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ORIGINAL ARTICLE

Selective reward responses to violent success events during video games

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Abstract

Aggressive behavior in violent video games activates the reward system. However, this effect is closely related to game success. Aim of the present study was to investigate whether aggressive behavior has a rewarding value by itself. Functional magnetic resonance imaging (fMRI) was measured in fifteen right-handed males while playing the video game *Carmageddon*. Neuroimaging data were analyzed based on violent and non-violent success and failure events. Correlations with subjective game experience measured brain-behavior and -affect relationships. Results revealed a differential involvement of the striatal reward system: non-violent success elicited activation of the ventral striatum, whereas violent success activated specifically the dorsal striatum. Subjective game experience correlated with putamen and medial prefrontal cortex activation specifically for violent success. These results emphasize a differential neural processing of violent and non-violent success events in dorsal and ventral striatum. Virtual violence seems to enable selective responses of the reward system and positive in-game experience.

Keywords Aggressive behavior · Neuroimaging · Nucleus accumbens · Play fighting · Putamen

Abbreviations

ACC	Anterior cingulate cortex
BOLD	Blood oxygenation level dependent
EPI	Echo-planar imaging
fMRI	Functional magnetic resonance imaging
FWHM	Full width at half maximum
GLM	General linear model
mOFC	Medial orbitofrontal cortex
mPFC	Medial prefrontal cortex
MPRAGE	Magnetization prepared rapid acquisition gradient
MR	Magnetic resonance
NAcc	Nucleus accumbens
TE	Echo time
TI	Inversion time
TR	Repetition time

Introduction

Aggression is a part of natural human behavior and serves numerous important purposes including obtaining food and territory, establishing status hierarchies, and fighting rivals to assure access to mates (Kramer et al. 2007). Neuropsychological studies describe a network encompassing orbitofrontal (OFC) and prefrontal cortex (PFC), anterior cingulate cortex (ACC), and the amygdala to mediate aggressive behavior (Hoptman 2003; Bufkin and Luttrell 2005; Paus 2005; Patrick 2008; Sterzer and Stadler 2009; Yang and Raine 2009; Coccaro et al. 2011; for a review, see Anderson and Kiehl 2012). Recent studies, however, increasingly point to the role of the reward system (e.g. Buckholtz et al. 2010) in processing aggression. The involvement of the reward

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system, encompassing nucleus accumbens (NAcc), caudate nucleus, putamen, striatal dopaminergic system, the PFC, and the OFC (Chen et al. 2017; for a comprehensive review, see Haber and Knutson 2010), may be critical to understand learning of adaptive and non-adaptive aggression (Marsh 2013).

Neuroimaging studies on competitive encounters and provocation demonstrated the involvement of the reward system, mainly the dorsal striatum, in performing aggressive acts (de Quervain et al. 2004; Zink et al. 2008). Individuals with psychopathic traits showed higher dopamine release in nucleus accumbens (NAcc) as well as higher activation in this structure in response to reward anticipation (Buckholtz et al. 2010). Psychopathy was also associated with atypical striatum morphology (Boccardi et al. 2013). Marsh (2013) proposed that the reward system in psychopaths might be overactive, while the systems governing threat anticipation are dysfunctional. Thus, evidence suggests that involvement of the reward system may enhance learning of aggressive behavior.

Animal studies show that certain forms of aggression are part of normal behavior, e.g. in play fighting, which is a common form of play activity in young animals (Pellis et al. 1993). Animals exert considerable effort in order to be able to perform aggressive acts, and they seem to draw pleasure from engaging in aggressive encounters (Fish et al. 2002). Those rewarding properties of aggression are mediated via the striatal dopaminergic system (Couppis and Kennedy 2008), similar to reward processing patterns in humans. Thus, it is reasonable to assume that aggressive behavior may have a rewarding value by itself in humans as well.

Functional imaging studies on aggression in healthy humans are rare since openly expressed aggressive behavior is difficult to induce experimentally during neuroimaging (Klasen et al. 2013). Moreover, research has differentiated between instrumental (i.e., proactive, goal-driven, and unemotional) and reactive (i.e., provocation-based, impulsive, and emotional) subtypes of aggression (Fontaine 2007). Blair et al. (2006) argue that reactive aggression (i.e. aggressive acts in response to frustrating or threatening events) and instrumental aggression (purposefully used acts to achieve a specific desired goal) are partly independent and mediated by at least partially dissociable neurobiological networks. Classic experimental approaches typically induce aggression via provocation, such as in the well investigated Taylor Aggression Paradigm (TAP; Taylor 1967). By definition, provocation approaches are well suited for reactive aggression forms; however, instrumental aggression forms may require different paradigms. Zaki and Ochsner (2012) stressed the necessity of naturalistic, ecologically valid, paradigms to complement well-controlled but simplified paradigms. One of such naturalistic solutions is virtual reality that opens new ways to study proactive aggressive behavior without negative consequences for the subjects (see Mathiak and Weber 2006; Slater et al. 2006; Zvyagintsev et al. 2016). Leading to high levels of immersion into the game world, video games are an interesting model to study neural correlates of complex behavior such as aggression (see Klasen et al. 2008; Mathiak et al. 2013; Mathiak and Weber 2006). Similar to the animal play fighting model (Delville et al. 2006), violent video games may provide insight into the neurobiology of aggressive behaviors, such as the killing of virtual opponents, which are otherwise difficult to study in humans.

