Trait Aggressiveness Modulates Neurophysiological Correlates of Laboratory-induced Reactive Aggression in Humans

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Abstract

■ Reactive aggression following provocation is a frequent form of human social behavior. The neural basis of reactive aggression, especially its control, remains poorly understood, however. We conducted an event-related potential (ERP) study using a competitive reaction time task that elicits aggression through provocation. Participants were selected from a larger sample because of extreme scores in trait aggressiveness, yielding high and low trait aggressive groups. As each trial in the task is separated into a decision phase, during which the punishment level for the opponent is set, and an outcome phase, during which the punishment is applied or received, we were able to disentangle provocation-related and evaluation-related modulations of the ERPs during the aggressive interaction. Specifically, we observed an enhanced frontal negativity during the decision phase under high provocation that was positively correlated with the participants' ability to refrain from retaliation. This held true for high trait aggressive participants only, pointing to a higher need for inhibitory and control processes in these people when provoked. During the outcome phase, we detected a mediofrontal negativity in loss compared to win trials, resembling previous ERP findings to negative feedback stimuli, which have been linked to the evaluation of an outcome's valence. This mediofrontal negativity was differentially pronounced in aggressive and nonaggressive participants: Nonaggressive participants showed only a slightly smaller mediofrontal negativity in win than in loss trials, suggesting that for them punishing the opponent had a similar negative valence as being punished. ■

INTRODUCTION

Aggression is a common behavior in both humans and animals that can be defined as ''any behavior directed toward the goal of harming or injuring another living being that is motivated to avoid such treatment'' (Baron, 1977). Although neuroscientific research on aggression has had a strong focus on the relationship between psychopathology (e.g., antisocial personality disorder) and brain regions (Blair, 2004; Anderson, Bechara, Damasio, Tranel, & Damasio, 1999), aggressive behavior is frequent also among neurologically and psychologically healthy people, asking for an explanation of its causes and underlying mechanisms.

In a recent review, Anderson and Bushman (2002) presented a general model of aggressive behavior (General Aggression Model [GAM]), stating that situational variables (for instance, provocation or pain), together with personal variables (such as traits, sex, etc.), influence aggressive behavior through the mediating effects of cognition, affect, and arousal. The outcome of these cognitive and affective processes are appraisal and decision processes, which finally lead to either thoughtful or impulsive actions, depending on the invested amount of cognitive control and effort. The GAM accounts for the differential effects of, for instance, cognitive primes (such as pictures of weapons) or pain on aggressive behavior and affective reactions (Lindsay & Anderson, 2000). However, simply observing an aggressive action reveals little about the underlying motivations and decision processes (Bushman & Anderson, 2001) or about its neural underpinnings. The present study is thus aimed at tapping into the decision and evaluation processes during an aggressive social interaction by taking a closer look at the time course of neurophysiological responses (event-related potentials, ERPs).

A reliable method to elicit aggression in an experimental setting is a competitive reaction time task, which entails punishment of the loser by the winner as suggested by Taylor (1967). Through provocation (selection of different punishment levels by the opponent), the Taylor Aggression Paradigm (TAP) seeks to elicit aggression, operationalized as the intensity of punishment administered to the opponent. The paradigm's convergent validity, as well as its discriminant and external validity, has been found to be high (Anderson, Lindsay, & Bushman, 1999; Giancola & Zeichner, 1995; Bernstein,

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Richardson, & Hammock, 1987). As each single trial comprises a decision phase during which the participant is required to set the prospective punishment for the opponent, and an outcome phase during which the punishment is applied or received, the paradigm in conjunction with neurophysiological recordings enables us to delineate the neural correlates of the different decision and evaluation processes underlying reactive aggression.

In two recent functional magnetic resonance imaging (fMRI) studies, modified versions of the TAP were used to characterize the neural correlates of social reactive aggression (Krämer, Jansma, Tempelmann, & Münte, 2007; Lotze, Veit, Anders, & Birbaumer, 2007). Presenting one opponent who turned from nice to unfair during the experiment, Lotze et al. (2007) could demonstrate different roles of the ventral and dorsal medial prefrontal cortex in reactive aggression, with the former thought to be related to affective processes such as compassion and the latter involved in cognitive processes elicited by more intense social interaction processes. By introducing two opponents—one highly and one less provoking—Krämer et al. (2007) were able to dissociate general social interaction processes and cognitive and motivational mechanisms specific for reactive aggression, reflected in activations in the dorsal and rostral parts of the anterior cingulate gyrus (ACC) and the striatum.

Although, up to now, no ERP study has taken a direct look at the temporal dynamics of an aggressive interaction, studies related to social interaction, inhibition, and the processing of feedback hint at the ERP components that could be of interest for the present study. In a first pilot study employing the TAP (unpublished data), we observed modulations of specific ERP components during the decision phase. In particular, we detected provocation-related differences in the time range of the N2 (maximum at 250–300 msec) and in a later right fronto-lateral negativity (400–600 msec).

The N2, a fronto-centrally distributed component, is observed in experimental settings calling for an inhibition of motor responses as in go/no-go tasks (Falkenstein, Hoormann, & Hohnsbein, 1999; Pfefferbaum, Ford, Weller, & Kopell, 1985) or in situations of stimulus–response incompatibility (van Veen & Carter, 2002). Evidence from animal and human studies suggest that the N2 in go/no-go tasks reflects inhibitory mechanisms emanating from areas in the prefrontal cortex (Pliszka, Liotti, & Woldorff, 2000; Sasaki & Gemba, 1986). Children with attention-deficit/hyperactivity disorder showed a reduced right frontal N2 in a stop-signal task, related to their core deficit in inhibitory control (Pliszka et al., 2000). Results of an fMRI study suggest a widely distributed cortical network including the dorsolateral and inferior frontal lobe underlying response inhibition (Garavan, Ross, & Stein, 1999). On the other hand, tasks calling for the monitoring for response competition, such as the Eriksen Flanker task, give rise to a fronto-central negativity that similarly has been termed N2 but for which the anterior cingulate gyrus has been identified as the likely generator (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002). In the paradigm used in the present study, provocation may lead to both a higher need for monitoring processes and inhibition of predominant aggressive reactions during the selection of the prospective punishment. This is suggested by the GAM, which refers to cognitive control and monitoring processes that might prevent an aggressive retaliation (Anderson & Bushman, 2002). Moreover, the results from a previous fMRI study on reactive aggression (Krämer et al., 2007) demonstrated a recruitment of cognitive control processes in response to a higher level of provocation. Therefore, N2-like negativities are of special interest in the decision phase.

For the outcome phase, the feedback-related negativity is of relevance (FRN, in the literature also referred to as mediofrontal negativity [MFN]) (Müller, Möller, Rodriguez-Fornells, & Münte, 2005; Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997). The FRN is an enhanced negative deflection following negative in comparison to positive feedback, with an onset at about 250 msec and a fronto-central maximum. Converging evidence suggests that the FRN reflects the evaluation of the valence of an outcome and is thus sensitive to the motivational significance of ongoing events. Source modeling studies have suggested the rostral anterior cingulate, the posterior cingulate gyrus (Müller et al., 2005; Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005), and right superior frontal gyrus (Nieuwenhuis et al., 2005) as generators of the FRN. The FRN can provide insight into the participants' motivational and evaluative processes in reaction to the provocation und punishment.

