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ERP indices for response inhibition are related to anxiety-related personality traits

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ABSTRACT

Anxiety is often associated with impaired cognitive control and avoidance behaviour. The aim of this study was to investigate the effect of anxiety-related personality traits, such as anxiety sensitivity and trait anxiety, on event-related potentials of response inhibition in a standard Go/Nogo-paradigm. We focused on the Nogo-N2 and Nogo-P3 components, which probably represent different sub-processes of response inhibition. The Nogo-N2 was mainly influenced by trait anxiety, while it was slightly affected by anxiety sensitivity. In contrast, the Nogo-P3 was significantly associated with anxiety sensitivity, but was less affected by trait anxiety. Thus, anxious subjects seem to maintain a higher level of cognitive control to prepare and to monitor the outcome of their actions, which is differentially reflected in Nogo-N2 and Nogo-P3 potentials. Our results show that anxiety-related personality traits modulate electrophysiological responses related to cognitive control processes and should be taken into consideration in studies investigating response inhibition.

1. Introduction

Executive functions control cognitive processes. According to the theoretical model of Norman and Shallice (1986), the executive system is especially involved in planning, error correction, and the adaptation to novel situations (Norman & Shallice, 1986; Posner & Dehaene, 1994). *Response inhibition*, another component of this control system (Mostofsky & Simmonds, 2008), is described as the suppression of actions that are inappropriate in a given context. It can be examined experimentally in a *Go/Nogo-task* using event-related potentials (ERPs). In such a paradigm, subjects should respond to one target stimulus in the *Go-condition* and withhold responses to the target stimulus in the *Nogo-condition*.

Two fronto-central event-related potentials (ERPs) have been associated with larger amplitudes in Nogo- than in Go-trials (Eimer, 1993; Falkenstein, Hoormann, & Hohnsbein, 1999). These components have been labelled as *Nogo-N2* and *Nogo-P3*, and are considered to represent different sub-processes of response inhibition. The Nogo-N2 is assumed to reflect inhibition or revision of a motor plan prior to motor execution. In contrast, the Nogo-P3 has been associated with motor inhibition (Falkenstein et al., 1999; Smith, Johnstone, & Barry, 2008; Zordan, Sarlo, & Stablum, 2008), but due to its long latency it has also been suggested that it reflects the monitoring of the outcome of inhibition (Righi, Mecacci, & Viggiano, 2009; Schmajuk, Liotti, Busse, & Woldorff, 2006). Furthermore, both components seem to be differentially modulated by distinct neurobiological systems (Beste, Baune, Domschke, Falkenstein, & Konrad, 2010; Beste, Willemssen, Saft, & Falkenstein, 2010) supporting the assumption of different sub-processes of response inhibition.

Response inhibition and cognitive control have been associated with activity within the anterior cingulate cortex (ACC) and other frontal brain areas (Beste, Saft, Andrich, Gold, & Falkenstein, 2008; Bokura, Yamaguchi, & Kobayashi, 2001; Falkenstein, 2006). Furthermore, the ACC is important for the integration of cognitive and emotional processes (Bush, Luu, & Posner, 2000), for the pathophysiology of psychiatric disorders (Damsa, Kosel, & Moussally, 2009), and is a crucial part of the human anxiety circuitry (Sehlmeyer et al., 2009). Patients with anxiety disorders may be characterized

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by neurocognitive deficits in inhibitory processing and response monitoring. While some studies observed smaller Nogo-N2 amplitudes (Herrmann, Jacob, Unterecker, & Fallgatter, 2003; Kim, Kim, Yoo, & Kwon, 2007), others found hyperactivation of the ACC (Ursu, Stenger, Shear, Jones, & Carter, 2003), enhanced Nogo-N2 and consequently increased response inhibition (Ruchsow et al., 2007).

While patients with anxiety disorders may show some degree of response over-inhibition, the question remains whether personality traits, which are closely related to pathological anxiety (Chambers, Power, & Durham, 2004; Naragon-Gainey, 2010; Schmidt, Mitchell, & Richey, 2008), can also modulate cognitive functions, such as response inhibition, and electrophysiology, such as Nogo-components. There are two major psychological concepts concerning anxiety-related personality traits that may be linked to response inhibition: *trait anxiety* (TA) and *anxiety sensitivity* (AS). TA describes the tendency to respond fearfully to a wide variety of unspecific stressors, and the need for both security and cognitive control (Fales et al., 2008). In contrast, AS represents the specific tendency to respond fearfully to one's own bodily sensations and anxiety-related symptoms, which is based on the belief that these symptoms are harmful (McNally, 2002). It has been a matter of controversial debates whether AS and TA represent common or different concepts of anxiety (Lilienfeld, 1996; McNally, 1996; McWilliams & Cox, 2001; Muris, Schmidt, Merckelbach, & Schouten, 2001). Actually, it is assumed that they both are related to each other and focus each on different facets of anxiety. While TA concentrates on cognitive anxiety symptoms, AS refers to physical and psychological anxiety symptoms.

