

Emotional reactivity to threat modulates activity in mentalizing network during aggression

Frederike Beyer,¹ Thomas F. Münte,¹ Christian Erdmann,^{1,2} and Ulrike M. Krämer¹

¹Department of Neurology and ²Department of Neuroradiology, University of Lübeck, RatzeburgerAllee 160, 23538 Lübeck, Germany

Aggression is a common response to provocation, albeit with considerable interindividual differences. In this fMRI study, we investigated emotional reactivity to threat as possible link between provocation and aggression, as well as the neural correlates of this relationship. We hypothesized that emotional reactivity, measured as fear potentiation (FP) of the startle response, would be negatively associated with aggressive behavior and would modulate neural activity during an aggressive interaction. In 30 healthy female participants, FP was measured as the difference between blink amplitudes while watching threatening vs neutral pictures. Participants subsequently engaged in a variant of the Taylor Aggression Paradigm (TAP), while being scanned. During the TAP, participants selected a punishment level for either a highly provoking or a nonprovoking opponent. There was no difference in aggressive behavior between participants high and low in FP. However, we found a negative correlation between FP and the neural provocation effect in several regions of a network previously associated with mentalizing including the medial prefrontal cortex, precuneus and the temporo-parietal junction. Independently of the FP variability, aggressive behavior correlated with the provocation effect on activity in the caudate nucleus. Our results indicate that during a provocative confrontation, high emotional reactivity to threat suppresses recruitment of the mentalizing network.

Keywords: aggression; empathy; fMRI; startle reaction; theory of mind

INTRODUCTION

As a source of both physical and emotional pain as well as financial burdens on society, aggression and its causes have long been of great interest to the social sciences. Early research on the neural basis of aggression mainly focused on pathological groups, such as psychopathic offenders (Lapierre *et al.*, 1995; Blair, 2001) and patients with lesions of the prefrontal cortex (Grafman *et al.*, 1996; Anderson *et al.*, 1999). More recently, investigations of the neural underpinnings of normal-range aggressive behavior in healthy samples have added to our understanding of the cognitive and emotional processes associated with it (Emery *et al.*, 2001; Halasz *et al.*, 2006; Krämer *et al.*, 2007; Lotze *et al.*, 2007). Nevertheless, there is not yet a definite answer on how personality and situational variables interact to cause aggression.

According to their General Aggression Model (GAM), Anderson and Bushman propose the subjective experience of affect as a direct precedent of behavioral choice in situations that potentially elicit aggressive reactions, such as interpersonal provocation (Anderson and Bushman, 2002). Thus, according to this model, a person's behavioral response to provocation will depend also on his/her emotional reaction. In the present study, we explore this aspect of the GAM by investigating how individual differences in emotional reactivity modulate behavioral and neural responses to a provocative situation.

Most researchers differentiate between reactive and instrumental aggression (Blair, 2004) or similar concepts (Barratt *et al.*, 1997; Bushman and Anderson, 2001). Reactive aggression is characterized as an aggressive response to provocation and in contrast to instrumental aggression is commonly driven by feelings of anger (Bushman and Anderson, 2001; Blair, 2004). Thus, it seems plausible that the likelihood of reacting aggressively to a provocation does not only depend on a person's ability to inhibit aggressive acts, as proposed by theories emphasizing the role of executive functions, but foremost on the

tendency of that person to react to provocation with feelings of anger. If, however, a person tends to interpret potentially harmful situations as threat, resulting in feelings of fear, this person should be less likely to aggress, and more likely to withdraw from that situation. Thus, the affective reaction to a provocation should determine whether approach-related aggressive or avoidance-related, nonaggressive escape tendencies are activated. Consistent with this idea, appetitive motivation has been associated with the tendency to experience anger (Carver, 2004), aggressive behavior (Cooper *et al.*, 2008) and alterations in the neural response to angry facial expressions (Passamonti *et al.*, 2008).

Using modulation of the startle eyeblink as a measure of affective state, Amodio and Harmon-Jones (2011) found further support for a connection between anger and approach-related rather than avoidant behavioral tendencies. As a defensive, avoidance-related reflex (Walker and Davis, 1997), the startle response is usually inhibited by positive stimuli and facilitated by aversive, such as threatening or injury-related stimuli (Bernat *et al.*, 2006; Caseras *et al.*, 2006). In participants high in trait anger, Amodio and Harmon-Jones (2011) found a stronger inhibition of the startle response by positive stimuli compared with participants low in trait anger, suggesting that participants high in trait anger also possess strong motivational approach tendencies. Contrarily, the facilitation of the startle response by threatening stimuli, generally referred to as fear potentiation (FP), is increased in participants scoring high on harm avoidance or fear (Corr *et al.*, 1995; Justus and Finn, 2007). FP is dependent on activation of the amygdala, which innervates the neural pathway of the startle response via hypothalamus and brainstem (Davis, 1986, 2006). In psychopathic individuals, modulation of the startle response by affective stimuli is reduced (Herpertz *et al.*, 2001), indicating a deficit in amygdala function in these patients, which is consistent with the observation of reduced amygdala volume in psychopathic individuals (Yang *et al.*, 2009). Similarly, healthy participants with psychopathic traits, especially high fearlessness-scores, show reduced startle potentiation by aversive stimuli (Benning *et al.*, 2005). These results consistently show that the strength of FP depends on an individual's proneness to activate avoidance tendencies in response to potentially threatening stimuli.