It is well established that playing games activate the dopaminergic reward system (Koepp et al. 1998; Hoeft et al. 2008; Cole et al. 2012). Recent studies have addressed contributions of particular events such as aggressive behavior to this neural pattern. In first-person shooter games, killing did not activate the reward system as compared to the ongoing gameplay (Mathiak and Weber 2006). Instead, decreased activity was observed after failure events such as virtual dying (Mathiak et al. 2011; Klasen et al. 2012; Katsyri et al. 2013a, b). Thus, neural effects and subjective perception of aggression in video games seem to be often confounded with the aspect of game success. In fact, in a meta-analysis of 32 game studies, success and failure events were among the most potent triggers for pleasant and unpleasant emotions, respectively (Nummenmaa and Niemi 2004). In the light of the rewarding value of game success (Przybylski et al. 2010), it is necessary to disentangle these aspects to identify a putative rewarding effect of aggressive behavior. However, so far none of the neuroimaging studies on violent gaming succeeded in revealing a direct rewarding component of aggression.

The present study sought to disentangle the reward system involvement in explicitly violent vs. non-violent playing behavior in a semi-naturalistic brain-imaging paradigm, using a violent racing game (*Carmageddon: TDR 2000*; Torus Games, Bayswater, Australia 2000). Carnagey and Anderson (2005) applied an experimental modification of the *Carmageddon* game to compare the impact of violent and non-violent events on physiological arousal and affect. In this study, they also demonstrated its high validity as a model to study aggression. While both killing pedestrians and collecting points produced similar effects on arousal (measured as blood pressure and pulse), playing the violent version of the game led to more hostile effect and to more aggressive cognitions than playing the non-violent version.

In the current study, functional magnetic resonance imaging (fMRI) measured neural activation during success and failure events that were associated with explicit violence, i.e. successful or failed killing of a virtual pedestrian, or did not contain violence, i.e. point collection or crash without involvement of virtual pedestrians (similarly as in Carnagey and Anderson 2005). We investigated whether the violent context modulated the responses in the reward system to success and failure events. Moreover, we investigated a putative relationship between event-related reward system patterns and subjective emotional response to the game. We hypothesized (1) a significant contribution of aggression to event-related reward system activation; and (2) a relationship between violence-related reward system patterns and a positive emotional game evaluation.

Materials and Methods

Participants

We studied 15 right-handed males (age range 21–28 years, mean 24.9 ± 2.6). All subjects played video games on average at least 5 h/week. They had no previous experience with the game *Carmageddon: TDR 2000* (Torus Games, Bayswater, Australia, 2000) that served as stimulus in this study. The subjects had normal or corrected to normal vision, normal hearing, and no contraindications against magnetic resonance (MR) investigations. All participants were systematically screened for physical illness, mental problems, past and present psychiatric and psychotherapeutic treatments, and medication. According to the screening, all participants were healthy. The experimental design was according to the Code of Ethics of the World Medical Association (Declaration of Helsinki, 1964) and the study protocol was approved by the local Ethics Committee. We obtained a written informed consent from each subject after providing a complete description of the study.

The sample size of the present study was determined based on a previous study by our lab (Mathiak and Weber 2006). This paper showed robust and strong event-related activation patterns in a violent video game in a sample of $N = 13$ players. Effect sizes in this data set were estimated to be rather large in magnitude, considering the abovementioned limitations. This was also true for midbrain structures of the reward system (see also Klasen et al. 2012).

Procedure

The subjects played the video game *Carmageddon: TDR 2000* (Torus Games, Bayswater, Australia, 2000) without restrictions over four scanning sessions of 10 min duration each. In the game, participants drove a racing car against other virtual drivers controlled by the computer. *Carmageddon* is a well investigated model to study aggressive behavior (Carnagey and Anderson 2005; Bushman and Anderson 2009) and has previously been applied to test for drug effects in virtual aggressive behavior (Klasen et al. 2013) and for extraction of cortical network related to aggression in an overlapping sample (Zvyagintsev et al. 2016). Similar to the above-mentioned studies, we employed a violent and a non-violent modification of the game. In the first version, the players gained points by killing pedestrians with their car. They were explicitly instructed to kill as many pedestrians as possible. This version of the game contained a considerable amount of violence with excessive depiction of blood splatter. In the second, non-violent condition, we introduced a modified version without pedestrians. Here, the players' task was to pick up the bonus points, i.e., colorful icons which can be found on the game map, without violent interactions. Picking up bonus points was accompanied by color explosion, with similar visual stimulation in both violent and non-violent game versions. All game play sessions (violent and non-violent) took place on the same game map, assuring a highly similar visual stimulation pattern. The map consisted of a post-apocalyptic landscape, with factories, a ghost city, a deserted fairground, a prairie landscape, etc. The size of the map was limited, and usually each player circled the map several times in the course of one game play session. Each subject played two violent and two non-violent sessions; the order of the sessions was randomized. Visual stimulation and game sound were delivered via MR compatible video goggles and headphones; sound levels were adjusted individually to a comfortable level. Both videos and sound were recorded for subsequent content analysis.

Game affect was measured using a German version of the Game Experience Questionnaire (GEQ; Ijsselsteijn et al. 2008). Ten items assessed positive and negative feelings during the game play on a 5-point Likert scale each. Game-related affect was calculated by subtracting the negative items from the positive ones. Internal consistency (Cronbach's alpha) was determined as 0.814. Other scales from the questionnaire such as game evaluation, tiredness, or returning to reality were of no relevance for the research question and therefore not considered for analysis.

Image acquisition

We conducted whole-brain fMRI with echo-planar imaging (EPI) sequences (echo time TE = 28 ms, repetition time TR = 2000 ms, flip angle = 77°, voxel size = 3 × 3 mm, matrix size = 64 × 64, 34 transverse slices, 3 mm slice thickness, 0.75 mm gap) on a 3 Tesla Siemens Trio scanner (Siemens Medical, Erlangen, Germany) with a standard 12-channel head coil. We acquired 310 volumes pro session (resulting in the sum of 1240 volumes for all four sessions). Following the functional measurements, high-resolution T1-weighted anatomical images were recorded using a magnetization prepared rapid acquisition gradient echo (MPRAGE) sequence (TE = 2.52 ms; inversion time TI = 900 ms, TR = 1900 ms; flip angle = 9°; FOV = 256 × 256 mm^2; 1 mm isotropic voxels; 176 sagittal slices).