In general, the interplay of anxiety traits, cognitive individual differences and electrophysiology has been investigated by recent research (Karch et al., 2008; Manly, Robertson, Galloway, & Hawkins, 1999; Roche, Garavan, Foxe, & O'Mara, 2005). For example, it has been shown that subjects with high trait anxiety or anxiety sensitivity display anxiety-related attentional biases (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van, 2007) and may thus show modified ERP components, cognitive performances (flanker task: Dennis & Chen, 2009; Moser, Hajcak, & Simons, 2005; n-back: Holmes, Nielsen, Tipper, & Green, 2009; stroop: Taake, Jaspers-Fayer, & Liotti, 2009 or processing of affective information Carretie, Mercado, Hinojosa, Martin-Loeches, & Sotillo, 2004; Dennis & Chen, 2007; Fox, Derakshan, & Shoker, 2008; Li, Li, & Luo, 2005; Mercado, Carretie, Tapia, & Gomez-Jarabo, 2006; Most, Chun, Johnson, & Kiehl, 2006; Rossignol, Philippot, Douilliez, Crommelinck, & Campanella, 2005). So far, only few studies emphasized the importance of monitoring anxiety traits with regard to response inhibition and Nogo-components (Karch et al., 2008; Righi et al., 2009). In particular, Righi et al. (2009) reported that, during a Go/Nogo-task, the N2-component was increased in trait and state anxious, healthy subjects, while the P3 was decreased in subjects who reported a higher frequency of cognitive failures.

To the best of our knowledge, this is the first study investigating the influence of two different anxiety-related personality constructs, such as TA and AS, on event-related potentials in a Go/Nogo-paradigm in healthy subjects. We hypothesize that individuals with high levels of TA and AS show a specific enhancement of executive control in this response inhibition task. We assume that persons with high anxiety are characterized by increased cognitive control and an enhanced evaluation of their behavioural outcomes, which may be reflected by increased Nogo-N2 and Nogo-P3 responses, and fewer false alarm rates. Moreover, with respect to each anxiety construct (AS, TA), we expect differential effects on Nogo-N2 and -P3 components.

2. Methods

2.1. Subjects

Subjects were 54 right-handed undergraduates at the University of Muenster without any medical, neurological and psychiatric disorders (39 female, 15 male; mean age = 22.58 years, standard deviation (*S.D.*) = 2.03, range 19–28 years). They all gave written informed consent in accordance with the guidelines of the ethical standards of the Declaration of Helsinki. All procedures were approved by the local Institutional Ethical Review Board.

2.2. Self-reports

Personality traits were determined on the day of electroencephalography (EEG) recording. Participants completed the *Anxiety-Sensitivity Index* (ASI-Revised; Peterson & Reiss, 1987; Reiss, Peterson, Gursky, & McNally, 1986), a 16-item self-report questionnaire measuring the fear of bodily sensations associated with arousal. Trait anxiety was measured by the *State-Trait Anxiety Inventory* (*STAI*) (Laux, Glanzmann, Schaffner, & Spielberger, 1981) which consists of 40 statements differentiating between *trait anxiety* (TA) and the temporary condition of *state anxiety*. As we focus on stable emotional traits, the state-anxiety score was not further considered.

2.3. Stimuli and procedure

In a Go/Nogo-paradigm, two words were presented on a computer screen in randomized order while EEG was recorded. The stimuli were displayed for 300 ms. The whole experiment took 15 min and consisted of two blocks of 100 stimuli each. The subjects had to react upon appearance of the *Go-stimulus* (press) and to refrain from responding upon appearance of the *Nogo-stimulus* (stop). Responses were given by pressing a response button either with the right or left hand thumb, counterbalanced across subjects. The intertrial interval was 1600 ms. Subjects were asked to respond within a reaction-time (RT) deadline. When RTs exceeded this deadline, an auditory feedback stimulus (1000 Hz, 60 dB sound pressure level (SPL)) was given. Subject's responses to Go- and Nogo-stimuli, and RTs were recorded. During the task, participants did not receive any feedback on their performance.

2.4. Data processing

EEG data were recorded from 24 Ag-AgCl electrodes (Fpz, Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, FC3, FC4, FC5, FC6, C3, C4, C7, C8, Pz, P3, P4, P7, P8, Oz, O1, O2, left mastoid – M1, right mastoid – M2) against a reference electrode located at Cz. Eye movements were monitored and recorded by means of two lateral and four vertical EOG electrodes. The sampling rate of all recordings was 500 Hz, applying a filter bandwidth 0–80 Hz to the EEG. Electrode impedances were kept below 5 kΩ. EEG was filtered off-line from 0.5 to 16 Hz and re-referenced to linked mastoids. Artefact-rejection procedures were applied twice: automatically, using an amplitude-threshold of ±80 μV, and visually, rejecting all trials contaminated by technical artefacts. Horizontal and vertical eye movements of the accepted trials were corrected by means of a linear regression for EOG correction (Gratton, Coles, & Donchin, 1983).

2.5. Data analysis

The following electrodes were selected for statistical analysis: Fz, FCz and Cz (Falkenstein et al., 1999). Components of interest were the N2 and P3. After averaging, amplitudes in Go- and Nogo-trials were evaluated using correct trials only. After digital low-pass filtering, the amplitudes were assessed relative to a 200 ms pre-stimulus baseline. The N2 was defined as the most negative peak occurring 200–300 ms after stimulus onset and was measured relative to baseline. The P3 was defined as the most positive peak occurring 300–500 ms after stimulus onset and was measured relative to baseline. For linear regression analyses mean amplitudes and latencies were determined averaging across electrode positions. This scoring method is comparable to that of other studies (Beste et al., 2008).

