

Does Playing Violent Video Games Induce Aggression? Empirical Evidence of a Functional Magnetic Resonance Imaging Study

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This study aims to advance the media effects debate concerning violent video games. Meta-analytic reviews reveal a small but noticeable association between playing violent video games and aggressive reactions. However, evidence for causal associations is still rare. In a novel, event-related functional magnetic resonance imaging study, 13 male research participants were observed playing a latest-generation violent video game. Each participant's game play was recorded and content analyzed on a frame-by-frame basis. Onscreen activities were coded as either "passive/dead, no interactions"; "active/safe, no imminent danger/no violent interactions"; "active/potential danger occurs, violent interactions expected"; "active/under attack, some violent interactions"; and "active/fighting and killing, many violent interactions." Previous studies in neuroscience on aggressive thoughts and behaviors suggested that virtual violence would suppress affective areas of the anterior cingulate cortex (ACC) and the amygdala subsequent to activity variations at cognitive areas of the ACC. Comparison of game play activities with and without virtual violence in 11 participants confirmed the hypothesis. The rather large observed effects can be considered as caused by the virtual violence. We discuss the applicability of neuroscience methodology in media effects studies, with a special emphasis on the assumption of virtuality prevalent in video game play.

In 2003, more than 239 million computer and video games were sold in the United States; that is almost two games for every household in the United States (Entertainment Software Association, 2004). More than 90% of all U.S. children and adolescents play video games, on average for about 30 min daily (Kaiser Family Foundation, 2002). The National Youth Violence Prevention Resource Center (2004) has stated that a 2001 review of the 70 top-selling video games found 49% contained serious violence. In 41% of all games, violence was necessary for the protagonists to achieve their goals. In 17% of the games, violence was the primary focus of the game itself (Children Now, 2001). “Mature” rated games are extremely popular with pre-teen and teenage boys who report no trouble buying the games (Federal Trade Commission, 2000, 2002; National Institute on Media and the Family, 2003). There is no doubt, violent video games are among the most popular entertainment products for teens and adolescents, especially for boys (cf. Vorderer, Bryant, Pieper, & Weber, 2006).

New generation violent video games contain substantial amounts of increasingly realistic portrayals of violence. Elaborate content analyses revealed that the favored narrative is “a human perpetrator engaging in repeated acts of justified violence involving weapons that result in some blood shed to the victim” (Smith, 2006; Smith, Lachlan, & Tamborini, 2003, p. 73). Smith et al. found that the amount and context of violence presented in state-of-the-art video games rated “M” (Mature) or “T” (Teens), and even “E” (Everyone) poses risks for negative effects such as the development of aggressive scripts for social problem solving (see also Haninger & Thompson, 2004).

Violent video games frequently have been criticized for enhancing aggressive reactions such as aggressive cognitions, aggressive affects, or aggressive behavior. Multiple theories have been developed to explain how exposure to violence in media—and video games in particular—could cause both short- and long-term increases in human aggression and violence (for reviews, see Anderson, 2003; Anderson et al., 2003; Anderson & Bushman, 2002a; Anderson et al., 2004; Carnagey & Anderson, 2003; Dill & Dill, 1998; Griffiths, 1999; Weber, Ritterfeld, & Kostygina, 2006). Early theories used notions of aggressive instinct, catharsis, and frustration to explicate potential origins of human aggression (Bushman, 2002; Feshbach, 1955). Recent theorizing, however, explains the long-term effects of media violence on aggression as originating from observational learning of cognitions related to aggressive behavior (Anderson & Huesmann, 2003; Bandura, 1973, 2001, 2002; Berkowitz, 1993; Huesmann, Moise-Titus, Podolski, & Eron, 2003; Krahe & Moeller, 2004), desensitization or emotional habituation (Funk, Baldacci, Pasold, & Baumgardner, 2004; Rule & Ferguson, 1986), and cultivation processes (Gerbner, Gross, Morgan, Signorielli, & Shanahan, 2002; van Mierlo & van den Bulck, 2004). Short-term effects may be based on processes like priming and imitation (Jo & Berkowitz, 1994) as well as arousal processes and excitation transfer (Bryant & Miron, 2003; Zillmann, 1978, 2003). The General Aggression

Model by Anderson and Bushman (2002b) can be considered an integration of different theories (besides catharsis theory) trying to explain the effects of media- and video game violence (see also Anderson & Huesmann, 2003; Buckley & Anderson, 2006; Carnagey & Anderson, 2003).

Though some studies found no association between violent video game play and aggressive reactions (see, e.g., Collwell & Payne, 2000; Durkin, 1995; Durkin & Aisbett 1999; Durkin & Barber, 2002; Kestenbaum & Weinstein, 1985; Scott, 1995), the majority reports antisocial effects. Explanations for the inconsistent findings are manifold. Besides methodological reasons (e.g., Olson, 2004), one explanation is that players understand and interpret the same games they play differently. Depending on how players read a game and its violent content, playing might have a greater or lesser impact on the players' attitudes, emotions, and behaviors (cf. Potter & Tomasello, 2003). Meta-analytic reviews (Anderson, 2004; Anderson & Bushman, 2001; Sherry, 2001) and literature reviews (e.g., Anderson & Dill, 2000; Dill & Dill, 1998; Griffith, 1999; Gunter, 1998) came to the same conclusion in attributing negative effects to violent video game playing. Overall and according to Cohen's (1988) classification of effect sizes, there is a small, but noticeable correlation between playing violent video games and aggressive reactions ($r \approx 0.26$). Though the average effect seems to be rather small in size, it is important to consider the aforementioned dosage of the potential risk factor "video game playing." Abelson (1985), Prentice and Miller (1992), and Rosenthal (1986) demonstrated that even small effects can result in high societal costs or damage under high exposure conditions.

