EEG theta/beta ratio: a marker of executive control and its relation with anxiety-linked attentional bias for threat

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Introduction

Anxiety disorders are among the most prevalent forms of psychopathology (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005; Kessler, Chiu, Demler, & Walters, 2005) and are associated with a significant impairment in overall functioning and reduction in quality of life (American Psychiatric Association, 2013). Research into the mechanisms that underlie anxiety disorders and/or maladaptive anxiety states is critical to help in treatment consideration.

Anxiety has often been related to certain attentional deficits (e.g. Mogg & Bradley, 1998; 2016; Derryberry & Reed, 2002; Eysenck, Derakshan, Santos and Calvo, 2007), more specifically, anxious individuals were found to often have an enduring automatic tendency to attend preferentially to threat related information (e.g. see Derryberry & Reed, 2002; Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg & van IJzendoorn, 2007). These tendencies are also called '*attentional biases'* favoring threatening information. Attentional biases to threat were for example suggested to maintain anxiety complaints, as anxious (distracting) information is not well regulated in, for example anxiety disorders (Mogg & Bradley, 1998; 2016). Moreover, attentional biases to threat are considered to causally relate to heightened trait anxiety (Bar-Haim et al., 2007; Mathews & MacLeod, 2002; van Bockstaele, Verschuere, Tibboel, De Houwer, Crombez, & Koster, 2014).

Critically however, several lines of evidence argued that the attentional processes that underlie attentional biases to threat fluctuate across time and context and depend on individual trait-differences (e.g. Mogg & Bradley et al., 1998; 2016; Iacoviello, Wu, Abend, Murrough, Feder, Fruchter et al., 2014; Kruijt, Field & Fox, 2016; Notebaert, Clarke & MacLeod, 2016). One example is individuals' capacity to control their attention, in other words, *attentional control*. Attentional control can be defined as the ability to control attention in relation to thought and reaction patterns (Derryberry & Reed, 2002). Impaired attentional control was found to be a predictor for certain anxiety disorders, which will be further outlined in subsequent sections of this introduction. Several studies furthermore found low attentional control to directly relate to stronger attentional biases/ disturbed threat processing (e.g. Derryberry & Reed, 2002; Eysenck et al., 2007; Koster, Crombez, Verschuere, & De Houwer, 2004) making attentional control an important concept to further investigate. In the current doctoral thesis, the specific role of attentional control in processing threatening information will be reviewed, along with the use of a related physiological marker; *EEG theta/beta ratio*.

Attentional control

Attention is the means by which the 'limited-capacity brain' allocates processing resources (Posner & Petersen, 1990). Giving attention to some features of our environment may cause partial or full exclusion of attention to other features (Driver, 2001). In other words, attention denotes concentration or distractibility (Lawrence, Ross, Hoffmann, Garavan, & Stein, 2003). *Attentional control,* as a key concept in this thesis, is the ability to effortfully control attention to support tasks and goals.

Attentional control has been described by different theoretical models in psychological research, including different component processes, contributing to the broader construct of 'cognitive control'. Miyake, Friedman, Emerson, Witzki, Howerter and Wager (2000) described several processes involved in cognitive 'executive' control functions. They used a latent variable analysis to identify the basic control functions for attention by selecting cognitive tasks on lower level functions. Three major functions were identified: 1) Inhibition: one's ability to deliberately inhibit dominant, automatic responses when necessary; this involves using attentional control to resist disruption or interference from task-irrelevant stimuli or responses, e.g., assessed by anti-saccade and Stroop tasks. 2) Shifting: shifting back and forth between multiple tasks, operations, or mental sets; this function involves adaptive changes in attentional control based on task demands. 3) Updating: updating and monitoring of working memory representations e.g. assessed by working memory tasks. Miyake et al. (2000) noted that correlations between shifting, updating and inhibition measures have an underlying commonality. This definition of executive functions underlying attentional control may reflect a shared requirement to maintain goals in working memory and/ or a common inhibitory process. In an update of their model, Miyake and Friedman (2012) propose that 'inhibition' can be seen as a common factor that is fundamental for all aspects of executive control. In this revised model, two factors are proposed to be subordinate of the common 'inhibition' factor; a separate shifting-specific and updating-specific factor.