Variability in Go- and Nogo-components attributable to personality traits was assessed by hierarchical linear regression analyses (Tabachnik & Fidell, 2007). The continuous quantitative variables AS and TA which were correlated were introduced as independent variables in the model. To examine whether age and gender accounted for additional variance in the N2 and P3, these variables were included as additional regressors in the analyses. Go- and Nogo-N2 and -P3 components were used as dependent variables in separate analyses. In order to highlight the differences in waveshapes between the ERPs of Go- and Nogo-trials and to further illustrate the anxiety-related effect on Nogo-potentials, we conducted additional *analyses of variances* (ANOVAs). First, we grouped subjects according to the STAI trait median score (Trait median = 32) into either a *low TA-group* or a *high TA-group*. *Second, we* defined a *low AS-group* and a *high AS-group* according to the ASI median score (ASI median = 14). The factors *AS-group* (low, high) and *TA-group* (low, high) were included as between-subject factors in separate repeated-measure analyses of variance with *electrode* (three levels: Fz, FCz, Cz) and *condition* (two levels: Go/Nogo) as within-subject factors. According to Mauchly's test, sphericity cannot be assumed and the Greenhouse–Geisser correction was employed to correct for sphericity.

Table 1
Regression coefficients (R^2, ΔR^2) and statistical results of hierarchical linear regression analyses on reaction times and false alarm rates with respect to the influence of trait anxiety, anxiety sensitivity, age and gender are shown. Trait anxiety and anxiety sensitivity were significantly associated with reduced false alarm rates.

Variables	RTs			False alarm rates		
	R^2	ΔR^2	$p<$	R^2	ΔR^2	$p<$
TA alone	0.000		0.91	0.246		0.001
TA added second		0.000	0.99		0.163	0.003
AS alone	0.001		0.81	0.096		0.03
AS added second		0.001	0.83		0.013	0.36
Age added third		0.000	0.89		0.002	0.75
Gender added fourth		0.101	0.03		0.001	0.81
Full model	0.103		0.26	0.261		0.006

AS: anxiety sensitivity; TA: trait anxiety; RTs: reaction times. R^2 illustrates the regression model, whereas ΔR^2 illustrates the improvement of the regression model when additional independent variables are considered.

Because of the Bonferroni correction for multiple comparisons, we only reported *p*-values exceeding 0.001.

3. Results

3.1. Behavioural data

The mean ASI score was 18.63 (*standard deviation S.D.* = 11.42, range = 3–57), the mean STAI trait score 33.90 (*S.D.* = 8.36, range = 21–64). The ASI and STAI trait scores were significantly correlated ($r = 0.38$; $p < 0.01$; $R^2 = 0.14$). Mean reaction times were 287.04 ms (*S.D.* = 21.77) and the mean false alarm rate 7.1% (*S.D.* = 2.24). To assess the effects of anxiety on the behavioural performance (RTs, false alarm rates), hierarchical regression analyses with AS, TA, age and gender as regressors were performed (see Table 1). None of the independent variables had significant influence on RTs ($F(4, 48) = 1.38$; p = n.s.; $R^2 = 0.103$). In combination, the four predictors accounted for 26% of the variance in false alarm rates ($F(4, 48) = 4.25$; $p < 0.01$). The predictors were then each examined with the variance associated with the other predictors removed. As hypothesized, TA and AS had significant influence on the criterion variable (TA: $R^2 = 0.246$; AS: $R^2 = 0.096$). Gender and age did not account for additional variance. Particularly, a higher level of anxiety indicates a decrease of false alarms.

3.2. Neurophysiological data – multiple regression analyses

The grand means of the ERP waveforms are shown in Fig. 1.

3.3. N2

A hierarchical regression analysis was employed for Nogo-N2 amplitudes. The inclusion of all predictor variables accounted for 58.5% of the variance in the criterion variable ($F(4, 48) = 34.39$; $p < 0.001$) (see Table 2). Then, each predictor was examined with the variance associated with the other predictors removed. Whereas TA accounted for 57.8% of the variance in the Nogo-N2 (see Fig. 2a for scatter plot), AS predicts 14.5%. No significant correlations were obtained for age and gender. Hierarchical regression analysis on Go-N2 amplitudes revealed that none of the regressors had significant influence ($F(4, 48) = 0.98$; p = n.s.; $R^2 = 0.075$). Regression analyses on Go- and Nogo-N2 latencies showed that TA, AS, gender and age had no significant impact on the criterion variables (Nogo-N2 latencies: $F(4, 48) = 0.59$; p = n.s.; $R^2 = 0.047$; Go-N2 latencies: $F(4, 48) = 0.58$; p = n.s.; $R^2 = 0.046$). To sum up, trait anxiety was principally associated with Nogo-N2 amplitudes.