As suggested by the GAM, both personality (as traits) and situative factors (as provocation) influence the cognitive and motivational processes associated with aggressive acts. Personality traits that have been shown to affect someone's proneness to aggression include her or his trait aggressiveness, narcissism, impulsivity, or emotional susceptibility (Bettencourt, Talley, Benjamin, & Valentine, 2006; Anderson & Bushman, 2002). Trait aggressiveness has been shown to affect aggressive behavior both under neutral and provoking conditions (Bettencourt et al., 2006). It is thus together with irritability the personality variable with the most reliable influence on laboratory-induced aggressive behavior. For the present study, we thus selected student participants on the basis of their score in an aggression questionnaire (Hampel & Selg, 1975), yielding two groups of high (HT) and low trait (LT) aggressive participants. This design affords us with the possibility to delineate the impact of both personality (trait aggression) and situative factors (provocation) on cognitive and motivational processes during an aggressive interaction. Besides the impact of traits, the search for sex differences in aggression has generated a considerable

amount of research literature, with controversial results, however (Bettencourt & Miller, 1996; Eagly & Steffen, 1986; Frodi, Macaulay, & Thome, 1977). Although researchers largely agree on a preponderance of men in physical aggression (Anderson & Bushman, 2002; Eagly & Steffen, 1986), they acknowledge a dramatic reduction of sex differences under conditions of provocation (Bettencourt & Miller, 1996; Frodi et al., 1977; Taylor & Epstein, 1967). In the present study, we therefore included both women and men to be able to control for putative sex differences.

METHODS

Participants

Data of the trait aggressiveness questionnaire were obtained from 231 economics students (129 women; mean age = 22.6 years, *SD* = 1.9 years). As psychology students are familiar with this kind of experiment and can be expected to get suspicious, we decided to select subjects from a sample of economics students, who were unfamiliar with psychological experiments. Based on their general aggression score (see below for further explanation), we selected participants with extreme values (low/high) for the brain potential experiment.

Forty-nine young, healthy students (24 women; mean age = 22.9 years, *SD* = 1.9 years) participated in the electroencephalogram (EEG) study after giving informed consent. All had normal or corrected-to-normal vision and were free of neuropsychiatric disorders. Nine participants were excluded due to extensive eye movement or muscle artifacts. Thus, 40 participants (21 women) were included in the analyses. The study was performed in agreement with the Declaration of Helsinki and approved by the ethics committee of the University of Magdeburg. Participants received money or course credit for taking part.

Questionnaire

Participants for the ERP study were selected based on their trait aggressiveness, assessed with a German inventory for the assessment of factors of aggression (FAF, Fragebogen zur Erfassung von Aggressivitätsfaktoren) (Hampel & Selg, 1975). With this questionnaire, five subscales (spontaneous aggression, reactive aggression, impulsiveness, autoaggression, aggression inhibition) and a control scale (openness) can be obtained. Spontaneous aggression (19 items) refers to unrestrained verbal or physical aggression, a typical item is, ''I sometimes like to taunt others.'' Items of the reactive aggression scale (13 items) ask for aggressive reactions to some kind of provocation or unfairness, such as. ''If someone provokes me, I want to punish him badly.'' Items of the impulsivity scale (13 items) deal with the affective component of aggression, as ''I flare up quickly, but get over it quickly.'' The sum of the scales ''spontaneous aggression,'' ''reactive aggression,'' and ''impulsiveness'' gives a reliable measure for outwardly directed aggression (internal consistency Cronbach's alpha = .85) and was thus used for selection of HT and LT aggressive participants. The sum score has been proven to be significantly different between both adolescent and adult violent criminals on the one hand and nonviolent controls on the other hand (Hampel & Selg, 1975), providing evidence for its external validity.

Task and Procedure

Aggression was elicited and assessed using a modified version of the TAP (Taylor, 1967). Participants were instructed that they were playing successive competitive reaction time trials against one of two opponents in alternating blocks. The opponents (one man, one woman), confederates of the experimenters, met the participant prior to the experiment to jointly listen to the instructions: They were told that whoever lost would be punished by the opponent with a highly aversive noise. The severity of the punishment, that is the volume of the noise, had to be selected for each trial on a range from 1 to 8. Additionally, participants would lose a corresponding amount of money (lowest noise—10 cents, highest noise—40 cents) to assure the participants' motivation to avoid the punishment. In fact, selections of the putative opponents and outcome of the trials (50% winning and losing trials in each block) were under the control of the experimenter. The experiment comprised eight blocks of 40 trials each, yielding a total of 320 trials. Participants were told that the opponents would play in alternating blocks and rest during the others. At the end of the experiment, participants were completely debriefed about the deception and the experiment's motivation. We always introduced one man and one woman as opponents, as the sex of the target is known to affect the level of aggressiveness (Taylor & Epstein, 1967). As the respective target's sex was not revealed during the experiment, this effect could not influence the behavior, however.

At the beginning of each trial, participants were asked to consider the punishment for the opponent in the upcoming trial (in the following referred to as decision phase; duration of 1.5 sec). A prompt to press the respective keyboard button followed. After the reaction time task proper (a visual cue, which prompted the players to press a button as fast as possible), the selection of the opponent was presented: In half of the blocks, the opponent mainly selected a low punishment (level 1–4 in 80% of the trials; blocks of low provocation); in the remaining blocks, the opponent selected mainly a high punishment (level 5–8 in 80% of the trials; blocks of high provocation). Thus, the participant got the impression of playing blockwise against one fair and one rather unfair opponent. Finally, feedback was given

whether the participant had won or lost (in the following referred to as outcome phase, duration of 500 msec). On win trials, they had to elicit the noise punishment for the opponent by button press; on loss trials, they were exposed to the aversive noise via headphones. In 10% of the reaction time task, ''no-go'' trials were introduced to ensure the participants' attention. These trials were followed by feedback on the successful or failed inhibition. False alarms were punished by the experimenter with monetary subtraction, whereas successful inhibitions did not yield any consequences. The feedback phase of these trials was excluded from any further analyses, as it was unrelated to any punishment or retaliation. Prior to the experiment, the volume of the loudest noise was adapted to the participant's individual threshold for perceiving noises as aversive. Intertrial interval was 3 sec. After each of the eight blocks, they were allowed to rest. Stimulus presentation and behavioral data acquisition were controlled with Presentation software (www.neurobehavioralsystems.com). The experiment, including preparation and debriefing, had a duration of 90 min (for the timing of each trial, see also Figure 1).

EEG Recordings

The EEG was recorded from 27 tin electrodes mounted in an elastic cap (Easycap; positions: Fp1/2, F3/4, C3/4, P3/4, O1/2, F7/8, T7/8, CP1/2, P7/8, FC1/2, FC5/6, CP5/6, Fz, Cz, Pz, with reference electrodes placed on the right and left mastoids. During recording, all scalp electrodes were referenced against the right mastoid and off-line rereferenced against the algebraic mean of the activity at the two mastoid processes. Electrode impedances were kept below 5 kΩ. To monitor horizontal eye movements, electrodes were placed on the outer canthus of the right and left eyes. Vertical eye movements and blinks were monitored by an electrode placed below the right eye referenced to the right mastoid. EEG and electrooculogram were recorded continuously with a band pass of 0.095 to 70 Hz and digitized with a sampling rate of 250 Hz.