In line with Miyake and Friedman (2012), Derryberry and Reed, (2002) describe attentional control as the top-down command over different components of attention. They point out that attentional control consists of two dimensions. *Attentional Focus* is the ability to maintain attentional engagement in the face of distraction, while *Attentional Shifting* is the ability to execute attentional disengagement, in other words to shift attention away from a distraction or toward a new task. Individual differences in attentional control can be measured reliably, both by self-report and performance measures (Derryberry & Reed, 2002). Attentional control may be conceptualized as a trait capturing the control of information processing (Derryberry & Reed, 2002).

Eysenck et al. (2007) proposed an attentional control theory, in which working memory and attention are controlled by two attentional systems: a bottom-up, stimulus-driven system, and a goal-directed top-down system. During anxious states for example, the bottom-up processing of threatening stimuli is automatically increased. This causes misbalance between both systems and bottom-up processes become favoured. The goal-directed top-down system on the other hand, supports two key functions as part of attentional control: inhibition of task-irrelevant information and responses, and switching between tasks (Berggren & Derakshan, 2013; Snyder, Miyake & Hankin, 2015).

Attentional control and anxiety disorders

As briefly mentioned before, low capacity of attentional control has repeatedly been associated with a broad spectrum of anxiety disorders (for a review see Cisler & Koster, 2010). Post-Traumatic Stress Disorder (PTSD) for example, may be characterized by trauma related impaired attentional control, that was most apparent when the threat cue was in the patient's domain of concern (PTSD relevant threat; see Bomyea, Risbrough, & Lang, 2012). PTSD patients' persistence of distressing intrusive thoughts may also stem from ineffective utilization of cognitive systems – specifically aspects of attentional functioning – to inhibit or down-regulate all information (e.g., Anderson & Levy, 2009; Joormann, Yoon, & Siemer, 2010; Verwoerd, de Jong, & Wessel, 2008). Hagenaars and Putman (2011) provided further evidence for the relationship between intrusive memories and attentional control. Healthy participants who were low in attentional control showed a self-perceived tonic immobility when viewing an aversive film, which in turn predicted intrusion frequency during the subsequent week. People with better attentional control were more resilient to this relationship.

General Anxiety Disorder (GAD) has also been associated with impaired attentional control (Amir, Beard, Burns, & Bomyea., 2009). In GAD, anxiogenic cognitions take the form of perseverative worry, which consists of repeated thoughts about everyday concerns (Armstrong, Zald, & Olatunji, 2011; Burns, Keortge, Formea, & Sternberger, 1996) and are thought to start as uninhibited selective thought-processing, akin to automatic attentional processing of threat-information (Hirsch & Mathews, 2012). Individuals suffering from GAD moreover experience related impairments, such that one's ability to manage attentional resources toward the prevention of 'unwanted negative thoughts' is undermined.

Similarly, social anxiety disorder (SAD) is characterized by a biased internal activation system of threatening thoughts such as fears of evaluation by others (Schmidt, Richey, Buckner, & Timpano, 2009). These threatening thoughts impact working memory and other attentional mechanisms (Hirsch & Mathews, 2012). Adapting and performing well in one's environment depends on capacity-limited attention and on executive functions like working memory. Individuals suffering from cognitive performance anxiety (also a sub-classification of SAD; American Psychiatric Association, 2013) often encounter comparable intrinsic thoughts that disturb cognitive functions and thus performance (Osborne & Franklin, 2002). Cognitive performance anxiety is generally defined as experienced fears about some domain of one's cognitive performance and about others' evaluations thereof. Test anxiety is a clear example of cognitive performance anxiety. Although moderate levels of stress may increase performance, severe cognitive performance anxiety will have a detrimental effect on actual cognitive performance (Cassady & Johnson, 2002; Eum & Rice, 2011).

These described lines of research lead to the hypothesis that enhancing attentional control may be helpful for persons who suffer from anxiety disorders and who are characterised by disrupted attentional processing. Enabling patients to exert more cognitive control over their attentional resources may help them direct attention to cognitive tasks instead of to distracting intrusions. Therefore, it is important to understand the underlying mechanisms of attentional processing, in particular threat selective attention.

