



# Trait anger in relation to neural and behavioral correlates of response inhibition and error-processing



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## ABSTRACT

Effortful control is considered to be an important factor in explaining individual differences in trait anger. In the current study, we sought to investigate the relation between anger-primed effortful control (i.e., inhibitory control and error-processing) and trait anger using an affective Go/NoGo task. Individuals low (LTA;  $n = 45$ ) and high (HTA;  $n = 49$ ) on trait anger were selected for this study. Behavioral performance (accuracy) and Event-Related Potentials (ERPs; i.e., N2, P3, ERN, Pe) were compared between both groups. Contrary to our predictions, we found no group differences regarding inhibitory control. That is, HTA and LTA individuals made comparable numbers of commission errors on NoGo trials and no significant differences were found on the N2 and P3 amplitudes. With respect to error-processing, we found reduced Pe amplitudes following errors in HTA individuals as compared to LTA individuals, whereas the ERN amplitudes were comparable for both groups. These results indicate that high trait anger individuals show deficits in later stages of error-processing, which may explain the continuation of impulsive behaviors in HTA individuals despite their negative consequences.

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## 1. Introduction

Anger is a universal, and in general an adaptive emotion that people experience regularly (Averill, 1983; Kassirer et al., 1997). Some individuals, however, experience anger with such an excessive frequency and intensity that their anger starts to interfere with daily life and becomes dysfunctional. For instance, high trait anger (HTA) is associated with a diversity of adverse outcomes, including aggressive behavior (Bettencourt et al., 2006; Tafrate et al., 2002), domestic violence (Barbour et al., 1998), poor psychosocial functioning (McDermut et al., 2009), health problems (Smith et al., 2004), and interpersonal problems (Baron et al., 2006; Roberts et al., 2007). Given the dysfunctional nature of HTA, several studies have been conducted to gain a deeper understanding of the factors underlying this harmful personality trait (for reviews, see Owen, 2011; Wilkowski and Robinson, 2008b, 2010).

### 1.1. Trait anger and effortful control

Studies on individual differences in trait anger have identified reduced effortful control as one of the key cognitive factors involved

(Bresin and Robinson, 2013; Denson, 2015; Wilkowski and Robinson, 2008b, 2010). Effortful control (cf. cognitive control; Luna et al., 2004) refers to “the efficiency of executive attention — including the ability to inhibit a dominant response and/or to activate a subdominant response, to plan, and to detect errors” (Rothbart and Bates, 2006, p. 129). Effortful control is proposed to mitigate anger through several means. For example, it allows individuals to reinterpret hostile interpretations in favor of non-hostile interpretations, to disengage from hostile information, and to suppress angry expressions and aggressive behavior tendencies (Wilkowski and Robinson, 2010). According to the theory of Wilkowski and Robinson (2010) individuals low in trait anger (LTA) recruit effortful control resources in potential hostile situations, whereas individuals high in trait anger allow these resources to lay dormant. Importantly, effortful control is underlain by several separate but interacting subcomponents, including (but not restricted to) inhibitory control, and performance monitoring (Luna et al., 2004). The current study focuses on these two effortful control processes (i.e. response inhibition and performance monitoring; cf. error-processing) with respect to trait anger.

### 1.2. Trait anger and inhibitory control

The first process, inhibitory control, refers to the ability to suppress automatic, inappropriate, goal-incompatible behaviors (such as aggression)

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in favor of adaptive, deliberate, goal-oriented behaviors (Luijten et al., 2014; Luna et al., 2004). A series of studies conducted by Wilkowski et al. (Wilkowski et al., 2010; Wilkowski and Robinson, 2007, 2008a; Wilkowski, 2011) have shown that high trait anger individuals (HTA) demonstrate lower inhibitory control compared to low trait anger individuals (LTA), especially following exposure to hostile stimuli, such as hostile words and angry expressions, on behavioral tasks (e.g., Stop Signal Task and Flanker Task). To our knowledge, there is only one study that assessed inhibitory control in HTA and LTA individuals with the inclusion of recording electroencephalographic (EEG) activity. Including electroencephalographic measures (e.g., Event-Related Brain potentials, ERPs) is important as they may provide information about the underlying neurophysiological mechanisms and can additionally inform about the time course of response inhibition. In this particular study, Liu et al. (2014) found faster reaction times and a decreased P3 component using difference waveforms for HTA individuals compared to LTA individuals, which is consistent with the idea of impaired response inhibition in HTA individuals. Notably, however, no differences were found between LTA and HTA individuals on the N2 component and the P3 component on NoGo trials, which are both event-related potentials often proposed to reflect inhibitory control related changes in brain activity (Luijten et al., 2014). Since effortful control deficits seem to become especially apparent when anger is primed at the behavioral level (Wilkowski and Robinson, 2008b, 2010), perhaps inhibitory deficits on the neurophysiological level for NoGo trials remained undetected as the study of Liu and colleagues included an affectively neutral Go/NoGo task. That is, given that HTA are hypothesized to allow effortful control processes to lay dormant under potentially hostile situations, whereas LTA do not (Wilkowski and Robinson, 2010), inhibitory control deficits for HTA on the neurophysiological level should also become more apparent following the activation of hostile thoughts. To test this idea, we sought to compare anger-primed inhibitory control in HTA and LTA individuals using an affective Go/NoGo task with the inclusion of an electrophysiological measure.

### 1.3. Trait anger and error-processing

The second process, error-processing, refers to the optimization of goal-directed behaviors by monitoring and evaluating ongoing behavior (Luijten et al., 2014). Error-processing is vital in order to adapt behavior to situational demands and in order to optimize goal-directed behavior (Botvinick et al., 2001; Ridderinkhof et al., 2004). As such, deficits in error-processing are hypothesized to lead to the continuation of inappropriate behaviors (e.g., outward expressions of anger), despite their negative consequences (Luijten et al., 2011b). Indeed, several studies have found reduced error-processing, as indexed by both reduced ERN and Pe amplitudes, while using different tasks in populations with externalizing psychopathology that are characterized by impulse control problems. However, mixed results have been obtained as to what component drives these error-processing deficits. Whereas some studies found evidence for both reduced ERN and Pe amplitudes (Franken et al., 2007; Luijten et al., 2011b; Ruchow et al., 2005), other studies either found evidence for reduced ERN amplitudes both in community (Dikman and Allen, 2000; Hall et al., 2007; Heritage and Benning, 2012; Potts et al., 2006) and patient samples (Munro et al., 2007a; Vilà-Balló et al., 2014; von Borries et al., 2010) or for reduced Pe amplitudes in patient samples (Brazil et al., 2009; Chen et al., 2005; Maurer et al., 2015). Interestingly, studies that found evidence for reduced Pe amplitudes have been mostly conducted in impulsive violent patient samples, such as female incarcerated psychopaths (Maurer et al., 2015), violent offenders with psychopathy (Brazil et al., 2009), and impulsive-violent offenders (Chen et al., 2014). Error-processing deficits on the neurophysiological level in HTA individuals have not yet been investigated, even though there are

reasons to expect that impaired error-processing may be present in high trait anger individuals as well (Robinson et al., 2012). Hence, to our knowledge this would be the first study to compare error-processing in HTA and LTA individuals on the neurophysiological level.

### 1.4. The present study

In short, the goal of the present study was to investigate effortful control (i.e. inhibitory control and error-processing) in HTA and LTA individuals on both the behavioral level and the neurophysiological level, because there is a need to better understand the (neuro)cognitive processes associated with trait anger. To this end, we recorded EEG activity while participants high or low on trait anger performed an affective Go/NoGo task including both anger-related pictures and neutral pictures. Go/NoGo tasks are frequently used to measure inhibitory control as well as error-processing (Luijten et al., 2014). Regarding inhibitory control, event-related potential studies have repeatedly shown increased N2 amplitudes in the frontal region and increased P3 amplitudes in the frontocentral region on NoGo trials compared to Go trials (Falkenstein et al., 1999; Luijten et al., 2014; Rietdijk et al., 2014). With respect to error-processing, two error-related brain waves are of interest, namely the Error-Related Negativity (ERN), and the error positivity (Pe). The ERN arises shortly after making commission errors and reflects initial error detection (Bernstein et al., 1995). The Pe follows the ERN, and reflects the more conscious processing or awareness of the motivational significance of an error (Luijten et al., 2014; Overbeek et al., 2005). Based on the literature, we expected HTA individuals to demonstrate less inhibitory control as reflected in more commission errors on NoGo trials, especially for anger-primed NoGo trials. Moreover, we expected reduced N2 and P3 NoGo amplitudes for HTA compared to LTA individuals; with more pronounced effects on trials including anger-related pictures. Finally, we explored whether HTA individuals showed deficits in error-processing as reflected by reduced amplitudes on the ERN and the Pe compared to LTA individuals.