However, a correlation does not indicate a causal relation. Though long-term studies in the realm of TV could demonstrate a causal relation between TV viewing and aggressive behavior (see, e.g., Huesmann et al., 2003; Paik & Comstock, 1994), the assumption of causality between playing video games and the tendency to show aggressive reactions is still controversial (Savage, 2004; Weber et al., in press). The crucial question is, "Do violent video games affect aggression (effect hypothesis) or do people with already aggressive traits prefer violent video games (selection hypothesis)?" Though initial longitudinal video game studies favor the effect hypothesis or propose mutually enforcing effects (e.g., Slater, Henry, Swaim, & Anderson, 2003), long-term video game studies with a panel design are still rare.

In a nutshell, teens and adolescents play video games frequently, and a significant portion of the games contain increasingly realistic portrayals of violence. Numerous inconsistent findings do exist, but theories that posit, and empirical evidence that demonstrates a positive correlation between playing violent video games and aggressive tendencies dominate the scientific literature. However, effect sizes are rather small, and the question of causality has not yet been answered sufficiently.

Therefore, further research is necessary to better understand the mechanisms for how playing violent video games could affect humans. Our study is grounded

on the propositions of the communibiology paradigm (Beatty & McCroskey, 2001) and neurophysiology perspective (Sherry & Weber, 2005), which we apply to video game effects. These propositions suggest that psychological processes depend on brain activity and that brain activity precedes psychological experience. Hence, common operationalizations of aggressiveness are substituted with neural brain activity patterns that are supposed to reflect aggressive cognitions and affects.

What does neuroscience know about aggression and its neural representation? Davidson, Putnam, and Larson (2000) introduced functional imaging to localize precursors of aggression. Their findings suggest that a circuit consisting of the orbital frontal cortex (OFC), amygdala, and anterior cingulate cortex (ACC) displays activation variations that Davidson et al. (ebd.) consider a neural correlate of aggression. Support for this hypothesis can be derived from studies in forensic psychology demonstrating that individuals with tendencies towards criminal and aggressive behavior show altered activity in the neural circuitry of emotion regulation (Bierbaumer et al., 2005; Schneider et al., 2002; Veit et al., 2002). In particular, the neural activity of patients studied was characterized by reduced involvement of affective networks.

The OFC, ACC, and amygdala belong to the emotional network, which is responsible for emotions (the bodily side of affection) and feelings (the representation of emotion in the mind; Damasio, 2003). The ACC in particular seems to play a major role in the evolution of feelings, by linking cognitive and affective processing. The ACC is part of the medial frontal cortex, which extends from premotor to basal structures around the corpus callosum. As such it has been considered an interface between cognition and emotion (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001). It is divided into the dorsal cognitive part (dACC) and the rostral affective part (rACC; Bush, Luu, & Posner, 2000). During cognitive interference tasks, increased brain activation was observed in the dACC in most studies, whereas the rACC was found activated if affective information was involved. Pietrini, Guazzelli, Basso, Jaffe, and Grafman (2000) used positron emission tomography (PET) to study neural correlates of imagined aggressive behavior. Their findings indicated that imagined scenarios involving aggressive behavior are associated with significant activity reductions in the ventromedial prefrontal cortex structure. Kimbrell et al. (1999) found comparable results. Based on functional magnetic resonance imaging (fMRI) analysis, Sterzer, Stadler, Krebs, Kleinschmidt, and Poustka (2003) found reduced ACC activity in adolescents with antisocial conduct and aggressive behavior disorders. Sterzer et al. (ebd.) concluded that differential anterior cingulate cortex activation may link emotional processing and aggressive reactions. Most recently, Mathews et al. (2005) reported similar brain activity patterns in adolescents exposed to higher levels of violent content in TV and video games in the 12 months preceding the experiment. They observed reduced activity in the frontal lobe structures of research participants who had higher exposure to

virtual violence. Moreover, Mathews et al. (ebd.) report that after high violent media exposure, adolescents with no psychopathological history demonstrated the same reduced brain activity in the frontal lobe as a control group of adolescents diagnosed with aggressive/disruptive behavior disorders.

Based on these findings, it seems that dynamic activity changes in the frontal lobe structure indicate aggressive cognition and affects. Specifically, aggression seems to be associated with activity variations in the dACC in combination with activity reductions in the rACC and the amygdala. If virtual violence does, in fact, result in aggression, the aforementioned neural patterns should be observable in individuals playing violent video games. Because neural activity is always an immediate response to a stimulus, any empirical evidence of an association of virtual violence with neural activity must be causal by nature. Thus, our research hypothesis is:

Involvement in virtual violence during video game play causes activity variation in the dorsal anterior cingulate cortex, reduced activity in the rostral anterior cingulate cortex, and reduced activity in the amygdala.

To date, previous findings do not justify a hypothesis on the direction of activity variations in the dACC. In addition, the a priori definition of a neural effect size can not be achieved due to the exploratory status of this approach. Therefore, effect direction in the dACC and neural effect sizes need to be determined a posteriori.

