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Frontal and parietal EEG asymmetries interact to predict attentional bias to threat

Gina M. Grimshaw [a,*], Joshua J. Foster [a,1], Paul M. Corballis [b]

[a] School of Psychology, Victoria University of Wellington, PO Box 600, Wellington 6140, New Zealand
[b] School of Psychology, University of Auckland, Private Bag 92019, Auckland 1142, New Zealand

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ABSTRACT

Frontal and parietal electroencephalographic (EEG) asymmetries mark vulnerability to depression and anxiety. Drawing on cognitive theories of vulnerability, we hypothesise that cortical asymmetries predict attention to threat. Participants completed a dot-probe task in which bilateral face displays were followed by lateralised targets at either short (300 ms) or long (1050 ms) SOA. We also measured N2pc to face onset as an index of early attentional capture. At long SOA only, frontal and parietal asymmetry interacted to predict attentional bias to angry faces. Those with leftward frontal asymmetry showed no attentional bias. Among those with rightward frontal asymmetry those with low right parietal activity showed vigilance for threat, and those with high right parietal activity showed avoidance. Asymmetry was not related to the N2pc or to attentional bias at the short SOA. Findings suggest that trait asymmetries reflect function in a fronto-parietal network that controls attention to threat.

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1. Introduction

Trait asymmetries in frontal activity, most commonly measured in EEG alpha power, have been identified as a marker of vulnerability to depression and anxiety (Coan & Allen, 2004; Thibodeau, Jorgensen, & Kim, 2006). Because alpha power decreases with increasing cognitive activity (Klimesch, 1999; Pfurtscheller, Stancak, & Neuper, 1996), inverse measures of alpha have been used to provide relative measures of left and right frontal activity (Allen, Coan, & Nazarian, 2004; Coan & Allen, 2004). While healthy individuals display a pattern of greater left than right frontal activity, a shift toward greater right than left activity is seen in those with current (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991) and remitted depression (Gotlib et al., 1998; Henriques & Davidson, 1990; Stewart, Coan, Towers, & Allen, 2011). The same pattern is seen in infants of depressed mothers (Field & Diego, 2008) and in those with genetic or familial risk of disorder (Bismark et al., 2010; Feng et al., 2012; Smit, Posthuma, Boomsma, & De Geus, 2007). Most importantly, a rightward pattern of frontal asymmetry prospectively predicts future depression (Mitchell & Pössel, 2012; Nusslock et al., 2011). Such findings indicate that frontal asymmetry is a marker of *vulnerability* to depression, and not a correlate of the disorder itself. A rightward shift in frontal asymmetry is also seen in current anxiety (Mathersul, Williams, Hopkinson, & Kemp, 2008; Tomarken & Davidson, 1994), and prospectively predicts future anxiety (Blackhart, Minnix, & Kline, 2006). Frontal asymmetry shows high internal reliability (Hagemann, 2004) and reasonable stability across time (Allen, Urry, Hitt, & Coan, 2004), making it useful as a trait marker for neurological vulnerability to depression and anxiety.

Most research on the relationship between asymmetry and emotional disorders has focused on frontal asymmetry, but relations between parietal asymmetry and both depression and anxiety have also been reported. Although depression and anxiety are highly co-morbid, they show diverging patterns of parietal asymmetry. Depression (particularly if it is not co-morbid with anxiety) is associated with relatively low right parietal activity (that is, a leftward shift in parietal asymmetry; Bruder et al., 1997; Kentgen et al., 2000; Stewart, Towers, Coan, & Allen, 2011). Reduced right parietal activity is also seen in those with familial risk of depression (Bruder et al., 2012; Bruder, Tenke, Warner, & Weissman, 2007). In contrast, anxiety is associated with relatively high right parietal activity, regardless of co-morbid depression (Bruder et al., 1997; Metzger et al., 2004). Studies that have focused on subtypes of anxiety indicate that increased right parietal activity is specifically correlated with anxious arousal (the somatic component of anxiety) and not with anxious apprehension

* Corresponding author. Fax: +64 4 463 5402.
E-mail addresses: gina.grimshaw@vuw.ac.nz (G.M. Grimshaw), jfoster2@uoregon.edu (J.J. Foster), p.corballis@auckland.ac.nz (P.M. Corballis).
[1] Address: Department of Psychology, 1227 University of Oregon, Eugene, OR 97403-1227, USA.

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(i.e., worry; Heller, Nitschke, Etienne, & Miller, 1997; Nitschke, Heller, Palmieri, & Miller, 1999).