## 2. Methods and materials

### 2.1. Participants

In total, 133 undergraduate students were recruited to take part in our experiment in return for course credits or a financial compensation of 10 euros. From this group, 94 participants were selected as high or low in trait anger. HTA participants scored 21 or higher and LTA scored 15 or lower on the Dutch version of the Trait Anger Subscale (TAS) from the State Trait Anger Expression Inventory-2 (Hovens et al., 2014) respectively at the time of testing. The TAS has adequate psychometric qualities (Hovens et al., 2014; Spielberger, 1999) and similar cut-off criteria have been used in previous studies to select subclinical angry samples (e.g., Eckhardt and Cohen, 1997; van Honk et al., 2001). The resulting LTA group consisted of 45 participants (71.7% women;  $M$  age = 20.76,  $SD$  = 2.44) with a mean score of 13.13 ( $SD$  = 1.42) on the TAS. The HTA group (79.6% women;  $M$  age = 20.88,  $SD$  = 3.11) consisted of 49 participants with a mean score of 24.57 ( $SD$  = 3.23), reflecting high scores (above the 85th percentile) on the TAS. One participant was excluded from the analyses on the Go/NoGo task because he failed to comprehend the instructions. All participants had normal or corrected-to-normal vision. This study was conducted according to the rules of the Helsinki Declaration on informed consent and confidentiality (World Medical Association, 2001). All procedures were carried out with adequate understanding and written informed consent of the participants and with permission of the local ethics committee.

## 2.2. Instruments

The Dutch version of the State Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999; Dutch translation: Hovens et al., 2014) measures the experience, expression, and control of anger. The STAXI-2 contains 57 items that are scored on a 4-point Likert scale (1 = “almost never”, 2 = “sometimes”, 3 = “often”, 4 = “almost always”). The measure comprises six distinct scales, i.e. State Anger, Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control-In, and Anger Control-Out. In the current study we focused on the Trait Anger scale (T-Ang; range 10–40), which assesses the general tendency to experience frequent, intense, and longer episodes of anger. The STAXI-2 has adequate psychometric properties (Hovens et al., 2014; Spielberger, 1999).

## 2.3. Anger-related Go/NoGo task

An anger-related version of a Go/NoGo task, adapted from Luijten et al. (2011a), was used. In this task, participants viewed a series of pictures with an anger-related or neutral content. Each picture was displayed for 200 ms and had a blue or yellow frame. The frame color indicated whether a stimulus was a Go or a NoGo trial. Response assignments were randomized across participants. Each stimulus was followed by a black screen for a randomly varying duration between 1020 ms and 1220 ms. Participants were explicitly instructed to respond as fast and as accurately as possible to the pictures in Go trials by pressing a button with their index finger, and to withhold their response for the NoGo trials. The task consisted of 112 different anger-related pictures and 112 neutral pictures selected from the International Affective Picture System (IAPS; Lang et al., 2008) and Google Images. Anger-related pictures displayed scenes of angry and/or fighting people, whereas neutral pictures showed similar scenes of people engaged in non-angry behaviors. Both picture types were matched for color, gender and number of people displayed on the pictures. Each picture was presented four times, resulting in a total of 896 trials, of which 25% were NoGo and 75% were Go trials. The amount of NoGo trials was equally divided over picture categories (i.e., 112 NoGo trials and 336 Go trials). We used a blocked design with four blocks consisting of 224 trials each. The first two blocks consisted of neutral pictures and the last two blocks consisted of anger-related pictures. This fixed order was chosen to prevent priming and carry-over effects of the anger-related pictures onto the neutral pictures. After each block, participants were given the opportunity to take a short break. The order of Go and NoGo trials was quasi randomized such that at most two NoGo trials were presented consecutively. Before starting the actual task participants performed 23 practice trials involving additional neutral pictures. Total task duration was about 22 min.

The accuracy rates for Go and NoGo trials as well as the median reaction times (RT) for the Go trials only were used as performance measures for the Go/NoGo task. The reaction time data for the Go trials were calculated after the deletion of incorrect responses and outliers for each individual, i.e., reaction times below 150 ms or above 1500 ms.

## 2.4. Procedure

Each patient was tested individually in a silent, secluded room. Participants were seated in a comfortable chair, and received a brief general introduction on the EEG measurements and the procedures. Next, electrodes were placed and participants were instructed to sit relaxed and to minimize eye-blinks and body movements during the experiment. Following these instructions, participants completed the Go/NoGo task along with two other tasks during EEG recording. E-Prime software (Version 2.0; Psychology Software Tools, Pittsburg, PA) was used to administer the tasks. Task instructions were provided before the start of each task. After completion of the three tasks, electrodes were removed and participants were seated behind a computer

to complete the questionnaires.<sup>1</sup> After having completed the experiment, participants were thanked for their participation. In total, testing lasted approximately 1 h and 45 min.

## 2.5. EEG recording and data reduction

The electroencephalogram (EEG) was recorded using the Biosemi Active-Two amplifier system (Biosemi, Amsterdam, The Netherlands) from 34 scalp sites mounted in an elastic cap according to the international 20/10 system (ACNS, 2006), and with two additional electrodes at FCz and CPz. Six additional electrodes were placed for offline referencing and for recording horizontal (HEOG) and vertical electro-oculogram (VEOG) to correct for eye-movements and blinks. The reference electrodes were attached to the left and right mastoids. The other electrodes were placed on the outer canthi of both eyes (HEOG), and the infraorbital and supraorbital regions of the right eye (VEOG). BrainVision Analyzer 2 (Brain products GmbH, Munich, Germany) was used to process the offline data. All signals were digitized with a sample rate of 512 Hz and 24-bit A/D conversion with a bandpass of 0–134 Hz, and were filtered offline. Data were offline-referenced to the average of the left and right mastoids. Off-line, EEG and EOG activity was filtered using phase shift-free Butterworth filters (24 dB/Octave roll off) with a bandpass of .15 Hz to 30 Hz. The Go/NoGo task EEG data were segmented in epochs from 200 ms before stimulus presentation to 1450 ms after stimulus presentation and 100 ms before the response onset to 600 ms after the response onset. Ocular correction was applied using the Gratton and Coles algorithm (Gratton et al., 1983). The mean 100 ms pre-stimulus period served as baseline. Artifacts were rejected by excluding epochs exceeding  $\pm 75 \mu\text{V}$  from the average.

For the N2 and P3 components the average ERP waves were calculated for artifact free trials for neutral Go, neutral NoGo, anger-related Go, and anger-related NoGo stimuli separately. Moreover, for calculating these components segments with incorrect responses (miss for Go trials or false alarms for NoGo trials) were excluded from the analyses. The N2 was defined as the average activity within the 200–300 ms time interval after stimulus onset and was studied at a cluster of frontocentral electrodes, including FC1, Fz, FC2, FCz, and Cz (Liu et al., 2014; Luijten et al., 2011a) as the N2 is predominantly examined and observed over anterior scalp sites (Falkenstein et al., 1999). The P3 was defined as the average value within the 300–500 ms time interval after stimulus onset and was studied at C3, Cz, C4, FCz, and CPz (Luijten et al., 2011a; Rietdijk et al., 2014). The mean number of analyzable Go and NoGo epochs for the N2 and P3 components after removal of the artifacts was 270 and 68 for anger-related pictures and 278 and 73 for neutral pictures respectively. Eight participants (3 LTA and 5 HTA individuals) in total were excluded from these ERP analyses because of less than 20 artifact free ERP epochs in at least one of the task conditions, which is required to obtain a reliable N2 and P3 (Rietdijk et al., 2014).