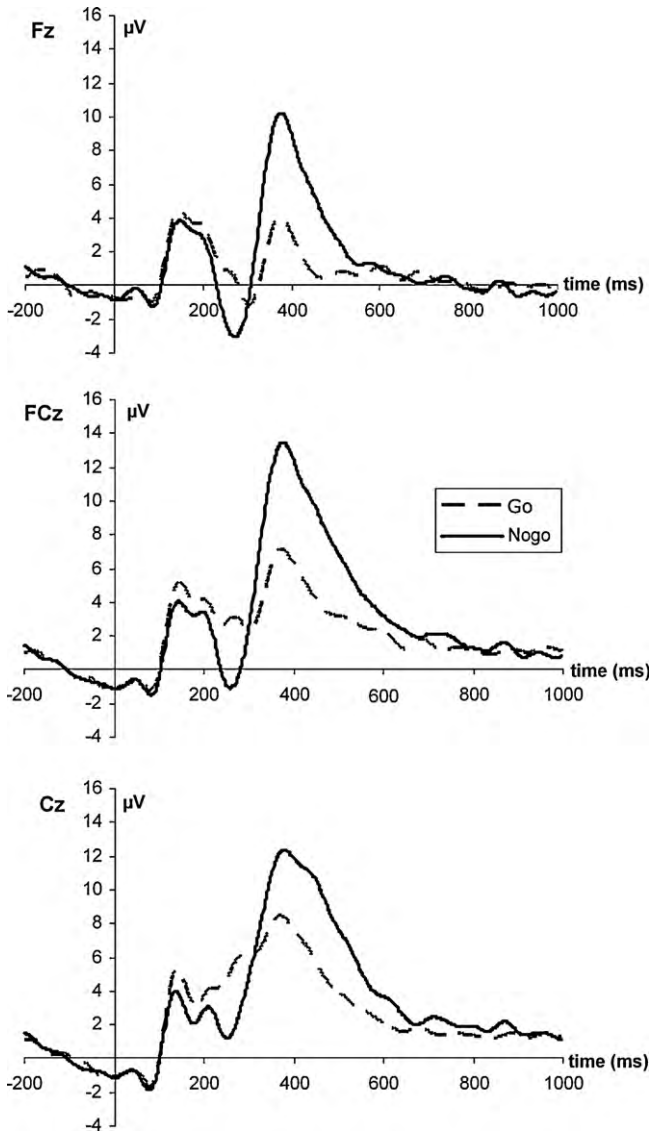

Fig. 1. Grand average waveforms of the ERPs across all subjects at electrodes Fz, FCz and Cz. Dashed lines denote the ERP on Go-trials, solid lines denote the ERP on Nogo-trials.

3.4. P3

Moreover, the hierarchical regression analysis on Nogo-P3 amplitudes showed that 35% of the variability in the criterion variable can be explained by the statistical model ($F(4, 48) = 6.478$; $p < 0.001$) (see Table 2). More than two thirds of the variability in Nogo-P3 amplitudes is predicted by AS (see Fig. 2b for scatter plot). Adding TA to the prediction, results in a small increment of 6% in R^2. The inclusion of age and gender had no additional impact on the dependent variable. Another hierarchical regression analysis revealed that the inclusion of all predictors did not accounted significantly for variance in the Go-P3 amplitudes ($F(4, 48) = 0.129$; p = n.s.; $R^2 = 0.011$). Regression analyses on Nogo-P3 latencies showed that 19% of the variance in the criterion variable was accounted for by the statistical model ($F(4, 48) = 2.88$; $p < 0.05$). Anxiety sensitivity captured 14% of the variance while the addition of TA, age and gender to the equation did not result in a significant increment of R^2. In the hierarchical regression analysis of Go-P3 latencies, 16.6% of the variance were explained by the full model ($F(4, 48) = 2.32$; p < n.s.). AS predicted 11% of the variance, while the inclusion of TA, age and gender had no significant impact on Go-P3

Table 2
Regression coefficients (R^2, ΔR^2) and statistical results of hierarchical linear regression analyses on Nogo-N2 and -P3 amplitudes with respect to the effects of trait anxiety, anxiety sensitivity, gender and age are given. AS specifically affects the Nogo-P3, whereas TA mainly influences the Nogo-N2.

Variables	Nogo-N2 amplitude			Nogo-P3 amplitude		
	R^2	ΔR^2	$p<$	R^2	ΔR^2	$p<$
TA alone	0.577		0.001	0.000		0.95
TA added second		0.437	0.001		0.055	0.05
AS alone	0.144		0.006	0.284		0.001
AS added second		0.005	0.47		0.338	0.001
Age added third		0.003	0.58		0.001	0.81
Gender added fourth		0.000	0.97		0.011	0.37
Full model	0.584		0.001	0.351		0.001

AS: anxiety sensitivity; TA: trait anxiety. Probability values are two-tailed. R^2 illustrates the regression model, whereas ΔR^2 illustrates the improvement of the regression model when additional independent variables are considered.