Threat selective attention and threat avoidance

Selective attention is the means by which certain features in the environment are selected by individuals for attentional focus (e.g. Driver, 2001; Derryberry & Reed, 2002; Koster et al., 2004). As already mentioned, anxious individuals (scoring high on *trait-anxiety*) selectively and automatically attend to emotional, mainly threat-related, stimuli, compared to neutral stimuli (for reviews see Bar-Haim et al., 2007; Cisler, Bacon & Williams, 2009; Cisler & Koster, 2010; Koster et al., 2004; Mogg & Bradley, 1998; 2016). In other words, anxious individuals have '*attentional biases*' favoring threatening information. These biases are important in that the attention selectively facilitates early threat processing, thereby influencing the cognitive and emotional processes related to anxiety (further referred to as '*threat selective attention*'; Mathews, May, Mogg & Eysenck, 1990; Williams, Mathews, & MacLeod, 1996).

Mogg and Bradley (1998; 2016) described threat selective attention as that being vulnerable to anxiety disorders stems mainly from a bias caused by automatic threat evaluation. This idea includes that an intrinsic stimulus threat value might be automatically assessed by a *valence evaluation system*, influenced by several variables (e.g. stimulus features, context, prior learning, state- and trait-anxiety). This intrinsic system is more reactive to threat cues in individuals prone to anxiety disorders. In the absence of threat, the system processes goal-relevant stimuli, but inhibits processing of minor task-irrelevant negative cues. However, if the *valence evaluation system* judges a stimulus to have a high threat value, this triggers automatic attention to the threat and interrupts all goal-related activity. Because anxiety prone individuals tend to evaluate mildly threatening stimuli as having a high motivational salience, they are more likely to direct attention to those stimuli. Hence, threat selective attention, specifically to mildly threatening stimuli may be an index of anxiety-proneness. In this way, threat selective attention actually maintains anxiety, as anxiety-prone individuals are more likely to notice minor threat cues in the environment, which enhances their perception of the world as aversive and unsafe, and increases their state anxiety (Bardeen & Orcutt, 2011; Derryberry & Reed, 2002; Putman, Arias-Garcia, Pantazi, & van Schie, 2012; Schoorl, Putman, van der Werff & van der Does, 2014; Taylor, Cross, and Amir., 2016; Peers & Lawrence, 2009).

Among others, Mogg et al., (1987) reported that initial threat selective attention may be opposed by *threat avoidance* in controlled attention-related strategies. Avoidance was proposed to reflect an attempt to reduce subjective discomfort or perceived danger, making avoidance possibly more apparent at higher levels of threat or anxiety (Mogg, Weinman, & Mathews, 1987). Whereas avoidant attention-related strategies may reduce immediate distress, they may not be useful on the long-term by causing habituation and thus persistence of anxiety (Mogg & Bradley, 2016). These considerations carry possible implications for the currently popular attentional bias modification (ABM) paradigm and its attempts to train attentional bias away from threat with the objective of effecting more adaptive, healthy attentional processing styles. The ABM-threat-avoidance training may cause strategic avoidance of threat, making it unhelpful for anxious individuals.

Neural mechanisms of attentional control and threat selective attention

Understanding the neural underpinnings of attentional control and threat selective attention may be fundamental for improvement of maladaptive anxiety states. Scientific research on these mechanisms points out that the bottom-up sensory and top-down control processes interact to determine how much 'attention' threat-related stimuli receive (e.g. Bishop, 2008; Hermans, Henckens, Joels & Fernandez, 2014). Top-down attentional control and inhibition of such stimulus-driven attention seems to be carried out by e.g. the dorsal anterior cingulate cortex and the dorso-lateral prefrontal cortex (DL-PFC; Fani, Jovanovic, Ely, Bradley, Gutman, et al., 2012). Bottom up processes of salient and threat-related distracters (which have also been classified as the 'salience network') seem to be mediated by sub-cortical brain areas like the ventral anterior cingulate cortex, the medial prefrontal cortex (mPFC), parahippocampal gyrus and the angular gyrus (e.g. Bishop, 2008).