For the ERN and Pe components the average ERP waves were calculated for artifact free trials for correct Go trials (hits for Go trials) and for incorrect NoGo trials (false alarms for NoGo trials). The ERN was defined as the average value in the 0–100 ms range after response onset (e.g., Ladouceur et al., 2006; Vocat et al., 2008). The Pe was defined as the average value in the 150–350 ms range after response onset (Alexopoulos et al., 2007; Rollnik et al., 2004). Both the ERN and the Pe were studied at FCz, Cz, and CPz as these midline electrodes are typically examined (Luijten et al., 2011b; Rietdijk et al., 2014). To obtain reliable information for the ERN and Pe at least 6 trials are needed (Olvet and Hajcak, 2009; Pontifex et al., 2010). In total, five participants were excluded from these ERP analyses because of fewer than 6 artifact free

<sup>1</sup> The Aggression Questionnaire (Buss and Perry, 1992), the Barratt Impulsiveness Scale (Patton et al., 1995), the Anger Rumination Scale (Sukhodolsky et al., 2001), and the Ambiguous Hostile Stories Task were also administered for different research purposes, but are not reported here as they go beyond the goals of the present study.



ERP epochs due to too few errors or due to too many artifacts. The mean number of analyzable epochs for the ERN and Pe components after removal of the artifacts was 591 for correct Go trials and 45 for incorrect NoGo trials.

## 2.6. Statistical analyses

Data were analyzed using SPSS 22.0. Repeated Measures Analyses of Variance (RM-ANOVA with Greenhouse–Geisser adjusted  $p$ -values) were used to analyze the accuracy rates and reaction time data on the Go/NoGo task, as well as the ERP indices of response inhibition (i.e., N2 and P3) and error monitoring (i.e., ERN and Pe). The between-subjects factor in all RM-ANOVAs was Group (HTA versus LTA). Several two-level within-subjects factors were of interest, specifically (a) Inhibition (Go versus NoGo), (b) Picture (anger-related versus neutral), and (c) Accuracy (Correct Go versus Incorrect NoGo). An Inhibition RM-ANOVA was conducted to analyze behavioral accuracy on the Go/NoGo task and a Picture RM-ANOVA was performed to analyze reaction time data in order to investigate general performance. Moreover, a Group  $\times$  Picture RM-ANOVA was chosen to analyze group differences with respect to the behavioral accuracy on the NoGo trials, and a Group  $\times$  Picture RM-ANOVA was conducted to analyze group differences regarding the reaction time data on Go trials. A Group  $\times$  Inhibition  $\times$  Picture  $\times$  Electrode (FC1, Fz, FC2, FCz, and Cz for N2; C3, Cz, C4, FCz, and CPz for P3) RM-ANOVA was performed for the stimulus-locked ERP analyses, and a Group  $\times$  Accuracy  $\times$  Electrode (FCz, Cz, and CPz) was conducted to analyze the response-locked ERP waves. Picture type was not included as a within subject variable in the analyses of the response-locked ERPs due to too few segments for each category. Post-hoc tests for interactions using Bonferroni correction for multiple comparisons were conducted only for interactions that included the between-subject factor Group.

## 3. Results

### 3.1. Behavioral data: general performance

As expected, participants performed less accurate on NoGo trials than on Go trials (75.6% versus 98.3% respectively),  $F(1, 92) = 349.97$ ,  $p < .001$ ,  $\eta_p^2 = .79$ .

### 3.2. Behavioral data: trait anger and performance on the anger-related Go/NoGo task

Results show that the reaction times did not differ between both groups nor was there a significant Group  $\times$  Picture interaction, both  $F_s < 1$  (see Table 1). With regard to accuracy, participants were less accurate on anger-related NoGo trials than on neutral NoGo trials,  $F(1, 91) = 27.81$ ,  $p < .001$ ,  $\eta_p^2 = .23$ . However, in contrast to predictions, no group differences were found on accuracy,  $F(1, 91) < 1$ , and there

was also no significant Group  $\times$  Picture interaction,  $F(1, 91) = 1.35$ ,  $p = .248$ ,  $\eta_p^2 = .02$ .

### 3.3. N2 amplitudes

Fig. 1 shows the grand average waveforms for neutral and anger-related pictures at Fz and Cz for the HTA and LTA groups. As expected, a robust main effect was found for Inhibition,  $F(1, 83) = 17.53$ ,  $p < .001$ ,  $\eta_p^2 = .17$ , on the N2 component at the frontocentral electrode cluster, meaning that on average the N2 amplitude elicited by NoGo stimuli was larger than by Go stimuli.<sup>2</sup> Moreover, there was a main effect for Picture,  $F(1, 83) = 14.96$ ,  $p < .001$ ,  $\eta_p^2 = .15$ , indicating that the N2 amplitudes were larger for neutral pictures than for anger-related pictures. The main effect for Electrode was also significant,  $F(4, 332) = 31.38$ ,  $p < .001$ ,  $\eta_p^2 = .27$ , with the largest N2 ( $-7.44 \mu V$ ) at Fz. The Electrode  $\times$  Inhibition interaction effect was significant as well,  $F(4, 322) = 12.58$ ,  $p < .001$ ,  $\eta_p^2 = .13$ . Follow-up analyses showed that for each electrode the difference in N2 amplitudes for Go and NoGo trials was significant, all  $t_s > 3.08$  and all  $p_s < .003$ , although this difference was somewhat smaller for the Electrode FC1. The Picture  $\times$  Inhibition interaction effect, however, was not significant,  $F(1, 83) = 3.64$ ,  $p = .060$ ,  $\eta_p^2 = .04$ . Importantly, contrary to expectations, no Group main or interaction effects were found, meaning groups did not differ regarding the N2 amplitude, all  $F_s < 3.36$  and all  $p_s > .070$ .

### 3.4. P3 amplitudes

The P3 waveforms for anger-related and neutral pictures in both groups are displayed in Fig. 2. Again, a robust main effect was found for Inhibition,  $F(1, 83) = 194.05$ ,  $p < .001$ ,  $\eta_p^2 = .70$ , indicating that the P3 amplitudes were on average larger for NoGo trials than for Go trials. No main effect of Picture was found,  $F(1, 83) = 1.81$ ,  $p = .182$ ,  $\eta_p^2 = .02$ , although the Picture  $\times$  Inhibition interaction was significant,  $F(1, 83) = 15.87$ ,  $p < .001$ ,  $\eta_p^2 = .16$ . Follow-up analyses revealed that the P3 amplitudes on Go trials were larger for neutral Pictures ( $1.11 \mu V$ ) than for anger-related pictures ( $0.14 \mu V$ ),  $F(1, 84) = 20.33$ ,  $p < .001$ ,  $\eta_p^2 = .20$ , whereas no differences were found with respect to picture content on NoGo trials ( $5.07 \mu V$  and  $5.45 \mu V$  for neutral and anger-related NoGo trials, respectively),  $F(1, 84) = 1.61$ ,  $p = .209$ ,  $\eta_p^2 = .02$ . Moreover, a main effect for Electrode was found,  $F(4, 332) = 15.35$ ,  $p < .001$ ,  $\eta_p^2 = .16$ , with the largest P3 at Cz and CPz ( $3.52 \mu V$  and  $3.51 \mu V$ , respectively). Again, a significant Electrode  $\times$  Inhibition interaction effect was found,  $F(4, 332) = 50.51$ ,  $p < .001$ ,  $\eta_p^2 = .38$ . Follow-up analyses showed that for each electrode the difference in P3 amplitudes for Go and NoGo trials was significant, all  $t_s > 10.58$  and all  $p_s < .001$ , whereby this difference was somewhat smaller for the Electrode C4. Finally, although a Group  $\times$  Electrode interaction was found,  $F(4, 332) = 50.51$ ,  $p < .001$ ,  $\eta_p^2 = .38$ , and the P3 on NoGo trials seemed lower in HTA individuals compared to LTA individuals yet not statistically significant ( $F[1, 83] = 2.76$ ,  $p = .100$ ), no other significant interaction or main effects including Group were found for the P3 amplitudes, all  $F_s \leq 2.76$  and all  $p_s \geq .100$ . Follow-up analyses regarding the Group  $\times$  Electrode interaction revealed no group differences on each electrode separately, all  $F_s \leq 2.376$  and all  $p_s \geq .14$ . In short, these results indicate that HTA individuals did not significantly differ from LTA individuals with respect to the P3 amplitude.