Content analysis and event definition

Game events were coded with an accuracy of 67 ms (according to one frame with 15 Hz frame rate) using the annotation software *Elan 3.8.0* (MPI for Psycholinguistics, Nijmegen, The Netherlands). Four different types of game events were annotated: violent success (killing a pedestrian; Fig. 1a), violent failure (missing to hit a pedestrian; Fig. 1b), non-violent success (collecting bonus points; Fig. 1c), and non-violent failure (crashing into an object; Fig. 1d). The coding schema is relatively simple and achieved inter-rater reliabilities above 0.9 in pretesting. Further, the coder received twenty hours of intensive and supervised training on material not used in the study. This type of content analysis with high time resolution has been described in detail before and was validated for fMRI analyses (e.g. Weber et al. 2009).

Image analysis

Image analyses were performed using *BrainVoyager QX 2.6* (Brain Innovation, Maastricht, The Netherlands) with standard preprocessing and slice scan time correction, 3D motion correction, Gaussian spatial smoothing (4 mm FWHM (full width at half maximum)), and high-pass filtering including linear trend removal. In order to avoid T1 saturation effects, we discarded the first five images of each session. Functional images were co-registered to the anatomical data and transformed into Talairach space. Data was time-synchronized to the coded game events and analyzed in an event-related fashion. We constructed a general linear model (GLM) from the events and convolved it with the hemodynamic response function. Statistical parametric maps were created using random effects GLM with predictors according to the four stimulus types. Contrasts between different predictors (game event types) were investigated using t-statistics. Time courses of blood oxygenation level dependent (BOLD) signal intensity values were extracted from cluster peak voxels, normalized to the ongoing gaming baseline value, and averaged over subjects, resulting in event-related BOLD response plots to the four different stimulus types.

To investigate relationships between event-related brain activation and subjective game experience, we calculated correlations of the individual contrast values with the game affect scores. This was performed with the standard procedure implemented in *BrainVoyager QX 2.6*. For a given comparison (e.g. violent success vs. violent failure), the contrast values were voxel-wise correlated with an external variable (e.g., the game affect scores). In this example, the resulting map revealed brain areas where the neural response to violent success (as compared to violent failure) was associated with game-related affect. Similar correlations with game affect were calculated for the contrasts "non-violent

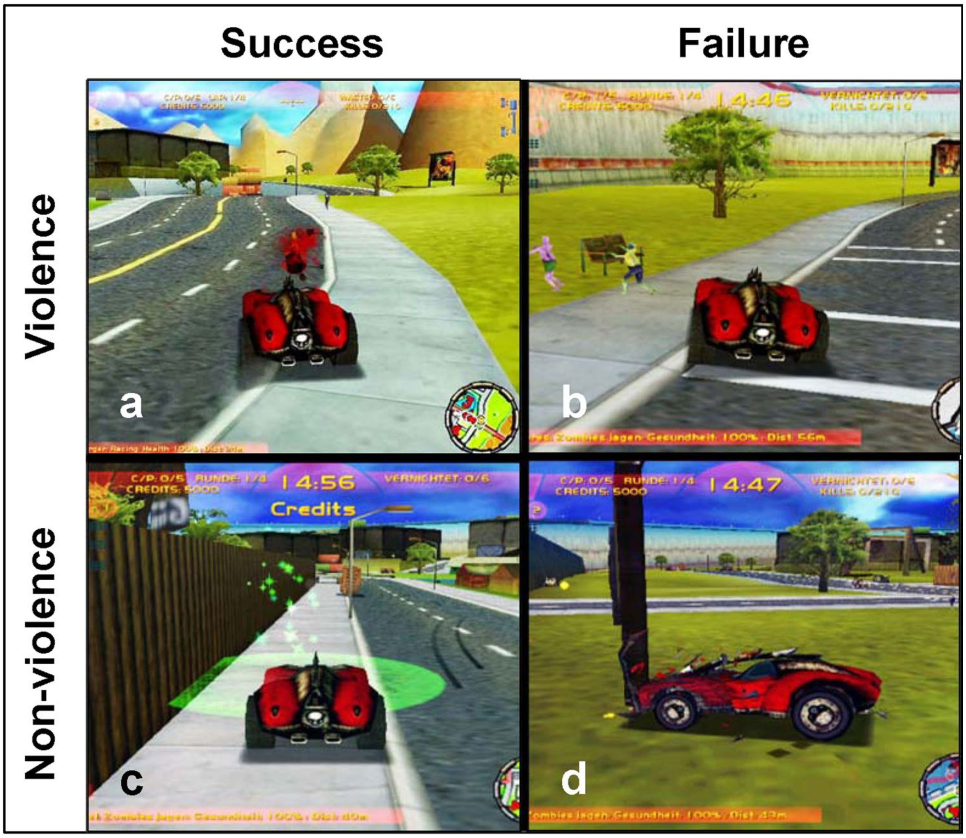

Fig. 1 In the violent game condition, the participants killed virtual pedestrians by hitting them with a car (**a** violent success events; the killed pedestrian is covered with the blood splatter and matching sounds are heard) while narrowly missing the human target constituted failure events (**b** non-violent failure events; here two pedestrians are running to the left escaping from the target point of the vehicle). In the non-violent modification of the game, players collected colorful icons by driving over them (**c** non-violent success events; colorful rain and sounds mark the successful hit of bonus points). Non-violent failure events were coded when the player crashed into objects in the game map (**d**)

success vs. non-violent failure", "violent success vs. non-violent success" and the interaction "success × violence". This method of linking brain activations to external measures (state, trait, or behavioral variables) is well established in social neuroscience and has been employed in numerous investigations (e.g., Mathiak et al. 2011).

All activations were thresholded at a voxel-wise $p < 0.05$ and cluster size $k > 45$ voxels, according to a $p < 0.05$ corrected for multiple comparisons after Monte Carlo simulations.