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Correspondence should be addressed to Ulrike M. Krämer, Department of Neurology, University of Lübeck, RatzeburgerAllee 160, 23538 Lübeck, Germany. E-mail: umkraemer@gmail.com

Accordingly, Vaidyanathan and colleagues (2009) showed that in healthy student populations, FP is a reliable measure of defensive reactivity and can thus be used as a biological marker of the emotional reactivity to threat. Implementing a startle paradigm in the MRI-scanner, Eippert and colleagues (2007) showed that deliberately increasing negative emotions while watching aversive pictures leads to an increase in startle response as well as in amygdala activity, further supporting the role of the amygdala in FP. Both up- and downregulation of emotion in this study was associated with increased activation of the anterior cingulate cortex (ACC), orbitofrontal cortex (OFC) and dorsolateral prefrontal cortex (DLPFC), underlining the importance of these structures in emotion regulation (Eippert *et al.*, 2007).

In the present study, we used the Taylor Aggression Paradigm (TAP; Taylor, 1967) to study aggression. The TAP is a method for eliciting and measuring reactive aggressive behavior in the laboratory. It is set up as a competitive reaction time (RT) task, during which the winning participant is given the opportunity to punish his opponent with some sort of aversive stimulus, which can be adjusted in intensity. The TAP has been shown to be a valid measure of aggression in the laboratory (Bernstein *et al.*, 1987; Giancola, 1998).

For the use of the TAP in a study with functional magnetic resonance imaging (fMRI), Krämer and colleagues (2007) adapted the TAP to include two opponents, one of whom acted highly provocative, selecting mostly high punishments, the other nonprovocative, selecting mostly low punishments. In order to differentiate the neural processes underlying the different elements of the TAP, Krämer and colleagues focused on two phases in each trial of the game: the decision phase, during which the participant had to select a punishment level in case he would win and the outcome phase, during which the participant was informed whether he won or lost. Contrasting the decision phases when playing against the highly vs the nonprovocative opponent yielded activation of the rostral and dorsal ACC and the anterior insula. Contrasting high and low punishment selections for the highly provocative opponent revealed activation of the dorsal striatum (caudate nucleus), suggesting that the decision for a high punishment of the provocative opponent is perceived as rewarding, regardless of the actual outcome of the game (Krämer *et al.*, 2007). This is in line with other imaging findings of increased activity in the caudate nucleus when deciding to punish unfairness (de Quervain *et al.*, 2004). In another study combining fMRI and the TAP, Krämer and colleagues (2011) showed that the provocation effect during early outcome phases in a prefrontal-subcortical network including the dorsal striatum predicted aggressive behavior later in the TAP. Thus, aggressive responses to provocation have repeatedly been linked to increased reactivity of the caudate. For the outcome phase, Krämer and colleagues (2007) found activation of the ventral striatum when contrasting win trials against lose trials. In a similar study involving fMRI and the TAP, Lotze and colleagues (2007) found activation of the medial prefrontal cortex (mPFC) during the decision phase, which was positively related to the punishment level selected. The authors argue that activity in the mPFC likely reflects regulation of emotional conflict, which results from opposing emotions of compassion and aversion towards the opponent (Lotze *et al.*, 2007).

In the current study, we investigated whether differences in the behavioral and neural response to provocation in the TAP are related to emotional reactivity to threat, measured as FP of the startle response. Since some previous studies found differences in startle response between men and women (Kofler *et al.*, 2001; McManis *et al.*, 2001), only female participants were included.

We hypothesized that FP would be negatively related to aggressive behavior in response to provocation. On a neural level, we expected that FP would be associated with increased activity in the amygdala during high provocation trials. We also expected modulation of neural

activity in aggression-related regions as OFC and ACC during high provocation in high FP participants, mediating the negative relationship between FP and aggressive behavior. Based on previous results in the TAP and independently of FP, we expected a positive relationship between activity in the caudate nucleus during high compared with low provocation in the decision phase and aggressive behavior.

METHODS

Forty healthy female participants (mean age = 23.2, s.d. = 2.7) were recruited from the local university. All but three participants were right-handed (self-report). All participants were free of any psychiatric or neurological disorder (self-report), gave informed written consent and received 7 Euro/h as compensation for participation. The study was approved by the local ethics committee and performed according to the Declaration of Helsinki.

Measurement of FP

The methods used for the measurement of FP of the startle response were adapted from previous studies (Caseras *et al.*, 2006; Conzelmann *et al.*, 2009).

Participants were presented with 48 pictures from the International Affective Pictures System (IAPS).¹ Half of these pictures were threatening stimuli (e.g. a gun pointed at the viewer, an attacking dog) the other half were neutral in content (e.g. a secretary on the phone; household objects). Pictures were presented in one of two different fixed orders which were set up randomly with the constraint that no more than two pictures of the same valence were presented consecutively. Each picture was presented for 6 s with a 12-s intertrial interval (ITI), during which a white central cross was presented on a black background. During 18 pictures of each valence, a short burst of white noise (50 ms, 95 dB), was presented over speakers 1.5, 2.8 or 4.0 s after picture onset. For the remaining 12 pictures, the startle probe was presented during the ITI and these trials were not analyzed.

Prior to the picture presentation, four startle probes were presented while participants watched the fixation cross to account for initial habituation of the startle response.

EMG measurement

EMG recordings of the musculus orbicularis oculi and analysis of EMG data were conducted according to the Guidelines for human startle eyeblink electromyographic studies (Blumenthal *et al.*, 2005). Two electrodes were placed below the left lower eyelid, one in line with the pupil and the other 1–2 cm to the left of the first. A ground electrode was positioned centrally on the forehead. Prior to electrode placement, the skin was treated with a peeling paste and alcohol. The EMG signal was amplified using an EEG amplifier (Quickamp; Brain Products).