Neural resources that are allocated towards this salience network seem to be highly influenced by catecholamines (e.g., norepinephrine and dopamine) in terms of mediating earliest responses to acute threat (Hermans et al., 2014). The goal oriented, top-down attentional control mediated by prefrontal cortex (PFC) networks is also dependent on adequate catecholamine action (Hermans et al., 2014; Arnsten, 2009a). Stress and anxiety trigger a variety of neurochemical changes, including increased influx of dopamine and nor-adrenaline into the PFC (Joëls & Baram, 2009). Both dopamine and nor-adrenaline influence PFC in a dose-dependent, inverted U-shaped manner (Arnsten, 2009a). While moderate levels are needed for good prefrontal executive control, dopaminergic and noradrenergic over-stimulation leads to decreased PFC function. In other words, increasing levels of catecholamines are associated with increasing performance until a tipping point is reached, after which further catecholamine stimulation will harm executive performance, including top-down attentional control (Arnsten, 2009a; Arnsten & Rubia, 2012; Hermans et al., 2014). This tipping point for the effects of stress-induced catecholamines (the apex of the inverted U-shape relation between catecholamines and cognitive performance) was found to be dependent on catecholamine-driven basal prefrontal function, and is therefore different for every individual (Arnsten, 2009a; Arnsten 2009b; Cools & D'Esposito, 2011). This implies that a well-dosed manipulation of catecholamine systems could increase attentional control over threat-bias, depending on individual differences in anxiety and baseline PFC function or catecholamine levels (Arnsten, 2006; Arnsten, 2011b).

In addition, it was found that the manipulation of attentional focus, threat-value, and stimulus presentation parameters (e.g. stimulus delay) have different influences upon subcortical areas and prefrontal activation to threatening stimuli (Bishop, 2008). For example, high perceptual load causes competition for perceptual resources and appears to inhibit the processing of threat distractors at an early stage, eliminating the amygdala response to these distractors. On the other hand, for low perceptual load the automatic bottom-up activity can be sufficient for such threat distractors to cause amygdala activity (Bishop, Jenkins & Lawrence, 2006; Pessoa, Padmala, & Morland, 2005). It can be argued that salient distractors compete for processing resources, such as entry to working memory and guidance of response selection. This is in line with the previously described attentional control theory (Eysenck et al., 2007); in low perceptual load, prefrontal cortical regions cause top-down attentional control to be electively activated in response to the occurrence of threat-related distractors (Bishop et al., 2006).

Moreover, individual differences in anxiety seem to modulate the strength of the amygdala signal to threat stimuli, even when participants are not attending to or consciously aware of these stimuli (Bishop, Duncan & Lawrence, 2004; Etkin, Klemenhagen, Dudman, Rogan, Hen, et al., 2004). Elevated anxiety is also associated with disrupted recruitment of prefrontal control mechanisms and thus executive control, in response to processing competition from threat-related distractors (Bishop et al., 2006; Bishop et al., 2004).

Taken together; (neural) individual differences of e.g. attentional control, catecholamine functioning and anxiety are important aspects when investigating threat selective attention and their contribution to threat processing should be further investigated. The concept of attentional control in particular seems to play a key role in a neural model of threat selective attention. Attentional control has mainly been measured by self-report, but research may benefit from using objective markers of attentional control (see also Bardeen & Daniel, 2018). An objective measure for attentional control, such as a psychophysiological marker, could prevent possible response biases of self-reports and provide a more accurate representation of attentional control (Kihlstrom, Eich, Sandbrand, & Tobias, 2000).

EEG theta/beta ratio as a marker of executive control

Electroencephalogram (EEG) measures represent the combined electrical fluctuations in membrane potentials generated from the interactions of the primary inhibitory and excitatory neurons (Gordon, 2000; Nunez, 1995) which reflects the number of neurons that discharge synchronously (Klimesch, 1999). Spectral analysis of a resting state EEG signal produces measures for power in different frequency bands, for example, the theta band, a low frequency band with signals oscillating between 4 and 7 Hz, or a high frequency band such as the beta band (13-30 Hz). Typically measured under resting conditions, the ratio between the theta and beta band (*theta/beta ratio; TBR*) has been utilized as a source of information about the baseline state of the brain (in terms of maturation and/or arousal) as well as a predictor for any subsequent brain activity that may be associated with increased cognitive demand (Barry, Clarke, & Johnstone, 2003).