The potential role of fronto-parietal interactions in emotional disorders was first highlighted by Heller's (1993) circumplex model of emotion. Based on the observed patterns of frontal and parietal asymmetry across disorders, Heller posited that asymmetries in frontal activity reflect the valence component of emotion (left/positive; right/negative) and the level of right parietal activity reflects arousal such that, when combined with a rightward frontal asymmetry, very low levels of right parietal activity are characteristic of depression, and very high levels of right parietal activity are characteristic of anxiety. Since Heller's original formulation of the model, the frontal valence component has been largely reconceptualised as an asymmetry in motivation (left/approach; right/withdrawal; Harmon-Jones, 2003; Harmon-Jones, Gable, & Peterson, 2010; but see Miller, Crocker, Spielberg, Infantolino, & Heller, 2013). Regardless of the exact nature of the frontal component (be it valence or motivation), the model provides a framework within which we can examine the relative roles of frontal and parietal activity in generating vulnerability to depression and anxiety.

Early studies of frontal (and to some extent, parietal) asymmetries focused on their relationship to emotional responding or affective style (e.g., Davidson, 1998). However, more recent research has focused on cognitive correlates. The search for candidate processes is largely motivated by cognitive theories of depression and anxiety which suggest that, under emotional stress, vulnerable individuals activate a constellation of negative biases in attention, interpretation, and memory that play a causal role in the onset and maintenance of disorder (Beck, 2008; Beck & Clark, 1997; Clarke, MacLeod, & Shirazee, 2008; Gotlib & Joormann, 2010; Mathews & MacLeod, 2005; Ouimet, Gawronski, & Dozois, 2009). Importantly, these cognitive biases are proposed to exist in some healthy individuals, and in the presence of sustained life stress can give rise to depression or anxiety. It is possible that frontal and parietal asymmetries are the neural instantiations of some aspects of this cognitive vulnerability. If so, individual differences in trait asymmetry should predict negatively-biased cognitive processing, even in individuals who are not currently anxious or depressed.

We focus here on the relationship between trait asymmetries and attentional bias to threat for two reasons. First, although alpha power has long been assumed to inversely reflect "cognitive activity" (e.g., Coan & Allen, 2004; Klimesch, 1999), current conceptualisations of oscillatory brain activity allow us to be much more specific (Grimshaw & Carmel, 2014; Miller et al., 2013). Studies that measure simultaneous resting EEG and fMRI find that alpha is correlated with activity in the dorsal fronto-parietal network (Laufs et al., 2003; Mantini, Perrucci, del Gratta, Romani, & Corbetta, 2007) that plays an important role in top-down modulation of visual processing (Corbetta & Shulman, 2002; Shomstein, Kravitz, & Behrmann, 2012; Suzuki & Gottlieb, 2013, Vossel, Geng, & Fink, 2014). Functionally, alpha appears to play an important role within this network in attentional control and the gating of perceptual awareness (Hanslmayr, Gross, Klimesch, & Shapiro, 2011; Mazaheri et al., 2013; Sadaghiani et al., 2012). And while few studies have applied source localisation procedures to EEG asymmetry data, those that have done so localise frontal asymmetry primarily to dorsal–lateral prefrontal cortex (dlPFC) which is a key node in the frontal–parietal network (Koslov, Mendes, Pajtas, & Pizzagalli, 2011; Pizzagalli, Sherwood, Henriques, & Davidson, 2005).

Linking alpha asymmetries to attentional processes in this way is consistent with an emerging consensus that cognitive processes like attention and executive control are core aspects of emotional processing (Ochsner & Gross, 2005; Pessoa, 2008). Thus, cortical asymmetries may predict personality traits and emotional vulnerabilities because they reflect attentional control processes that are affected in those traits. In support of this hypothesis, EEG studies have found that rightward frontal asymmetry predicts vigilance to angry faces in a spatial cueing task (Miskovic & Schmidt, 2010), and experimentally increasing left frontal activity through the manipulation of approach-related motivation results in a narrowing of attention on Navon-like tasks, potentially through effects on top-down attentional control (Gable & Harmon-Jones, 2008; Harmon-Jones & Gable, 2009). These associations between trait asymmetries and attention suggest that the relationship between attentional bias to threat and both frontal and parietal asymmetry warrants further investigation.