We analyzed EMG recordings using EEGLAB, a Matlab-based open-source toolbox for analysis of electrophysiological recordings (Delorme and Makeig, 2004). EMG signals were high-pass filtered at 10 Hz, low-pass filtered at 500 Hz and baseline-corrected using the 50 ms prior to onset of the startle probe as baseline. We then visually inspected each startle trial for artifacts. Trials with excessive noise or eyeblinks in the 50 ms baseline period were excluded. Since several participants showed strong initial startle responses with habituation during the first picture trials, we excluded the first three picture trials from analysis in all participants (Bernat *et al.*, 2006). Blink magnitude was measured as the maximum absolute amplitude in an interval of 20–160 ms

¹ Picture numbers: *aversive* 1050, 1201, 1300, 6370, 6250, 6020, 6213, 6230, 6300, 6350, 6821, 6940, 6510, 9120, 1930, 9490, 6242, 6244, 6260, 2130, 9800, 6243, 6241, 2681; *neutral* 1121, 1670, 2383, 5395, 2514, 2575, 5900, 7503, 7550, 7590, 7620, 7950, 7235, 7236, 7705, 7500, 5731, 2221, 2272, 2393, 5390, 5533, 5661, 7010.

following the startle probe. Blink scores were *z*-transformed separately for each participant over all trials to ensure comparability of scores between participants (Caseras et al., 2006). To obtain individual FP-scores, we subtracted the mean standardized blink amplitude for neutral pictures from the respective value for threatening trials.

Aggression paradigm

After completing the FP measurement, participants were introduced to their two female opponents for the TAP, who were confederates of the experimenter. The confederates did not actually participate in the TAP, but opponent's selections and outcome of the trials were controlled by a computer, according to the parameters described below. They received written instructions explaining the design of the TAP. Punishment in the TAP was implemented as an aversive noise which could be adjusted in terms of loudness. The minimum and maximum intensity of the noise was individually adjusted such that participants could still hear the lowest noise over the scanner noise and participants rated the loudest noise as unpleasant, but not painful. Participants viewed the computer screen through scanner-compatible goggles and heard the punishment noise over headphones.

Figure 1 shows the time course for a single trial of the TAP. In the beginning of each trial, a fixation cross was presented for a random interval of 8–12 s. Then, the participant was informed against which opponent she would be playing (duration 6 s). During this time, the participant had to select one of eight punishment levels by button press. This was followed by the RT task with a mean duration of 2.4 s, an interval of 1.6–6 s and a 6 s outcome phase, during which the participant was informed whether she won or lost and which punishment level her opponent had selected. In lost trials, the punishment noise was presented in the respective loudness at the end of the outcome phase. Outcome of the RT task as well as the opponents' selections were controlled by the computer. One opponent selected mostly low punishment levels with the highest selection being 4 (mean = 1.93), whereas the other opponent selected mainly high punishment levels with the lowest selection being 4 (mean = 6.57). The experiment was divided into three runs with 28 trials per run. In each run, participants played an equal number of trials against each opponent in randomized order and won 50% of the trials. Overall, the TAP took ~40 min.

MRI recording

We obtained structural (T1-weighted; FOV = 240 mm; matrix = 240 × 240; 175 sagittal slices of 1-mm thickness) and functional (Gradient-Echo-EPI-Sequence; TR = 2000 ms; TE = 30 ms; FOV = 192 mm; flip angle = 80; matrix = 64 × 63; slice thickness = 3.3 mm; 34 transversal slices; voxel size = 3.0 × 3.0 × 3.3 mm; three runs of 395 volumes each) images using a 3-Tesla Philips Achieva scanner.

Postexperimental assessment and personality measures

After the scanner session, participants filled out questionnaires to check the success of the provocation manipulation and the deception concerning the RT task. Participants then completed a series of personality questionnaires: the trait scale of a German adaptation of the state-trait anxiety inventory (Laux et al., 1981); a German version of the Behavioral Inhibition System and Behavioral Activation System scales (Strobel et al., 2001); a German version of the Buss and Perry Aggression Questionnaire (Herzberg, 2003); a German version of the Barratt Impulsiveness Scales–short version (Meule et al., 2011). Finally, participants were thoroughly debriefed, paid and thanked for their participation.

Data analysis

Of the 40 participants, three were excluded due to excessive noise on the EMG recordings, one due to excessive movement during the MRI-scan (>4 mm), five, because they were suspicious about the putative confederates in the TAP and one because she did not select any punishment level on the majority of trials. Thus, the data of 30 participants were included in the analysis.

Behavioral analysis and personality measures

To assess the success of the provocation manipulation, we compared mean punishment level selections for the provocative opponent to those for the nonprovocative opponent using a paired-samples *t*-test. To obtain individual scores of the behavioral provocation effect, we subtracted the mean punishment level selection for the nonprovocative opponent from that for the provocative opponent in each participant. Since the aim of this study was to elucidate the psychophysiological and neural basis of between-subjects variability in aggressive behavior, we focused on the mean provocation effect for each participant, instead of investigating within-subject variability in punishment selection across trials. We correlated individual provocation scores with FP and the personality measures as well as FP with the personality measures.

To analyze differences in the time needed for the selection of the punishment level depending on the provocation level, we compared RTs for the two opponents. Since overall RTs varied greatly between participants, we used *z*-standardized RTs in order to ensure comparability of the RT-differences for the two opponents between participants. To see whether RTs were modulated differently by provocation for participants high and low in behavioral provocation effect, we split participants into three groups of ten participants each, according to their behavioral provocation effect. We entered RTs for the two extreme groups into a repeated measures analysis of variance (ANOVA) with the within-group factor 'opponent' (high vs low provocation) and the between-group factor 'behavioral provocation effect' (high vs low). For analyses of behavioral and EMG data, we used a significance threshold of $P < 0.05$.