Results

Behavioral effects

Content analysis revealed a total of 1674 virtual violence events, i.e. killing of pedestrians (111.6 ± 29.7 (mean ± standard deviation) events per participant). Failed violence (failing to hit a pedestrian) was coded 596 times in total (39.7 ± 10.8 per participant). Non-violent success events (collecting of bonus points) were detected 1515 times (101.0 ± 24.3 per participant). Non-violent failure (crashing into an object) was coded 3847 times in total (256.5 ± 28.2 per participant). Participants reported a slightly higher average game affect after playing ($\Delta = 1.93 \pm 5.85$), which was statistically not significant. Game affect did not correlate with the frequency of the gaming events (all $p > 0.2$). Subjects who played the violent version of the game during their last fMRI session demonstrated lower negative affect as compared to those playing the non-violent game at the end (two-sample T test; $t(13) = 2.30$, $p = 0.04$).

fMRI

Violent success > violent failure

The direct comparison of violent success and violent failure events yielded a significant activity in bilateral putamen for the success events (Fig. 2a; Table 1). The event-related time courses of the BOLD signal revealed that both the violent success and failure events led to a strong initial activation

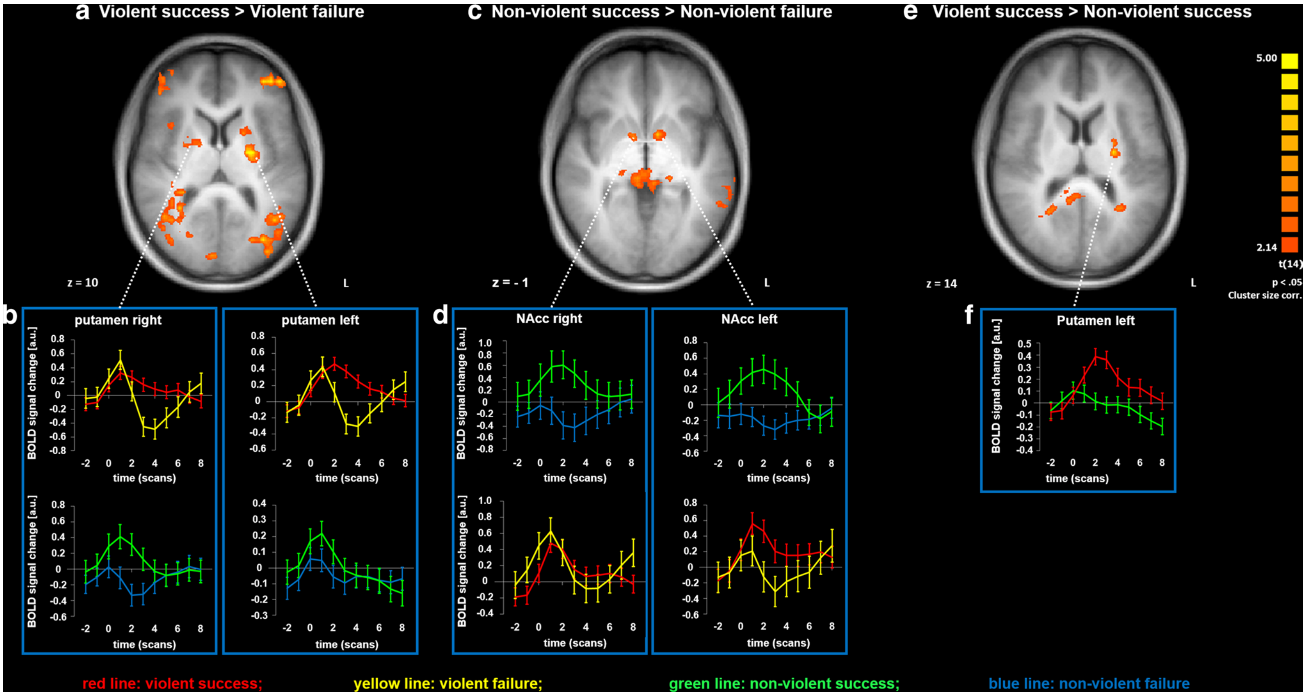

Fig. 2 Violent success compared to violent failure events led to significantly stronger putamen activity (**a**). The plots depict the event-related time courses of the BOLD signal during the violent condition in the right and left putamen (**b**, upper panels). The initial putamen activation occurring in anticipation of the violent event was followed by a rapid deactivation in the failure events (negative prediction error). This differential response pattern to violent success and failure partly manifested in left, but not right nucleus accumbens (**d**, lower panels). Non-violent success compared to non-violent failure events led to stronger activation in the nucleus accumbens (**c**). The plots depict the event-related time courses of the BOLD signal in the nucleus accumbens during non-violent condition (**d**, upper panels). While the non-violent success increased the NAcc activation, the non-violent failure led to deactivation. Some activation increase showed also in the right putamen in non-violent success, but not during non-violent failure (**b**, lower panel). Violent success events as compared to non-violent success activated the reward system (**e**). The event-related time courses of the BOLD signal in the left putamen (**f**) demonstrate increased activity during violent success (red line) but not during non-violent success (green line)

of the putamen anticipating the actual event. This peak was followed by sustained activity after success (upper panels of Fig. 2b; red curves), but a sharp and sudden activation decrease when the player failed in the violent task (upper panels of Fig. 2b; yellow curves). A similar trend emerged in the right NAcc (Fig. 2d, lower left panel) but not for the left NAcc (Fig. 2d, lower right panel).

Non-violent success > non-violent failure

The contrast map of non-violent success with non-violent failure events yielded a different response pattern: activity in the NAcc increased after success events (Fig. 2c; Table 2). Event-related time courses of the BOLD signal revealed that non-violent success events (green curve) led to the activation of the NAcc whereas non-violent failure (blue curve) deactivated this structure (upper panels of Fig. 2d). In the putamen cluster, non-violent success and failure events yielded responses that were weaker than in the violent condition (Fig. 2b, lower panels).

