BIOLOGICAL PSYCHOLOGY

Attentional bias modification facilitates attentional control mechanisms: Evidence from eye tracking

Nigel T.M. Chen [a,b,*], Patrick J.F. Clarke [b], Tamara L. Watson [c], Colin MacLeod [b,d], Adam J. Guastella [a]

[a] Brain & Mind Research Institute, University of Sydney, 100 Mallett Street, Camperdown, NSW 2050, Australia
[b] Centre for the Advancement of Research on Emotion, School of Psychology, University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia
[c] School of Social Sciences and Psychology, University of Western Sydney, Locked Bag 1797, Penrith, NSW 2751, Australia
[d] School of Psychology, Babeş-Bolyai University, Strada Mihail Kogălniceanu 1,Cluj-Napoca 400084, Romania

ARTICLE INFO

ABSTRACT

Social anxiety is thought to be maintained by biased attentional processing towards threatening information. Research has further shown that the experimental attenuation of this bias, through the implementation of attentional bias modification (ABM), may serve to reduce social anxiety vulnerability. However, the mechanisms underlying ABM remain unclear. The present study examined whether inhibitory attentional control was associated with ABM. A non-clinical sample of participants was randomly assigned to receive either ABM or a placebo task. To assess pre–post changes in attentional control, participants were additionally administered an emotional antisaccade task. ABM participants exhibited a subsequent shift in attentional bias away from threat as expected. ABM participants further showed a subsequent decrease in antisaccade cost, indicating a general facilitation of inhibitory attentional control. Mediational analysis revealed that the shift in attentional bias following ABM was independent to the change in attentional control. The findings suggest that the mechanisms of ABM are multifaceted.

© 2014 Elsevier B.V. All rights reserved.

1. Introduction

Social anxiety disorder (SAD) is a debilitating form of mental illness characterized by an excessive fear of negative social evaluation. Cognitive theories have emphasized the role of aberrant information processing in the maintenance and exacerbation of this condition, with particular emphasis on early attentional processing (Mogg & Bradley, 1998; Williams, Watts, MacLeod, & Mathews, 1997). A strong base of empirical literature suggests that SAD is characterized by an attentional bias favouring the processing of social threat stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007). That is, socially anxious individuals may preferentially allocate attentional resources to the detection of cues in their environment which may indicate social disapproval.[1] This attentional bias to threat serves to maintain

anxiety by disproportionately elevating arousal in social situations, falsely confirming various negative cognitions, and triggering maladaptive safety behaviours (Rapee & Heimberg, 1997).

A causal relationship between attentional bias and anxiety vulnerability has further been demonstrated through the use of attentional bias modification (ABM; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). The most commonly used ABM task involves the brief presentation of a threat and neutral stimulus pair, followed by a probe which appears in the location vacated by one of the two stimuli. Participants are required to respond to the probe. However, the probe location is contingent on the stimulus pair such that the probe is always presented in the location of the neutral stimulus. Efficient completion of this task therefore will be enhanced by reducing attentional bias towards threat. MacLeod et al. (2002) demonstrated that this task can indeed modulate attentional bias. This induction of a bias away from threat was further associated with reduced anxious reactivity in response to a subsequent stressor. Recent studies have additionally shown that ABM may be protective against anxiety during a naturalistic stressor (See, MacLeod, & Bridle, 2009), and may reduce social anxiety symptoms in clinically socially anxious individuals (Amir et al., 2009; Schmidt, Richey, Buckner, & Timpano, 2009). Taken together, recent research suggests a causal relationship between ABM and

---

* Corresponding author at: School of Psychology, University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia.
  E-mail address: nigel.chen@uwa.edu.au (N.T.M. Chen).
  [1] It is to be noted that previous research have additionally observed an attentional bias away from emotional information in social anxiety (e.g. Chen, Ehlers, Clark, & Mansell, 2002).

social anxiety vulnerability. While such findings may be suggestive of the potential therapeutic application of ABM in the treatment of SAD, the mechanisms which underlie the modulation of attentional bias are not well understood.

Neurocognitive models of attention suggest that two biasing signals, from a stimulus driven system and an attentional control system, determine selective attention to emotional stimuli (Corbetta & Shulman, 2002). The stimulus driven system is largely amygdala-centred, and encompasses bottom-up processing. This system functions to automatically deploy attention to salient stimuli, with particular regard for the detection of threat (Bishop, 2007). The latter attentional control system recruits regions such as the prefrontal cortex (PFC), and may subsequently provide a more flexible top-down control of attention relevant to current goals and task requirements, including for instance, the inhibition of task irrelevant information (Bishop, 2007, 2008; Desimone & Duncan, 1995; Vuilleumier, 2005).

It has been suggested that the anxiety-linked attentional bias to threat may represent an imbalance between the two attentional systems. Attentional Control Theory (Eysenck, Derakshan, Santos, & Calvo, 2007) posits that heightened anxiety is associated with an enhanced stimulus driven system and a relatively impaired attentional control system. Correspondingly, anxiety has been associated with amygdala hyperactivation in response to threatening stimuli (Bishop, Duncan, & Lawrence, 2004b; Phan, Fitzgerald, Nathan, & Tancer, 2006; Stein, Simmons, Feinstein, & Paulus, 2007; Straube, Mentzel, & Miltner, 2005; Yoon, Fitzgerald, Angstadt, McCarron, & Phan, 2007). Evidence further suggests that anxiety is associated with the impoverished recruitment of prefrontal areas suggesting a deficit in attentional control (Bishop, 2009; Bishop, Duncan, & Lawrence, 2004a). Taken together, anxious individuals may have difficulty exerting goal directed attention in the presence of task irrelevant stimuli. With regard to threat stimuli, such information may be particularly difficult to inhibit (Eysenck et al., 2007).