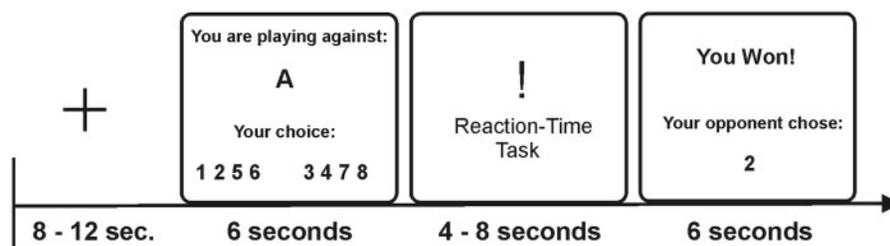


Fig. 1 Paradigm. Figure shows the time course for one trial of the TAP.

Analysis of fMRI data

We analyzed fMRI data using the Matlab-based toolbox SPM8 of the Wellcome Trust Centre for Neuroimaging, London. Preprocessing included temporal adjustment for differences in slice time acquisition, motion correction, coregistration of EPI images with T1-weighted anatomical images of each subject and segmentation, normalization into MNI-space and spatial smoothing with an 8-mm kernel. In order to improve the accuracy of the coregistration, T1-weighted images were first coregistered to a gradient echo EPI which was similar in contrast to the functional images, but less affected by artifacts. Both the gradient echo EPI and the T1-weighted image were then used as source images for the coregistration with the functional images.

We estimated a first-level general linear model (GLM) per subject, modeling decision and outcome phases for the two opponents and motion regressors included in the design matrix. On the second level, we estimated two flexible factorial designs, one for the decision phase with two factors (subject and provocation) and one for the outcome phase with three factors (subject, provocation and outcome). Subject factors were not included in the design matrices. To investigate the relationship between emotional reactivity to threat and neural activations associated with provocation, we defined the contrast high > low provocation for the decision phase on the first level and estimated a second-level regression model with individual FP scores as regressor. For the outcome phase, we estimated regression models using the contrasts high > low provocation for win and lose trials separately.

As mentioned above, Krämer and colleagues (2007) found increased activity in the dorsal striatum during high punishment selections under high provocation. Although this effect was not directly related to our research question, since the number of fMRI-studies on the TAP is still very limited, we were interested in the reliability and generalizability of this effect. To assess whether the relationship between caudate activity and aggressive behavior generalizes to the interindividual level, we correlated the behavioral provocation effect with the high > low provocation contrast value for the caudate. The regions of interest (ROIs) of left and right caudate were defined using Automated Anatomical Labeling (AAL; Tzourio-Mazoyer *et al.*, 2002).

For definition of functional ROIs and extraction of ROI activation values, we used the SPM-toolbox marsbar (Brett *et al.*, 2002). For statistical testing of fMRI data, we used a threshold of $q < 0.05$ with false discovery rate (FDR) correction for analyses of main effects and the regression analyses, unless otherwise stated. For interaction analyses, we used a threshold of $P < 0.001$, uncorrected.

RESULTS

EMG data

Z-standardized blink amplitudes were significantly higher for aversive than neutral pictures (means = 0.08 and -0.08 , $t_{29} = 2.58$, $P = 0.015$; Figure 2A) showing a potentiation of the startle reflex by threatening stimuli.

Behavioral data and personality questionnaires

Provocation in the TAP was successful as participants selected higher punishment levels under high than under low provocation (mean selections 4.12 and 3.18, $t_{29} = 3.77$, $P = 0.001$; Figure 2B). Analysis of RTs showed no significant main effect for opponent, but a significant interaction of opponent \times behavioral provocation effect ($F_{1,18} = 5.52$, $P = 0.03$). Participants in the highly aggressive group made faster selections for the provocative than the nonprovocative opponent ($z = -0.11$ and 0.11 ; $t_9 = 2.28$, $P = 0.048$), whereas there was no significant difference between opponents for the low aggressive group (Figure 2C). There was no significant correlation between the

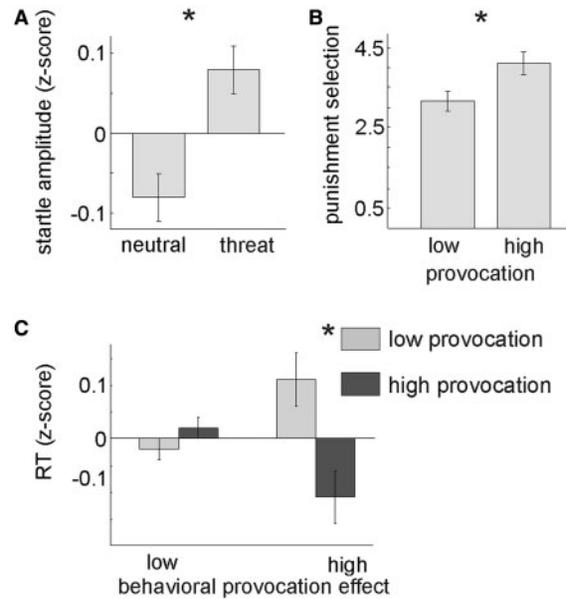


Fig. 2 Behavioral results. Mean standardized startle amplitudes are given for neutral and threatening trials (A). (B) depicts mean noise level selected for the nonprovocative and highly provocative opponent. (C) Mean standardized RTs for the punishment selections for the nonprovocative and highly provocative opponent are given separately for participants low and high in behavioral provocation effect.

behavioral provocation effect and FP ($r < 0.001$, $P > 0.998$) or any of the personality measures. There was also no significant correlation between FP and any of the personality measures.

Functional neuroimaging data

The focus of this study was on the regression of FP with neural activations during the TAP. Prior to these results, we report the main effects found for the decision phase. Main effects for the outcome phase are reported at the end of the results section.

