



Aberrant social and cerebral responding in a competitive reaction time paradigm in criminal psychopaths

Ralf Veit^{a,*}, Martin Lotze^{b,1}, Sven Sewing^a, Heiner Misenhardt^c, Tilman Gaber^{a,d}, Niels Birbaumer^{a,e}

^a Institute of Medical Psychology and Behavioral Neurobiology, University of Tübingen, Germany

^b Functional Imaging Unit, Center for Diagnostic Radiology and Neuroradiology, University of Greifswald, Germany

^c Centre of Psychiatry, Forensic Department, Bad Schussenried, Germany

^d Max Planck Institute for Biological Cybernetics, Tübingen, Germany

^e Ospedale San Camillo, Istituto di Ricovero e Cura a Carattere Scientifico, Venezia, Italy

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ABSTRACT

In a previous study (Lotze et al., 2007) we described dorsal medial prefrontal cortex (mPFC) activation in healthy subjects during retaliation in a competitive reaction time task. Interestingly, the less callous the subjects were, the more they responded with ventral mPFC-activation when watching the opponent suffering. In this study we used this paradigm to investigate behavioral and neural responding of ten criminal psychopathic individuals from a forensic psychiatric institution. In contrast to healthy subjects, who show reactive aggressive behavior of inflicting punishment with increasing intensity after experiencing an increasing amount of punishment from a yoked opponent, psychopathic participants did not react with comparable retaliation. However, when psychopaths punished with a high amount they showed increased activation in the hypothalamus, the lateral prefrontal cortex, the posterior cingulate cortex and the amygdala. The trait “physical aggression” showed a positive correlation with hypothalamic activation. Medial prefrontal areas, associated with emotional control and conflict management in healthy subjects performing this paradigm, were inactive in psychopathic subjects during retaliation. When psychopaths observed the yoked opponent being punished they showed increased activation in the dorsal and ventral medial prefrontal cortex, which was positively associated by impulsivity and antisocial behavior of Hare’s psychopathy construct. This finding supports the notion that reactive aggression is more related to antisocial behavior and anger management than with emotional and interpersonal characteristics of psychopathy and suggests that two separate brain activation patterns seem to account for these two behavioral dispositions.

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Introduction

The competitive reaction time paradigm (Taylor, 1967) is an excellent methodology employed in the laboratory to study direct physical aggression. This task is a widely used valid measure of aggressive behavior, appropriate to induce reactive aggression in a laboratory setting (Giancola and Zeichner, 1995). In a previous study we used the Taylor paradigm (Lotze et al., 2007) and demonstrated that healthy subjects show increased activity in the medial prefrontal cortex (mPFC) when punishing after being provoked. We interpreted this activation as associated with guilt during performing an aggressive act. Interestingly, the activity of the dorsal part of the mPFC was correlated positively with the strength of the selected

aversive stimulus during retaliation, whereas the ventral mPFC was activated independent of the applied stimulus strength. The ventral part was active while observing the suffering opponent. Subjects with higher total psychopathy scores based on the Levenson self report scale (LSRS; Levenson et al., 1995) exhibited less ventral mPFC activation.

In light of the above, we were interested in the behavior and functional activation of criminal psychopaths during the performance of this reactive aggression paradigm. These subjects have severe problems in emotional learning and show a failure of differential emotionally conditioned responses in the limbic-prefrontal circuit during Pavlovian classical aversive conditioning (Veit et al., 2002; Birbaumer et al., 2005), and impairment in emotion processing and empathy (Mueller et al., 2003). This callous unconcern for feelings of others is associated with repeated violation of the rights of others as well as a disregard of social norms.

It has been demonstrated that damage to orbital and ventrolateral frontal cortex is related to a heightened risk of aggression (Blair, 2006) and there are many studies showing a strong association between psychopathy and engagement in violent and aggressive

* Corresponding author. Institut für Medizinische Psychologie und Verhaltensneurobiologie, Eberhard-Karls-Universität Tübingen, Gartenstraße 29; D-72074 Tübingen, Germany. Fax: +49 7071 295956.

E-mail address: ralf.veit@uni-tuebingen.de (R. Veit).

¹ These authors contributed equally to the work.

behavior (Woodworth and Porter, 2002). Overall, two forms of aggression are distinguished: reactive aggression elicited in response to frustration or provocation and instrumental goal-directed aggression. These types of aggression are mediated by different neural systems. It has been shown that damage of the medial prefrontal lobe results in disinhibition of reactive aggressive behavior as can be found in “acquired sociopathy” (Blair and Cipolotti, 2000). Instrumental aggression, however, is a core feature of developmental psychopathy probably related to a dysfunctional socialization process.

Reactive aggression is mediated by medial amygdala, the medial hypothalamus, and the dorsal half of the periaqueductal gray (PAG) (Gregg and Siegel, 2001). The amygdala and orbital frontal cortex are parts of a neural circuitry involved in the modulation of reactive aggression and fear. It has been found that lower cerebral blood flow during rest in the lateral orbital frontal cortex (BA 47) is associated with a history of reactive aggression in patients with antisocial personality disorder (Goyer et al., 1994). Soderstrom et al. (2002) reported that reduced prefrontal functioning is more associated with reactive than instrumental aggression. However, little is known about the circuit responsible for instrumental aggression. This type of aggression is assumed to be regulated by cortical “cognitive” systems and less dependent on the “emotional” hypothalamic and limbic systems (Nelson and Trainor, 2007).

