression with a combination of psychosocial, endocrine, and psychophysiological methods.

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List of Abbreviations

ACC	anterior cingulate cortex
ACTH	adrenocorticotrophic hormone
Ag/AgCl	silver/silver chloride
BPAQ	Buss and Perry Aggression Questionnaire
CRH	corticotropin-releasing hormone
dB	decibel
EEG	electroencephalography
EOG	electrooculogram
ERP	event-related potential
fMRI	functional magnetic resonance imaging
GAM	General Aggression Model
GR	glucocorticoid receptor
Hz	Hertz
LC-NE	locus coeruleus norepinephrine
LPP	late positive potential
M	mean
mPFC	medial prefrontal cortex
MR	mineraloglucocorticoid receptor
OFC	orbitofrontal cortex
P1	refers to the first positive stimulus-locked ERP component
P2	refers to the second positive stimulus-locked ERP component
P3	refers to the third positive stimulus-locked ERP component
PAG	periaqueductal gray
PFC	prefrontal cortex
PSAP	Point Subtraction Aggression Paradigm
SD	standard derivation
SE	standard error
TAP	Taylor Aggression Paradigm
VMPFC	ventromedial prefrontal cortex

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Erklärung

Hiermit erkläre ich, dass ich die vorliegende Dissertation selbständig verfasst und keine anderen als die angegebenen Quellen und Hilfsmittel verwendet habe. Zudem wurde die Arbeit an keiner anderen Universität zur Erlangung eines akademischen Grades eingereicht.

Trier, 6. Januar 2010

Katja Bertsch

Erklärung zu den Kapiteln II-IV

Bei der in Kapitel II beschriebene und in Bertsch et al. (2009) veröffentlichte Studie war ich verantwortlich für die Ideengenerierung, Hypothesenbildung, Datenerhebung, Datenauswertung, Dateninterpretation sowie die schriftliche Ausarbeitung des Manuskripts. Die Ko-Autoren der Studie waren mitverantwortlich für die Erarbeitung und Planung des Experiments, die Datenerhebung sowie Auswertung der Verhaltensdaten (R. Böhnke) und lieferten Anregungen bei der Diskussion der Befunde (E. Naumann, M.R. Kruk).
Eben dies gilt auch für die in den Kapiteln III und IV beschriebenen Studien, welche zur Veröffentlichung bei wissenschaftlichen Zeitschriften eingereicht sind.

Trier, 6. Januar 2010

Katja Bertsch