Several lines of evidence further point out that the TBR is possibly of interest when investigating attentional control. A robust finding for example is that TBR scores are higher in patients diagnosed with attention-deficit/ hyperactivity disorder (ADHD; Barry et al., 2003). Also, administration of methylphenidate (a stimulant that is beneficial for ADHD) is effective through restoration of sub-optimal prefrontal cortical executive function via upregulation of post-synaptic PFC catecholamine function and normalizes the TBR (Clarke, Barry, McCarthy, Selikowitz & Johnstone, 2007). This fits the previously described inverted U-shape relation between catecholamines and cognitive performance. Furthermore, high TBR scores were related to poor prefrontal cortical mediated attentional and inhibitory functions, (i.e. Arns, Conners, & Kraemer, 2013; Barry et al., 2003) and likely reflects functional reciprocal cortical-subcortical interactions in both healthy and clinical populations (Knyazev, 2007; Schutter & Knyazev, 2012).

TBR has been used as an electrophysiological marker of (top-down, PFC-regulated) attentional control to investigate its effect on attentional bias and trait anxiety in healthy adults (Angelidis, Hagenaars, van Son, van der Does, & Putman, 2018). TBR was also found to negatively correlate with motivational decision making and the learning processes involved therein (Schutter & van Honk, 2005; Massar, Rossi, Schutter, & Kenemans, 2012; Massar, Kenemans, & Schutter, 2014; Schutte, Kenemans, & Schutter, 2017). Putman et al. (2010) found that TBR correlated negatively with the ability to modulate response inhibition in an emotional go/no-go task (Putman, van Peer, Maimari, & van der Werff, 2010). A similar correlation between TBR and self-reported attentional control was observed. The emotional go/no-go task utilizes emotional stimuli to induce a response bias in terms of longer response latencies and more errors. This response is in turn modulated by activity in the amygdala, as well as in the lateral orbitofrontal cortex (Schulz, Fan, Magidina, Marks, Hahn & Halperin, 2007). In other words, TBR may reflect voluntary top-down processes of executive control (including attentional control), mediated by (dorso-lateral) PFC, over bottom-up processes from limbic areas, such as the anterior cingulate cortex, hippocampus and amygdala; (Bishop, 2008; Gregoriou, Rossi, Ungerleider, Desimone, 2014; Knyazev, 2007; Schutter & Knyazev, 2012).

In sum, frontal TBR is considered to reflect PFC regulated executive control; it might therefore as well be related to individual differences of the catecholamine tipping point, as described before. The established model of inverted U-shape relations between prefrontal catecholamine activity and cognitive attentional control (Arnsten, 2006; Arnsten, 2009a; Cools and D'Esposito, 2011) would predict that if TBR indeed represents executive control, it is also regulated by prefrontal catecholamine levels (Arnsten, 2006; Hermans et al., 2014). Frontal TBR as an electrophysiological marker for executive control thus might be a useful approximation of individual differences in baseline prefrontal catecholamine function that could be used when investigating catecholamine manipulation, for example. Including other measures that reflect basal PFC regulated executive control can also improve outcomes of studies of effects of pharmaca on prefrontal cognitive processing. Likely, EEG TBR measurement is a useful tool in psychopharmacological studies.

Multiple studies to date found that TBR is (directly) negatively correlated with ACS scores (Angelidis, van der Does, Schakel, & Putman, 2016; Putman et al., 2010; Putman, Verkuil, Arias-Garcia, Pantazi, & van Schie, 2014; but see Morillas-Romero, Tortella-Feliu, Bornas & Putman, 2015; Angelidis et al., 2018). Also, TBR was found to correlate to task-based measures of attentional control in patients suffering from multiple sclerosis, having clinically impaired attention (Keune, Hansen, Weber, Zapf, Habich, Muenssinger et al., 2017). TBR was reported to have a very high one-week and two-week re-test reliability (Angelidis et al., 2016; Keune et al., 2017), supporting the validity of TBR as a reflection of trait attentional control. TBR thus seems to be a marker of executive control and attentional control and requires further investigation to explore its specific functions/applications. As attentional control plays an important role in threat processing, further research was needed to investigate TBR's relation to threat related and emotional processes, which will be further discussed in the next section.