Off-line eye and muscle artifacts were rejected automatically. For nine participants with extensive blinks, eye movements were corrected with the technique of Second Order Blind Identification (Joyce, Gorodnitsky, & Kutas, 2004). Stimulus-locked ERPs were obtained separately for the different phases of the trial (decision phase: time-locked to the prompt to consider punishment for the opponent in the upcoming trial; outcome phase: time-locked to the feedback). The epochs were 1024 msec long, including a 100-msec baseline.

Data Analysis

Unless otherwise specified, only those 80% of trials were analyzed that followed a block-congruent selection of the opponent, that is, a high selection in a block with 80% highly provoking trials and vice versa. Selections of the participants under high and low provocation and reaction times were scored and compared with repeated measures analysis of variance (ANOVA; within-subject factor provocation: high vs. low; between-subject factor FAF: high vs. low trait aggressive, i.e., HT vs. LT).

As we observed that some participants' behavior was contrary to their questionnaire score (i.e., LT participants showed highly aggressive behavior and vice versa), we tested whether the neural responses depended rather on the experimentally induced aggression. To this end, we performed a median-split in each of the two groups (LT and HT) based on participants' overall aggressive behavior in the paradigm (mean selection in all trials). We thus yielded groups with low and high experimentally induced aggression, referred to as LE and HE, respectively (see Figure 2C). Note that as the median of selections in the two groups were different (LT = 4.23 and HT = 5.20), the groups LT–HE and HT–LE are in fact overlapping with respect to their behavior. We will thus consider the between-subject factor behavior (LE vs. HE) only in interaction with the between-subject factor FAF (LT vs. HT).

Event-related potentials were analyzed separately for the decision and outcome phase. For the *decision phase*, ERPs were quantified by a mean amplitude measure at different electrode positions. Based on the results of our previous pilot study, we used 12 electrodes (F3/4, C3/4, P3/4, F7/8, T7/8, P7/8) to yield three factors:

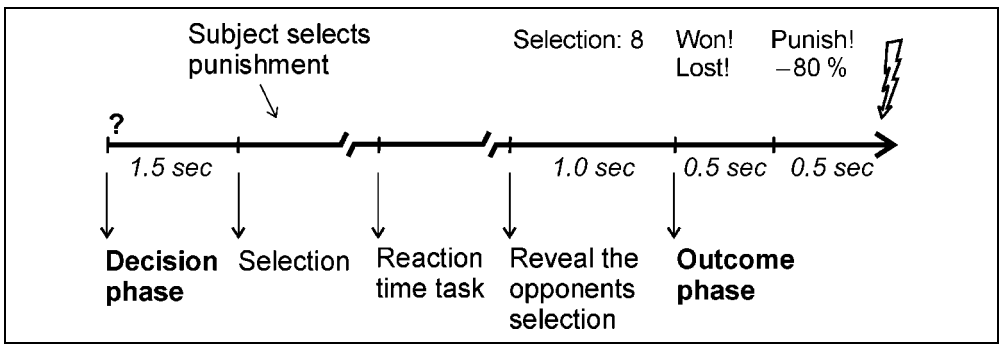

Figure 1. Time line for a single trial, a trial of high provocation (opponent selects 8) is depicted an example. The duration of the selection and reaction time task phases depended on the participants' reaction time. The intertrial interval had a duration of 3 sec.

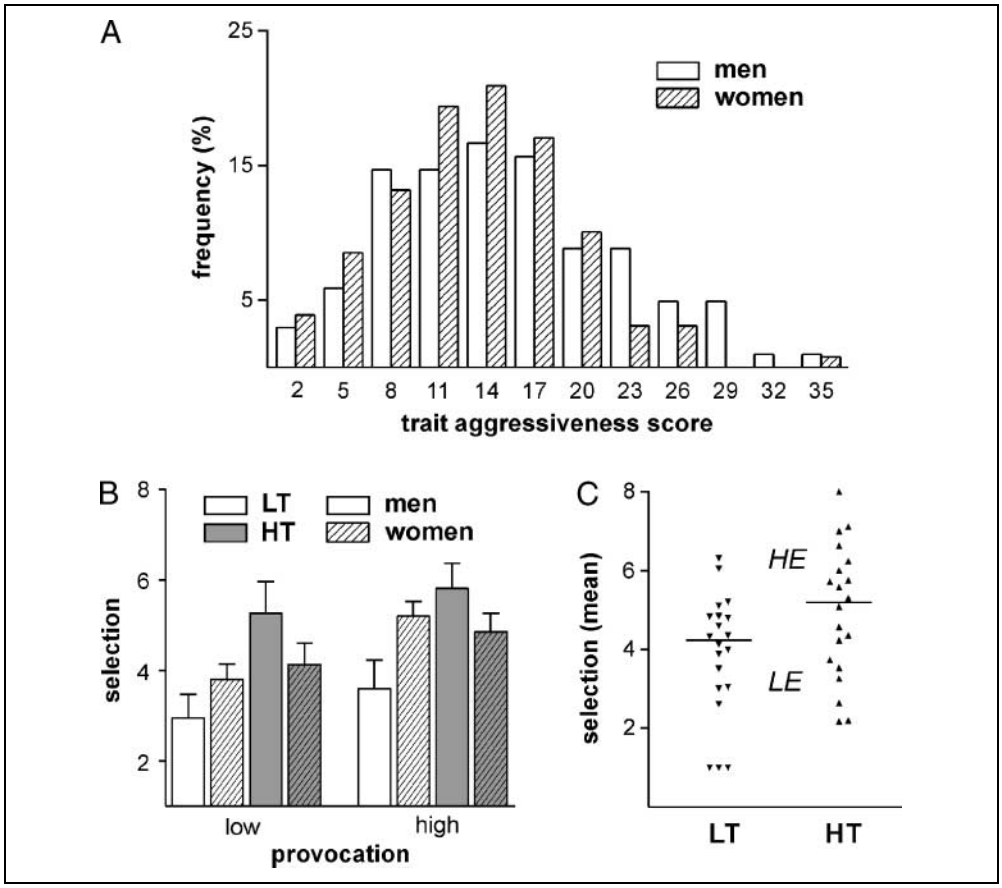

Figure 2. Behavioral results. Depicted first is the distribution of the aggressiveness scores of men and women in the questionnaire sample (n = 231). Each bar represents the percentage for a score range of three points, the numbers refer to the mean of the respective range (i.e., "2" means: aggression score of 1 to 3; A). Shown are the mean selections under low (left) and high provocation (right) in the different groups (B). White boxes show low trait aggressive (LT), gray boxes high trait aggressive (HT) participants. Blank boxes show men's data, whereas lined boxes show women's data. C shows the distribution of the mean punishment selection in both groups (HT and LT). The small lines mark the groups' median, and thus, the cut value to yield groups of low (LE) and high (HE) experimentally induced aggression.

anteriority (frontal, central, posterior; in the following referred to as ANT), hemisphere (right, left; referred to as HEM), and laterality (parasagittal and temporal; referred to as LAT). Two components were analyzed: a negative deflection around 300 msec ("N2," time window 270–300 msec) and a late frontal negativity quantified by a mean amplitude measure in the time window 350–600 msec. The ANOVA thus comprised the between-subject factors FAF (HT vs. LT) and behavior (HE vs. LE) and the within-subject factors provocation (high vs. low provocation trial), ANT, HEM, and LAT. For all statistical effects involving more than one degree of freedom in the numerator, the Huynh and Feldt (1976) correction was applied to correct for possible violations of the sphericity assumption. The corrected probabilities, together with the corresponding ε-values, are reported.