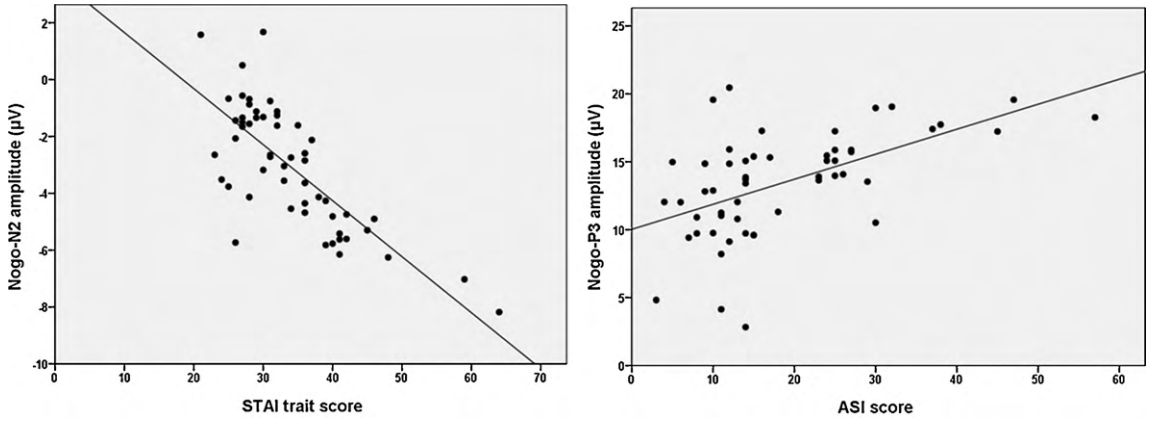

Fig. 2. (A) Scatter plot of the relation between Nogo-N2 amplitudes and trait-anxiety (TA) scores. (B) Scatter plot of the relation between Nogo-P3 amplitudes and anxiety sensitivity (AS) scores.

latencies. To conclude, anxiety sensitivity mainly affected Nogo-P3 amplitudes.

3.5. Neurophysiological data – ANOVA

To further illustrate this differential effect of TA on Nogo-N2 amplitudes and of AS on Nogo-P3 amplitudes we performed repeated-measure ANOVAs with electrode (Fz, FCz, and Cz) and condition (Go and Nogo) as within-subject factors. TA-group (high and low) and AS-group (high and low) were included as between-subject factors in separate analyses.[2] Statistical results with respect to the effects of electrode and condition are presented in Table 3. Effects of TA-groups and AS-groups on Nogo-N2 and -P3 are presented in Table 4.

3.6. Nogo-N2

The ANOVAs on Nogo-N2 amplitudes revealed main effects of electrode, condition and electrode × condition interactions. As shown in Fig. 3, the high TA-group revealed larger N2 amplitudes than the low TA-group in the Nogo-condition: condition × TA-group interaction ($F(1, 52) = 26.92$; $p = 0.001$; $\eta^2 = 0.34$) (Fig. 3a). With respect to AS, Nogo-N2 amplitudes were slightly enhanced in the high AS-group (-1.20 ± 0.21) compared to the low AS-group (-0.20 ± 0.21) ($F(1, 52) = 12.078$; $p < 0.001$; $\eta^2 = 0.188$).

3.7. Nogo-P3

The ANOVAs on Nogo-P3 amplitudes all revealed main effects of electrode, condition and electrode × condition interactions. As can be seen in Fig. 4, the high AS-group showed larger P3 amplitudes (11.27 ± 0.53) than the low AS-group in Nogo-trials: condition × AS-group interaction ($F(1, 52) = 11.96$; $p = 0.001$; $\eta^2 = 0.19$). P3 amplitudes were not different for the two TA-groups.

4. Discussion

We examined response inhibition in healthy individuals with respect to the influence of different anxiety-related personality traits. To the best of our knowledge, this study is the first showing that the Nogo-N2 and Nogo-P3, which reflect sub-processes of response inhibition (Falkenstein et al., 1999), are differentially modulated by trait anxiety and anxiety sensitivity in healthy subjects. In line with our prediction, the Nogo-N2 and Nogo-P3 were associated with enhanced anxiety. Particularly, the Nogo-N2 was differentially modulated, that is a higher level of trait anxiety was mainly related to larger Nogo-N2, while fewer effects were obtained for anxiety sensitivity. The Nogo-P3 was best predicted by anxiety sensitivity, while it was slightly affected by trait anxiety.

4.1. Behavioural performance

Behavioural data reflect and corroborate ERP findings as TA and AS contributed significantly to the variance in false alarm rates. Higher levels of trait anxiety and anxiety sensitivity were correlated

[2] In a first analysis, we incorporated the factors row (F-electrodes, FC-electrodes and C-electrodes), laterality (left, central, right) and condition (Go, Nogo) as within-subject factors and group as between subject-factor (Beste et al., 2008). We accounted for a significant row × laterality × condition × group interaction (F's > 3.1; $p < .01$). Subsequent repeated-measure ANOVAs for the left, middle and right electrode positions revealed a row × condition × group interaction for the middle electrodes, but not for the left- and right-sided electrodes for the Nogo-N2 and Nogo-P3 (all F's < 1; $p > .3$), suggesting that the effects obtained were not differentially lateralized in the different groups. Therefore, we restricted all further analyses on Nogo-N2 and Nogo-P3 to the middle line of electrodes (i.e. Fz, FCz and Cz).

Table 3
Statistical results of ANOVAs (test statistic, two-tailed) with respect to the effects of electrode and condition on Nogo-N2 and -P3 amplitudes.

	N2 amplitudes		P3 amplitudes	
	AS-groups	TA-groups	AS-groups	TA-groups
Electrode	$F(1.6, 80.7) = 4.03$ $p < 0.001$ $\eta^2 = 0.396$	$F(1.5, 77.2) = 33.20$ $p < 0.001$ $\eta^2 = 0.39$	$F(1.7, 88.9) = 31.86$ $p < 0.001$ $\eta^2 = 0.38$	$F(1.7, 89.2) = 32.19$ $p < 0.001$ $\eta^2 = 0.38$
Condition	$F(1, 52) = 82.81$ $p < 0.001$ $\eta^2 = 0.614$	$F(1, 52) = 116.64$ $p < 0.001$ $\eta^2 = 0.69$	$F(1, 52) = 195.04$ $p < 0.001$ $\eta^2 = 0.79$	$F(1, 52) = 162.40$ $p < 0.001$ $\eta^2 = 0.76$
Electrode × condition	$F(1.4, 72.9) = 34.13$ $p < 0.001$ $\eta^2 = 0.396$	$F(1.3, 69.2) = 34.33$ $p < 0.001$ $\eta^2 = 0.40$	$F(1.2, 59.6) = 22.29$ $p < 0.001$ $\eta^2 = 0.30$	$F(1.2, 59.7) = 22.65$ $p < 0.001$ $\eta^2 = 0.30$

AS: anxiety sensitivity; TA: trait anxiety.

