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EEG theta/beta ratio as a potential biomarker for attentional control and resilience against deleterious effects of stress on attention

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Abstract Anxious stress compromises cognitive executive performance. This occurs, for instance, in cognitive performance anxiety (CPA), in which anxiety about one's cognitive performance causes that performance to actually deteriorate (e.g., test anxiety). This is thought to result from a prefrontal cortically (PFC) mediated failure of top-down attentional control over stress-induced automatic processing of threat-related information. In addition, stress-induced increased catecholamine influx into the PFC may directly compromise attentional function. Previous research has suggested that the ratio between resting state electroencephalographic (EEG) low- and high-frequency power (the theta/beta ratio) is related to trait attentional control, which might moderate these effects of stress on attentional function. The goals of the present study were to test the novel prediction that theta/beta ratio moderates the deleterious effects of CPA-like anxious stress on state attentional control and to replicate a previous finding that the theta/beta ratio is related to self-reported trait attentional control. After recording of baseline frontal EEG signals, 77 participants performed a stress induction or a control procedure. Trait attentional control was assessed with the Attentional Control Scale, whereas stress-induced changes in attentional control and anxiety were measured with self-report visual analogue scales. The hypothesized moderating influence of theta/beta ratio on the effects of stress on state attentional control was confirmed. Theta/beta ratio explained 28% of the variance in stress-induced deterioration of self-reported attentional control. The negative relationship between theta/beta ratio and trait attentional control was replicated ($r = -.33$). The theta/beta ratio reflects, likely prefrontally mediated, attentional control, and should be a useful biomarker for the study of CPA and other anxiety–cognition interactions.

Keywords Test anxiety · Stress · Attentional control · Cognitive performance · Theta/beta ratio

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Anxiety and psychological stress are associated with suboptimal attentional control and cognitive performance. For instance, patients suffering from various anxiety disorders report problems with concentration, which is also a diagnostic criterion for posttraumatic stress disorder (PTSD) and generalized anxiety disorder (GAD; American Psychiatric Association, 2000). It has been shown in laboratory settings that acute stress manipulations can decrease executive cognitive performance, using for instance tasks measuring working memory (WM) or divided attention (for an overview, see Eysenck, Derakshan, Santos, & Calvo, 2007). In real life, test anxiety is associated with poorer academic performance and successful treatment of test anxiety leads to performance improvement, which also testifies to the causal effect of anxiety on cognitive function (Hembree, 1988). Test anxiety is an instance of performance anxiety, which has been defined as "a strong but delimited fear that severely compromises an individual's capacity to execute a task at a level that could be reasonably expected" (Powel, 2004, p. 804). We will use the term *cognitive performance anxiety* (CPA) to refer to anxiety that concerns specifically cognitive performance, including test anxiety. Although (cognitive) performance anxiety is associated with trait tendencies of fear of negative evaluation by others and social anxiety (see Bögels et al., 2010; but see also Blöte, Kint, Miers, & Westenberg, 2009), CPA can be a noticeably isolated phenomenon that manifests

only in delimited contexts, and as such is transient in nature (Powel, 2004).

A prominent current cognitive theory explaining why CPA is associated with poor cognitive performance is the attentional control theory of Eysenck et al. (2007). This theory holds that attentional control—the key executive cognitive function—is determined by a dual-process system wherein a bottom-up, stimulus-driven attention network is regulated by a top-down, strategic attention network. The top-down network is thought to reside in more anterior (prefrontal) areas of the brain and the stimulus-driven system is likely more posterior and subcortical in origin (Bishop, 2008; Eysenck et al., 2007; Rossi, Pessoa, Desimone, & Ungerleider, 2009). Good executive cognitive performance and attentional control require balance between these systems. During states of anxiety, increased influence of stimulus-driven attention allows for better detection and processing of salient and threat-related information. However, this disrupts this balance and taxes scarce executive resources, resulting in compromised attentional control and suboptimal cognitive executive performance. This increased stimulus-driven attention, or "threat biased attention," can concern internal as well as external information (Eysenck et al., 2007).