For the *outcome phase*, we examined the FRN. As the FRN is superimposed on the large positive deflection, we filtered the data with a band-pass filter of 4–10 Hz to remove the slow waves (see Luu & Tucker, 2001 for a similar procedure). As the FRN is known to be maximal at fronto-central sites, we subjected the participants' mean amplitude between 270 and 300 msec at Fz to a repeated measures ANOVA with the between-subject factors FAF and behavior and the within-subject factors feedback (won vs. lost) and provocation (high vs. low provocation trial). In this analysis, we included all trials and refer with high and low provocation to the actual selection of the opponent, presented directly before the feedback. Losing under high provocation thus means that the participant had to expect a high punishment and vice versa.

RESULTS

Questionnaire Data

The FAF mean score (see above for selected factors) was 13.19 (SD = 6.57), and higher for men (mean = 14.3, SD = 7.23) than for women [mean = 12.3, SD = 5.88; $t(229) = 2.32$, $p = .021$; see Figure 2A for the distribution]. From this sample, 24 HT and 25 LT aggressive participants were selected for the EEG study, of whom nine had to be discarded due to extensive artifacts. The mean score for the remaining 20 HT aggressive participants (11 women) was 23.8 (range 19 to 34, corresponding to a percentile > 82.0, with respect to the complete sample), and for the 20 LT aggressive participants (10 women) was 3.9 (range 0 to 7, percentile < 18.2). In this sample, only a small sex effect could be observed [main effect of sex: $F(1, 36) = 4.052$, $p = .052$], with no differences between high and low aggressive participants (interaction FAF × Sex: $F < 1$).

Behavioral Data

Participants selected higher punishments in blocks of high provocation compared to blocks of low provocation [main effect provocation: $F(1, 38) = 42.91, p < .001$]. HT aggressive participants showed more aggressive behavior [main effect FAF: $F(1, 38) = 4.42$, $p = .042$]. However, a weak-to-middle correlation of FAF score and average punishment selection confirmed the aforementioned observation of trait-incongruent behavior in some participants ($r = .317$, $p = .046$). The level of selected punishments increased over time, when comparing the four blocks in each condition, resulting in a significant main effect of block [repeated measures ANOVA with FAF, provocation, and block: main effect block, $F(3, 114) = 3.52$, $p = .023$]. No interaction with the level of provocation or the FAF was detected (both $p > .2$). The average level of selections under high provocation was in the first block 4.62 (±1.74) and in the last block 4.95 (±2.0), whereas under low provocation it increased from 3.76 (±1.65) to 4.19 (±2.09).

Interestingly, the effect of trait aggressiveness on the aggressive behavior was observed in the male participants only, whereas HT and LT aggressive women showed similar behavior. This was confirmed with a repeated measures ANOVA with the between-subject factors FAF and sex and the within-subject factor provocation [interaction FAF × Sex: $F(1, 36) = 5.37, p = .026$; main effect FAF in men: $p < .05$, in women: $p > .10$]. Women tended to react stronger on the provocation as indicated by a marginally significant interaction of Sex × Provocation [$F(1, 36) = 3.44$, $p = .072$; for the behavioral results, see also Figure 2B]. Participants' mean reaction time to select the noise level was 770.4 msec ($SD = 361.7$), with no differences between conditions and the groups (all main effects and interactions: $p > .05$). Participants' mean reaction time on the target was 257.3 msec ($SD = 82.8$), which again did not differ between the provocation level and the groups (all main effects and interactions, $p > .1$). During debriefing, participants reported to have been more aggressive under high than under low provocation [$F(1, 35) = 41.43$, $p < .01$]. Again, an interaction of FAF and sex indicated that trait aggressiveness was a significant predictor of reported aggression in men, but not so in women [$F(1, 35) = 4.79$, $p = .035$; main effect FAF in men: $p < .05$, in women: $p > .5$].

ERPs in the Decision Phase

ERPs in the decision phase were characterized by the typical N1 and P2 components, followed by an extended negativity. In high provocation trials, a right-lateralized, frontal negative deflection of the ERP was observable first in the time range of the N2 (250–300 msec) and then from 350 msec on. Visual inspection suggested that this held true for the high trait aggressive (HT) participants only (Figure 3A and B; see Figure 6 for the topography of these effects). Indeed, in the HT group, high provocation elicited an enlarged negativity compared to low provocation, indicated by the interaction Provocation × ANT × FAF [$F(2, 72) = 3.86, p = .045, \varepsilon = 0.6572$; see Figure 3A]. This effect was maximal at frontal sites and peaked around 270–300 msec. No such effect was detected in the LT group (HT: Provocation × ANT, $p < .01$; LT: Provocation × ANT, $p > .10$; see Figure 3B). Interestingly, this effect was seen only for those HT aggressive participants, who, nevertheless, did *not* behave aggressively in the experiment (i.e., the HT–LE group), as indicated by a (albeit marginal) significant interaction FAF × Behavior × Provocation × ANT [$F(2, 72) = 3.42$, $p = .0549$, $\varepsilon = 0.6572$; see Figures 4 and 6].

As mentioned previously, ERP effects resembling the N2 in the current experiment have repeatedly been linked

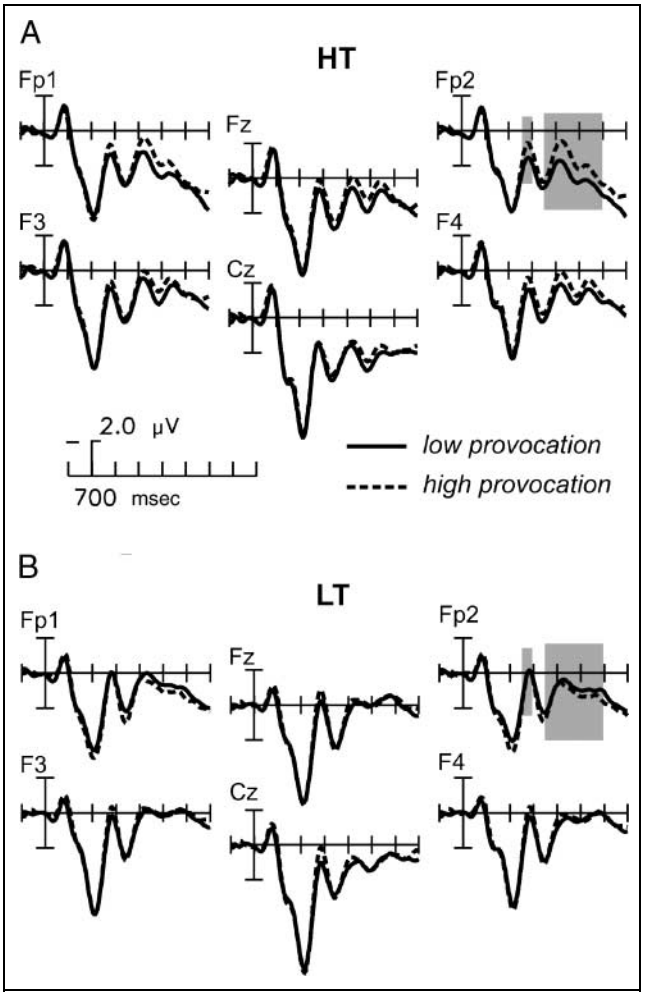

Figure 3. Grand-average ERPs in the decision phase. For illustration, the ERPs were low-pass filtered (high cutoff 12 Hz). Depicted are the ERPs in (A) the high trait aggressive group (HT) and (B) the low trait aggressive group (LT). Low provocation trials are shown with solid lines, high provocation trials are shown with dashed lines. Marked are the analyzed time windows for the two effects in the decision phase (early and late frontal negativity).