Table 4
Mean amplitudes (in μV) with standard errors of the means and statistical results for Nogo-components with respect to the effects of groups of high and low anxiety sensitivity and trait anxiety are given. AS specifically affects Nogo-P3, whereas TA significantly influences the Nogo-N2.

	Anxiety sensitivity		Test statistic (two-tailed)	Trait anxiety		Test statistic (two-tailed)
	Low	High		Low	High	
Nogo-N2 amplitude	−2.26 ± 0.39	−3.86 ± 0.39	$F(1, 52) = 4.03$ $p = 0.05$ $\eta^2 = 0.07$	−1.63 ± 0.31	−4.50 ± 0.31	$F(1, 52) = 26.92$ $p = 0.001$ $\eta^2 = 0.34$
Nogo-P3 amplitude	11.83 ± 0.68	15.09 ± 0.68	$F(1, 52) = 11.96$ $p = 0.001$ $\eta^2 = 0.19$	13.20 ± 0.75	13.72 ± 0.75	$F(1, 52) = 1.25$ $p =$ n.s. $\eta^2 = 0.02$

with fewer false alarms reflecting enhanced response inhibition. As no significant influences on RTs were obtained, the results are highly specific and unlikely to be biased by speed-accuracy-trade-off effects (SAT).

4.2. N2-effects

As expected, and in accordance with the behavioural data, Nogo-N2 amplitudes were significantly enhanced in anxious subjects,

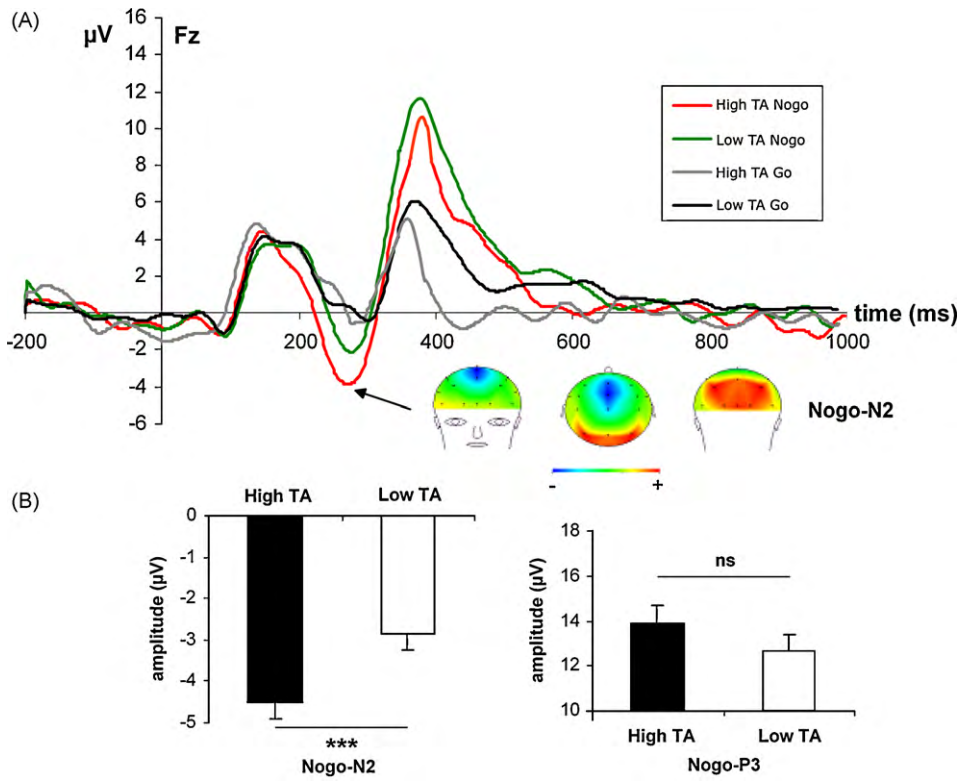

Fig. 3. (A) Grand average ERP waveforms for the low and high trait anxiety (TA) groups. Black and green lines denote the potentials on Go- and Nogo-trials for the low TA-group. Grey and red lines denote the potentials on Go- and Nogo-trials for the high TA-group. Additionally, the topography of the Nogo-N2 is given (collapsed over both groups). (B) Plot of the mean amplitudes of the Nogo-N2 and Nogo-P3 for the high and low TA-group. A difference is seen for the Nogo-N2, but not for the Nogo-P3.