Violent success > non-violent success

The hypothesis that aggressive behavior may yield activation in the reward system was confirmed by the contrast between violent and non-violent success in the left putamen (Fig. 2e; Table 3). BOLD signal time courses confirmed an increase in activity to violent success but not to failure (Fig. 2f; red and green curves, respectively). Finally, the interaction of success with violence did not yield a significant activation cluster.

To compare motion parameters between the game conditions, we extracted all 6 rigid-body motion parameters (3 translations (*x*, *y*, *z*) and 3 rotations (pitch, roll, yaw)) and

Table 1 Regions activated in response to violent success vs. violent failure

Region	Talairach coordinates			MNI coordinates			Cluster size (mm^3)	*T* value
	X	*Y*	*Z*	*X*	*Y*	*Z*		
Middle frontal gyrus R	41	55	15	42	56	15	2394	8.76
Angular gyrus L/R	−28	−56	33	−28	−59	34	72,029	7.48
Medial frontal gyrus L/R	−4	34	36	−4	34	40	1591	6.46
Medial frontal gyrus L/R	14	16	54	14	12	59	2039	6.27
Middle frontal gyrus R	53	−2	18	56	−1	16	2286	5.98
Putamen L	−25	−8	9	−26	−7	7	3439	5.70
Middle frontal gyrus L	−34	43	15	−35	45	16	2741	4.92
Putamen R	26	16	3	27	19	−1	2331	4.29

Table 2 Regions activated in response to non-violent success vs. non-violent failure

Region	Talairach coordinates			MNI coordinates			Cluster size (mm^3)	*T* value
	X	*Y*	*Z*	*X*	*Y*	*Z*		
Nucleus accumbens R	11	−56	18	11	−58	18	13,406	8.00
Middle frontal gyrus L	−52	13	36	−54	13	38	2748	6.48
Middle frontal gyrus L	−43	40	6	−44	43	5	1286	5.69
Superior parietal lobule L	−34	−62	33	−34	−65	34	3699	5.47
Nucleus accumbens L	−10	4	−3	−10	7	−7	2098	4.87
Posterior cingulate gyrus L/R	−1	−38	33	−1	−38	33	1805	4.44
Middle temporal gyrus L	−64	−35	−9	−69	−34	−16	1578	4.15
Cerebellum R	5	−74	−27	5	−75	−37	1919	4.02

Table 3 Regions activated in response to violent success vs. non-violent success

Region	Talairach coordinates			MNI coordinates			Cluster size (mm^3)	*T* value
	X	*Y*	*Z*	*X*	*Y*	*Z*		
Posterior cingulate gyrus R	17	−38	24	17	−40	24	1961	6.47
Parahippocampal gyrus L	−31	−41	3	−33	−41	−1	1870	6.44
Putamen L	−25	−11	15	−26	−10	14	1385	6.32
Cerebellum L/R	−1	−50	−21	−2	−49	−30	1400	5.03

calculated average volume-to-volume displacements (mm/°) for violent and non-violent game conditions. The conditions differed with respect to translation along the x axis ($t = 2.35$, $p = 0.03$) and pitch ($t = 2.65$, $p = 0.02$), but none of the other parameters. None of these differences remained significant after correction for multiple comparisons. To further exclude the possibility that the reported brain activations might reflect artefacts arising from head motion during the video game, we calculated an additional GLM with all 6 rigid-body motion parameters (3 translations, 3 rotations) as confound predictors. The activation clusters in putamen and NAcc remained very similar using the same threshold, suggesting little influence of head motion on the observed responses in the reward system. Finally, to further evaluate the possibility of motion confounds, we correlated the motion parameters with the event types across the different game conditions. Even without correction for multiple comparisons, none of the six motion parameters (x, y, z, pitch, roll, yaw) was correlated with any of the event types in any of the game conditions (all $p < 0.22$).

To evaluate a potential influence of the asymmetric success/failure frequency rates (see Behavioral effects) on reward system activation patterns, we calculated a subject-wise frequency asymmetry coefficient, which was defined as follows: (number of violent success events/number of violent failure events)/(number of non-violent success events/number of non-violent failure events). The values of the frequency asymmetry coefficient were then correlated with the peak contrast values in the putamen and NAcc clusters from Fig. 2a–e. This method allowed us to investigate whether interindividual differences in frequency asymmetry were associated with interindividual differences in the reward system activation patterns. No correlations with the frequency asymmetry coefficient were observed in any of the reward system regions (all $p > 0.16$). Similarly, the ratio of success to failure events was unrelated to the reward system clusters, both in the violent (all $p > 0.35$) and non-violent (all $p > 0.45$) conditions. Finally, the success/failure rates of the violent and non-violent conditions were also unrelated to each other ($r = -0.08$, $p = 0.78$).

Brain–emotion interaction

In the violent game condition, game affect (Game Experience Questionnaire) correlated positively with the responses to success (violent success > violent failure, see Fig. 2a) in the reward system, i.e. the putamen and the medial prefrontal cortex (mPFC; Fig. 3a). In other words, higher putamen and mPFC activity in this contrast predicted a more positive evaluation of the game play. This effect was absent for the non-violent game condition (non-violent success > non-violent failure, compare Fig. 2c): without violence, success-related activity in the reward system did not correlate with game affect (Fig. 3b). This difference was confirmed by the correlation analysis with the direct contrast of both success types (violent success > non-violent success); it revealed a significant cluster in the mPFC (Fig. 3c). Finally, both in mPFC and putamen, game affect correlated with the interaction *success × violence* ((violent success > violent failure) > (non-violent success > non-violent failure); Fig. 3d). Thereby, the data confirm a significant influence of violence-specific reward system activity on subjective game evaluation.