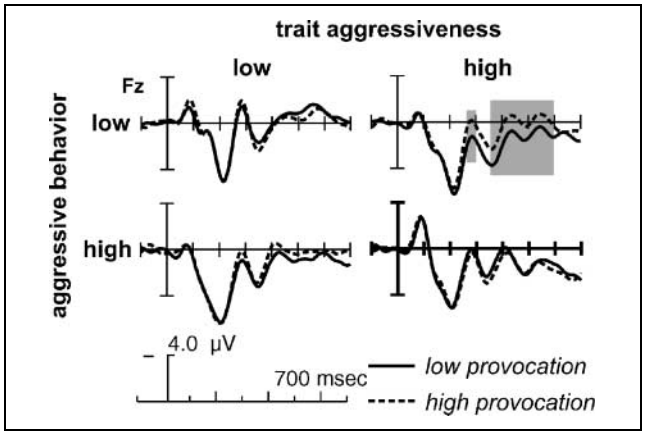

Figure 4. Grand-average ERPs at Fz in the decision phase. For illustration, the ERPs were low-pass filtered (high cutoff 12 Hz). Depicted are the ERPs in the four different groups: low (LE, upper part) and high (HE, lower part) aggressively behaving participants in the low trait aggressive group (LT, left) and the high trait aggressive group (HT, right). Low provocation trials are shown with solid lines, high provocation trials with dashed lines. Marked are the analyzed time windows for the two effects in the decision phase (early and late frontal negativity).

to inhibitory processes (Pliszka et al., 2000; Pfefferbaum et al., 1985). Assuming that the higher negativity reflects inhibitory processes, it should be largest for those HT aggressive participants who inhibit their predominant reaction to get back to the opponent, namely, those who show low aggressive behavior during the actual experiment. To further probe this hypothesis, we computed the mean amplitude of the difference waves (high minus low provocation) between 270 and 300 msec at Fz, where the maximum of the negativity was detected, and tested its relationship with the participants' mean selection, reflecting their average level of aggressive behavior (irrespective of the level of provocation). Although a clear correlation between aggressive behavior in the experiment and the enhanced negativity emerged for the HT aggressive participants ($r = .54$, $p = .014$), a similar relation was absent in the LT aggressive group ($r = .01$; see also Figure 5). The interaction effect in the moderator analysis (Cohen, Cohen, West, & Aiken, 2003) was marginally significant ($p = .060$). As the FAF questionnaire contains an inhibition subscale, we checked whether the participants' score on this scale did predict their behavior and/or the fronto-lateral negativity. Both the correlation with the aggressive behavior and with the magnitude of the neurophysiological effect were not significant, however (both $r < .1$, $p > .2$).

Following the early frontal negativity, we observed an enhanced late frontal negativity for high compared to low provocation trials. This effect was lateralized with a maximum over right frontal electrodes. Again, this effect was differentially affected by the participant's trait aggressiveness and experimentally induced aggressive behavior. We found in the HT aggressive group an enhanced frontal, right-lateralized negativity only for the HT–LE participants [high FAF: Behavior × Provocation × ANT × HEM: $F(2, 36) = 3.60$, $p = .038$, $\varepsilon = 0.9818$; see also Figure 4 and Figure 6]. The significant correlation of the participants' average aggressive behavior and the amplitude difference at Fz further supported this finding ($r = .48$, $p = .032$). No such relation was found for the LT aggressive participants (all interactions of involving Provocation × Behavior: $p > .1$; correlation $r = -.172$, $p > .1$), yielding a significant interaction effect in the moderator analyses ($p = .042$). Nevertheless, the interaction of the late frontal negativity effect with the between-subject factor FAF in the overall ANOVA was only marginally significant [Provocation × ANT × HEM × FAF × Behavior: $F(2, 72) = 2.50$, $p = .0992$].

The fact that the early and the later negativity during the decision phase had a similar fronto-lateral distribution and were similarly related to the participants' behavior suggests that it might be rather *one* underlying extended frontal negativity than two separate effects. If this holds true, no topographic differences should be detectable comparing the early and later effect. We therefore subjected the vector-normalized data to a repeated measures ANOVA to probe for interactions of time (270–300 msec vs. 350–600 msec) with provocation and electrode position (factors ANT, HEM, and LAT) (McCarthy & Wood, 1985). As both observed effects were strongest for the HT–LE participants, we performed those analyses with this group only. No significant interactions

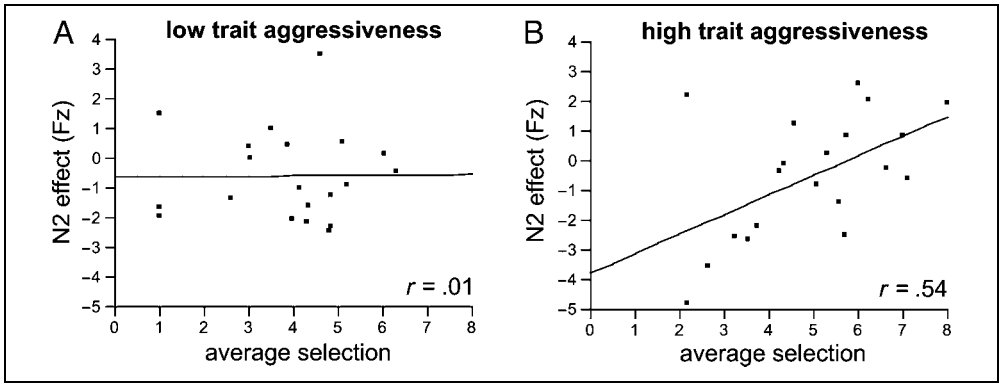

Figure 5. Correlation of aggressive behavior (mean selection over all blocks) and early negativity effect (amplitude of difference high minus low provocation trials). The left side shows the (nonsignificant) correlation in the LT aggressive group, the right side shows the correlation in the HT aggressive group.

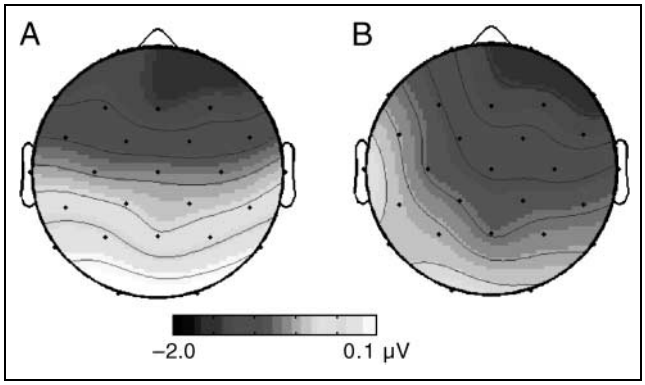

Figure 6. Maps showing the early (A) and the late frontal negativity (B) in the HT–LE group. The maps depict the mean amplitude of the difference between high and low provocation in the time window 270–300 msec and 350–600 msec.

were detected (all $p > .05$), further supporting the interpretation as one prolonged negativity.