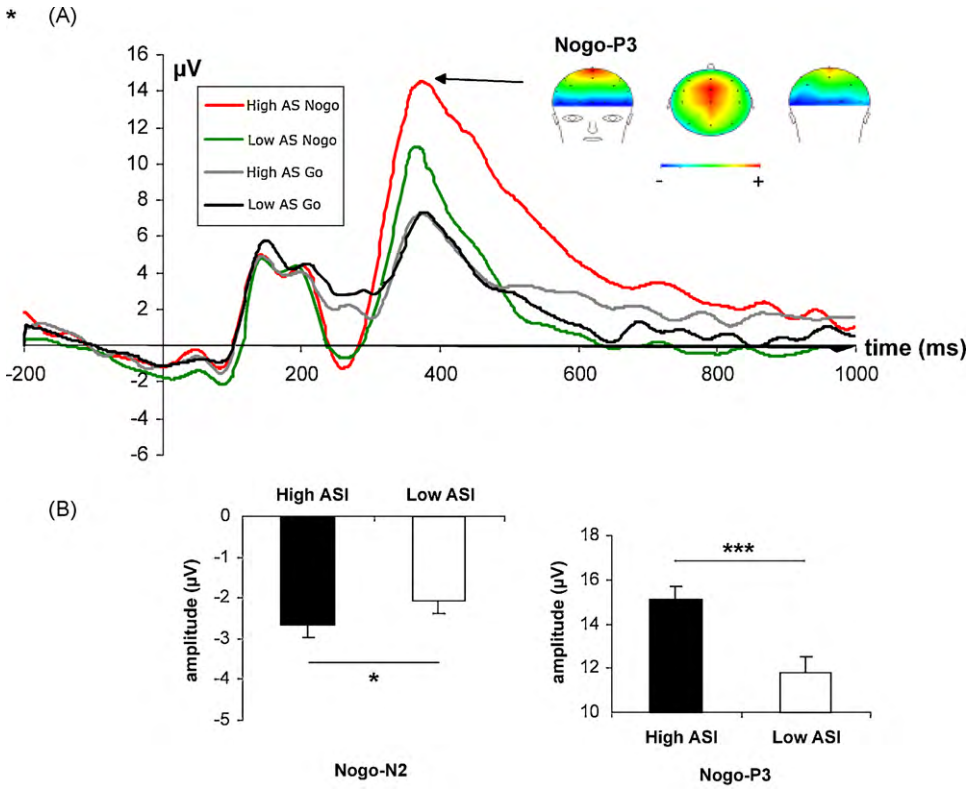

Fig. 4. (A) Grand average ERP waveforms for the low and high anxiety sensitivity (AS) groups. Black and green lines denote the potentials on Go- and Nogo-trials for the low AS-group. Grey and red lines denote the potentials on Go- and Nogo-trials for the high AS-group. Additionally, the topography of the Nogo-P3 is given (collapsed over both groups). (B) Plot of the mean amplitudes of the Nogo-N2 and Nogo-P3 for the high and low AS-group. A difference is seen for the Nogo-P3 and for the Nogo-N2.

particularly with respect to trait anxiety. This anxiety-related effect corroborates studies that reported enhanced Nogo-N2 amplitudes in patients with anxiety disorders (e.g. Ruchsow et al., 2007) and anxious subjects (Righi et al., 2009). Yet, others found reduced Nogo-N2 amplitudes in patients with anxiety disorders compared to healthy controls (Kim et al., 2007). High trait-anxious people are more cautious, and exert more cognitive control than low anxious people (McWilliams & Cox, 2001), for example to inhibit inappropriate motor actions (McNally, 2002), which may result in an increased inhibition-related Nogo-N2 response. This finding and the significant negative correlation of TA scores with N2 amplitudes and false alarm rates support the assumption that the Nogo-N2 specifically reflects (pre-) motor inhibition processes (Falkenstein et al., 1999). Anxiety sensitivity, which is rather related to a monitoring aspect of anxiety, had a smaller effect on the Nogo-N2.

Studies on the behavioural inhibition (BIS) and behavioural activation system (BAS) are in line with our current finding that anxiety traits are related to enhanced response inhibition. It is proposed that anxiety is an over-activation of the BIS that responds to aversive stimuli and produces behavioural inhibition, increased arousal and attention to outputs (Gray, 1982). Furthermore, it is assumed that high BIS levels represent a vulnerability factor for anxiety or depression (Johnson, Turner, & Iwaka, 2003; McDermott et al., 2009). This is relevant, as these psychiatric disorders are frequently accompanied by alterations in response inhibition processes.

Another effective way to measure response inhibition processes and mainly the capacity to maintain attention is the *Sustained Attention to Response Task* (SART) (Manly et al., 1999). The SART is a variant of a Go/Nogo-paradigm, in which the Nogo-stimuli were presented more rarely and more unpredictably than in a standard Go/Nogo design (Dockree, Kelly, Robertson, Reilly, & Foxe, 2005). Studies employing SART found that the amplitudes of Nogo-components were negatively correlated with the Nogo-stimulus probability (Braver, Barch, Gray, Molfese, & Snyder, 2001; Bruin & Wijers, 2002) and that the performance to sustain attention was modulated by individual differences in cognitive performance (Manly et al., 1999; Righi et al., 2009; Roche et al., 2005) or anxiety (Righi et al., 2009). However, compared to standard Nogo-paradigms, SART is determined to mainly investigate the capacity to sustain attention (Manly et al., 1999) and is more sensitive to variations in attentional performance, which may be due to the small Nogo-stimulus probability (Dockree et al., 2005; Manly et al., 1999; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997; Roche et al., 2005). Because of these attentional bias effects on ERPs, such as on Nogo-P3, we chose the standard Go/Nogo-paradigm to investigate response inhibition.