METHOD

Participants

Research participant recruitment ads were posted at the University of Tübingen, Germany and in local video game and computer stores. Inclusion criteria were age between 18 and 26 years, a minimum weekly video game playing time of 5 hr, and right-handedness. We excluded individuals with contraindication against magnetic resonance investigations; acute disorders; or anamnesis of neurological, psychiatric, or ophthalmologic history from the study.

Thirteen German male volunteers (age range 18–26, *Mdn* = 23) participated in the study. On average, participants played video games for 15.1 hr (\pm 9.0 hr) per week and started playing video games at the (*Mdn*) age of 12.

Experimental Procedure

Participants played the “Mature” rated first-person-shooter game *Tactical Ops: Assault on Terror* (Infogrames Europe, Villeurbanne, France; U.S. edition;

<http://www.tactical-ops.de/>) for five rounds, 12 min per round (an average of 60 min total), while in an fMRI scanner. Brain activity was measured throughout game play. Physiological measures (skin conductance response, heart rate, peripheral pulse amplitude) were also taken. These data, as well as audio data from the game, were recorded on an ADC DT9801 device (Data Translation, Inc., Marlboro, MA). In addition, we recorded the video display of the game play on a Super-VHS videotape and synchronized the video display with the fMRI signal.

Content Analysis

Game play recordings were content analyzed with a novel frame-by-frame method, which assessed whether virtual violence was involved at any moment during play. We designed an inductive, time-based content analysis with two independent coders (male graduate students at the University of Southern California) and one supervisor. The coders rated the extent of a participant's violent interactions within specific virtual game environments, based on recorded and digitized videos of the individual's game play.

The coding scheme consisted of five categories defining the ordinal play phases as (1) passive/dead, no interactions; (2) active/safe, no imminent danger/no violent interactions; (3) active/potential danger occurs, violent interactions expected; (4) active/under attack, some violent interactions; and (5) active/fighting and killing, many violent interactions. The play phases differ in the intensity of violent interactions from 1 (*no interactions*) to 5 (*many violent interactions*). In addition, 19 types of transitions between the 5 main play phases were defined. The transitions reflected how a participant decided to interact in a certain situation to switch between the play phases. The coding scheme also provided special codes to rate cases in which the research participants exhibited violent behavior without a need to score (e.g., committing suicide; shooting at team members, hostages, or inventory; shooting at already killed opponents again; looting).

The coders received about 16 hr coding training, in which they discussed the different playing phases with experienced video game players and learned to rate events and violent interactions according to the coding scheme. Training was based on one participant's recorded video, not used in the study. The entirety of each participant's recorded game play was analyzed with every single frame (25 frames/sec) as the unit of analysis. The coders coded the beginning and end of every main category and every transition with a precision of 0.1 sec. The entire coding procedure took about 120 hr per coder and yielded an overall intercoder reliability of 0.85. Inconsistent ratings were discussed with the supervisor and corrected according to the coding scheme (for a detailed description of the content analysis see Weber, Behr, Ritterfeld, & Mathiak, 2005).

fMRI Procedure

Simplified, fMRI defines a technology in which the amount of blood flow in circumscribed brain areas is calculated on the basis of magnetic responses of hemoglobin. For the calculations, fMRI utilizes primarily the blood-oxygenation level dependent (BOLD) effect. This effect can be measured by magnetic resonance tomography (for a virtual tour to explore fMRI scanning, see <http://www.cfn.upenn.edu/virtscan.htm>). Blood flow in brain areas is closely correlated with neural activity in those areas (Logothetis & Pfeuffer, 2004). Changes (increases or decreases) in blood flow indicate that certain neural regions are active or inactive while processing a specific mental task.

For this study, the fMRI procedure was conducted at a high magnetic field strength of 3 Tesla (Magnetom TRIO, Siemens, Erlangen, Germany). Multi-echo single-shot echo-planar imaging (echo times = 23, 40, and 62 ms) with dynamic distortion correction (Weiskopf, Klose, Birbaumer, & Mathiak, 2005) and dephasing compensation (Mathiak, Hertrich, Grodd, & Ackermann, 2004) reduced artifacts and increased sensitivity. Whole brain coverage with 24 interleaved slices (repetition time = 2.25 sec) and spatio-temporal oversampling reconstruction resulted in an apparent time resolution of 1.13 sec after spatial filtering. For reference, we acquired anatomical data of each participant before the functional sessions (T1-weighted 3D-MPRAGE, 256 × 224 × 160 matrix with 1mm isotropic voxels).

Measures of Arousal and Subjective Experience

To control for arousal and to monitor the research participants during game play, physiological measures were taken throughout the scanning procedure. These measures combined peripheral pulse (pulseoxymetry) and skin conductance, derived from the left foot. In addition, at the conclusion of fMRI scans, participants were asked to fill in a questionnaire about their experience of the procedure, using Likert scales that ranged from 1 (*totally disagree*) to 9 (*totally agree*). The questionnaire included four items on compliance and four items on immersion during the game play experience (which will be explained in the following).