Automatic attentional biases to threat-related external stimuli occur in all anxiety disorders, for visual as well as auditory stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Harvey, Watkins, Mansell, & Shafran, 2004; Mogg & Bradley, 1998). Automatic attention to internal salient information manifests as worry, that is, perseverative negative thoughts about future events that are initially automatically generated and are experienced as uncontrollable (Hirsch & Mathews, 2012). Worry and related processes play a prominent role in test anxiety and CPA. A distinction has been made between two components of the experience of test-anxiety. *Emotional* anxiety concerns mainly the experience of the visceral anxiety response (i.e., increased sympathetic autonomous nervous system activity), whereas *cognitive* anxiety refers to intrusive and perseverative worrying thoughts such as negative projections about the feared cognitive underperformance and evaluation thereof by others (see Cassady & Johnson, 2002; Sarason, 1984). It is this cognitive component of anxiety (e.g., intrusive worries about failing a test), that has been most clearly associated with affected academic performance (Hembree, 1988; Sarason, 1984). The occurrence of worry and intrusions is related to individual differences in trait attentional control capacity (Hagenaars & Putman, 2011; Koster, Soetens, Braet, & De Raedt, 2008; Verwoerd, de Jong, & Wessel, 2008) just as attentional control is related to automatic processing of external threatening stimuli (Bardeen & Orcutt, 2011; Derryberry & Reed, 2002; Putman, Arias-Garcia, Pantazi, & van Schie, 2012; Schoorl, Putman, van der Werff, & van der Does, 2013). Because of the assumed role of automatic threat processing in the effects of stress on cognitive executive function (Eysenck et al., 2007; Sarason, 1984), such findings imply that reduced attentional control can be a risk-factor factor for the cognitive deleterious effects of CPA.

Biological psychology offers additional explanations for effects of anxious stress such as CPA on attentional control and cognitive performance. The top-down attentional network is based in the prefrontal cortex (PFC), most notably the (dorso)lateral PFC (Arnsten, 2009; Arnsten & Rubia, 2012; Bishop, 2008). Under stress, the influx of the catecholamines noradrenaline (NA) and dopamine (DA) into the PFC is immediately increased. Although moderate increases of these catecholamines are beneficial to attentional and WM performance, too high concentrations of either will compromise functioning of prefrontal attentional networks (Arnsten, 2009). In the case of prolonged stressors, increased activation of glucocorticoid receptors, resulting from the slower release of the hormone cortisol, might cause additional disruption of PFC executive functioning (e.g., Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; but see also Oei, Tollenaar, Spinhoven, & Elzinga, 2009).

Resting-state electroencephalography (EEG) provides a potential biomarker for basal PFC-mediated attentional control. Poor functioning of PFC mediated attentional and inhibitory functions as seen in attention deficit disorder (ADD) and attention deficit/hyperactivity disorder (ADHD) is robustly related to an increased ratio between power in the slow theta frequency band and the fast beta band (see Barry, Clarke, & Johnstone, 2003). This increased theta/beta ratio is observed in ADD and more pronounced in ADHD (Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002). Administration of drugs that increase PFC attentional network function through enhancement of postsynaptic catecholamine function (Arnsten, 2009), normalizes the theta/beta ratio in AD(H)D (Clarke, Barry, McCarthy, Selikowitz, & Johnstone, 2007). It has been speculated that an increased theta/beta ratio (relatively greater theta than beta power) may reflect more involvement of subcortical (limbic) as opposed to cortical structures in inhibitory cortical control over subcortically driven motivational tendencies and automatic responses (Schutter & van Honk, 2005). Taken together, these lines of research imply that theta/beta ratio should be related to attentional control and affect regulation (Bishop, 2008; Buhle et al., 2013). We (Putman, van Peer, Maimari, & van der Werff, 2010) previously reported that theta/beta ratio is also increased in healthy young students with lower scores on a self-report measure of trait attentional control (the Attentional Control Scale, or ACS; Derryberry & Reed, 2002). This suggests that the relationship between attentional performance and theta/beta ratio might reflect a continuum of brain-behavior correlation of which AD(H)D represents but an extreme end, rather than suggesting a qualitative difference between the neural correlates of attentional control in AD(H)D patients and healthy individuals. Baseline resting state theta/beta ratio has been

related to stimulus evoked behavior (e.g., Massar, Rossi, Schutter, & Kenemans, 2012; Putman et al., 2010; Schutter & van Honk, 2005), self-reported emotional and motivational traits (Putman et al., 2010) and psychiatric diagnosis reflecting dynamic behavior over extended periods (Clarke et al., 2002). Interindividual variance of the ratio thus seems to reflect the interindividual variance of some default brain state or organization predisposing for differential responding to environmental challenges. All in all, there are indications that the theta/beta ratio provides a measure to study individual differences in attentional control and as such might be a useful biomarker in the study of CPA. As we mentioned, CPA is a transient state and it would be of interest to determine if theta/beta ratio is only related to trait attentional control, or if it can indeed also predict the stress-induced transient decline of attentional control as seen in CPA.