Robustness of fMRI results

Previous findings suggest that the BOLD responses to violent success in video games are robust across participants (Mathiak and Weber 2006). To evaluate whether this was also the case for the data in the present study, we created an exemplary group probability map of unthresholded 1st level maps for the contrast Violent success > Violent failure (see Fig. 2a, b). The results of the mapping are depicted in Fig. 4. Positive t values in right and left putamen, almost identical to the clusters from Fig. 2 a, were observed for > 75% of all participants (single voxels up to 100%; Fig. 4). Thus, the effects were robust across the group and clearly not driven by single participants or outliers.

Discussion

We studied the neural correlates of reward processing during success and failure events in a violent (killing vs. missing pedestrians) and a non-violent (bonus points vs. crashes) version of a racing game. The involvement of the striatal reward system depended on the violent content: violent events activated putamen (dorsal striatum) whereas non-violent success affected NAcc (ventral striatum). Moreover, the activity of the putamen and the mPFC correlated with the affective game experience only in the violent version of the game. These results show a differential neural processing of violent and non-violent success events and emphasize the rewarding aspects of aggressive interactions in video games.

The NAcc (ventral striatum) has a key role in reward-based acquisition and maintenance of behavior and in the motivational control of actions (for a review, see Ena et al. 2011; Hart et al. 2014). In our study, ventral striatum activation was observed in a non-violent context. For the violent events, no difference between success and failure emerged; nevertheless, time series suggest that both violent and non-violent success events may activate NAcc in a comparable fashion. The putamen (dorsal striatum), in turn, is implicated in goal-directed instrumental actions and reflects reward obtaining and reward probability (Breiter et al. 2001; Knutson et al. 2001, 2003; Preuschoff et al. 2006; Tobler et al.

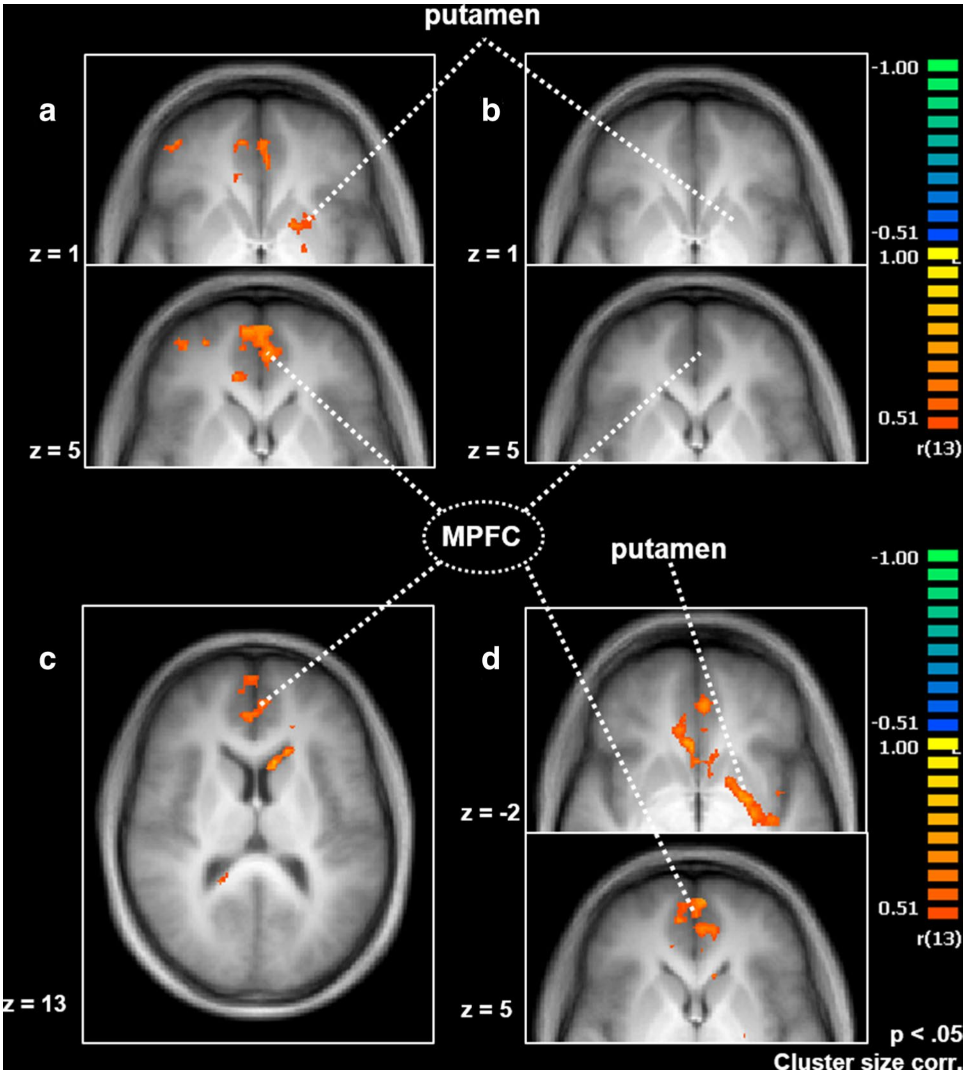

Fig. 3 The game affect correlated positively in the left putamen and the medial prefrontal cortex (mPFC) with the responses to violent success vs. violent failure (**a**) but not the non-violent success vs. non-violent failure (**b**). The correlation analysis with the direct contrast of violent success vs. non-violent success revealed a significant cluster in the mPFC (**c**). Both in mPFC and putamen, game affect correlated with the interaction contrast success × violence (**d**)