Analytical fMRI Paradigm

To evaluate and interpret the results of this study correctly, it is important to differentiate between two distinct analytical fMRI paradigms. The first is called *statistical parametric mapping* (SPM) and the second *region of interest* (ROI) analysis. SPM explores brain areas that, based on a stimulus, show a statistically significant activation or deactivation. For the measurements, the brain is segmented into about

30,000 voxels. Each voxel serves as a dependent variable in a general linear model with the stimulus (here: virtual violence) folded with the expected hemodynamic response function as independent variable. After correction for multiple testing, significant models indicate activated or deactivated brain areas. This approach can be used for exploration. Mathiak and Weber (2005) provided a detailed analysis and discussion of the study's SPM procedures.

The ROI analyses that we report here rely on theoretically or empirically derived a priori assumptions about the brain areas and networks involved. Thus, the ROIs can be localized in anatomic structures, and hypotheses about activity patterns may be deduced. For the ROI analyses presented in this article, we chose the following coordinates within the Montreal Neurological Institute system (Collins, 1994): $(x,y,z) = (\pm 6, -13, +40)$ mm for the dACC, $(\pm 6, +38, -4)$ mm for the rACC, and $(\pm 24, +6, -20)$ mm for the amygdala (coordinates with negative/positive x -value are localized within the left/right hemisphere). For comparisons between the different violent activity conditions, correlational analysis, and effect estimation, we extracted time-series data from the normalized and smoothed functional images.

RESULTS

Participants' Experience of Procedure

Questions on participants' experience of the procedure indicated high means for compliance¹: (a) The study was fun ($M = 7.4, SD = 1.6$); (b) The study was interesting ($M = 7.9, SD = 1.4$); (c) I felt bad during the measurement ($M = 2.2, SD = 1.5$); (d) I would participate in a similar study again ($M = 7.6, SD = 2.1$). Immersion in the game play was rated above scale mean: (e) I felt like I was acting in the environment rather than controlling a game ($M = 4.7, SD = 2.4$); (f) I felt present in the game environment ($M = 5.7, SD = 2.3$); (g) From time to time I was not aware of my real environment ($M = 5.6, SD = 3.5$); (h) The game required all of my attention ($M = 5.6, SD = 2.3$). Overall, the participants felt more or less comfortable playing the game in the scanner and could play the game like they do in their normal environment.

Neural Effects of Violent Game Play

The research hypothesis postulated in this study has two aspects. The first aspect contends an association between virtual violence in a violent video game and brain activity. The second aspect suggests a causal relation between the two. Because the focus is on time series of coordinates (ROIs) for the dACC, rACC, and amygdala defined a priori, that is, not on SPM analyses, we tested both aspects through

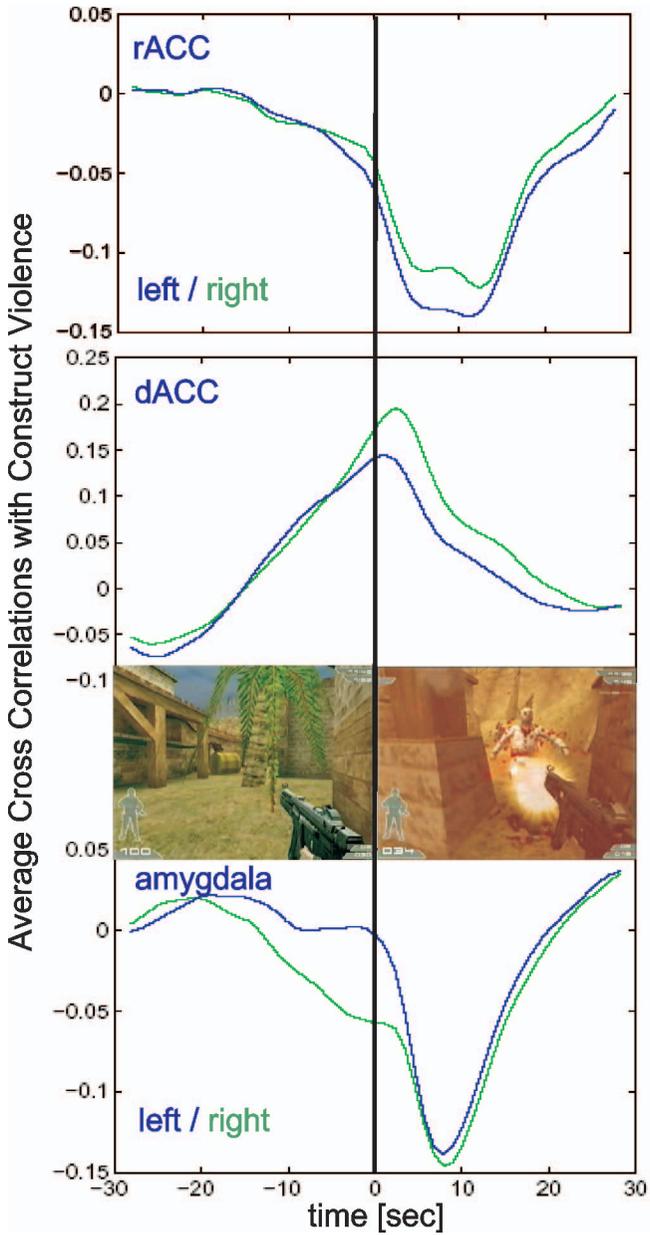


FIGURE 1 Average cross-correlations of violence construct with rACC, dACC, and amygdala.

cross-correlation analyses. SPM analyses of the whole brain with a primarily explorative focus are discussed elsewhere (Mathiak & Weber, 2005).