Most imaging studies investigating violence and aggression at a behavioral level used structural neuroimaging techniques. Only a few studies using functional neuroimaging exist. Blair et al. (1999) demonstrated that the paracingulate cortex was activated when subjects viewed aggressive facial expressions using fMRI. Pietrini et al. (2000) conducted a PET study using script driven imagery and found a pronounced deactivation of the ventral mPFC during evoked aggressive emotions.

The present study focused on the question, which cerebral areas associated with aggression and aggression control are active in psychiatric inmates with psychopathy during retaliation and opponent observing. In these patients we expected a deficit in mPFC activation. For retaliation we focused on the dorsal mPFC, associated with decreased emotion control and feelings of guilt, while for opponent observation we expected decreased ventral mPFC-activity, associated with the known deficit in empathic responding.

Materials and methods

Participants

10 male psychopathic patients (mean age: 31.0 years; SD 5.8 years) from two forensic psychiatric institutions in Germany participated in the study. Psychopathy in the sample was diagnosed by experienced clinicians using the PCL:SV (Hart et al., 1995). The screening version of the PCL (PCL:SV, Hart et al., 1995) was developed to measure psychopathy in civic or forensic settings. The screening version consists of a 12-item scale on the basis of the PCL-R. The PCL:SV has comparable validity and reliability as the full PCL-R version. Psychopaths had a mean PCL:SV total score (score ranges from 0 to 24) of 16.11 (SD 3.62). This is slightly lower than the standard value (cutoff: 18) reported for U.S. populations of psychopaths, but in accordance with German and European norms (Cooke et al., 2004).

Most of the participants committed a wide range of criminal acts including homicide, rape, assault, burglary and more. Their criminal careers reached back to childhood. Each participant was accompanied by members of the forensic psychiatric department. In addition, we used the self report scale (LSRS) of Levenson et al. (1995) to compare our results with our previous study (Lotze et al., 2007) of non-clinical individuals, as well with other studies using self report ratings of psychopathic traits (Rilling et al., 2007). This scale was developed for assessing psychopathic characteristics or traits in non-institutionalized samples. The questionnaire contains 26 items in a 4-point scale

divided into two factors (primary and secondary psychopathy), similar to the factor 1 and factor 2 of the PCL-R (Hare, 1991). The psychopathic subjects had a total score in the LSRS of 62.50 (SD 9.98), in line with Brinkley et al. (2001), who investigated prison inmates and considered participants with scores of 58 and more as psychopathic. The Buss-Perry (BP) aggression questionnaire (Buss and Perry, 1992) was administered to assign different components of aggression (physical and verbal aggression, anger and hostility). This scale comprises 29 items using a 5-point scale from 1 (extremely uncharacteristic of me) to 5 (extremely characteristic of me). Participants were paid 200 € in arrangement with the psychiatric institutions. The study was approved by the Ethics Committee of the local Medical Faculty. Written informed consent was obtained according to the guidelines of the Declaration of Helsinki.

Experimental design

After providing informed consent, the experimenter briefly introduced the alleged participant to the opponent (a confederate of the lab), but no further social interaction was allowed. Thereafter, the participants were told that the opponent would participate in the reaction time competition in a different room. A modified version of the Taylor Aggression Paradigm (TAP) was used to measure physical aggression. Participants were told that if they react faster than the opponent they were allowed to administer physical punishment with a “shot” of a projectile to the opponent’s middle finger. If they would loose, because of the slower reaction times the opponent would be allowed to treat them with the same punishing stimulus. In order to provoke reactive aggression the intensity of the pain stimulus increased from an average of 2.33 points on a 5-point scale during the first run to an average of 3.92 during the last run of scanning. Details of stimulus design and the time course of the trials are given in the [Supplementary Material](#) and [Suppl. Figure 1](#). After the experiment, debriefing revealed that all participants believed to play against a real opponent.

Mechanical aversive stimuli

The mechanical aversive stimuli were applied using a plastic cylinder (diameter of 7 mm) moved by air pressure modulated by a pneumatic device (Dokoh-Pneu, Erlangen; velocities: 2 m/s to 20 m/s). The individual pain threshold was determined by increasing and decreasing pressure velocities of the plastic cylinder. Prior to the experiment the participants were asked to rate the stimulation intensity on a scale from one (only touch) over three (uncomfortable) to five (very unbearable) on a visual analogue scale. A rating of four was used as the individual pain threshold. This procedure was repeated until the pain threshold was consistently in a range of four.

fMRI acquisition and functional imaging procedure

All subjects were investigated with a 3 Tesla MR-scanner (Siemens Trio) using T2*-weighted echo-planar imaging (EPI, TE = 30 ms; TR = 1.5 s; 22 slices of 3 + 1 mm thickness in a tilted transversal orientation; matrix size 64 × 64) and a T1-anatomy (MP-Rage; 176 slices, 1 × 1 × 1 mm). In addition we acquired a static field map after the second functional session to unwarp geometrically distorted EPIs. Four sessions with 305 scans each were conducted.

The scanning comprised four runs with 20 trials each. Each trial started with a written cue followed by a visual signal prompting the subject to press a button with the right index finger as quickly as possible. After 2–4 s a smiley symbol indicated trial outcome (the corner of the mouth-up or down-symbolized “win” or “lost” trials). After losing trials, a visual five-point scale appeared for 3 s, showing the subject the intensity of the aversive stimulus he was about to receive (the subject saw an upwards moving bar on a scale pretending

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