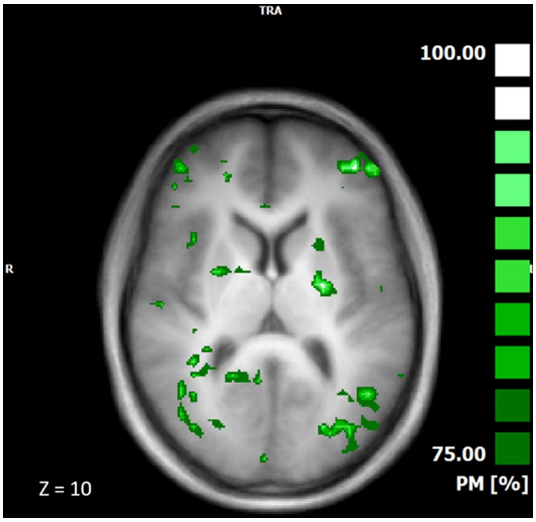

Fig. 4 Probability map of positive *t* values for the contrast Violent success > Violent failure, revealing robust effects across participants in the reward system

2006). This neural system for goal-directed motor behaviour and reward-based learning may also mediate competitive and instrumental aggression (Blair et al. 2006; Hillman 2013). Indeed, the putamen has been implicated in the processing of threatening stimuli (Sinke et al. 2010) as well as in aggression-related emotional states, such as contempt (Sambataro et al. 2006) and hate (Zeki and Romaya 2008). Animal studies also demonstrated higher dopamine turnover in the putamen of aggressive mice (Tizabi et al. 1980). Zeki and Romaya (2008) proposed that the putamen may be involved in the planning of aggressive motor acts, being mobilized in a socio-emotional context. This view is supported by our data; putamen activity increased when the player planned and successfully performed an aggressive motor act.

In addition to the putamen, the mPFC was implicated in the positive experience of specifically violent game content. The mPFC is a target region of projections from the dopaminergic mesolimbic pathway of reward processing and is consistently activated during the processing of rewarding stimuli (for a review, see Fareri and Delgado 2014). It encodes affective stimulus properties (Grabenhorst et al. 2008) and plays a central role in updating the neural representation of incentive values in the adjacent orbitofrontal cortex (Kennerley and Walton 2011). In view of those findings, recent models consider the mPFC an interface area between affective evaluation and decision making, integrating reward-related information with potential actions and their outcome probabilities (Alexander and Brown 2011). Moreover, mPFC is central for the regulation of aggression (for a review, see Blair 2004); the control of reactive aggressive behavior is frequently impaired after lesions to the mPFC (Grafman et al. 1996). Regulation abilities of the mPFC have been ascribed to cognitive control (Ochsner et al. 2004) and self-monitoring of emotions (Lane et al. 1997). This extends also to mental states of others and the detection of social norm violations, which prevents socially inacceptable behavior (Berthoz et al. 2002). With respect to aggression, Lotze et al. (2007) suggested that the mPFC suppresses conflicting feelings of compassion and aversion towards the opponent and fosters adequate responses.

Bringing these notions together, we suggest an integrative model for the role of the mPFC in virtual aggression. According to this model, the mPFC may support the suppression of conflicting feelings towards the victim (compassion and aggression) by activating the rewarding value of violence. This view is consistent with a model of Haber and Knutson (2010), hypothesizing an information flow from the ventral to the dorsal striatum: the NAcc is recruited early during reward prediction and responds to information about reward magnitude, whereas the activity of subsequent processing moves dorsolaterally through the striatum, conceivably as a result of information integration from prefrontal circuits. In our study, this cortical mediation of the reward processing via mPFC—in accordance with Lotze et al. (2007)—may have suppressed the feelings of compassion and allowed killing, resulting in the activity of putamen and mPFC, which led to a more positive evaluation of the gameplay. In a similar vein, in our previous study the temporal pole ameliorated the experience of failure events during game play (Mathiak et al. 2011). We found no correlation of affect with gaming behavior, which demonstrates that the enjoyment of game play and conceivably the reward sensitivity to violence was not influenced by game performance. Further studies will help to verify the role of mPFC in suppressing the feelings of compassion in violent video games.

Ventral and dorsal striatum are involved in the development of goal-directed behavior particularly via coding reward expectation and reward prediction errors (Cohen 2007; Garrison et al. 2013). Phasic increases in activity encode the positive prediction error, i.e. when the outcome is better than anticipated, whereas activity decreases to negative prediction error are observed when outcomes are worse than expected (for a review see Schultz 2004). Neuroimaging studies repeatedly demonstrated the involvement of both putamen and NAcc in the coding of negative prediction errors (McClure et al. 2003), e.g. in instrumental conditioning paradigms (Mattfeld et al. 2011). The present study found a reduction of striatal activity particularly after violent failure events. An early onset of putamen activation, anticipating the actual event, was followed by an immediate deactivation after the observed failure (Fig. 2b). Failure game events can thus be perceived as negative prediction errors in an instrumental conditioning paradigm. The game events encompass the anticipatory and appetitive phase of motivated behavior: the player anticipates a successful action leading to gaining reward points (killing a target in the violent version and bonus collection in a non-violent one). According to the principles of operant conditioning, violent game events may thus foster procedural learning processes.

Over the years, there has been a substantial amount of research dedicated to the question whether there is a relationship between virtual aggression and “real” social aggressive interactions. It has been suggested that playing violent games is causal for real-life aggression (e.g. Anderson and Bushman 2002), but this view has also been challenged (e.g. Ferguson 2013). Apart from the debate about such transfer effects to the real world, it has been shown that violence against virtual characters seems to share psychological and neural features of real-life aggression. As an example, Cheetham et al. (2009) demonstrated that applying pain to an innocent virtual character in a virtual replication of the Milgram experiment involved brain networks of personal distress—although the participant was fully aware that the “victim” was no real person. Remarkably, virtual violence in games seems to share features of “hot” (i.e. reactive) and “cool” (i.e., instrumental) forms of aggression. On the one hand, game violence evokes emotional and physiological reactions, resembling “hot” aggression (Anderson and Bushman 2001; Gentile et al. 2016); on the other hand, emotional reactions seem to be actively suppressed during virtual violence, resembling “cool” aggression (Mathiak and Weber 2006). In summary, it seems justified to consider game violence an aggression form of its own, with some more and some less established connections to its real-life counterpart. In particular, a possible relationship between rewarding effects of real and virtual aggression forms is still unclear and needs further systematic investigation. First evidence for a possible connection comes from fMRI research using the Point Subtraction Aggression Paradigm (PSAP; Cherek 1981). In this paradigm, reactive aggression towards the opponent leads to activation of the striatal brain reward system (Skibsted et al. 2017). Striatal activation in the PSAP is stronger in violent offenders and positively related to trait aggression (da Cunha-Bang et al. 2017). There are two tentative conclusions from these observations. First, social aggression can be rewarding, just as virtual violence. Second, these rewarding effects seem to generalize beyond the laboratory setting into real-world aggression. However, further studies are needed to establish these connections empirically.