In the present study, we sought to replicate the previous finding of a negative correlation between theta/beta ratio and trait attentional control, as measured with the ACS. In addition and most importantly, we tested the novel predictions that ACS score and specifically theta/beta ratio moderate the deleterious effects of an acute psychosocial stress manipulation on state attentional control, such that higher theta/beta ratio or lower ACS score predict stronger stress-induced decline of attentional control. Putman et al. (2010) reported relations between non-specific frontally recorded theta/beta ratio and attentional control in unselected participants. To replicate that study and because of above outlined assumptions that theta/beta ratio may reflect involvement of the prefrontal cortex, we were primarily interested in theta/beta ratio recorded from frontal locations. To induce stress, we employed a bogus mental arithmetic test using negative feedback and threat of negative evaluation by others.

Method

Participants

A group of 80 participants who were recruited from campus were randomly allocated (stratified by gender) to a stress or control group (both $n = 40$). All participants provided written informed consent. The experimenters initially withheld information concerning the stress manipulation that was employed, but an extensive debriefing was done to ensure that participants understood how and why they had been deceived. The study was approved by the local review board.

Self-report trait questionnaires

Participants completed the trait version of the State–Trait Anxiety Inventory (STAI-t; Spielberger, 1983; Van der Ploeg, Defares, & Spielberger, 1980) and the Attentional Control Scale (ACS; Derryberry & Reed, 2002; Verwoerd, de Jong, & Wessel, 2006). The STAI-t assesses anxiety (range 20–80), and the ACS assesses attentional inhibition, attentional shift, and the capacity to flexibly generate new thoughts (range 20–80). The internal consistency of these questionnaires is generally good, and in the present study, Cronbach's alpha was .88 for STAI-t and .80 for ACS.

Visual Analogue Scales (VAS)

The VAS contained items to measure state anxiety and state attentional control, mixed with filler items. Participants had to indicate their acquiescence to the items by marking 100 mm lines, anchored "1" and "100" to the left and right end. Scores on the items "How anxious do you feel?," "How stressed do you feel?," and "How tense do you feel?" were averaged as the variable State Anxiety (SA; Cronbach's alpha = .753). Scores on the items "How focused are you?," "How concentrated are you?," and "How fast can you think?" were averaged as the variable state attentional control (state AC; Cronbach's alpha = .816).

General procedure

Participants were tested individually as part of a larger study that also assessed relations between EEG delta–beta coupling and threat-biased attention at rest (see Putman et al., 2012). EEG was measured at the beginning of the procedure. VAS scales were completed before and after the stress/control procedure and the STAI-t and ACS were completed one day later. Debriefing about the deceitful nature of the stress procedure took place at the end of the first day.

Stress/control procedures In the stress condition, participants performed a scripted bogus verbal mental arithmetic test, administered by a cold and stern experimenter in front of a supposedly running camera. Participants were first asked to introduce themselves to the camera, with emphasis on information about their academic performance. The experimenter then proceeded to explain that they were to perform a mental arithmetic test, tailored to suit the abilities of university students. The experimenter explained that the camera would record their performance so that later, other students could "rate and judge several other dimensions of your cognitive performance." Participants were told that they were going to perform blocks of slowly increasing difficulty levels if they passed a certain criterion level on the previous block and that a student of their academic background was expected to reach a considerably high level of performance. A stopwatch was noticeably used to emphasize time pressure (participants had to answer by multiple choice 6 s after the question was read out to them). After passing the "easy first block" and moving on to the "slightly more difficult block," they were told: "You

did not pass this block. We will do a really easy block again." This went back and forth between the first and second levels twice more, until participants were finally told that "You did not pass this block. There is no point in continuing so we will stop this test. We will continue with something else and come back to this at the end." This procedure took approximately 20 min. In the control condition, participants performed a computerized mental arithmetic task alone and without a camera. These sums were designed to be fairly easy, and no feedback was given at any time.

EEG recording

EEG was recorded for 8 min continuously in alternating 1-min blocks of eyes open/eyes closed recording. Scalp EEG recordings were acquired from the F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 10/20 positions using the ActiveTwo BioSemi system (BioSemi, The Netherlands). Electrodes placed at the left and right mastoids were used for offline re-referencing of the scalp signals to the average of the mastoid signals. Offline data processing was done in Brain Vision Analyzer V2.02 (Brain Products GmbH, Germany). A 0.1-Hz high-pass filter, 100-Hz low-pass filter, and 50-Hz notch filter were applied. Data were analyzed in four-second segments. The data were automatically corrected for ocular artifacts (Gratton, Coles, & Donchin, 1983) and segments containing remaining artifacts were removed. A fast Fourier transformation (with a resolution of 0.25 Hz, using a hamming window of 10%) was applied to calculate area power density for the beta (13–30 Hz), and theta (4–7 Hz) frequency bands. We were primarily interested in the average of the frontal signals (cf. Putman et al., 2010). The power densities for the three frontal electrodes were averaged into measures for frontal beta and frontal theta power density (and likewise for central and parietal electrodes). Because of nonnormality, power densities were log-normalized before calculation of the measures of interest, the theta/beta ratios.