Prior to the analyses, we prefiltered and adjusted the dACC, rACC, and amygdala time-series by arousal (heart rate). All time series were stationary in mean and variance. Figure 1 shows averaged cross-correlation functions (based on all 13 participants).

Over time, rACC and amygdala correlate negatively and dACC positively with the violence construct, which indicates a down-regulation of rACC and amygdala and an up-regulation of dACC after play phases involving violence. Due to the fMRI-related hemodynamic response, the observed effects are delayed by about 6 sec. Effects occur exclusively in the immediate vicinity of violent game play and predominantly follow virtual violent activities in time. This finding provides evidence for a causal relation between the violence stimulus and the neural activity pattern.

Cross-correlation functions with lags up to ± 600 sec and higher reveal that, outside a window of about -20 to $+20$ lags (sec), no effects are visible. Smaller effects shortly before violent activities indicate effects in expectation of active fighting phases (the experienced players can anticipate upcoming fight episodes). The observed cross-correlations are significant. However, we prefer not to argue with the concept of significance in this article, simply because of the extended length of time series. In detail, between 3000 and 3600 time points were measured per participant. Therefore, even cross correlations outside the previously mentioned window and below practical significance (e.g., cross-correlations of 0.01) are of statistical significance.

The observed patterns were stable across 11 of the 13 research participants cooperating in the study. Maximal average cross-correlations are $+0.17$ for the dACC, -0.13 for the rACC, and -0.14 for the amygdala. In 1 of the 13 participants, noticeably higher correlations of $+0.19$ (dACC), -0.54 (rACC), and $+0.33$ (amygdala) with involvement in virtual violence were observed. In 2 other participants, the opposite pattern occurred, that is, signal decrease in dACC and increases in the rACC and the amygdala. The consistency within the research participants suggests that the intersubject variability is not caused by variable signal quality only. However, the small sample size does not allow for conclusive statistics on the nature of these deviances.

Further, Figure 1 shows that the responses within the cognitive part of ACC precede the affective suppression and partially even anticipate violent interactions. This pattern suggests active suppression of affective processing in favor of the cognitive operation. Simple connectivity analyses in Figure 2 by means of cross-correlation functions across the entire time course of the game demonstrate inhibitory actions of the cognitive dACC on affective rACC and amygdala after a delay of 1 to 20 sec and of the affective rACC on the amygdala after a delay of about 10 sec.

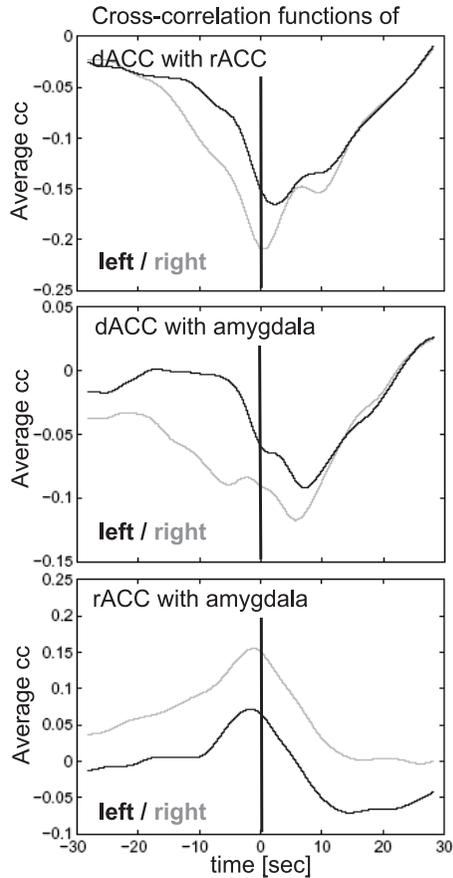


FIGURE 2 Average cross-correlations between dACC, rACC, and amygdala.

Effect Sizes

The BOLD response of fMRI does not allow for absolute quantification. However, the relative signal change expressed as a percent value is frequently used as a semiquantitative measure for effect size. As elaborated, ROIs were defined a priori and localized in the neural anatomy. Signal changes in a priori defined areas will not reflect maximal signal changes but can be considered as average change within the target structure across the participants. Figure 3 displays average percentage BOLD signal changes in the dACC, rACC, and in the amygdala for the two nonviolent (1, 2) and all violent phases of game play (3, 4, 5). Again, the expected neural activity pattern is prevalent, i.e. an up-regulation of the dACC, and a down-regulation of the rACC and the amygdala in conditions that involve virtual violence. The

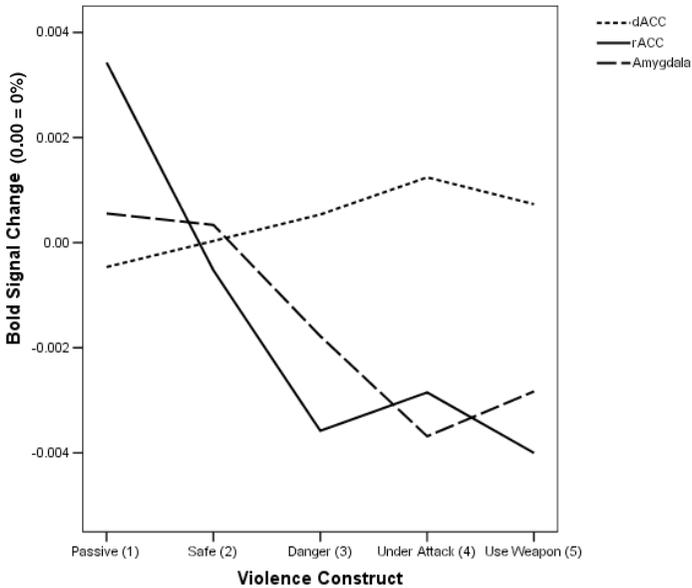


FIGURE 3 Blood-oxygenation level dependent signal changes at dACC, rACC, and amygdala in percent (0.00 = 0%).