Decision phase

The contrast high > low provocation for the decision phase showed no activation at the chosen significance threshold. Using a more liberate threshold of $P < 0.001$ uncorrected and a clustering threshold of 20 voxels, we found activity in the right parahippocampal gyrus, as well as activity of the right globus pallidus and caudate nucleus. The correlation analysis of the high > low provocation contrast value for the AAL-defined caudate ROI and the behavioral provocation effect showed a positive correlation ($r = 0.48$, $P = 0.008$; Figure 3 and Table 1).

Emotional reactivity

For the decision phase, regressing individual scores of FP against the contrast high > low provocation showed a negative relationship between FP and activation of the bilateral mPFC, cuneus, precuneus, temporo-parietal junction (TPJ), the left middle and inferior frontal gyrus, bilateral clusters in superior and middle temporal gyrus, extending to the temporal poles, and the right insula, thalamus and caudate head (Figure 4). To demonstrate the linearity of this effect, in Figure 4 we also plot individual contrast values for two of these regions (precuneus and left TPJ) against FP scores. For this purpose, we averaged the contrast values across all voxels in the two clusters. Note, that these plots serve visualization purposes only and we did not estimate correlation coefficients for these data. As these results were derived from a regression analysis on contrast values (i.e. difference values), they do

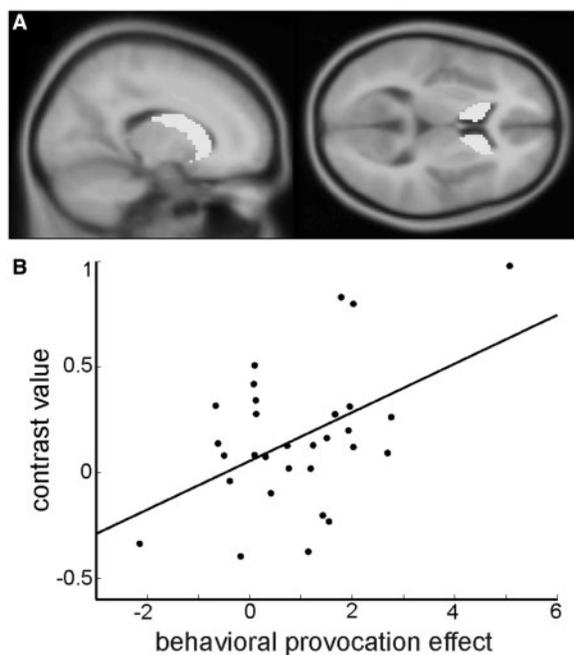


Fig. 3 Decision phase. Displayed is the region of interest defined for the correlation of provocation effect in the nucleus caudatus and the behavioral provocation effect (A); (B) shows the correlation map for the behavioral provocation effect and the high > low provocation contrast value for the bilateral caudate ROI.

Table 1 Neural activations for the decision phase

Region	Hemisphere	X, Y, Z	T
<i>High > low provocation</i>			
Globus pallidus	R	12, 0, 2	3.75
Caudate	R	12, 22, -4	3.45
<i>Regression FP x high > low provocation, negative</i>			
Inferior frontal gyrus	L	-46, 40, 2	4.69
Middle frontal gyrus	L	-50, 18, 36	5.80
Temporo-parietal junction	L	-46, -60, 30	5.37
	L	-38, -68, 48	5.00
	R	54, -56, 36	3.75
Cuneus/precuneus	L	-4, -70, 22	5.24
	R	16, -86, 12	4.33
	R	12, -80, 36	4.03
	L	0, -48, 36	4.89
	L	-12, -52, 30	4.29
	R	12, -52, 36	3.62
Superior temporal gyrus	L	-36, 16, -22	4.38
	R	54, -4, -8	3.95
Middle temporal gyrus	L	-62, -36, -4	4.25
Inferior frontal gyrus	L	-32, 26, -20	4.05
Medial frontal gyrus	R	2, 54, 20	5.22
	L	-4, 58, 32	4.81
	L	0, 40, 46	4.74
Lingual gyrus	R	24, -78, -16	4.79
Superior frontal gyrus	R	6, 22, 60	4.67
	L	-4, 20, 62	4.30
Insula	R	40, 16, -16	4.38
	R	30, 22, -20	4.25
Thalamus	R	12, -26, 16	4.12
Caudate head	R	6, 18, 4	4.05

R = right; L = left; coordinates and *t*-values are given for the peak of the activation according to Montreal Neurological Institute (MNI) space.

not reveal how the activity level in the decision phase differed across participants. To further examine this, we defined functional ROIs for the above mentioned regions and calculated for each participant the mean activity in each ROI for the high provocation vs baseline and low

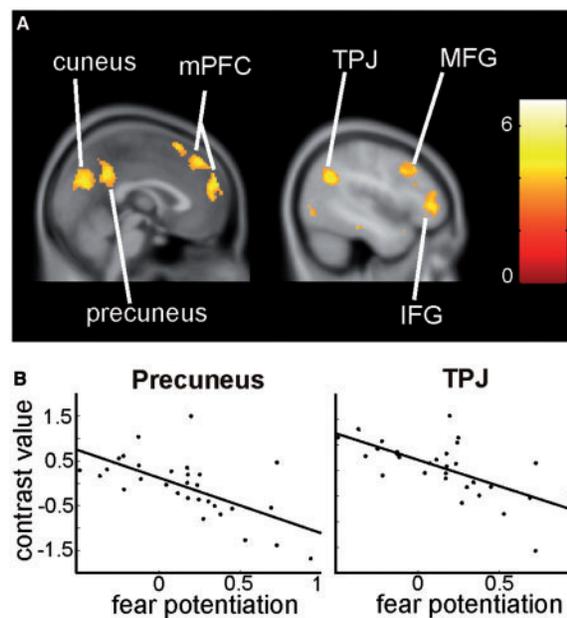


Fig. 4 Regression analysis. Figure shows contrast images for the negatively weighted regression $FP \times high > low$ provocation for the decision phase (A; displayed at $P < 0.001$ uncorr., corresponding to $q < 0.02$ FDR corrected, clustering threshold 20 voxels). MFG = middle frontal gyrus; IFG = inferior frontal gyrus. (B) For visualization purposes, scatter diagrams are shown for the relationship between FP and high > low provocation contrast values averaged across the precuneus and TPJ clusters.

provocation vs baseline contrasts. We then plotted these values averaged for the ten participants with the lowest and highest FP-scores. The different regions showed the same general pattern of a relative decrease in activation during high provocation trials in the high-FP group and a relative increase in activation during high provocation trials in the low-FP group (Figure 5).