As we observed significant sex differences in the behavioral data, we conducted post hoc analyses to test whether these behavioral effects were reflected in the neurophysiological data. To this end, we subjected the mean amplitude for both the early (270–300 msec) and late time window (350–600 msec) to a repeated measures ANOVA with the factors sex, FAF, provocation, ANT, HEM, and LAT. In contrast to the behavioral data, we did not observe any sex differences in the neurophysiological data, neither regarding the early nor the late frontal negativity (main effects of factor sex and all interactions, $p > .05$).

ERPs in the Outcome Phase

ERPs following negative feedback in both high and low provocation trials showed a clear MFN peaking around 300 msec in comparison to positive feedback [main effect of feedback at Fz: $F(1, 36) = 39.45$, $p < .001$; see Figure 7]. The FRN was independent of the level of provocation, that is, the magnitude of punishment to be expected (interaction Feedback × Provocation, $p > .1$).

The difference between positive and negative feedback was differentially pronounced in the different groups, reflected by the interaction of Feedback × FAF × Behavior [$F(1, 36) = 4.40$, $p = .043$; see Figure 8]. In fact, for the participants in the HT–LE group, the difference between negative and positive feedback was even absent (main effect of feedback, $p > .1$). Interestingly, it seemed that for both the HT–LE and LT–LE participants, positive feedback also elicited an MFN that was only slightly smaller than after negative feedback. This was confirmed by the interaction of Feedback × Behavior [$F(1, 36) = 7.15$, $p = .01$]. Although HE participants showed a clear difference between negative and positive feedback [Feedback: $F(1, 18) = 29.58$, $p < .001$], LE participants showed a smaller, albeit still significant, difference for losing and winning trials [Feedback: $F(1, 18) = 10.09$, $p = .005$]. This observation suggests that punishing the opponent had a similar negative valence for nonaggressive participants as being punished.

However, as argued previously, a group analysis based on a median-split is problematic, as it assigns participants to different groups who show, in fact, quite similar behavior, and thus, may yield misleading results. This is also true for the present set of data as the median of punishment selection was different in the HT and LT aggression groups. Thus, when comparing the HE and LE participants, the groups are, in fact, overlapping with respect to their behavior.

To therefore further probe the relation between the participants' aggressive behavior and their feedback evaluation, we compared the upper and lower third regarding the aggressive behavior (each group with $n = 13$, average selection >5.2 and <3.8, respectively). This was done irrespective of the participants' trait aggressiveness. This analysis confirmed our finding of a modulated FRN [Feedback × Third: $F(1, 24) = 7.53$, $p = .011$]. Although the negativity had a similar amplitude for win and loss trials in the lowest third (amplitude at

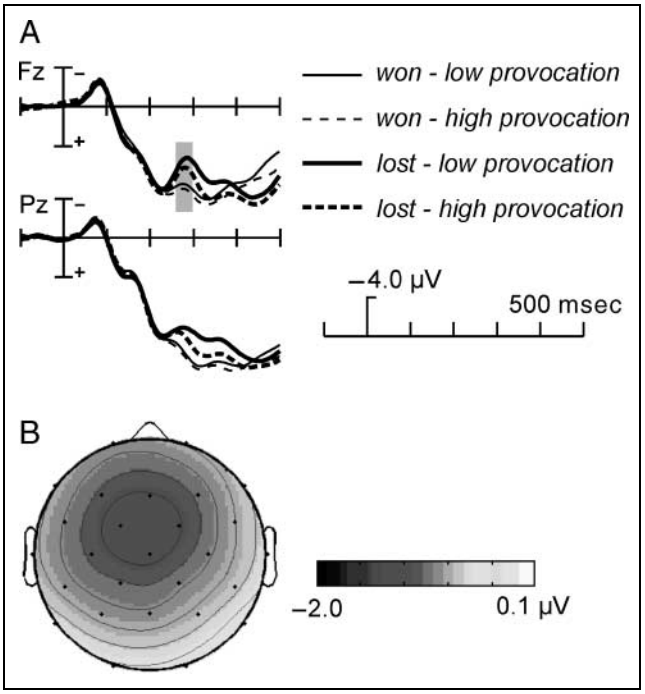

Figure 7. (A) Grand-average ERPs at Fz and Pz in the outcome phase. For illustration, the ERPs were low-pass filtered (high cutoff 12 Hz). Depicted are the ERPs in the whole sample ($n = 40$). At Fz, the time window of the maximum of the FRN is marked in gray. Depicted are the ERPs for the four different conditions: win (thin lines) and lost trials (thick lines), high provocation (dashed lines) and low provocation trials (solid lines). (B) Map showing the FRN effect in the complete sample (band-pass filtered 4–10 Hz). The map depicts the mean amplitude of the difference between negative and positive feedback in the time window 270–300 msec.

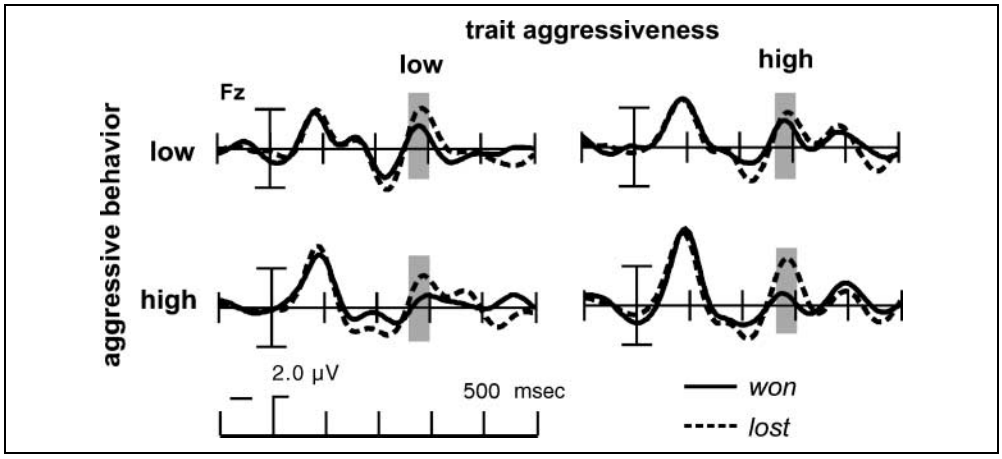

Figure 8. Grand-average ERPs at Fz in the outcome phase, separately for the four groups. The ERPs were band-pass filtered (4–10 Hz) to remove the underlying slow positive waves. Depicted are the ERPs in the different groups: low (LT, left) and high (HT, right) trait aggressive, separately for low (LE, upper part) and high (HE, lower part) aggressive behavior. ERPs following positive feedback are shown with solid lines, following negative feedback with dashed lines. The time window used for the FRN analyses is marked in gray.

Fz, wins: −0.95 μV; losses −1.36 μV; $p > .1$), it showed a significant modulation by the feedback in the most aggressive group (highest third; wins −0.25 μV; losses −1.83 μV; $p < .005$).

