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).

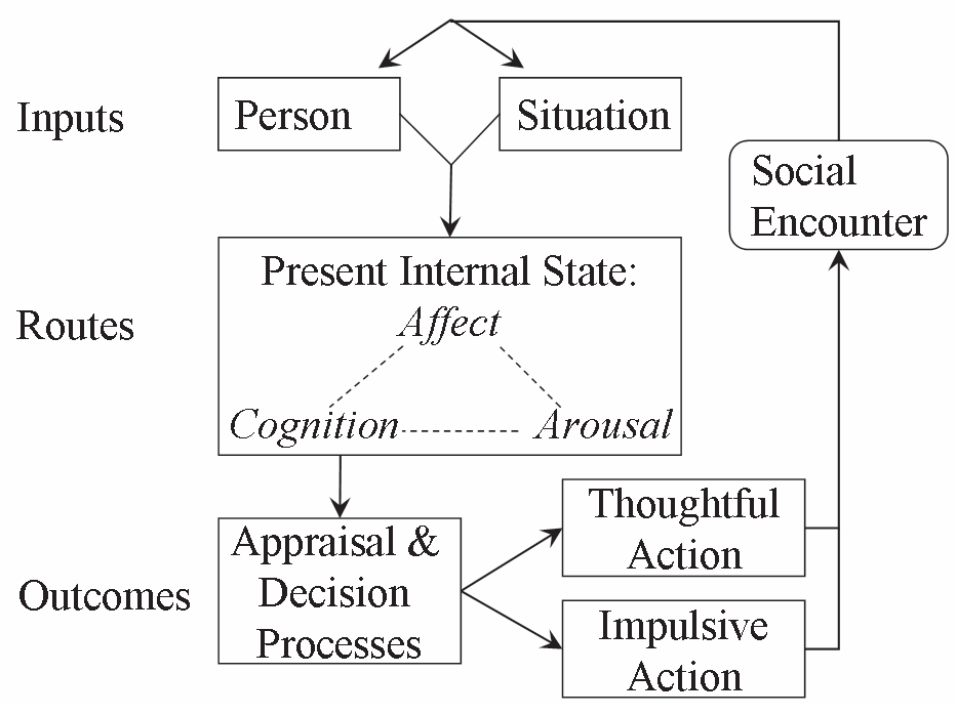

Figure 1. The General Aggression Model Episodic Processes (Anderson & Bushman, 2002, p. 34).

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.