As further exploratory analysis, we correlated the individual high > low provocation contrast values for these ROIs with the behavioral provocation effect. There was no significant correlation between behavioral provocation effect and the high > low provocation activation difference for any of these ROIs.

Outcome phase

Contrasting win against lose trials yielded activation of the bilateral caudate head and body, middle frontal gyrus, TPJ and precuneus (Figure 6A; Table 2). The opposite contrast showed activation of Heschl's gyrus as well as the bilateral insula, supplementary motor area (SMA) and right middle frontal gyrus (Figure 6B; Table 2). The interaction of opponent and outcome showed activation in the bilateral anterior cingulate and middle cingulate gyrus, bilateral SMA, left middle frontal gyrus, left precuneus, bilateral superior frontal gyrus and right TPJ. The cingulate gyrus and the left precuneus were activated more strongly for win trials against the nonprovocative compared with the provocative opponent. Comparing high vs low provocation for lose trials showed increased activation of the SMA and anterior cingulate and reduced activation of the right TPJ and right superior frontal gyrus. Importantly for our research question, we found no significant correlation between neural activations during the outcome phase and FP.

DISCUSSION

In the current study, we investigated the relationship between emotional reactivity to threat, measured as FP of the startle response, and behavioral and neural responses to provocation. On the behavioral

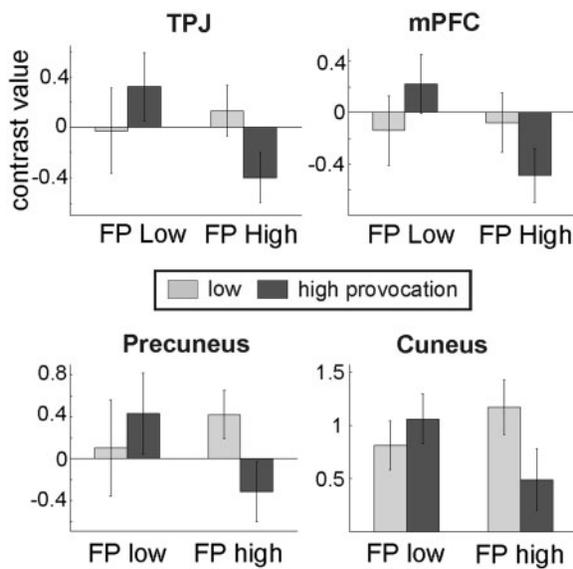


Fig. 5 ROI values. Shown are the contrast values for ROI derived from the regression FP × high > low provocation for the decision phase, displayed for high and low provocation in participants high and low in FP.

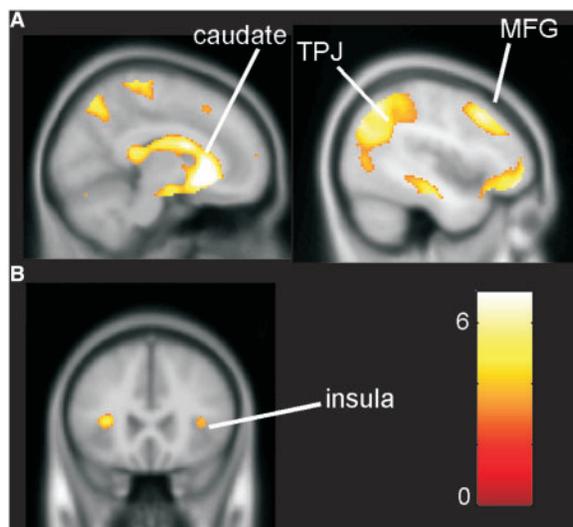


Fig. 6 Outcome phase. Figure shows the contrast images for win > lose (A; displayed at $P < 0.001$ uncorr., corresponding to $q < 0.005$ FDR corrected, clustering threshold 20 voxels) and lose > win (B; displayed at $P < 0.001$ uncorr., corresponding to $q < 0.05$ FDR corrected, clustering threshold 20 voxels).

level, we found no direct relationship between FP and aggressive retaliation. In terms of neuronal response, we found that during the punishment selection for the provocative compared with the nonprovocative component, FP was negatively associated with activity in precuneus, cuneus, TPJ, mPFC and temporal poles. These regions are associated with mentalizing tasks such as inferring another’s intentions or beliefs (Völlm *et al.*, 2006; Krämer *et al.*, 2010; Dodell-Feder *et al.*, 2011; Sebastian *et al.*, 2012), indicating that participants high in emotional reactivity to threat showed reduced mentalizing during the confrontation with the provocative opponent.