Limitations

Our study has a number of limitations that have to be taken into account for the interpretation of the results.

First, the present study did not record psychophysiological data that may have been associated with the game events. In particular, mPFC seems to be an important node for the regulation of psychophysiological markers such as heart rate variability (Sakaki et al. 2016). In fact, previous research has shown a stable association between the exposure to violent games and physiological arousal parameters such as heart rate (Anderson and Bushman 2001; Gentile et al. 2016). In line with the view of Thayer et al. (2012), the mPFC may be a node in a neurovisceral system of emotion regulation and experience. Thus, anticipation and/or experience of reward may lead to activation of this system, possibly supporting the hedonic component of reward experience. Future studies are needed to clarify the brain–heart interplay during the experience of complex, close-to-reality scenarios.

Second, striatal activity may also reflect motoric or sensory processes. Since aggression in the present game requires motor performance, a confound cannot entirely be ruled out, particularly since the controlled movement (button presses on the keyboard) was not recorded. In fact, two types of motion-related effects may influence the data: (1) head motion, which may cause substantial artefacts in fMRI data and can be a serious confound in functional neuroimaging experiments, and (2) brain activity resulting from event-related hand motion. As for (1), it seems unlikely that striatal activation patterns can be explained by motor processes. Only two out of six motion parameters differed between game conditions, and none of them remained significant after correction for multiple comparisons. Also, including motion parameters as confound predictors had little influence on the results. Moreover, motion parameters were uncorrelated to all event types in all game conditions. As for (2), we suggest that hand motion-related brain activity should less be regarded as a confound, but more as an integral part of the game. The concepts "success" and "failure" are by definition consequences of an individual's actions, and actions in a video game require hand motor activity. Thus, the neural processing of success and failure events is inextricably linked to button presses on the keypad and thus also to motor activity. Sensory processes, in turn, also offer only limited explanatory power since putamen activity in our study increased already in the anticipatory phase, i.e. before hitting the pedestrian. Thus, it seems justified to assume that striatal activity is substantially related to reward processing.

Third, another limitation concerns the assessment of subjective game evaluation. Since the GEQ was presented after the end of the gameplay, there was no separate evaluation of the violent and non-violent gameplay sessions. As for the overall game evaluation, the correlation with event-related brain activity suggests that game enjoyment was associated with the rewarding effects of violent success. This is also supported by the fact that the subjects who played the violent version of the game in their last session demonstrated lower negative affect.

Fourth, a limiting aspect concerns the game events in violent and non-violent conditions. Whereas the violent targets were mobile, the non-violent targets were static. Clearly, this can be considered a confounding factor. However, this violence-free modification was a gaming mode of the game Carmageddon: TDR2000 and thus ecologically valid with respect to real-life game play. This is directly related to a core challenge of semi-naturalistic designs: finding a trade-off between a well-controlled (but artificial) situation and a "real-life" (but uncontrolled) situation (reliability-validity dilemma, Lienert and Raatz 1998). In the case of semi-naturalistic designs, it will be difficult to avoid this kind of associated additional variables. Thus, they can as well be considered part of an ecologically valid setup. Nonetheless, we sought to investigate whether the aspect of target motion had an influence on reward system activity. For this purpose, we calculated the contrast (violent success + violent failure) > (non-violent success + non-violent failure). This contrast reflected the difference between moving and non-moving targets. At the predefined threshold ($p < 0.05$, Monte–Carlo-corr.), the mapping revealed relative deactivations for moving targets in many brain areas, including anterior insula, inferior frontal gyrus, dorsomedial prefrontal cortex, posterior cingulate cortex, precuneus, somatosensory cortex, as well as primary and higher visual areas. Relative activations were limited to one cluster in the left hippocampus. No differences were observed in regions of the reward system. Thus, there is no evidence for an influence of target motion on reward system activity in the present study.

Finally, although the visual and auditory stimulation was matched between the game conditions, allover sound intensities differed across the participants since the sound level was adjusted individually. It seems possible that these differences may constitute a potential confounder, which cannot be ruled out since the individual volume settings for the scanner headphones were not recorded. However, since the volume adjustment took place already before the first game session, it seems unlikely that these individual differences had a substantial influence on the comparison of the different game conditions or event types.

Conclusions

The present study gives insight into the neural mechanisms during violent and non-violent video game playing. The aspect of violence modified neural processing of the game in several ways. First, violent actions led to a marked response pattern in the dorsal striatum which points at reward-based learning processes. Second, reward system activation specifically during virtual violence predicted a positive emotional response to the game. Together, these findings are a clear indication that game violence is rewarding even beyond the aspect of success. Violence itself thus seems to be a driving factor for gameplay motivation—and may even promote video game addiction, which is as well characterized by game-induced reward system hyperactivation (Ko et al. 2009). Further research may thus clarify the role of violence in the development of excessive game consumption.

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Compliance with ethical standards

Conflict of interest All authors (M.K., K.A.M., M.Z., P.S., R.W., K.M.) report no further conflicts of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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