Results

Participants

The EEG recordings of three participants were of poor quality, and their data were discarded. The mean age for the remaining 77 participants (30 males and 47 females; 39 in the control condition and 38 in the stress condition) was 19.6 years, SD = 2.5. We found no significant group differences at baseline for the gender, age, ACS, state AC or SA, and theta/beta ratio measures (all ps > .1). A group difference did emerge for trait anxiety, which was higher in the experimental than in the control condition [$t(75) = 2.123$, $p = .037$, $d = 0.479$]. See Table 1.

Table 1 Gender frequencies and means (and standard deviations) for other group characteristics, for the control (n = 39) and stress (n = 38) conditions

	Control	Stress	p
Gender	16m/23f	14m/24f	.707
Age	19.3 (1.9)	19.9 (2.9)	.256
STAI-t	37.1 (7.2)	41 (9.0)	.037
ACS	53.6 (7.9)	55.1 (7.6)	.411
Baseline state AC	61.5 (14.9)	56.7 (15.1)	.166
Baseline SA	24.6 (14.3)	27.6 (20.6)	.465
T/B ratio	1.11 (.19)	1.05 (.17)	.108

STAI-t = trait version of the State–Trait Anxiety Inventory, ACS = Attentional Control Scale, state AC = state attentional control, SA = state anxiety, T/B ratio = frontal theta/beta ratio

Bivariate relations between baseline self-report measures and frontal theta/beta ratio

STAI-t and ACS were negatively correlated (r = –.47, p < .001), as is commonly reported. Baseline SA was positively correlated to STAI-t (r = .35, p = .002), and baseline state AC was positively correlated to ACS (r = .34, p = .002). Frontal theta/beta ratio was not significantly correlated to STAI-t (r = .09, p = .438), but—replicating our previous finding—it was negatively correlated to ACS (r = –.33, p = .004), even when controlling for STAI-t (r = –.32, p = .004; cf. Putman et al., 2010). We observed no significant correlation between baseline state AC and frontal theta/beta ratio (r = –.07, p = .510). For comparison with a previous report (Putman et al., 2010), we also looked at the correlation between ACS and age, which showed a trend-level positive correlation (r = .22, p = .060), and at the correlation between age and frontal theta/beta ratio, which was not significant (r = –.15, p = .202; cf. Putman et al., 2010).

Effects of the stressor on state AC and SA

A repeated measures (rm)ANOVA for SA showed a significant Condition × Time interaction: $F(1, 75) = 18.175$, $p < .001$, $\eta_p^2 = .195$. The interaction remained significant when controlling for STAI-t: $F(1, 74) = 17.381$, $p < .001$, $\eta_p^2 = .190$. The interaction reflected that in the control group SA did not change significantly from the first measurement (M = 24.6, SD = 14.3) to the second measurement (M = 24.0, SD = 17.8), $t(38) < 1$, but did significantly rise in the stress group from M = 27.6 (SD = 20.6) to M = 44.7 (SD = 20.9): $t(37) = 4.871$, $p < .001$, $d = 0.824$. SA levels after the manipulation differed significantly between the groups: $t(75) = 4.668$, $p < .001$, $d = 1.067$. Thus, the stress manipulation was successful at elevating state anxiety levels.

An rmANOVA for state AC showed a significant Condition × Time interaction: $F(1, 75) = 24.185$, $p < .001$,

η_p^2 = .244. The interaction remained significant when controlling for STAI-t: $F(1, 74) = 21.928$, $p < .001$, $\eta_p^2 = .229$. The interaction reflected that in the control group state AC did not change from the first measurement ($M = 61.5$, $SD = 14.9$) to the second measurement ($M = 62.8$, $SD = 17.4$), $t(38) < 1$, but did significantly drop in the stress group from $M = 56.7$ ($SD = 15.1$) to $M = 37.7$ ($SD = 22.8$): $t(37) = 5.788$, $p < .001$, $d = 1.003$. State AC levels after the manipulation differed significantly between the two groups: $t(75) = 5.451$, $p < .001$, $d = 1.240$. Thus, the stress manipulation was successful at reducing state attentional control.