### 3.5. ERN

The response-locked ERP waveforms at FCz for correct Go and incorrect NoGo trials for both groups are depicted in Fig. 3. As expected,

**Table 1**  
Accuracy rates (in proportions) and reaction times (in ms) for both groups on the anger-related Go/NoGo task.

	LTA (N = 44)		HTA (N = 49)	
	M	SD	M	SD
Acc Go Agr	.98	.02	.98	.03
Acc Go Neutr	.98	.02	.99	.02
Acc NoGo Agr	.74	.16	.73	.14
Acc NoGo Neutr	.78	.12	.78	.12
RT (ms) Go Agr	304	66	306	70
RT (ms) Go Neutr	307	63	305	52

<sup>2</sup> Peak measures yielded similar results regarding the N2, P3, ERN, and Pe.

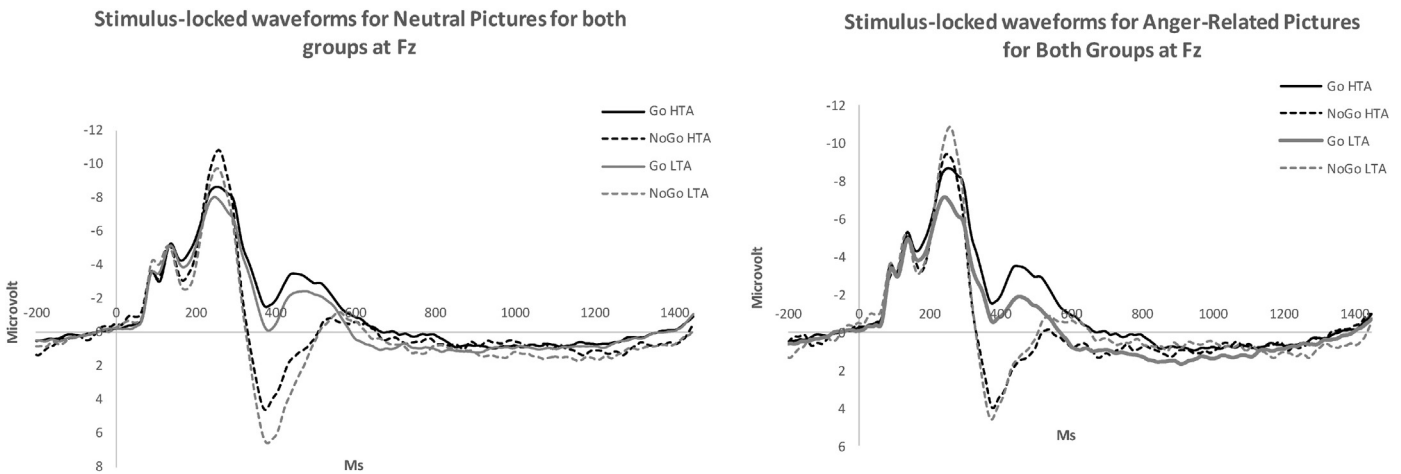


Fig. 1. Grand-average stimulus-locked waveforms for neutral and anger-related pictures at Fz for correct Go and NoGo trials in high (HTA) and low trait anger individuals (LTA).

the ERN was larger for errors than for correct responses,  $F(1, 86) = 258.40$ ,  $p < .001$ ,  $\eta_p^2 = .75$ . There was also a main effect of Electrode,  $F(2, 172) = 50.51$ ,  $p < .001$ ,  $\eta_p^2 = .38$ , with the largest ERN at FCz ( $-2.52 \mu V$ ). The Electrode  $\times$  Accuracy interaction effect was significant as well,  $F(2, 172) = 9.57$ ,  $p < .001$ ,  $\eta_p^2 = .10$ . Follow-up analyses revealed that the difference in ERN amplitudes between accurate and incorrect trials was significant for each electrode, all  $t$ s  $> 15.37$  and all  $p$ s  $< .001$ , whereby the difference was slightly smaller on CPz. Most importantly, no main or interaction effects including Group were found for the ERN, meaning that HTA individuals and LTA individuals did not differ regarding the ERN, all  $F$ s  $\leq 1.05$  and all  $p$ s  $\geq .309$ .

### 3.6. Pe

Similar to the ERN, the Pe amplitudes were larger for errors than for correct responses,  $F(1, 86) = 82.31$ ,  $p < .001$ ,  $\eta_p^2 = .49$ . There was also a main effect for Electrode,  $F(2, 172) = 15.71$ ,  $p < .001$ ,  $\eta_p^2 = .15$ , with the largest Pe at Cz ( $9.18 \mu V$ ), and a significant Electrode  $\times$  Accuracy interaction effect,  $F(2, 172) = 17.98$ ,  $p < .001$ ,  $\eta_p^2 = .17$ , whereby the difference in Pe amplitudes between accurate and incorrect trials was smallest for the electrode FCz,  $t = 6.82$ ,  $p < .001$ . No main effect was found for Group,  $F(1, 86) = 3.61$ ,  $p = .061$ ,  $\eta_p^2 = .04$ , nor was there a significant Group  $\times$  Accuracy  $\times$  Electrode interaction,  $F(2, 172) =$

$3.01$ ,  $p = .079$ ,  $\eta_p^2 = .03$ . Most importantly, there was a significant Group  $\times$  Accuracy interaction,  $F(1, 86) = 4.34$ ,  $p = .040$ ,  $\eta_p^2 = .05$ . Follow-up analyses indicated that the Pe amplitudes for errors, but not for correct responses, were significantly reduced in HTA individuals as compared to LTA individuals,  $F(1, 86) = 4.97$ ,  $p = .028$ .

## 4. Discussion

The main goal of the current study was to compare anger-primed inhibitory control and error-processing in HTA and LTA individuals. An affective Go/NoGo task was used to assess both processes in the HTA and LTA group. Based on prior research, we expected reduced inhibitory control in HTA individuals compared to LTA individuals as reflected in more commission errors and reduced N2 and P3 amplitudes on NoGo trials, especially for anger-related trials. Regarding error-processing, we explored whether reduced ERN and Pe amplitudes could be found for HTA individuals as compared to LTA individuals. Contrary to our predictions, we found no group differences regarding inhibitory control. That is, HTA and LTA individuals made comparable numbers of commission errors on NoGo trials and no significant differences were found on the N2 and P3 amplitudes. We did find consistently larger N2 and P3 amplitudes across groups for NoGo trials compared to Go trials, indicating that the affective Go/NoGo task showed the typical Go/NoGo effect.