EEG theta/beta ratio, stress, threat and emotional processes

The prefrontal cortex (PFC) is often viewed as the control center, exerting executive control over various bottom-up processes, e.g. regulating fearful responses driven by the amygdala (Bishop et al., 2004). The finding of TBR being negatively related to attentional control indicates that the relationship between PFC regulated executive control and theta/beta ratio might reflect a continuum of brain-behaviour correlation of which, for example, attentional deficits represent a far end of the spectrum. Baseline resting state TBR has been similarly related to stimulus evoked behaviour (e.g., Massar et al., 2012; Putman et al., 2010; Schutter & van Honk, 2005), self-reported emotional and motivational traits (Putman et al., 2010) and psychiatric diagnosis reflecting dynamic behaviour over extended periods (Clarke, Barry, McCarthy & Selikowitz, 2002). The inter-individual variance of TBR thus seems to reflect the inter-individual variance of a certain brain state that specifically determines one's response to environmental challenges. These are indications that TBR may be useful in the study of, for example, performance in the presence of environmental stressors. Putman et al., (2014) actually tested the prediction that TBR moderates the deleterious effects of anxious stress on state attentional control. As expected, resting state TBR did moderate the effects of stress on change in state attentional control, which favours the idea that TBR predicts resilience/vulnerability to the effects of performance anxiety-like stress on self-reported state attentional control.

Furthermore, a number of associations have been found between attentional control and emotion regulation capability, suggesting that the two functional mechanisms are related (e.g. Rothbart & Rueda, 2005). Cognitive reappraisal, which is an emotion regulation strategy premised on reinterpretation of the meaning of a stimulus in order to regulate the emotional response to it, has been a particularly successful strategy in terms of regulating subjective and physiological responses (Gross, 1998). Ochsner and Gross (2005) reported that 'cold' forms of emotional control are strikingly similar to the consistently found activations observed in cognitive reappraisal. If cognitive reappraisal relies on the same functional mechanism as 'cold' forms of cognitive control, it is reasonable to expect that individuals with biased cognitive control, accompanied by an elevated TBR will be similarly poorer in their capability to apply cognitive reappraisal. TBR can therefore be indirectly related to emotion regulation which is also supported by more recent studies (Morillas-Romero et al., 2015; Tortella-Feliu, Morillas-Romero, Balle, Llabrés, Bornas, & Putman, 2014). Spontaneous emotion down regulation has for example previously been found to be predicted by attentional control and a slowing down of heart rate (Morillas-Romero et al., 2015). Also, TBR as a measure of attentional control was suggested to be associated with discomfort ratings and time needed to downregulate negative emotion after subjects were exposed to negative pictures (Tortella-Feliu et al., 2014). These results contribute to a better understanding of the involvement of TBR in emotion regulation processes, however more studies are needed to further extend and elaborate these findings.

TBR might therefore be a useful electrophysiological marker of emotion-attentional control interactions for research on anxiety and should be studied further.

The role of mind wandering as involuntary, distracting thought patterns

Lower TBR has consistently been linked to 'on task' processes and the processes that accompany performance on a task. Importantly, Braboszcz & Delorme (2011) found that increased theta and decreased beta (in other words, increased TBR) was specifically present during mind wandering episodes (uncontrolled thought), and the opposite (decreased TBR) during controlled thought periods. We therefore assumed that TBR might be a marker for these uncontrolled thought/ controlled thought processes. Since mind wandering is described as a deficit in working memory and attention (McVay & Kane, 2009) and a predictor for performance errors (Smallwood & Schooler, 2006), poor attentional control might cause a higher tendency to mind wander. In other words, more frequent and lengthy occurrences of mind wandering episodes during the standardized ~8 minutes "resting state" assessment of spontaneous TBR might be responsible for the negative correlation between the average TBR during such measurements and attentional control.