Moderation analyses for the role of ACS score

To test if ACS moderated the effect of the stressor on state AC, a hierarchical regression analysis was performed (Aiken & West, 1991) with ΔAC (state AC at $t = 1$ minus state AC at $t = 2$) as dependent variable; larger values of ΔAC indicate larger decreases in state AC. In the first step, condition and centered ACS score were entered as predictors. STAI-t was also entered, to control for the group difference in trait anxiety. In the second step, the Condition × Centered ACS Score interaction term was added to test for moderation. This showed significant independent effects of Condition (β = .51, $p < .001$) and ACS (β = –.19, $p = .05$) in the first step, but adding the Condition × ACS interaction term in the second step did not explain more variance than the second step (ΔR^2 was .011, $p = .292$). Explorative post-hoc testing showed a near significant negative correlation between ACS and ΔAC in the stress group ($r = –.310$, $p = .058$). In sum, although ACS and condition independently predicted ΔAC, we found no convincing evidence that ACS moderated the effect of the stress manipulation on state AC.

Moderation analyses for the role of frontal theta/beta ratio

To test whether frontal theta/beta ratio moderated the effect of the stressor on state AC, a separate hierarchical regression analysis was performed, with ΔAC again as the dependent variable. See Table 2.

In the first step, condition and centered frontal theta/beta ratio were entered as predictors. STAI-t was also entered, to control for the group difference in trait anxiety. This model showed $R^2 = .291$, $F(3, 73) = 10.009$, $p < .001$. In the second step, the Condition × Centered Frontal Theta/Beta Ratio interaction term was added, to test for moderation. This model showed $R^2 = .373$, $F(4, 72) = 10.697$, $p < .001$, with the addition of the interaction term explaining significantly more variance ($\Delta R^2 = .081$), $F(1, 72) = 9.334$, $p = .003$, confirming that the effect of condition on ΔAC was moderated by frontal theta/beta ratio.

The Condition × Frontal Theta/Beta Ratio interaction term also still added significantly to the model when we controlled for ACS and the ACS × Condition interaction ($\Delta R^2 = .07$, $p = .006$), showing that frontal theta/beta ratio explains unique additional variance in addition to self-reported trait attentional control.

Table 2 Regression model ($n = 77$) showing that frontal theta/beta ratio moderates the effect of the manipulation on cognitive stress sensitivity (ΔAC)

Predictor	β	*t*	sr^2	R^2
Step 1				.291***
STAI-t	–.003	0.034	.000	
Condition	.536	5.160	.258***	
T/B ratio	.222	2.198	.047*	
Step 2				.373***
STAI-t	–.011	0.116	.000	
Condition	.544	5.529	.266***	
T/B ratio	–.038	0.297	.001	
Condition × T/B ratio	.388	3.055	.081**	

sr^2 = semipartial correlation squared, STAI-t = trait version of the State–Trait Anxiety Inventory, T/B ratio = frontal theta/beta ratio. * $p < .05$, ** $p < .005$, *** $p < .001$.

The correlation between ΔAC and frontal theta/beta ratio in the stress group was $r = .53$, $p = .001$ ($r = –.05$, $p = .75$, in the control group). See Fig. 1 for scatterplots of these relations.

Finally, to further illustrate the nature of the interaction, a simple slope analysis was performed (Aiken & West, 1991). As can be observed in Fig. 2, state AC decreased (i.e., ΔAC increased) in the stress condition. This decrease in state AC was most pronounced for high frontal theta/beta ratio (+1 *SD*: β = .834, $t = 6.153$, $p < .001$), and to a lesser extent for mean frontal theta/beta ratio (β = .541, $t = 5.736$, $p < .001$), whereas there was only a trend for a reduction of state AC for low frontal theta/beta ratio (–1 *SD*: β = .248, $t = 1.866$, $p = .066$). Higher frontal theta/beta ratios are associated with stronger declines in state AC due to stress.

We exploratively analyzed the relations between theta/beta ratio and stress-induced changes in state anxiety. Using the same regression model as for ΔAC, but now with ΔSA as the dependent variable, we found no evidence for moderation by theta/beta ratio of the effect of condition on ΔSA (ΔR^2 for Step 2 of the model was .000, $p = .653$). At the statistical trend level, the model did show that, controlling for STAI-t and condition, larger theta/beta ratios predicted trend-level larger increases in state anxiety (β = –.203, $t = –1.933$, $p = .057$).