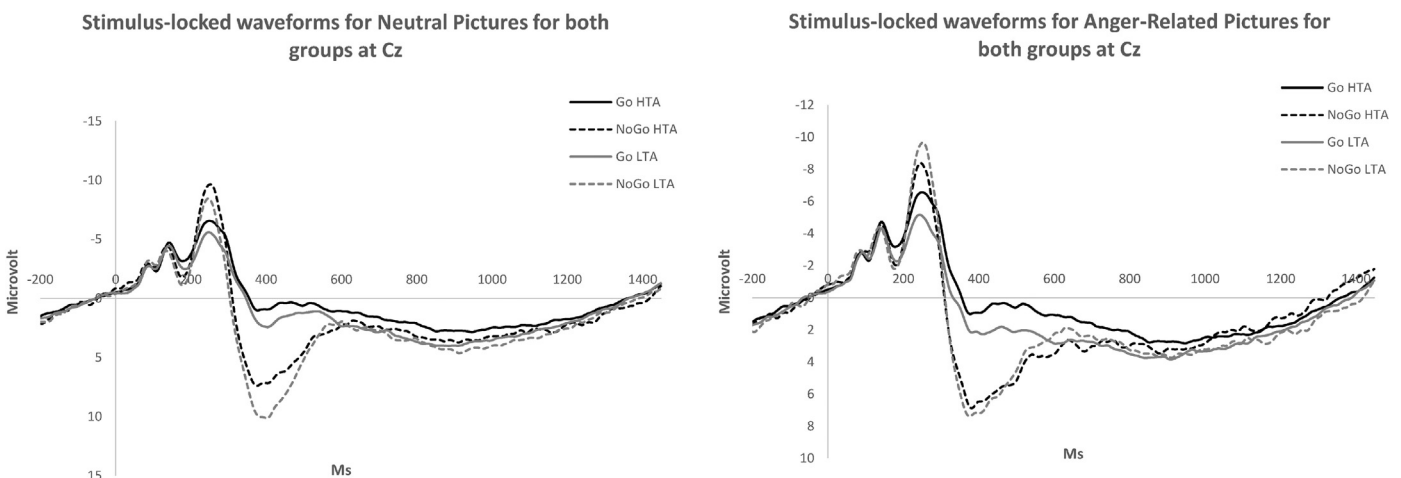
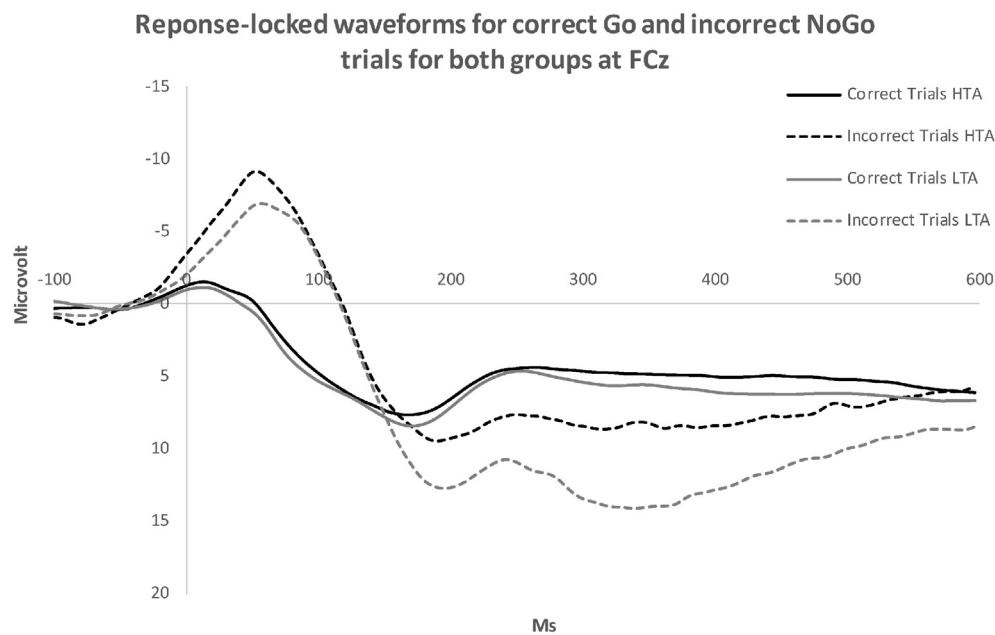


Fig. 2. Grand-average stimulus-locked waveforms for neutral and anger-related pictures at Cz for correct Go and NoGo trials in high (HTA) and low trait anger (LTA) individuals.



**Fig. 3.** Grand-average response-locked waveforms at FCz for correct Go and incorrect NoGo trials in high (HTA) and low trait anger individuals (LTA).

With respect to error-processing, we found reduced Pe amplitudes following commission errors on NoGo trials in HTA individuals as compared to LTA individuals, whereas the ERN amplitudes were comparable for both groups.

Given that the Pe is supposed to reflect motivational salience to errors (Overbeek et al., 2005), whereas the ERN is thought to reflect a more automatic process of error detection (Bernstein et al., 1995; Yeung et al., 2004), it seems that HTA and LTA individuals do not differ in automatic error detection. In contrast, HTA individuals show deficits in later stages of error-processing (i.e., the Pe component) compared to LTA individuals. In other words, the current study is the first study to indicate that HTA individuals show diminished motivational salience to errors compared to LTA individuals. Importantly, these deficits in later stages of error-processing, may provide an explanation for the continuation of impulsive behaviors in HTA individuals despite the negative consequences. The combination of reduced Pe amplitudes and intact ERN amplitudes found in HTA individuals is in accordance with previous ERP studies showing error-processing deficiencies related to the more conscious and later processing of errors in populations characterized with externalizing psychopathology, including dysfunctional anger (Brazil et al., 2009; Chen et al., 2005; Maurer et al., 2015). Moreover, our results are in line with several fMRI studies showing reduced activation in brain regions related to error-processing (i.e., the anterior cingulate cortex and orbitofrontal cortex) in dysfunctional angry populations (Blair, 2012; Coccaro et al., 2007; Davidson et al., 2000; Fulwiler et al., 2012; Siever, 2008). This study adds to this literature by showing that error-processing deficits in later stages of processing are related to dysfunctional anger in healthy populations as well. However, it should be noted that, we found no group differences on the ERN, which is in contrast with previous studies that did find reduced ERN amplitudes in impulsive, externalizing populations (Dikman and Allen, 2000; Hall et al., 2007; Heritage and Benning, 2012; Munro et al., 2007a; Potts et al., 2006; Vilà-Balló et al., 2014; von Borries et al., 2010). Although there is no straightforward explanation for these contradictory findings, it should be noted that the Pe seems to be more consistently identified as a marker in impulsive dysfunctional angry samples (Brazil et al., 2009; Chen et al., 2005; Maurer et al., 2015). Moreover, it seems that our finding on the Pe was not confounded by an overall reduced cognitive ability (e.g., deficits in earlier perceptual or attentional memory

processes) as groups did not differ on the stimulus-locked N2 and P3 components.

Our finding that HTA and LTA individuals performed equally well on our task measuring inhibitory control was in contrast with previous studies that showed reduced hostility-primed inhibitory control in HTA individuals (Bresin and Robinson, 2013; Wilkowski et al., 2010; Wilkowski and Robinson, 2008a), but in line with the study of Hull et al. (2003) who found no relation between trait anger and inhibitory control. Interestingly, studies conducted with individuals high on trait aggressiveness, a concept distinct from but closely related to trait anger (Bettencourt et al., 2006; Wilkowski and Robinson, 2010), have also shown mixed results (Denny and Siemer, 2012; Krämer et al., 2011; Pawliczek et al., 2013). One explanation could be that the anger-related pictures used in the current study were not salient enough to elicit sufficient recruitment of effortful resources in contrast to previous studies (Bresin and Robinson, 2013; Wilkowski et al., 2010; Wilkowski and Robinson, 2008a). For instance, one difference between our study and these previous studies is that we used anger-related pictures instead of anger-related words to prime anger. Perhaps individuals are more likely to ruminate on anger in response to processing personally relevant anger-related words compared to anger-related pictures. Thus, perhaps stronger effects are to be found when relevant threat-related words are used instead of general hostile pictures (also see Verona and Bresin, 2015; Siegle et al., 2002). However, in contrast to this explanation, we did find reduced accuracy rates and N2 amplitudes for anger-related pictures compared to neutral pictures, indicating reduced response inhibition for anger-related pictures compared to neutral words.