range (maximum–minimum) of observed BOLD signal changes was of the order of 0.17% for the dACC, 0.74% for the rACC, and 0.42% for the amygdala, and did not change significantly after correction for arousal. For this structure, this is a rather large effect (see Discussion). The translation into Cohen's (1988) effect size measures results in $d = 0.3$ (dACC), $d = 0.92$ (rACC), and $d = 0.70$ (amygdala). According to Dunlop, Cortina, Vaslow, and Burke (1996) original standard deviations were used to calculate the effect sizes rather than paired t -test value or the within-subject's F value which leads to a slight underestimation of the actual effect sizes.

The ANOVA procedure for repeated measurement designs yield significant results for the dACC (Wilk's $\Lambda = 0.33$, $F = 4.59$, $p < .027$, $\eta^2 = 0.67$), rACC (Wilk's $\Lambda = 0.19$, $F = 9.55$, $p < .003$, $\eta^2 = 0.81$), and amygdala (Wilk's $\Lambda = 0.28$, $F = 5.75$, $p < .014$, $\eta^2 = 0.72$). Tests for linear trends were significant in the three ROIs (dACC: $F = 8.28$, $p < .014$; rACC: $F = 17.97$, $p < .001$; amygdala: $F = 30.02$, $p < .001$), but not for higher order trends.

Again, with the exception of two research participants who showed the opposite pattern, individual effect sizes are remarkable. Single participants reached effect sizes of +0.56% BOLD signal change for the dACC, -1.56% for the rACC, and -0.81% for the amygdala under violent conditions.

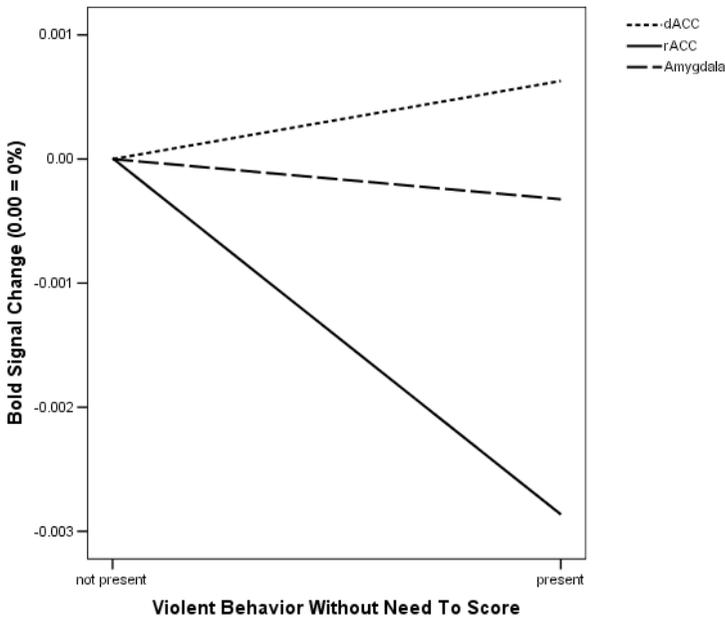


FIGURE 4 Blood-oxygenation level dependent signal changes at dACC, rACC, and amygdala during violent behavior without need to score in percent (0.00 = 0%).

This article contains no in-depth analyses regarding specific behavior within the virtual environment. However, initial analysis on violent behavior unrelated to in-game goals (e.g., shooting at team members, hostages, or inventory; shooting dead opponents again) reveals different patterns of neural activity. Figure 4 contrasts the aforementioned behavior with all other behavior to win the game. When violence was applied to situations where no opponent was present and, therefore, no threat to the participant's avatar existed, the otherwise noted down-regulation of the amygdala's activity could not be observed.

DISCUSSION

Drawing from the constraints of previous scientific efforts that have tried to capture the antisocial effects of violent video game playing, we combined methodologies from communication (micro content analysis of game play) and neuroscience (micro fMRI analysis of neural patterns during game play). In this study, we applied a within-subject design in which brain activity patterns were matched with the game play, which allowed for distinguishing between actions involving virtual violence and actions in which virtual violence was absent. Consequently, the de-

sign enabled us to avoid game play as a confounding variable, which would be the case if different games (nonviolent vs. violent game) were compared. In addition, time series secured reliability of findings because stimulus and response associations could be observed multiple times.

In accordance with previous findings on the neural correlates of aggression, we found an active suppression of affective areas such as the rACC and the amygdala, as well as increased activity in the dACC. Our results indicate that virtual violence in video game playing results in those neural patterns that are considered characteristic for aggressive cognition and behavior. It is possible that the neural patterns of the ACC represent a mechanism to suppress positive emotions (such as empathy) so that the individual can play the game successfully.