4.3. P3-effects

Consistent with previous studies, greater P3 amplitudes were observed in Nogo- than in Go-trials (e.g. Eimer, 1993). In contrast to the N2-component, trait anxiety had only an additional influence on the P3-amplitude. Instead, anxiety sensitivity contributed significantly to the variance in Nogo-P3 amplitudes. Anxiety sensitivity is specifically associated with the evaluation and fear of one's own bodily sensations (Domschke, Stevens, Pfleiderer, & Gerlach, 2010; McNally, 2002). We suggest that the tendency to monitor behavioural outcomes leads to the enhanced Nogo-P3 response, as reflected in our data. In this way, the finding supports the assumption that the Nogo-P3 reflects the evaluation of response inhibition (Beste, Dziobek, Hielscher, Willemssen, & Falkenstein, 2009; Beste et al., 2008; Righi et al., 2009; Schmajuk et al., 2006). Moreover, our results are consistent with other data showing a magnifying effect of anxiety or cognitive control on P3 amplitudes (Karch et al., 2008; Ruchsow et al., 2007).

Hierarchical regression showed that 19% of the variance in Nogo-P3 latencies could be explained by AS, TA, age and gender. Particularly AS predicted 14% of the variance in the dependent variable. These findings are in line with the *processing efficiency theory* of Eysenck and Calvo (1992), which mainly provides an explanation of the effects of anxiety on task performance (Eysenck & Calvo, 1992; Eysenck, Derakshan, Santos, & Calvo, 2007; Murray & Janelle, 2007). According to this theory, anxious subjects are thought to be cautious, diligent and ruminative. Moreover, high levels of anxiety are assumed to activate a control system that provides extra processing resources to the task to improve performance. This may be reflected by lengthened processing times (Eysenck & Calvo, 1992) and the above reported prolonged latencies in anxiety. Furthermore, behavioural data show that high levels of anxiety are associated with high quality of task performance, as anxious subjects exhibit fewer false alarms than low anxious subjects. Thus, both behavioural performance and enhanced Nogo-amplitudes illustrate that anxiety is related to an over-inhibition of responses. This is also in line with Eysenck and Calvo (1992) who assume that anxious subjects show enhanced cognitive effort to avoid aversive states and to reach a certain level of performance. To conclude, these findings point to a dysfunctional cerebral activation in anxious people when response inhibition is required (Huang et al., 2009).

The relationship between AS and TA has been a matter of controversial debates (McWilliams & Cox, 2001). In our study, TA and AS are moderately intercorrelated ($r = 0.38$), which provides an argument for the hypothesis that these personality factors represent a common concept (Lilienfeld, 1996). In contrast, we found that AS and TA are related to different neurophysiological processes, namely Nogo-N2 and -P3. TA, which primarily refers to cognitive symptoms of anxiety and a tendency to respond fearfully in general (McWilliams & Cox, 2001), was primarily associated with the Nogo-N2, reflecting pre-motor response inhibition. AS, focusing on self-evaluation of physical and psychological symptoms (McWilliams & Cox, 2001), was mainly correlated with the Nogo-P3 which represents the evaluation of the preceding response (Roche et al., 2005) and of the successful outcome of the inhibition process (Schmajuk et al., 2006). Thus, both AS and Nogo-P3 comprise an evaluative component, which is reflected by the strong relationship between AS and Nogo-P3 in our data. To conclude, although the concepts of TA and AS overlap phenotypically in our study, we found a neurophysiological dissociation. This finding provides support for the assumption that TA and AS represent "related, but distinct concepts" of anxiety (McNally, 1996; McWilliams & Cox, 2001; Muris et al., 2001) differentially associated with distinguishable neuronal processes.

4.4. Common neuronal network underlying anxiety and response inhibition

The reported functional relation between anxiety and ERPs may well be based on a common neuronal network. Emotional traits and cognitive functions, such as response inhibition, are related to the same neuroanatomical region, i.e. the ACC (Bokura et al., 2001; Bush et al., 2000; Sehlmeyer et al., 2009). Moreover, response inhibition and anxiety-related processes share neurochemical substrates, such as the dopaminergic (DA) and serotonergic system (Beste et al., 2009; Beste, Willemssen, et al., 2010; Fallgatter, Jatzke, Bartsch, Hamelbeck, & Lesch, 1999; Segman et al., 2002; Yoon, Yang, Lee, & Kim, 2008). For example, the association between enhanced Nogo-N2 and -P3 and anxiety-related personality traits might be interpreted as an expression of increased dopaminergic activity during response inhibition in anxious subjects. Common underlying factors might influence both characteristics observed here, thus personality traits and electrophysiological components might be affected by common biochemical or genetic factors. Identification of these underlying factors requires further examination.

4.5. Conclusion

In summary, the results show that anxiety-related personality traits, such as anxiety sensitivity and trait anxiety, differentially modulate dissociable psychophysiological sub-processes of response inhibition. Even non-affective stimulus material may do so, suggesting a strong generalizability of the examined personality traits and its influence on executive functions. ERPs yielded the psychophysiological correlate of an over-inhibition in anxious people. Finally, our data demonstrate that the assessment of anxiety traits may be important for studies investigating response inhibition functions.

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