The findings regarding the N2 and P3 components were in line with a previous study from Liu et al. (2014), who also failed to find reduced N2 and P3 components for HTA individuals compared to LTA individuals. The current study adds by showing that the N2 and P3 component were comparable in HTA and LTA individuals, even under conditions when anger is primed. Although response inhibition (i.e., accuracy, N2, and P3 amplitudes) seemed somewhat reduced when anger was primed, this effect was evident for both groups and not specific for HTA individuals. Interestingly, Munro et al. (2007b) employed a Go/NoGo task in violent offenders and found that violent offenders did not differ from healthy controls regarding the N2 and P3 components as well, although there seemed to be some evidence for reduced N2



NoGo effects in impulsive violent offenders low in psychopathy. Notably, Chen et al. (2005) did find lower N2 amplitudes in impulsive-violent offenders compared to offenders without an impulsive-violent criminal record. Moreover, studies conducted in juvenile violent offenders without psychopathy also revealed reduced N2 and P3 amplitudes compared to controls (Guan et al., 2015; Vilà-Balló et al., 2014). Hence, one potential explanation for these mixed findings is that, whereas neural indicators of response inhibition are related to impulsive aggressive behaviors, they are not specifically related to trait anger or to instrumental aggressive behaviors. Finally, one could argue that our task was not sensitive enough to detect individual differences in inhibitory control. We consider this explanation unlikely, however, since there was considerable interindividual variance in this study and prior studies have shown effects with the same task (Luijten et al., 2011a). Interestingly, using the same task, we did find trait anger to be inversely related with performance accuracy as well as error-processing in a forensic psychiatric sample, although no relation was found between trait anger and the N2 and P3 components (Lievaert, Veen van der, Huijding, Hovens, & Franken, 2015). Hence, an interesting avenue for future research would be to test the boundary conditions regarding the relation between inhibitory control, trait anger, and aggression. For instance, further research is warranted that takes in account related factors such as impulsive violent behaviors and anxiety, and by specifying subgroups.

An important strength of the current study is that it allowed us to investigate underlying processes of trait anger, without addressing confounding comorbid psychopathology that is often present in violent samples, such as substance abuse. However, extension of the present study to clinical samples, including samples with problematic anger, should provide further insight regarding anger-related response inhibition and error-processing on the neurophysiological level. Future studies should also investigate the moderating impact of motivation on the relation between trait anger on the one hand and response inhibition and error-processing on the other hand (Hall et al., 2007). Furthermore, studies might benefit from including trait anger as a covariate rather than dichotomizing into two groups, as this approach may be less susceptible to being influenced by confounds (e.g., anxiety). Another interesting avenue for future research would be to induce state anger and to investigate its impact on high and low trait anger individuals while performing tasks measuring inhibitory control. Finally, the reduced N2 and P3 amplitudes as well as the diminished performance on anger-related trials may be due to stimulus-order effects, given that participants always received the neutral blocks first. Other outcomes may have been obtained if the neutral and hostile pictures were presented using a random design (i.e., if picture type had been varied trial-by-trial).

In summary, our hypothesis that high trait anger individuals demonstrate impaired hostility-primed inhibitory control on both the behavioral level as well as the neurophysiological level could not be confirmed. Importantly, we did find initial evidence for impaired conscious error-processing in high trait anger individuals. This deficit may shed light on the neurobiological underpinnings of trait anger and may explain the continuation of inappropriate behaviors (e.g., outward expressions of anger), despite their negative consequences. Future studies are needed to evaluate whether anger management results in reduced error-processing deficits. Another interesting avenue for research would be to investigate whether these error-processing deficits may predict treatment effects.

## Conflicts of interest

The authors report no conflicts of interest.

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## References

- ACNS, 2006. American clinical neurophysiology society guideline 5: Guidelines for standard electrode position nomenclature. *J. Clin. Neurophysiol.* 23, 107–110.
- Alexopoulos, G.S., Murphy, C.F., Gunning-Dixon, F.M., Kalayam, B., Katz, R., Kanellopoulos, D., ... Foxe, J.J., 2007. Event-related potentials in an emotional go/no-go task and remission of geriatric depression. *Neuroreport* 18 (3), 217–221. <http://doi.org/10.1097/WNR.0b013e328013ceda>.
- Averill, J., 1983. Studies on anger and aggression: implications for theories of emotion. *Am. Psychol.* 38 (11), 1145–1160. <http://doi.org/10.1037/0003-066X.38.11.1145>.
- Barbour, K.A., Eckhardt, C.I., Davison, G.C., Kassiove, H., 1998. The experience and expression of anger in maritally violent and maritally discordant-nonviolent men. *Behav. Ther.* 29 (2), 173–191. [http://doi.org/10.1016/S0005-7894\(98\)80001-4](http://doi.org/10.1016/S0005-7894(98)80001-4).
- Baron, K.G., Smith, T.W., Butner, J., Nealey-Moore, J., Hawkins, M.W., Uchino, B.N., 2006. Hostility, anger, and marital adjustment: concurrent and prospective associations with psychosocial vulnerability. *J. Behav. Med.* 30 (1), 1–10. <http://doi.org/10.1007/s10865-006-9086-z>.
- Bernstein, P.S., Scheffers, M.K., Coles, M.G.H., 1995. “Where did I go wrong?” A psychophysiological analysis of error detection. *J. Exp. Psychol. Hum. Percept. Perform.* 21 (6), 1312–1322. <http://doi.org/10.1037/0096-1523.21.6.1312>.
- Bettencourt, B.A., Talley, A., Benjamin, A.J., Valentine, J., 2006. Personality and aggressive behavior under provoking and neutral conditions: a meta-analytic review. *Psychol. Bull.* 132 (5), 751–777. <http://doi.org/10.1037/0033-2909.132.5.751>.
- Blair, R., 2012. Considering anger from a cognitive neuroscience perspective. *Wiley Interdiscip. Rev. Cogn. Sci.* 3 (1), 65–74. <http://doi.org/10.1002/wcs.154>.
- Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., Cohen, J.D., 2001. Conflict monitoring and cognitive control. *Psychol. Rev.* 108 (3), 624–652. <http://doi.org/10.1037/0033-295X.108.3.624>.
- Brazil, I.A., de Bruijn, E.R.A., Bulten, B.H., von Borries, A.K.L., van Lankveld, J.J.D.M., Buitelaar, J.K., Verkes, R.J., 2009. Early and late components of error monitoring in violent offenders with psychopathy. *Biol. Psychiatry* 65 (2), 137–143. <http://doi.org/10.1016/j.biopsych.2008.08.011>.
- Bresin, K., Robinson, M.D., 2013. Losing control, literally: relations between anger control, trait anger, and motor control. *Cogn. Emot.* 27 (6), 995–1012. <http://doi.org/10.1080/02699931.2012.755119>.
- Buss, A.H., Perry, M., 1992. The aggression questionnaire. *J. Pers. Soc. Psychol.* 63 (3), 452–459. <http://doi.org/10.1037/0022-3514.63.3.452>.
- Chen, C.-Y., Tien, Y.-M., Juan, C.-H., Tzeng, O.J.L., Hung, D.L., 2005. Neural correlates of impulsive-violent behavior: an event-related potential study. *Neuroreport* 16 (11), 1213–1216. <http://doi.org/10.1097/00001756-200508010-00016>.
- Chen, C.-Y., Muggleton, N.G., Chang, J.-R., 2014. Inefficiency of post-error adjustment in impulsive violent offenders. *Neuroreport* 25 (13), 1024–1029. <http://doi.org/10.1097/WNR.0000000000000212>.
- Coccaro, E.F., McCloskey, M.S., Fitzgerald, D.A., Phan, K.L., 2007. Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biol. Psychiatry* 62 (2), 168–178. <http://doi.org/10.1016/j.biopsych.2006.08.024>.
- Davidson, R., Putnam, K., Larson, C., 2000. Dysfunction in the neural circuitry of emotion regulation—a possible prelude to violence. *Science* 289 (5479), 591–594. <http://doi.org/10.1126/science.289.5479.591>.
- Denny, K.G., Siemer, M., 2012. Trait aggression is related to anger-modulated deficits in response inhibition. *J. Res. Pers.* 46 (4), 450–454. <http://doi.org/10.1016/j.jrp.2012.04.001>.
- Denson, T.F., 2015. Four promising psychological interventions for reducing reactive aggression. *Curr. Opin. Behav. Sci.* 3, 136–141. <http://doi.org/10.1016/j.cobeha.2015.04.003>.
- Dikman, Z.V., Allen, J.B., 2000. Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology* 37 (1), 43–54. <http://doi.org/10.1017/S0048577200980983>.
- Eckhardt, C.I., Cohen, D.J., 1997. Attention to anger-relevant and irrelevant stimuli following naturalistic insult. *Personal. Individ. Differ.* 23 (4), 619–629. [http://doi.org/10.1016/S0191-8869\(97\)00074-3](http://doi.org/10.1016/S0191-8869(97)00074-3).
- Falkenstein, M., Hoormann, J., Hohnsbein, J., 1999. ERP components in Go/NoGo tasks and their relation to inhibition. *Acta Psychol.* 101 (2–3), 267–291. [http://doi.org/10.1016/S0001-6918\(99\)00008-6](http://doi.org/10.1016/S0001-6918(99)00008-6).
- Franken, I.H.A., van Strien, J.W., Franzek, E.J., van de Wetering, B.J., 2007. Error-processing deficits in patients with cocaine dependence. *Biol. Psychol.* 75 (1), 45–51. <http://doi.org/10.1016/j.biopsycho.2006.11.003>.
- Fulwiler, C.E., King, J.A., Zhang, N., 2012. Amygdala–orbitofrontal resting-state functional connectivity is associated with trait anger. *Neuroreport*. <http://doi.org/10.1097/WNR.0b013e3283551cfc>.
- Gratton, C., Coles, M., Donchin, E., 1983. A new method for off-line removal of ocular artifact. *A New Method for off-Line Removal of Ocular Artifact* 55, pp. 468–484.
- Guan, M., Liao, Y., Ren, H., Wang, X., Yang, Q., Liu, X., Wang, W., 2015. Impaired response inhibition in juvenile delinquents with antisocial personality characteristics: a preliminary ERP study in a Go/NoGo task. *Neurosci. Lett.* 603, 1–5. <http://doi.org/10.1016/j.neulet.2015.06.062>.
- Hall, J., Bernat, E., Patrick, C., 2007. Externalizing psychopathology and the error-related negativity. *Psychol. Sci.* 18 (4), 326–333. <http://doi.org/10.1111/j.1467-9280.2007.01899.x>.
- Heritage, A.J., Benning, S.D., 2012. Impulsivity and response modulation deficits in psychopathy: evidence from the ERN and N1. *J. Abnorm. Psychol.* 122 (1), 215–222. <http://doi.org/10.1037/a0030039>.
- Hull, L., Farrin, L., Unwin, C., Everitt, B., Wykes, T., David, A.S., 2003. Anger, psychopathology and cognitive inhibition: a study of UK servicemen. *Personal. Individ. Differ.* 35 (5), 1211–1226. [http://dx.doi.org/10.1016/S0191-8869\(02\)00329-X](http://dx.doi.org/10.1016/S0191-8869(02)00329-X).