As the FRN has been related to the perceived valence of a stimulus (Yeung, Holroyd, & Cohen, 2005), our findings suggest that winning (and thus punishing the opponent) was associated with negative feelings for the less aggressive participants. This could reflect their empathic feelings for the punished opponents (Lotze et al., 2007). One might thus speculate that this emotional modulation of the punishment's evaluation is driving the regulation of aggressive behavior in less aggressive participants. This should cause lower punishment selections especially after winning trials in these participants. Indeed, nonaggressive participants selected lower punishments after win than after loss trials, in high provocation blocks at least (mean selection after winning: 2.74, after losing: 3.11; $p < .05$). This difference was not observable in low provocation blocks, most probably due to a floor effect (main selection after winning: 2.11, after losing: 2.19). Highly aggressive participants did not exhibit this modulation of aggressiveness after winning, resulting in the threefold interaction of Feedback × Provocation × Third on the selection in the consecutive trial [$F(1, 24) = 4.14$, $p = .046$].

Similarly to the decision phase, we performed post hoc analyses to probe for sex differences in the neurophysiological data. To this end, we subjected the mean amplitude at Fz for the FRN to a repeated measures ANOVA with the factors FAF, sex, feedback, and provocation. Again, no differential neurophysiological responses comparing men and women were detected.

DISCUSSION

The present study is the first to record neurophysiological responses during an aggressive interaction with the aim to tap into the decision processes and evaluation processes involved and to demonstrate their modulation by trait aggressiveness. Provocation-related modulations of specific ERP components that were differentially pronounced for participants showing HT and LT aggressiveness may provide insight into the mechanisms subserving aggression and expand findings from mere behavioral studies.

Behavioral Data

Both behavioral effects and results of the postexperimental questionnaire showed a clear provocation effect in the study, despite the fact that a modified version of the TAP with an increased number of trials was employed. We also made behavioral observations that are not directly reflected by neurophysiological data. In contrast to the male participants, the trait aggressiveness was not predictive of the actual aggressive behavior in women. In fact, the women's behavior appeared more driven by the provocation level in the experiment rather than by their trait aggressiveness. This is in line with previous studies which, in general, have found a weaker relation between trait aggressiveness and actual behavior for women (Giancola, 2003). The tendency of a stronger provocation effect in women is corroborated by earlier studies on laboratory-induced aggression (Bettencourt & Miller, 1996; Taylor & Epstein, 1967). These behavioral differences were not directly reflected by neurophysiological differences. Thus, further studies are needed to specify the neural correlates of sex differences in aggressive behavior.

Decision Phase

During the decision phase, we observed an enhanced frontal negativity (''N2'') in high compared to low provocation trials. This effect was detectable only in HT aggressive participants and was correlated with the

actual aggressive behavior, such that HT aggressive participants who, nevertheless, behaved nonaggressively showed the highest effect. No such effect was seen in the LT aggressive participants. Negativities of a similar temporal onset and distribution have been observed in a number of situations and have been interpreted as reflecting response conflict (van Veen & Carter, 2002) or inhibitory processes (Falkenstein et al., 1999; Pfefferbaum et al., 1985). As participants did not have to respond during the decision phase, the observed negativity can neither be due to the detection of a conflict between two motor responses nor be related to the inhibition of a predominant motor response. Rather, we propose that this effect is reflecting general monitoring or inhibition processes unrelated to the motor output itself (Bruin & Wijers, 2002). The lack of significant correlations between the inhibition subscale of the FAF and the participants' behavior or neurophysiological response might argue against this association with inhibitory processes. However, the FAF inhibition score has failed to differentiate between violent criminals and controls, and rather assesses the knowledge of moral rules and degree of conscientiousness (Hampel & Selg, 1975). It might thus be unrelated to the inhibitory mechanisms necessitated in a social provocation.

Following this early provocation-related effect, we observed an enhanced late fronto-lateral negativity under high compared to low provocation. This effect showed a similar distribution and relation to the participants' aggressiveness as the earlier effect. It was also observable for HT aggressive participants only and correlated with their level of aggressive behavior. Further analyses of the topography confirmed the similarity of both effects. This indicates that both effects are probably reflecting an extended right frontal negativity, although further studies will be needed to probe possible functional and anatomical differences. It might well be that this extended negativity reflects the prolonged activity of those same neural generators that are involved in response competition and inhibitory processes. However, for the present study, we will, for descriptive purposes only, refer to this extended frontal negativity as decision-related negativity (DRN), in parallel to the later discussed FRN.

The fact, that the DRN was largest in HT aggressive participants, who behaved nonaggressively in the actual experiment (see Figures 4 and 5), suggests that a conflict between different motivations might drive this effect: having the predisposition to get back to a provoking opponent, but willing to prevent escalation at the same time. As no self-report data were assessed that could confirm this association with a conflict, future studies will be needed to further substantiate this argument. However, given the similarity of the neurophysiological response with data from ''pure'' cognitive tasks, this goes in line with recent observations that similar (although not necessarily the same) mechanisms are engaged by cognition–emotion competition as by conflicts within the cognitive domain (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003). In an fMRI study on the neural basis of decision making in the ultimatum game, for instance, unfair offers yielded a higher blood oxygenation level-dependent response in ACC (Sanfey et al., 2003). The authors explain this with a detection of a conflict between the emotional response to unfairness and the cognitive motivation to maximize money. Results in an ''emotional'' Stroop task also point to a similar role of ACC in emotional conflict resolution (Etkin et al., 2006), although the activation was in more rostral parts of ACC, compared to the dorsal activations found typically in situations entailing stimulus–response incompatibilities (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). Similar to these findings, the DRN in the present study might reflect a conflict between the emotional response to provocation and the cognitive motivation to refrain from retaliation. Moreover, these results might be useful to extend abovementioned fMRI data, as they can speak also to the similarities or differences in the temporal dynamics of the neural correlates in purely cognitive tasks or in tasks entailing emotion–cognition interferences.

However, the DRN might also reflect inhibitory processes and self-regulation that takes place in these participants (Knoch & Fehr, 2007), as suggested by the negative correlation between the DRN and aggressive retaliation. This would be in line with the aforementioned studies of the N2 in the context of response inhibition (Pliszka et al., 2000; Falkenstein et al., 1999). As pointed out previously, the present experimental setting was quite different to the usual response inhibition tasks. Moreover, although the DRN had a similar onset as the classical N2, a prolonged duration was observable, which distinguishes it from results in go/no-go or stop-signal studies. Recent fMRI studies have extended these results by demonstrating a role for the right PFC also in self-control and inhibition within the scope of complex decision making and social interactions (Knoch & Fehr, 2007; Knoch et al., 2006). More research is clearly needed to establish the interpretation of the DRN in terms of inhibition and its relation to social decision making in the right PFC.