Within this framework, it is possible that ABM may modulate selective attention by affecting either the stimulus driven or attentional control systems. Recent imaging research has observed altered lateral PFC activation in response to the presentation of threat stimuli following ABM training away from threat (Browning, Holmes, Murphy, Goodwin, & Harmer, 2010). Additional electrophysiological research has found that ABM may modulate the event-related potential (ERP) complex (Eldar & Bar-Haim, 2010). In this study, participants who were administered ABM exhibited a subsequent increase in N2 amplitude and reductions in P2 and P3 amplitudes, while no change was observed for early P1 and N1 ERP components. It was inferred that ABM is associated with modulation of later information processing, including increased attentional control, and a reduction in the neurocognitive resources allocated to the processing of the emotional properties of stimuli. Further ERP studies have similarly suggested that ABM may influence later stages of information processing (Osinsky, Wilisz, Kim, Karl, & Hewig, 2014; Suway et al., 2013), although the modulation of earlier attentional processing following ABM has also been observed (O'Toole & Dennis, 2012). While recent findings generally implicate that ABM may facilitate attentional control, no direct assessment of changes in attentional control performance following ABM has been conducted.

However, such potential ABM-related changes in attentional control may be examined by changes in antisaccade task (Hallett, 1978) performance. The antisaccade task is a well established method for the assessment of inhibitory attentional control, and has been previously applied to range of psychopathology (Hutton & Ettinger, 2006). This task involves the presentation of a peripheral stimulus. Participants are required to either prosaccade (look towards) or antisaccade (look away) from the stimulus. While the prosaccade is largely a reflexive response, central to the execution

of the antisaccade is the inhibition of the prepotent prosaccade response, prior to the subsequent generation of a volitional saccade (Munoz & Everling, 2004). Critically, correct antisaccade performance necessitates the recruitment of attentional control-related brain regions, such as the lateral PFC, thus supporting its use as an index of inhibitory attentional control (Ettinger et al., 2008; Hutton & Ettinger, 2006; Munoz & Everling, 2004). Saccade latency and error rates are typically measured.

Indeed, the antisaccade task has been a useful tool in the elucidation of the attentional anomaly marked in anxiety. Attentional Control Theory (Eysenck et al., 2007) further posits that while anxiety is characterized by a deficit in attentional control, such a deficit may primarily impair the *efficiency* domain of processing, which is typically indexed by speed or reaction time measures. In contrast, the *effectiveness* of processing, inferred from measures such as error rates, is considered to be relatively unimpaired. Consistent with this notion, high trait anxious individuals, in comparison to low anxious individuals, have typically been found to exhibit longer antisaccade latencies, while no differences in error rates were observed (Ansari & Derakshan, 2009, 2011; Ansari, Derakshan, & Richards, 2008). Such findings suggest that anxiety may be associated with a deficit in the efficiency of attentional control.

In order to further examine whether attentional control deficits may be general or more specific with regard to the inhibition of threatening information, an emotional variant of the antisaccade task has previously been employed (Derakshan, Ansari, Hansard, Shoker, & Eysenck, 2009). While the standard antisaccade task uses solely a neutral stimulus, the emotional antisaccade task additionally presents socially relevant emotionally valenced stimuli. Anxious individuals have exhibited longer latencies to antisaccade away from threat stimuli (Derakshan et al., 2009; Reinholdt-Dunne et al., 2012), suggesting a specific impairment in inhibiting the attentional deployment to threatening information. Given the findings in high trait anxious individuals, social anxiety may be associated with a deficit in inhibitory attentional control, and such a deficit may be particularly apparent for the inhibition of threat stimuli.

The present study sought to assess whether ABM may modulate inhibitory attentional control in a non-clinical sample, as assessed by performance changes in an emotional antisaccade task administered before and after ABM. It is well established that antisaccade latencies are longer than prosaccade latencies (Munoz & Everling, 2004). This difference is primarily thought to reflect the inhibition of the prepotent saccade needed in order to perform an antisaccade (Olk & Kingstone, 2003), and is typically referred to as the antisaccade *cost* (Godijn & Kramer, 2008; Reinholdt-Dunne et al., 2012). Given recent findings (Browning et al., 2010; Eldar & Bar-Haim, 2010), it was hypothesized that ABM, in comparison to a placebo task, would subsequently increase inhibitory attentional control, indicated by a relative reduction in the antisaccade cost. In light of previous antisaccade research suggesting both general (e.g., Ansari & Derakshan, 2009) and threat-specific (e.g., Derakshan et al., 2009) inhibitory impairments in anxiety, two alternate hypotheses were formed. It was first predicted that ABM would result in a general facilitation of inhibitory attentional control, inferred from a relative reduction antisaccade cost across all levels of stimulus valence following ABM. Alternatively, it was predicted that ABM may result in a threat-specific facilitation of inhibitory attentional control, indexed by a reduction in antisaccade cost for only threat stimuli.

A secondary aim of the present study concerned the relationship between attentional bias and inhibitory attentional control. ABM has previously been shown to augment attentional bias (e.g., MacLeod et al., 2002) and for the present study, it was hypothesized that ABM may additionally modulate attentional control. However, the relationship between such ABM-related changes remains to be determined. Hence, the present study sought to examine the