- Van Honk, J., Tuiten, A., van den Hout, M., Putman, P., de Haan, E., Stam, H., 2001. Selective attention to unmasked and masked threatening words: relationships to trait anger and anxiety. *Personal. Individ. Differ.* 30 (4), 711–720. [http://doi.org/10.1016/S0191-8869\(00\)00160-4](http://doi.org/10.1016/S0191-8869(00)00160-4).
- Hovens, J.E., Lievaart, M., Rodenburg, J.J., 2014. *STAXI-2: Vragenlijst over boosheid. Hogrefe, Amsterdam*.
- Kassinove, H., Sukhodolsky, D.G., Tsytarev, S.V., Solovyova, S., 1997. Self-reported anger episodes in Russia and America. *J. Soc. Behav. Pers.* 12, 301–324.
- Krämer, U.M., Kopyciok, R.P.J., Richter, S., Rodriguez-Fornells, A., Münte, T.F., 2011. The role of executive functions in the control of aggressive behavior. *Front. Psychol.* 2 (July), 152. <http://doi.org/10.3389/fpsyg.2011.00152>.
- Ladouceur, C.D., Dahl, R.E., Birmaher, B., Axelson, D.A., Ryan, N.D., 2006. Increased error-related negativity (ERN) in childhood anxiety disorders: ERP and source localization. *J. Child Psychol. Psychiatry* 47 (10), 1073–1082. <http://doi.org/10.1111/j.1469-7610.2006.01654.x>.
- Lang, P.J., Bradley, M.M., Cuthbert, B.N., 2008. *International Affective Picture System (IAPS): Affective Ratings of Pictures and Instruction Manual. Technical Report A-8. University of Florida, Gainesville, FL*.
- Lievaart, D.M., Veen, F.M.van der, Huijding, J., Hovens, J.E., Franken, I.H.A., 2015, augustus 28. *The Relation between Trait Anger and Impulse Control in Forensic Psychiatric Patients: an EEG Study. Insri Meeting, Amsterdam*.
- Liu, Y., Zhan, X., Li, W., Han, H., Wang, H., Hou, J., ... Wang, Y., 2014. The trait anger affects conflict inhibition: a Go/NoGo ERP study. *Front. Hum. Neurosci.* 8, 1076. <http://doi.org/10.3389/fnhum.2014.01076>.
- Luijten, M., Littel, M., Franken, I.H.A., 2011a. Deficits in inhibitory control in smokers during a Go/NoGo task: an investigation using event-related brain potentials. *PLoS One* 6 (4), e18898. <http://doi.org/10.1371/journal.pone.0018898>.
- Luijten, M., van Meel, C.S., Franken, I.H.A., 2011b. Diminished error processing in smokers during smoking cue exposure. *Pharmacol. Biochem. Behav.* 97 (3), 514–520. <http://doi.org/10.1016/j.pbb.2010.10.012>.
- Luijten, M., Machielsen, M.W.J., Veltman, D.J., Hester, R., de Haan, L., Franken, I.H.A., 2014. Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *J. Psychiatry Neurosci.* 39 (3), 149–169. <http://doi.org/10.1503/jpn.130052>.
- Luna, B., Garver, K.E., Urban, T.A., Lazar, N.A., Sweeney, J.A., 2004. Maturation of cognitive processes from late childhood to adulthood. *Child Dev.* 75 (5), 1357–1372. <http://doi.org/10.1111/j.1467-8624.2004.00745.x>.
- Maurer, J.M., Steele, V.R., Edwards, B.G., Bernat, E.M., Calhoun, V.D., Kiehl, K.A., 2015. Dysfunctional error-related processing in female psychopathy. *Soc. Cogn. Affect. Neurosci.* <http://doi.org/10.1093/scan/nsv070>.
- McDermut, W., Fuller, J.R., DiGiuseppe, R., Chelminski, I., Zimmerman, M., 2009. Trait anger and Axis I disorders: implications for REBT. *J. Ration. Emot. Cogn. Behav. Ther.* 27 (2), 121–135. <http://doi.org/10.1007/s10942-009-0092-2>.
- Munro, G.E.S., Dywan, J., Harris, G.T., McKee, S., Unsal, A., Segalowitz, S.J., 2007a. ERN varies with degree of psychopathy in an emotion discrimination task. *Biol. Psychol.* 76 (1–2), 31–42. <http://doi.org/10.1016/j.biopsycho.2007.05.004>.
- Munro, G.E.S., Dywan, J., Harris, G.T., McKee, S., Unsal, A., Segalowitz, S.J., 2007b. Response inhibition in psychopathy: the frontal N2 and P3. *Neurosci. Lett.* 418 (2), 149–153. <http://doi.org/10.1016/j.neulet.2007.03.017>.
- Olvet, D.M., Hajcak, G., 2009. The stability of error-related brain activity with increasing trials. *Psychophysiology* 46 (5), 957–961. <http://doi.org/10.1111/j.1469-8986.2009.00848.x>.
- Overbeek, T.J.M., Nieuwenhuis, S., Ridderinkhof, K.R., 2005. Dissociable components of error processing. *J. Psychophysiol.* 19 (4), 319–329. <http://doi.org/10.1027/0269-8803.19.4.319>.
- Owen, J.M., 2011. Transdiagnostic cognitive processes in high trait anger. *Clin. Psychol. Rev.* 31 (2), 193–202. <http://doi.org/10.1016/j.cpr.2010.10.003>.
- Patton, J.H., Stanford, M.S., Barratt, E.S., 1995. Factor structure of the Barratt impulsiveness scale. *J. Clin. Psychol.* 51 (6), 768–774. [http://doi.org/10.1002/1097-4679\(199511\)51:6<768::AID-JCLP2270510607>3.0.CO;2-1](http://doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1).
- Pawliczek, C.M., Derntl, B., Kellermann, T., Gur, R.C., Schneider, F., Habel, U., 2013. Anger under control: neural correlates of frustration as a function of trait aggression. *PLoS One* 8 (10), 1–10. <http://doi.org/10.1371/journal.pone.0078503>.
- Pontifex, M.B., Scudder, M.R., Brown, M.L., O'Leary, K.C., Wu, C.-T., Themanson, J.R., Hillman, C.H., 2010. On the number of trials necessary for stabilization of error-related brain activity across the life span. *Psychophysiology* 47, 767–773. <http://doi.org/10.1111/j.1469-8986.2010.00974.x>.
- Potts, G.F., George, M.R.M., Martin, L.E., Barratt, E.S., 2006. Reduced punishment sensitivity in neural systems of behavior monitoring in impulsive individuals. *Neurosci. Lett.* 397 (1–2), 130–134. <http://doi.org/10.1016/j.neulet.2005.12.003>.
- Ridderinkhof, K.R., Ullsperger, M., Crone, E.A., Nieuwenhuis, S., 2004. The role of the medial frontal cortex in cognitive control. *Science (New York, N.Y.)* 306 (5695), 443–447. <http://doi.org/10.1126/science.1100301>.