Emotional reactivity modulates neural response to provocation

Our results suggest that although emotional reactivity to threat shows no direct influence on aggressive behavior in the TAP, on a neural level

Table 2 Neural activations for the outcome phase

Region	Hemisphere	X, Y, Z	T
<i>Win > Lose</i>			
Caudate Head	R	12, 20, -8	10.61
	L	-12, 17, -6	9.48
Postcentral gyrus	R	10, -34, 72	5.00
Medial frontal gyrus	L	-8, -30, 68	4.58
Caudate body	R	14, 8, 20	7.96
	L	-8, -12, 22	6.51
Inferior parietal lobule	R	50, -56, 42	6.75
	L	-48, -60, 44	6.91
Precuneus	L	0, -66, 48	5.74
Middle frontal gyrus	R	4, -66, 50	5.73
	L	40, 20, 44	6.43
	L	-40, 18, 40	6.68
	<i>Lose > Win</i>		
Heschl’s gyrus	R	48, -24, 10	8.31
Superior temporal gyrus	L	-36, -34, 14	6.43
	R	-40, -24, 6	6.14
Middle frontal gyrus	L	54, 0, 46	5.54
Insula/inferior frontal gyrus	L	-32, 26, 10	5.15
	R	38, 22, 10	3.93
Medial frontal gyrus	R	10, -1, 64	4.73
<i>Interaction Outcome × Opponent</i>			
Anterior cingulate gyrus	R	6, 16, 26	4.32
	R	10, 12, 36	4.08
	L	-6, 14, 36	3.77
Medial frontal gyrus	R	6, 0, 58	3.81
	L	-12, -26, 38	3.43
Cingulate gyrus	R	12, -24, 42	4.21
	L	-12, -26, 38	3.43
Inferior parietal lobule	R	54, -56, 38	4.45
	R	58, -32, 34	3.58
	L	-64, -32, 32	3.74
	L	-38, 12, 50	3.73
Middle frontal gyrus	L	-8, -48, 54	4.09
Precuneus	R	36, 16, 56	4.52
	L	-12, -2, 68	4.2
Superior frontal gyrus	R	16, 32, 54	4.17
	L	-6, 60, 28	3.84
	R	12, 48, 40	3.44
	R	54, -28, -8	3.58
Middle temporal gyrus	R	30, -82, -4	3.51
Middle occipital gyrus	R		

R = right; L = left; Coordinates and t-values are given for the peak of the activation according to Montreal Neurological Institute (MNI) space.

it modulates the reaction to a provocative situation in a highly specific manner. The majority of regions for which we found a negative relation to FP for the high > low provocation contrast in the decision phase (TPJ, precuneus, mPFC, temporal poles) form the so-called mentalizing network, which is reliably found to be activated in studies involving theory of mind and empathy (Völlm, *et al.*, 2006; Krämer, *et al.*, 2010; Dodell-Feder, *et al.*, 2011; Sebastian, *et al.*, 2012).

Processes previously associated with activity in the mentalizing network include inferring the intentions of another person and predicting their behavior, as well as experiencing empathy for another’s emotional distress (Völlm, *et al.*, 2006; Ciaramidaro *et al.*, 2007; Sebastian *et al.*, 2012). Activation of the cuneus and inferior frontal gyrus, which we also found to be modulated by FP, has been associated with social competition (Polosan *et al.*, 2011). Our results indicate that participants that showed little fear in response to threatening stimuli activated mentalizing processes when confronted with a provocative opponent. In participants who showed a highly fearful reaction to threatening stimuli, these processes were suppressed during the confrontation with the provocative opponent. Avoiding mentalizing processes, thus not thinking about the actions, intentions and emotions of the threatening opponent, might constitute a protective mechanism. Similarly, it has been shown that individuals high in anxiety initially

direct their attention towards threat cues, which is followed by redirection of attention away from these cues, referred to as attentional avoidance (Cisler and Koster, 2010). Thus, the neural deactivation-patterns we observed may reflect cognitive disengagement from the aggressive interaction, prompted by high fear arousal. One has to keep in mind, however, that this interpretation is solely based on the neural activation patterns we observed, as we collected no behavioral measure of mentalizing processes during the TAP. Such reverse inference is especially problematic for brain areas that are activated by many different tasks and processes. While the regions we found to be modulated by FP individually serve a number of different functions, their joint activation appears to be specifically associated with social information processing, however. Nevertheless, at this point our interpretation of the results has to be considered preliminary. Mentalizing is difficult to measure directly, as it constitutes, by definition, an internal process. However, in future studies, our interpretation might be supported by the collection self-report data concerning participants' strategies during the decision-making in the TAP.

Contrary to our hypothesis, we found no effect of FP on amygdala activity during the decision phase. One might speculate that as participants high in fear reactivity mentally disengaged from the threatening interaction to reduce or prevent fear arousal, this might have possibly reduced activity in the amygdala, too. Alternatively, we might not have had enough power to detect amygdala effects with the current paradigm. The amygdala response is known to habituate rapidly (Breiter et al., 1996; Yang, et al., 2009) and as the amygdala is close to air-filled sinuses, it is especially susceptible to artifacts caused by local inhomogeneity.

Furthermore, FP did not, as expected, modulate activity of the OFC and ACC in high compared with low provocation trials. Based on findings from studies on patients with brain lesions (Grafman et al., 1996), we expected activity in the OFC to be negatively related to aggressive behavior, mediating the proposed negative relationship between FP and aggressive behavior. We did not observe orbitofrontal activity in our previous studies with the TAP (Krämer et al., 2007, 2011), either. While studies on psychiatric and brain lesion patients have emphasized the importance of the OFC in social learning and the regulation of aggressive behavior (Grafman et al., 1996; Anderson, et al., 1999; Antonucci et al., 2006), these results argue against a strong role of the OFC in controlling aggressive behavior in the context of the TAP, also in relation to interindividual differences in emotional reactivity. In fact, it has recently been argued that the OFC is a crucial structure for reversal learning (Kringelbach and Rolls, 2003), pointing rather to its importance for the development of socially appropriate behavior rather than the inhibition of anger or aggression on a trial-wise basis (Blair, 2012). As for the ACC, Krämer and colleagues (2007) previously found a positive connection between activity in this region and aggression in the TAP. The ACC has been associated with conflict monitoring as well as decision making (see Botvinick, 2007, for a review), but also with more unspecific regulation of autonomic functions (Critchley et al., 2005). Depending on whether aggression is seen as intuitive or counterintuitive response to provocation, one could argue for both a positive or negative relationship between conflict monitoring and aggressive behavior. From the present data, we can only conclude that emotional reactivity to threat does not seem to be directly related to the degree of cognitive conflict evoked by provocation.