However, one might argue that the neural activity observed does not reflect aggressiveness due to violent action, but rather fear elicited in a virtual environment endangering the player's virtual life. Consistent with this line of arguing are meta-analytic findings by Phan, Wager, Taylor, and Liberzon (2002), which attributed fear reactions to the amygdala. BOLD changes in the amygdala observed in our study may, in fact, indicate fear and thus support the claims of gamers who justify their virtual aggressiveness in violent games as necessary for their virtual survival. However, the data obtained also contain information on virtual violence that is not justified by game logic, namely violence without being endangered. First results indicate that in such "fear free" environments, almost no BOLD changes in the amygdala occur, but that the suppression of the rACC in particular is heavily pronounced. We may conclude that virtual violent interactions involving a defensive component, and ultimately fear of losing a virtual life, elicit neural patterns that are distinguishable from the pure lust for aggressive behavior.

One of the advantages of this study lies in the neurobiological causal link found: cross-correlation analyses demonstrate the consistency and the exclusiveness of responses—the indicative brain activity pattern results almost after every virtual violent action, and only after those. The unequivocal relation between stimulus and response leaves no room for alternative explanation for the observed activity patterns. Thus, the research hypothesis claiming that violent interactions in video games cause the specified brain activity patterns can be confirmed. However, the limitation to the main contrast violence and to three ROIs does not allow a description of the entire neural network involved in the complex task of playing violent video games. In particular, the role of the OFC and executive control areas must be specified further.

The effect direction in the dACC and the sizes of effect were not defined a priori. Based on our data, we posit increased activity in the dACC shortly prior and during virtual violent interactions. Most interestingly, the reported effect sizes (% BOLD signal change) are larger than usually expected in comparable fMRI studies with a priori defined and anatomically localized ROIs. For instance, in a previous biofeedback study, signal changes at the level of the rACC were targeted for maxi-

mization (Weiskopf et al., 2003). Even after extensive training, the participants achieved a median signal change of only 0.13%. Obviously, we were able to differentiate systematic variance from error to a large extent and the activity pattern evoked through virtual violence are pronounced and not heavily moderated by third factors. The coding scheme of the applied content analysis and its underlying violence construct seems to be highly relevant for neural activity in the focused a priori defined ROIs. Furthermore, the magnitude of the obtained signals results from virtual immersion, in which information processing is constrained by the aspect of virtuality. Virtual environments may lead to a higher and more complex involvement of neuronal structures compared to standard stimulation paradigms. Given that high immersion in virtual environments suspends sensory processing from nonvirtual physical stimuli to some extent (Ritterfeld & Hünnerkopf, in press), the virtual experience of video game play controls external distractions even more. Support for this assumption is provided by the endurance of the unsavory scanning procedure of all 13 participants. Not only did they spend an unheard average of about 60 min in the scanner without interruption or complaints; most of the participants even deliberately expressed their will to cooperate in similar studies again. Consequently, the observed high immersion in game play obviously minimized disturbance of the undoubtedly unnatural context of data collection (game play does usually not happen in a scanner) which, in principle, would question ecological validity of the study.

This study presents a twofold novelty—first, in applying neuroscience methods to communication research, and second, using a novel analytical fMRI design for hypothesis testing rather than exploration. In the following, we address the more fundamental question of whether neuroscience methodologies can contribute to the understanding of media effects.

Though neuroscience sometimes seems to exude an aura of objectivity, questions of reliability and validity of the results still need to be addressed. Researchers measure the reliability of independent data (media content) in a content analysis by comparing how multiple coders code the same material. Reliability of dependent measures (brain activity) in fMRI procedures is typically approximated in individual error corrections for head and body movements. However, the necessary data normalization and transformation (such as standardizing the individual participant's brain to the Montreal Standard Brain), spatial filtering and smoothing, hemodynamic response folding, and the selection of brain areas assumed to be representative for the ROI under consideration, make fMRI a less reliable measure than often assumed. Thus, the resulting brain patterns serve as the best possible measures for stimulus response, but they are not error free.

The need to create one average brain does usually result in exclusion of some of the participants (see also Murray et al., this issue). In our case, data from 2 of 13 participants did not comply with the expected patterns, which accounts for approximately 15% of the sample size. These individual differences deserve—without doubt—closer attention in future studies. Hostility traits that are prone to utilize

aggression for mood management (Arsenio, Gold, & Adams, 2004) should especially be investigated more closely.

Individual differences impact not only reliability but validity as well. The selected sample included only young men who were experienced game players, to reduce moderating effects and usability issues during data collection. Moreover, it was expected that video game experts are less reluctant to use virtual violence than novices would be. However, we do not know whether novices would show similar effects. Smaller effects should be observed in novices compared to experts if we apply the hypothesis of pronounced violent script salience to experienced compared to nonexperienced players. Alternatively, the desensitization hypothesis suggests larger effects in novices if experts already have decreased sensitivity towards virtual violence. Because this study revealed effect sizes in experts that are already much higher than those fMRI studies with a similar analytical design usually report, we assume the desensitization hypothesis is less likely.