Relations to frontal, central, and parietal theta/beta ratios

Although our primary interest was with the frontal theta/beta ratio, we exploratively repeated the most important analyses with the central and parietal theta/beta ratios (cf. Putman et al., 2010; for one participant, the central and parietal recordings were lost due to technical problems). To test a moderating role

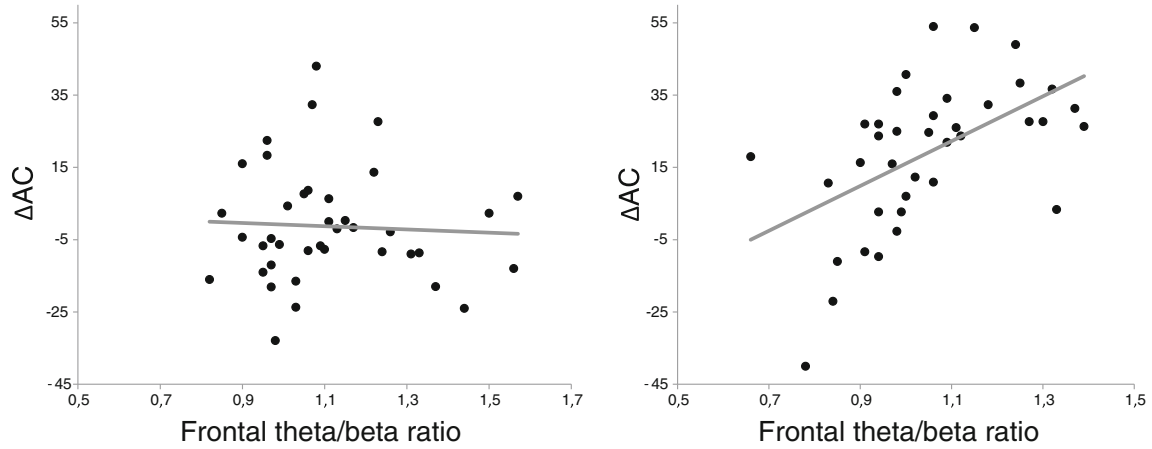

Fig. 1 Scatterplots for the relations between ΔAC (state attentional control at $t = 1$ minus $t = 2$) and frontal theta/beta ratio in the control group (left panel) and the stress group (right panel)

of theta/beta ratio on the effect of the stressor on ΔAC, the previously described moderated regression model (controlling for trait anxiety) was repeated for central theta/beta ratio and parietal theta/beta ratio. The model for central theta/beta ratio showed similar evidence of moderation, with the addition of the Condition × Centered Central Theta/Beta Ratio term adding significantly to the model ($\Delta R^2 = .045$, $p = .035$), but for the parietal recording, no significant evidence of such a moderation effect emerged ($\Delta R^2 = .026$, $p = .112$). Table 3 presents correlations between the three theta/beta ratios and ACS scores in the entire group and between the ratios and ΔAC in the stress group.

Steiger tests for comparison of dependent correlation coefficients showed that the correlations between theta/beta ratios and ACS scores were not significantly different for the frontal, central, or parietal recordings. However, the correlations between theta/beta ratios and ΔAC were significantly stronger (uncorrected p values) for frontal than for central [$t(34) = 2.251$, $p = .03$] and for frontal than for parietal [$t(34) = 2.234$, $p = .03$] recordings.

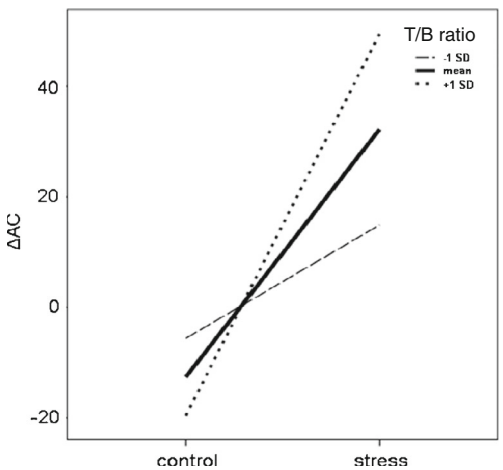

Fig. 2 Simple slopes for frontal theta/beta ratio's moderation of the effect of stress on attentional control (ΔAC = state attentional control at $t = 1$ minus $t = 2$). T/B ratio = frontal theta/beta ratio. A higher frontal theta/beta ratio is associated with a greater decline of attentional control

Discussion

We aimed to study the effect of CPA-analogue stress on the subjective experience of attentional control and how this might be moderated by attentional control. Although ACS scores could not be shown to moderate the effect of the stressor, the EEG data showed that theta/beta ratio did moderate the effects of stress, as expected. We also replicated a previously reported negative correlation between ACS and theta/beta-ratio. These results are discussed in more detail below.