Along these lines, it will be interesting to investigate whether mind wandering is the underlying responsible mechanism of the TBR – attentional control relationship as previously described, and will possibly provide better understanding of the functional and neural mechanisms that are responsible for the relationship between TBR and executive control. An effective way of measuring mind wandering episodes is to include the underlying neural mechanisms such as functional connectivity of mind wandering related brain regions. Mind wandering was mainly found to activate the Default Mode Network (Karapanagiotidis, Bernhardt, Jefferies & Smallwood, 2017; Smallwood, Beach, Schooler & Handy, 2008); a network of interacting neural regions, known to have activity that is highly correlated within this network during task unrelated thoughts (Stawarczyk, Majerus, Maquet, & D'Argembeau, 2011). Also, it was found that regions within the 'Executive Control Network', which consists of the DL-PFC, dorsal anterior cingular cortex (dACC) and posterior parietal regions (Seeley, Menon, Schatzberg, Keller, Glover, Kenna et al., 2007), became active during awareness of mind wandering, attentional shifting and sustained attention (Hasenkamp, Wilson-Mendenhall, Duncan, & Barsalou, 2012; Christoff, Ream, Geddes, & Gabrieli 2003). Additional insights can thus be obtained when looking at functional connectivity within the Default Mode Network and the Executive Control Network during mind wandering episodes, and what role it plays in the hypothesized underlying responsible mechanism of mind wandering on the TBR and attentional control relation.

Manipulating EEG theta/beta ratio

Increased slow-wave activity coupled with decreased fast wave activity was first observed in ADHD in the early 1970s (Loo & Makeig, 2012; Satterfield, Cantwell & Satterfield, 1974). This work primed the interest in determining EEG abnormalities in ADHD children, and more research has been carried out since, to replicate the findings (e.g. Barry et al., 2003; Hermens, Kohn, Clarke, Gordon, & Williams, 2005; Lazzaro, Gordon, Whitmont, Meares, & Clarke, 2001; Monastra, Lubar, & Linden, 2001; Ogrim, Kropotov, & Hestad, 2012). Main findings were that ADHD was associated with increased EEG theta and decreased beta bands; in other words; increased theta/beta ratio (for review and meta-analysis, see Arns et al., 2013; Barry et al., 2003). Several studies reported that manipulating TBR using Neurofeedback training (NFT) could successfully reduce the TBR and ADHD-related symptoms in individuals diagnosed with ADHD (e.g. hyperactivity, impaired attention; e.g. Kouijzer, de Moor, Gerrits, Congedo and van Schie, 2009, for a review see Vernon, 2005).

The study of the potential beneficial effects of reducing TBR with NFT in healthy adults seems warranted, given the abovementioned relations between TBR and various psychological regulatory constructs. However, some studies reported that no changes in EEG were actually observed when applying a commonly used NFT method (Janssen, Bink, Weeda, Geladé, van Mourik, Maras, & Oosterlaan, 2017; Shönenberg, Wiedemann, Schneidt, Scheeff, Logemann, Keune et al., 2017; Doppelmayr & Weber, 2011). Further replications and extensions of studies on exact changes therefore seem imperative to ascertain the effects of TBR NFT in healthy adults, and whether it can provide a tool to study causality in this relation and possibly even enhance human performance.

Integration and scientific relevance

The above-mentioned relationships between TBR, attentional control and emotional processes further reinforce the notion that frontal TBR has a unique and independent predictive value in the study of executive control and threat selective attention, specifically, control over information in an emotional context. These relationships may provide valuable information for investigating emotion-regulation related disorders, as these disorders have previously been linked to executive or attentional problems in anxiety. The role of attentional control and its relation with aberrant attentional threat-processing in the diagnosis, maintenance and treatment of anxiety disorders should not be underestimated (Mogg & Bradley, 2016). TBR seems to provide a promising variable of interest for such research.

Aim of this thesis

Considering that TBR is a potentially useful marker of executive/attentional control, both in healthy and clinical samples, we designed studies in healthy adults to further investigate the relation of TBR with threat selective attention. We manipulated threat value and attentional stages; catecholamine functioning; uncontrolled thought (mind wandering) and the executive control brain network. We also explored if TBR in a healthy adult sample is affected by NFT. In summary, we aimed to answer the following research questions.

General research questions

1) What is the role of TBR in attentional processing? (Chapters 1 and 2)

2) What is the role of TBR and attentional control in effects of catecholamine-related pharmacological manipulations, such as caffeine administration? (Chapter 2).

3) Is the TBR - attentional control relationship as observed possibly driven by mind wandering episodes? (Chapters 3 and 4).

4) Is the TBR – mind wandering relationship related to functional connectivity in the default mode network and the executive control neural network? (Chapter 4).

5) Can TBR be manipulated by means of neurofeedback training to possibly further enable future clinical interventions and to enable studies assessing causal effects of TBR? (Chapter 5).

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