Although the exact functional meaning of the observed DRN needs to be specified in further studies, the involvement of executive processes in the control of aggressive behavior is clearly implicated by this effect. As we observed the DRN in the HT–LE group only, the association of this effect with executive functioning might be counterintuitive, given the usually reported *lower* executive functions in HT aggressive people (Giancola, 2004; Morgan & Lilienfeld, 2000). However, an inverse relationship between trait aggressiveness and executive functions has been reported particularly regarding clinical groups as persons with antisocial personality disorder or conduct disorder (Morgan &

Lilienfeld, 2000). With respect to healthy people, an effect of executive functions on laboratory-induced aggression has been demonstrated, which was differentially pronounced for men and women and under intoxicated and sober conditions (Giancola, 2004; Hoaken, Shaughnessy, & Pihl, 2003). The participants of the present study were students (i.e., mentally healthy, high-functioning individuals), thus a weaker correlation of executive functions and trait aggressiveness can be expected. Moreover, the current results do not exclude that the LT group had, in general, better executive functions. However, the DRN suggests that only the participants prone to aggressive behavior had to recruit executive functions when provoked to refrain from retaliation.

This allows us to make differential predictions of the effects of manipulations that interfere with executive functions (e.g., alcohol intoxication, dual-task settings). As the LT aggressive group stayed at ease during the provocation without higher cognitive effort, interference with their executive functions should have little effect on aggressive behavior. In contrast, one would predict higher aggression also in the HT–LE participants under conditions interfering with their cognitive control processes. In fact, a similar behavioral effect has been observed by Giancola (2002), who showed that alcohol consumption led to a more pronounced increase of aggressive behavior in men with higher as opposed to lower levels of trait anger. There is evidence suggesting that the alcohol–aggression relationship is mainly driven by a disruption of executive functions (Hoaken, Giancola, & Pihl, 1998). We propose that the DRN effect in the current study is the neural correlate of aggression-controlling executive processes. This is in line with an fMRI study employing the TAP (Krämer et al., 2007), which reported prefrontal activations during the decision phase, reflecting the enhanced cognitive processing associated with aggressive retaliation.

Outcome Phase

During the outcome phase, when participants got feedback whether they could administer the punishment to the opponents or were exposed to the aversive noise themselves, again, specific modulations of the neurophysiological responses by situational and personality factors were observed.

Negative feedback, that is, the warning that the participant lost and would be punished, elicited a frontocentrally located enhanced negativity, peaking at about 300 msec. This FRN was independent of the actual level of provocation (i.e., the to-be-expected punishment). This is consistent with previous studies showing that the FRN is unaffected by the punishment's magnitude (Yeung & Sanfey, 2004). However, the difference between ERPs following positive and negative feedback was related to the participants' trait aggressiveness and their aggressive behavior in the experiment. Although highly aggressive participants showed a clear difference between losing and winning trials, this difference was smaller in the nonaggressive participants. In fact, an FRN, although slightly smaller than in the loss trials, seemed to be present in win trials also in the nonaggressive participants. To the extent to which the FRN can be seen as a direct reflection of the perceived valence of a stimulus, this suggests that winning (and thus being able to punish the opponent) is associated with negative feelings for these participants.

This interpretation is supported by recent reports of an FRN elicited by observing another person losing money in a gambling task, depending on the affective engagement of the observer (Fukushima & Hiraki, 2006; Yu & Zhou, 2006). This suggests that the FRN can be driven by empathic involvement in the outcome of actions for another person, thus extending earlier studies showing a modulation of the FRN by the person's motivation and emotional involvement (Yeung et al., 2005). As the FRN is believed to be generated in ACC, this fits well with the hypotheses that this region is critical for the integration of emotional and cognitive information in the control of actions (Paus, 2001). The finding of an enhanced FRN in win trials in nonaggressive participants might thus be taken as an indicator of their empathy with the to-be-punished opponent. This dovetails with the results of the aforementioned fMRI study of reactive aggression (Lotze et al., 2007), demonstrating higher activation in the ventromedial PFC in less callous (thus supposedly more empathic) participants during the observation of the opponent suffering. This observation is especially interesting, as one might argue that such an emotional modulation of the punishment's evaluation is causing the regulation of aggressive behavior in participants. Indeed, in the present study, less aggressive subjects selected lower punishments, in particular, in trials following wins. This further supports the impact of the outcome's emotional evaluation on the aggressive behavior. Further studies relating this effect with, for instance, self-report data on individuals' empathy will be needed to clarify the kind of emotional evaluation that exerts an impact here.

Implications for a Model of Aggression

The neurophysiological findings can be used to further specify the General Aggression Model (Anderson & Bushman, 2002). A trivial prediction is, of course, that people with HT aggressiveness should show more aggressive behavior, especially when provoked. Although this was true for our participants' average behavior, we also observed participants showing nonaggressive behavior irrespective of their high predisposition for aggression. Although the model acknowledges that individual differences in decision and evaluation processes may modulate aggressive behavior, the present data provide evidence for differences in executive control processes

instigated by the frontal cortex that modulate aggressive behavior under provocation in HT aggressive participants. No such provocation effect on cognitive control processes was found in the LT aggressive group, likely because their predisposition to react in a nonaggressive way does not necessitate the recruitment of executive control functions to ameliorate aggressive reactions.

Our data thereby specify the role of executive functions in modulating aggressive behavior that has been postulated in previous studies (Hoaken et al., 2003; Blair, 2001). Based mainly on studies with sociopathic patients, different models have been proposed that tap into the regulatory impact of executive functions on reactive aggression (Blair, 2001). Thus, some models stress the executive function of ''inhibitory control,'' explaining aggressive outbursts as failed inhibitions of violent impulses (Krakowski, 2003). This is corroborated by ERP studies demonstrating reduced inhibitory control in psychopathics, reflected in a diminished no-go-related N2 component or impaired performance (Munro et al., 2007; Kiehl, Smith, Hare, & Liddle, 2000). Other models rather point at a disturbed interplay of executive functions and emotion (Blair, 2001). The social response reversal model, for instance, states, that the orbito-frontal cortex is modulating social behavior on the basis of social cues such as angry facial expressions (Blair, 2004). Accordingly, aggressive behavior in patients with orbito-frontal lesions is ascribed to their deficits in processing angry facial expressions.

Although inhibitory control models cannot account for a range of psychopathological observations, especially concerning impulsivity, the present data stress the relevance of inhibition in the control of aggressive tendencies in healthy participants. This is implicated by the relation of the DRN and the participants' ability to refrain from retaliation. Inconsistent findings on inhibition deficits in sociopathic patients might well be due to differences in pathology or experimental tasks employed. With the present data, we suggest one way to clarify the relation of inhibitory processes and aggression and extend it to healthy, high-functioning people.

The modulation of the FRN by empathy for the opponent, on the other hand, points to a mechanism how emotional evaluation affects the regulation of aggressive behavior. As pointed out previously, it might well be that the outcome's evaluation is driving the enhanced executive control processes during the decision phase, triggered by a change in reward expectancies (Blair, 2004; Rolls, 2000). Further studies will have to clarify the interaction of other personality traits (namely empathy) and executive functions in the regulation of reactive aggression. This extends results from an fMRI study on reactive aggression (Krämer et al., 2007). The authors report enhanced blood oxygenation level-dependent responses in the dorsal striatum and in the dorsal part of ACC during the decision for retaliation. The dorsal ACC activation is related to the processing of reward expectations on the one hand and possible behavioral costs (by risking higher punishments) on the other hand. This emphasizes the role of action monitoring and processing of reward contingencies in the control of aggression. Moreover, our data indicate that not only angry facial expressions, as suggested by Blair (2001), but also empathic feelings might have an impact on social response reversal mechanisms, in healthy humans at least.

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