- Rietdijk, W.J.R., Franken, I.H.A., Thuri, A.R., 2014. Internal consistency of event-related potentials associated with cognitive control: N2/P3 and ERN/Pe. *PLoS One* 9 (7), e102672. <http://doi.org/10.1371/journal.pone.0102672>.
- Roberts, B.W., Kuncel, N.R., Shiner, R., Caspi, A., Goldberg, L.R., 2007. *Power Pers.* 2 (4).
- Robinson, M. D., Wilkowski, B. M., Meier, B. P., Moeller, S. K., & Fetterman, A. K. (2012). Counting to ten milliseconds: low-anger, but not high-anger, individuals pause following negative evaluations. *Cogn. Emot.*, 26(2), 261–81. <http://doi.org/10.1080/02699931.2011.579088>
- Rollnik, J.D., Schröder, C., Rodríguez-Fornells, A., Kurzbuch, A.R., Däuper, J., Möller, J., Münte, T.F., 2004. Functional lesions and human action monitoring: combining repetitive transcranial magnetic stimulation and event-related brain potentials. *Clin. Neurophysiol.* 115 (1), 145–153. <http://doi.org/10.1016/j.clinph.2003.05.001>.
- Rothbart, M., Bates, J., 2006. *Temperament*. In: Damon, W., Eisenberg, N. (Eds.), *Handbook of Child Psychology: Vol. 3. Social, Emotional, and Personality Development*, sixth ed. Wiley, New York, NY, pp. 105–176.
- Ruchow, M., Spitzer, M., Grön, G., Grothe, J., Kiefer, M., 2005. Error processing and impulsiveness in normals: evidence from event-related potentials. *Cogn. Brain Res.* 24 (2), 317–325. <http://doi.org/10.1016/j.cogbrainres.2005.02.003>.
- Siegle, G.J., Steinhauser, S.R., Thase, M.E., Stenger, V.A., Carter, C.S., 2002. Can't shake that feeling: event-related fMRI assessment of sustained amygdala activity in response to emotional information in depressed individuals. *Biol. Psychiatry* 51 (9), 693–707. [http://doi.org/10.1016/S0006-3223\(02\)01314-8](http://doi.org/10.1016/S0006-3223(02)01314-8).
- Siever, L.J., 2008. Neurobiology of aggression and violence. *Am. J. Psychiatr.* 165 (4), 429–442. <http://doi.org/10.1176/appi.ajp.2008.07111774>.
- Smith, T., Glazer, K., Ruiz, J., Gallo, L., 2004. Hostility, anger, aggressiveness, and coronary heart disease: an interpersonal perspective on personality, emotion, and health. *J. Pers.* 72 (6), 1217–1270.
- Spielberger, C.D., 1999. *The State-Trait Anger Expression Inventory-2 (STAXI-2): Professional Manual. Psychological Assessment Resources, Inc., Odessa, FL*.
- Sukhodolsky, D.G., Golub, A., Cromwell, E.N., 2001. Development and validation of the anger rumination scale. *Personal. Individ. Differ.* 31 (5), 689–700. [http://doi.org/10.1016/S0191-8869\(00\)00171-9](http://doi.org/10.1016/S0191-8869(00)00171-9).
- Tafra, R.C., Kassinove, H., Dundin, L., 2002. Anger episodes in high- and low-trait-anger community adults. *J. Clin. Psychol.* 58 (12), 1573–1590. <http://doi.org/10.1002/jclp.10076>.
- Verona, E., Bresin, K., 2015. Aggression proneness: Transdiagnostic processes involving negative valence and cognitive systems. *Int. J. Psychophysiol.*
- Vilà-Balló, A., Hdez-Lafuente, P., Rostan, C., Cunillera, T., Rodríguez-Fornells, A., 2014. Neurophysiological correlates of error monitoring and inhibitory processing in juvenile violent offenders. *Biol. Psychol.* 102C, 141–152. <http://doi.org/10.1016/j.biopsycho.2014.07.021>.
- Vocat, R., Pourtois, G., Vuilleumier, P., 2008. Unavoidable errors: a spatio-temporal analysis of time-course and neural sources of evoked potentials associated with error processing in a speeded task. *Neuropsychologia* 46 (10), 2545–2555. <http://doi.org/10.1016/j.neuropsychologia.2008.04.006>.
- Von Borries, A.K.L., Brazil, I.A., Bulten, B.H., Buitelaar, J.K., Verkes, R.J., de Bruijn, E.R.A., 2010. Neural correlates of error-related learning deficits in individuals with psychopathy. *Psychol. Med.* 40 (09), 1559–1568. <http://doi.org/10.1017/S0033291709992017>.
- Wilkowski, B.M., 2011. Responding to social signals for response inhibition: a psychological process underlying trait anger. *Soc. Psychol. Personal. Sci.* 3 (1), 72–79. <http://doi.org/10.1177/1948550611409246>.
- Wilkowski, B.M., Robinson, M.D., 2007. Keeping one's cool: trait anger, hostile thoughts, and the recruitment of limited capacity control. *Personal. Soc. Psychol. Bull.* 33 (9), 1201–1213. <http://doi.org/10.1177/0146167207301031>.
- Wilkowski, B.M., Robinson, M.D., 2008a. Guarding against hostile thoughts: trait anger and the recruitment of cognitive control. *Emotion* 8 (4), 578–583. <http://doi.org/10.1037/1528-3542.8.4.578>.
- Wilkowski, B.M., Robinson, M.D., 2008b. The cognitive basis of trait anger and reactive aggression: an integrative analysis. *Personal. Soc. Psychol. Rev.* 12 (1), 3–21. <http://doi.org/10.1177/1088868307309874>.
- Wilkowski, B.M., Robinson, M.D., 2010. The anatomy of anger: an integrative cognitive model of trait anger and reactive aggression. *J. Pers.* 78 (1), 9–38. <http://doi.org/10.1111/j.1467-6494.2009.00607.x>.
- Wilkowski, B.M., Robinson, M.D., Troop-Gordon, W., 2010. How does cognitive control reduce anger and aggression? The role of conflict monitoring and forgiveness processes. *J. Pers. Soc. Psychol.* 98 (5), 830–840. <http://doi.org/10.1037/a0018962>.
- World Medical Association, 2001. *Declaration of Helsinki – World Medical Association Declaration of Helsinki*. *Bull. World Health Organ.* 79 (4), 373–374.
- Yeung, N., Botvinick, M.M., Cohen, J.D., 2004. The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychol. Rev.* 111 (4), 931–959. <http://doi.org/10.1037/0033-295X.111.4.931>.