Our results replicate a previously reported negative correlation between theta/beta ratio and self-reported trait attentional control. Increased theta/beta ratio in (childhood) ADHD patients is a robust phenomenon (Barry et al., 2003; Loo & Makeig, 2012) and likely reflects suboptimal functioning of the PFC. The dorsolateral PFC, ventromedial PFC and (right) inferior PFC are importantly involved in goal-directed attentional control, emotion regulation and inhibition, respectively,

Table 3 Pearson correlation coefficients between theta/beta ratios and ACS scores in the entire sample ($n = 77$ for frontal and $n = 76$ for central and parietal theta/beta ratios) and between theta/beta ratios and ΔAC in the stress group ($n = 38$ for frontal and $n = 37$ for central and parietal theta/beta ratios)

	ACS	ΔAC
Frontal theta/beta ratio	–.33**	.53**
Central theta/beta ratio	–.28*	.35*
Parietal theta/beta ratio	–.25*	.28

Predicted relations appear strongest for the frontal recordings and weaker for more posterior recordings. ACS = Attentional Control Scale, ΔAC = state attentional control at $t = 1$ minus $t = 2$. * $p < .05$, ** $p < .005$.

functions that are affected in ADHD (Arnsten, 2009; Arnsten & Rubia, 2012; Conzelmann et al., 2009). The observation that NA and DA agonists have potent therapeutic effects (Arnsten, 2009; Arnsten & Rubia, 2012) supports the hypothesis that the attentional control problems in ADHD are related to PFC dysfunction. It has also been reported that psychopharmacological treatment with an NA/DA agonist normalizes the theta/beta ratio (Clarke et al., 2007), further supporting the idea that the theta/beta ratio might be a biomarker of PFC-mediated attentional control. Others have suggested that increased slow wave/fast wave ratios may reflect reduced cortical inhibition of subcortically mediated approach motivation or reward sensitivity (Schutter & van Honk, 2005). In agreement, increased theta/beta ratio has been associated with increased reward responsiveness, risk taking and impulsiveness (Massar et al., 2012; Schutter & van Honk, 2005; van Dongen-Boomsma et al., 2010). Together, these lines of research suggest that increased theta/beta ratio may reflect compromised attentional control functioning and behavioral inhibitory processes, which both rely on the PFC. Our previous study (Putman et al., 2010) suggested that individual differences in theta/beta ratio reflect similar functions in healthy young adults by showing that theta/beta ratio was positively correlated to a self-report measure of reward-oriented motivation and negatively correlated to self-reported trait anxiety and trait attentional control. The present data do not confirm the relation with trait anxiety, but do replicate the finding of a negative relation between theta/beta ratio and attentional control (also when controlling for anxiety). All in all, we conclude that in healthy and young adults, individual differences in theta/beta ratio reflect individual differences in, likely PFC-mediated, trait attentional control capacity.

We aimed to extend this previously suggested notion and tested if theta/beta ratio also predicts a transient stress-induced decline in attentional control. In order to test effects of CPA-like stress, we developed a new stress-induction procedure that is practically easier to perform than the often used Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993). In order to maximize its effect (Dickerson & Kemeny, 2004), our procedure incorporated elements inducing a socially evaluative threat to the self-identity (the camera, judgment by others and emphasis on the expected relation between cognitive performance and the students' social identity as a person of intellectual capabilities). Manipulation checks showed that the stress induction procedure worked well. Self-reported anxiety increased by 62%, attentional control decreased by 66%, and both changes had strong effect sizes. Informal debriefing and observation did suggest that the procedure might be made even more effective by excluding one easy/hard block rotation, since it seemed as some participants "gave up" after the second failure. We conclude that this stress induction procedure is effective and likely robust and can be applied in future studies, including but not limited to, studies of test anxiety and CPA.

The results confirmed the hypothesis that theta/beta ratio moderates the effect of the stressor on self-reported attentional control: people with elevated ratios showed stronger declines of subjectively experienced attentional control. Theta/beta ratio has previously been related to trait anxiety and punishment-sensitivity (Putman et al., 2010; Schutter & van Honk, 2005) so it is important to note that the moderating effect of theta/beta ratio on state attentional control was independent from trait anxiety and so does not likely reflect merely increased anxious sensitivity to the stressor. The moderation was also independent from effects of trait attentional control as measured with the ACS. This shows that theta/beta ratio has additional value over a self-report measure of attentional control when predicting CPA-analogue responses to stress. As such, the theta/beta ratio biomarker has unique predictive qualities that can be utilized in the study of CPA and anxiety–cognition interactions. In the stress condition, the ratio explained about 28% of variance in the CPA-like cognitive response to the stressor, which is considerable. Test anxiety has a high prevalence (estimates range from 10% to 52%), seems to have increased over the last few decades (Beidel, Turner, & Trager, 1994; MacDonald, 2001; Putwain, 2007), and has negative effects on academic performance and the predictive validity of academic aptitude tests (Bonacio, Reeve, & Winford, 2012; Hembree, 1988). As such, test anxiety, just one of many expressions of CPA, brings about much human suffering as well as economic costs (by threatening the output of expensive educational systems). Obviously, CPA is a serious problem, and theta/beta ratio may provide a measure that could help our understanding and prediction of CPA, as well as the attentional dysfunction that patients with various anxiety disorders present with. Future studies should attempt to establish test–retest reliability and familial relations to assess if theta/beta ratio might be considered a useful endophenotype for CPA or test anxiety (Gottesman & Gould, 2003).