Our main finding suggests parallel neural patterns between highly immersive virtual environments and real experiences. Here, virtual violence is able to activate the same brain patterns that are present when people have aggressive thoughts or a pronounced tendency to act aggressively. But even if the neural patterns in real life and virtual experiences are identical, the experiences may not be. Thus, the equivalence of subjective experiences and brain activity is questionable. According to identity theory, subjective experiences (cognitions, feelings) are in fact identical to brain activity (Pauen & Stephan, 2002). Under this assumption, identity theory declines the concept of dualism of body and mind, giving consciousness a material basis (Heidelberger, 2004). However, critics of identity theory have long pointed to pain as an example of neural patterns that do not tell us much about the subjective experience (e.g., Searl, 1980). Thus, even if it is possible to associate neural activity with affective states, parity of the two qualities is not necessarily implied. Therefore, virtual experiences may look the same as nonvirtual experiences, but feel different from them.

For example, one might argue that the observed effects do not allow us to distinguish between pretense (play) and nonpretense experiences in the virtual environment. Pretense would be defined as an experience in which the player is constantly aware of the role-playing aspect of the game. Without question, game players do not consider game play a real² life event—ultimately, they are not shooting; they are playing a game. In fact, Gallese and Goldman (1998) defined the play as a highly immersive state of pretense and Rothmund, Schreier, and Groeben (2001) considered the ability to distinguish between fictionality and reality a core aspect of media literacy that, according to Rakoczy, Tomasello, and Striano (2004), may even be seen in children 3 years of age. In concordance with Rothmund et al. (2001), media experts, who are by definition more media literate than novices, should express a higher ability for pretense awareness. In contrast, Ritterfeld and Hünnerkopf (in press) argued that the conscious awareness of fictionality (a term used largely in the context of humanities) or virtuality (a term mostly applied to

new media) is metacognitive by nature and may be suspended during the use of highly immersive media, such as video games. To our knowledge, there is no indication yet that pretense and nonpretense experiences can be distinguished on the basis of neurological data. Subjective measures, on the other hand, can only be applied post hoc and are therefore lacking in validity (Kahnemann, 2003). Thus, an empirically sound answer to the status of pretense in virtual play is still beyond the scope of this study. We can only speculate whether the sense of pretense is a result of context variables and the absence of unparalleled experiences. Following this thread, pretense metacognition would be more suppressed the more realistic the simulation is.

The parallelism in neural activity evoked through virtual and physical stimulation that our findings suggest is also supported by a few other studies. For example, virtual spatial navigation results in neural patterns comparable to physical spatial navigation (Mraz, Hong, Quintin, Zakzanis, & Graham, 2003). One might argue that spatial navigation is a purely cognitive function not necessarily involving affect and emotion. But more recently, Morris, Pelphey, and McCarthy (2004) observed neural activity typical of social perception in virtual encounters. Undoubtedly, social perception is not exclusively cognitive but involves affective responses as well. Consistent with this line of argumentation, J. Blascovitch (personal communication, June 8, 2005) recently reported data indicating that automatic processing, such as fear in virtual environments, is not controlled through the awareness of virtuality: Participants above a virtual cliff behaved as though a physical one existed. Consequently, being immersed in a virtual environment might result in experiences that are—from a subjective point of view—indistinguishable from experiences in physical environments. In the case of virtual violence, however, one might argue that the pain of being killed by a virtual opponent is missing and thus, simulation of physical environments is significantly constrained. But this does not imply that being under virtual attack and virtually defending oneself does not result in the same fear and fight reactions that one would experience in actual combat. In sum, virtual and nonvirtual experiences may be identical in relevant mental dimensions (such as aggressive states), but surely not in all aspects (such as pain).

In other words, the neural equivalence of virtual and nonvirtual encounters that some studies have observed indicates equivalence of cognition or emotions, but the subjective experience, the feelings, might be different. Correspondingly, feelings might be the same (e.g., aggression in violent games), though the cognitive (e.g., a virtual cliff does look different from a cliff in the Grand Canyon) and emotional experience is different (i.e., nobody is actually killing me in a video game).

Taken together, the ancient effort of philosophy, more recently aided by psychology, and even more recently biology including neurosciences, in trying to solve the mind–body problem and the implied relation of mental processes (metacognition, cognition, emotions, and feelings) and material neural activity is supplemented by equally challenging question regarding the equivalence of virtual and nonvirtual experiences. This question goes far beyond video game play; it also

involves such nonphysical states as dreams, hallucinations, and imagination. We are convinced that the interdisciplinary endeavor will eventually provide answers. However, there is still a long way to go.

In the meantime, the results of this study should give reason for concern. Though the design supposedly only illustrates short-term effects, relevance of repeated activation should not be underestimated. Video games especially are assumed to provide all the necessary tools for an optimized learning environment (Buckley & Anderson, 2006). Moreover, Koepp et al. (1998) showed that video games activate the neural reward system quite effectively. They reported that dopamine is released during playing video games and dopamine plays a key factor for learning and reinforcement of behavior (see also Robbins & Everitt, 1992). Most recently, Kirsh, Olczak, and Mounts (2005) found that violent video game playing results in an attentional bias toward negatively valenced stimuli.

Therefore, the strong effect sizes in our sample of experienced game players may well be a consequence of repeated elicitation of neural activity associated with aggression and, thus, already reflect a long-term impact.

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NOTES

¹Translated from German into English, 1 (*totally disagree*) to 9 (*totally agree*).

²The popular distinction of “real” versus “virtual” is problematic. If a person is immersed in a sad narrative, s/he might be crying real tears. Thus, distinguishing between “physical” and “virtual” environments is more precise.

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