Emotional reactivity and aggressive behavior

Although FP was associated with reduced activity in the mentalizing network in the provocative situation, we found no correlation between FP or activity in the mentalizing network and aggression. It is plausible that participants low in emotional reactivity to threat thought about

possible reasons for the opponent's aggressive behavior and tried to understand their opponent's cognitive and affective state. While these processes would all activate the mentalizing network, the conclusion drawn from them—whether it is reasonable to select high punishments—depends also on the participant's belief system and on her expectations of how high or low selections will influence the opponent's behavior. The TAP also offers no information about the motivation behind an opponent's behavior, leaving room for a variety of possible interpretations. Thus, a nonfearful response to a provocation would not necessarily have to be an aggressive one.

On the other hand, a possible explanation of why participants high in FP did not consistently show nonaggressive behavior lies in the nature of the TAP. In naturalistic settings, the two common options of reacting to an aggressive opponent are fight and flight. Reduced activity in the mentalizing network when being provoked might indicate that participants high in FP avoided thinking about the provocative opponent's intentions and affective state. On a behavioral level, however, physically escaping the situation was not an option. It is possible that some participants high in FP deemed an aggressive response most likely to change their opponent's behavior and avoid future provocation. Alternatively, participants high in FP might have adopted a heuristic tit-for-tat strategy in lack of a flight alternative. Future studies investigating connections between emotional reactivity to threat and behavioral responses to provocative situations could test this by implementing a real flight alternative to the aggressive option.

Main effects in decision and outcome phase

Behavioral analysis of the decision phase showed that participants with a strong behavioral provocation effect took longer to select a punishment level for the nonprovocative opponent than for the provocative one. This suggests that in these participants, selecting high punishments for the provocative opponent was a rather automatic response, whereas selecting low punishments for the nonprovocative opponent may have been a more controlled and thus slower process. No such difference was found for participants low in behavioral provocation effect, suggesting that for them, selecting punishments for the provocative and nonprovocative opponents required similar levels of consideration.

On a neural level, we found a positive correlation between caudate activity in the high > low provocation contrast during the decision phase and the behavioral provocation effect. Caudate activity is associated with feedback-driven learning, especially reward learning (Tricomi et al., 2006) and positive feedback, as well as the processing of reward in general (Delgado et al., 2004). De Quervain and colleagues (2004) observed increased activity in the caudate when participants could punish a defector in a socioeconomic game, even if this punishment came at financial cost for the participant. Activity of the caudate in their study was correlated with the amount of money participants were willing to spend on punishing the defector. Similar results on a neural level were obtained by Krämer et al. (2007), who found a positive relationship between caudate activity in the decision phase of the TAP and punishment level selection on a trial-wise basis. Furthermore, comparing the neural provocation effect for early outcome phases in the TAP across participants, Krämer and colleagues (2011) found a positive relationship between activity in several brain regions, including the dorsal striatum, and aggressive behavior in later trials. This suggests that aggression is also related to activity in the caudate during those phases, where participants learn about their opponent's behavior. Our finding that the contrast value for the caudate during the decision phase was positively correlated with aggressive behavior across participants, lends further support to a connection between caudate activation and aggression. Thus, activation of reward learning circuits during

the selection of a high punishment may have caused preferential activation of this behavioral pattern in participants with a high provocation effect. When these participants played against the nonprovocative opponent, this predominant response (selecting high punishment levels) had to be suppressed in favor of a nonaggressive response, leading to longer RTs in these trials.

For the outcome phase, we found activity in the caudate nucleus, TPJ, middle frontal gyrus and precuneus when contrasting win vs lose trials. The opposite contrast showed activity in the insula, among other regions. These results replicate previous findings of increased striatum activity in win trials (Krämer *et al.*, 2007) and underline the importance of this structure in reward processing and the role of the insula in the processing of aversive events (Nitschke *et al.*, 2006). Note that Krämer and colleagues (2007) found increased activity in the insula when comparing high against low provocation for win trials, which we could not replicate with the current study. Of interest for our research question, we found no connection between FP and the neural provocation effect for win or lose trials. This suggests that emotional reactivity to threat selectively influences the neural response to provocation when a behavioral decision is required, as opposed to the outcome phase, where the participant remains passive.

CONCLUSIONS

The present study shows that the neural structures and—by extension—the mental processes recruited during an aggressive interaction depend on the preposition of an individual to react fearfully to threatening stimuli. High fear reactivity suppressed activity in a brain network supporting mentalizing processes during the confrontation with a provocative opponent, probably reflecting a cognitive disengagement from the threatening situation. However, we did not find an effect of mentalizing activity or FP on aggressive behavior. According to the GAM by Anderson and Bushman (2002), personal and situational factors influence how affect, arousal and cognition interact to come to a decision how to respond to provocation. The results of the current study reveal the effect fear arousal has on social cognitive processes. In line with the GAM, the behavioral outcome of activating or deactivating mentalizing processes likely depends on additional situational and personality variables that should be investigated in future studies. Finally and supporting previous findings, RT data and neural activation patterns during the punishment selection suggest that reacting aggressively to provocation is associated with activity in reward circuits, leading to a strengthening of the aggressive response.

Conflict of Interest

None declared.

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