Unexpectedly, no evidence for a moderating role for trait attentional control (ACS score) was apparent in the relationship between stress and attentional function. We expected ACS to perform a similar moderator function as the theta/beta ratio on the basis of theoretical considerations as put forth above. We have no ready explanation for the fact that AC as measured with the ACS and as reflected in the theta/beta ratio show such different results. Obviously, objective measures of current neural activity (our baseline theta/beta ratio) and self-reported measures of a trait (ACS score) are both imperfect approximations of the pure construct of baseline AC. ACS and theta/beta ratio were currently moderately related ($r = -.33$), so empirically it should not be considered all too surprising that the results for theta/beta and ACS diverged like this. Concerning the lack of support for the theoretical prediction that trait AC should moderate effects of stress on attentional function, it should be noted that the correlation between ACS and the attentional response to the stressor was near

significant. We consider these results inconclusive, and future studies should revisit the role of AC as measured with the ACS.

We aimed to test our hypotheses specifically for frontal recording of theta/beta ratio. Some authors assume that the ratio reflects an interplay of signals originating from cortical (beta) as well as subcortical limbic cortical areas (theta; Knyazev, 2007; Massar et al., 2012; Schutter & van Honk, 2005). Because of such speculated involvement of subcortical areas, and because of the difficulty with estimating source localization for scalp-recorded EEG signals, it is not possible to make strong a priori predictions about which recording position should yield the strongest relationships between theta/beta ratio and behavior or cognitive function. However, since relations between theta/beta ratio and attentional control likely reflect some involvement of the PFC, focusing on frontal recordings seems a reasonable choice that limits the problem of multiple statistical testing, which is inherent to EEG research measuring signals from multiple electrode positions. Also, in a previous article, frontal recordings seemed somewhat more clearly related to self-reported attentional control and behavioral inhibition than did central or parietal recordings (Putman et al., 2010). Additional analyses in the present study showed that the results were strongest for frontal recordings, with central recordings yielding weaker, and parietal recordings yielding even weaker, nonsignificant results. It must be noted though that some of these differences were too small to be significant when formally tested and others were significant only at statistical trend level when corrected for multiple comparisons. All in all, the data from this study now allow for firm conclusions concerning the question of whether the (pre)frontal cortex is indeed critically involved in relationships between theta/beta ratio and attentional control. Ideally, EEG measurements should be combined with imaging techniques such as (f)MRI to approach this issue.

Several aspects of this study call for further attention. First, many studies have indirectly verified that self-report instruments of trait attentional control can validly measure this construct, by demonstrating the predicted relationships with various objective measures of resistance against automatic processing of goal-unrelated distraction from emotionally salient stimuli (e.g., Bardeen & Orcutt, 2011; Derrybery & Reed, 2002; Peers & Lawrence, 2009; Putman et al., 2012). Nevertheless, future studies should attempt to replicate our findings using objective measures of (aspects of) attentional control that have been shown to be sensitive to stress manipulations—for instance, tasks that measure divided attention or working memory (see Eysenck et al., 2007). Also, relationships between theta/beta ratio and effects of stress on more ecologically valid measures of cognitive performance should be studied (e.g., testing the hypothesis in academic or otherwise real-life cognitively demanding settings). Second, a noteworthy aspect of our method is that our self-report state anxiety instrument makes no distinction between the emotional and cognitive components of CPA. The cognitive approach to CPA (see Cassady & Johnson, 2002; Eysenck et al., 2007; Sarason, 1984) leads to the hypothesis that theta/beta ratio should predict stress-induced changes in cognitive anxiety level (i.e., increased worry about performance), but not emotional anxiety (i.e., awareness of increased sympathetic activity). Using our undifferentiated state anxiety measure, explorative analysis showed no statistically significant and evidently meaningful effects. A future study should make the distinction between emotional and cognitive anxiety and analyze relations between theta/beta ratio and effects of stress on these separable constructs.

In sum, these data confirm and extend a previous suggestion that theta/beta ratio may be a biomarker for attentional control capacities in healthy individuals. This study has also shown that theta/beta ratio predicts resilience/vulnerability to the effects of CPA-like stress on self-reported state attentional control. The theta/beta ratio seems a useful biomarker in the study of CPA and other anxiety–cognition interactions.

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