The second reason that we focus on attentional bias to threat is that, although negative biases have been implicated in both depression and anxiety, the nature of the bias differs in the two disorders. These differences are most apparent on a common measure of attentional deployment, the dot-probe task (MacLeod, Mathews, & Tata, 1986). In a typical dot-probe paradigm, two cue stimuli (usually words or faces; one neutral and one emotional) are presented briefly, followed by a probe stimulus at the location of one of the cues. Importantly, the emotionality of the cue does not predict the location of the probe and is therefore irrelevant for the task. Faster responses to probes that are co-located with the emotional stimulus indicate an attentional bias to emotion. A key variable in dot-probe studies is stimulus onset asynchrony (SOA), the time between the onset of the cues and the onset of the probe. Biases at different SOAs reflect biases in different stages of processing (Cisler & Koster, 2010). Biases at short SOAs (typically less than 500 ms) reflect rapid orienting to the threat stimulus, and are ubiquitously observed in individuals who are clinically anxious or high in trait anxiety (see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007, for a meta-analysis), but not typically in depressed or healthy individuals (Bar-Haim et al., 2007; De Raedt & Koster, 2010). Rapid orienting to a threat stimulus is thought to arise through hypersensitivity of a largely stimulus-driven threat detection mechanism that involves the amygdala, coupled with a failure of control mechanisms in left lateral prefrontal cortex that normally suppress processing of task-irrelevant threat stimuli (Bishop, Duncan, Brett, & Lawrence, 2004; Bishop, Jenkins, & Lawrence, 2007).

With longer durations between cues and targets, healthy individuals again show no systematic bias (Bar-Haim et al., 2007). However, two different biases that reflect top-down attentional control mechanisms can be observed in some individuals. One is a bias toward threat, which is thought to reflect difficulty in disengaging attention from a threat stimulus, and may reflect failure of a dorsal fronto-parietal network to control the deployment of attention (Corbetta & Shulman, 2002; Eysenck, Derakshan, Santos, & Calvo, 2007; De Raedt & Koster, 2010). Attentional bias to threat at long SOAs has been associated with anxiety (Bradley, Mogg, Falla, & Hamilton, 1998; Mansell, Ehlers, Clark, & Chen, 2002), depression (Kircanski, Joormann, & Gotlib, 2012; Leyman, De Raedt, Schacht, & Koster, 2007), and familial vulnerability to depression (Joormann, Talbot, & Gotlib, 2007). Alternatively, some studies show anxiety to be related to an attentional bias *away* from threat at long SOAs (e.g., Chen, Ehlers, Clark, & Mansell, 2002; Koster, Verschuere, Crombez, & van Damme, 2005; Onnis, Dadds, & Bryant, 2011). Bias away from threat is a key feature of the vigilance–avoidance hypothesis of anxiety (e.g., Mogg, Bradley, Miles, & Dixon, 2004; Weinberg & Hajcak, 2011), which posits that anxious individuals show an involuntary rapid orienting toward threat, followed by a strategic shifting of attention away from threat in order to reduce or control their anxiety. Avoidance is thus an emotion regulation strategy, and likely mediated by frontal emotional control systems (Cisler & Koster, 2010). In support of this claim, Judah, Grant, Lechner, and Mills (2013) recently showed that anxiety-related avoidance in the dot-probe paradigm depends

on the availability of cognitive resources. They found that socially-anxious participants showed avoidance of disgusted faces at a long SOA. However, when the dot-probe trials were completed under working memory load (taxing control mechanisms rooted in frontal cortex), anxious participants showed vigilance for the disgusted faces instead (Judah et al., 2013).

Two studies have previously examined the relationship between cortical asymmetry and attentional bias to threat in the dot-probe task. In an early study, Schutter, Putman, Hermans, and van Honk (2001) found that attentional bias to angry faces (with a 500 ms SOA) was associated with parietal asymmetry in the beta band (13–20 Hz), but not in alpha. Given that there is no clear delineation of the functional significance of beta asymmetry, this finding is difficult to interpret. However, a more recent study (Pérez-Edgar, Kujawa, Nelson, & Zapp, 2013) showed that resting parietal asymmetry in the alpha band predicted attentional bias to angry faces at a 500 ms SOA. Participants with high right parietal activity showed a bias away from emotional faces, and those with low right parietal activity showed a bias toward emotional faces. Pérez-Edgar and colleagues also used a stress manipulation (preparing a speech) to assess changes in cortical asymmetry in response to stress. Frontal asymmetry before the stressor did not predict attentional bias, however, changes in frontal asymmetry as a result of the stressor did. Those individuals who showed increased left frontal activity during the stressor showed no attentional bias, either toward or away from, angry or happy faces. However, those who responded to the stressor with an increase in right frontal activity showed attentional biases toward angry and away from happy faces. Their findings are consistent with a role for left frontal cortex in cognitive control in the face of emotional distraction.

In the present study, we extend this previous research to test the hypothesis that trait asymmetries in frontal and parietal activity are neural correlates of attentional bias to threat. We further consider not just the independent roles of frontal and parietal asymmetry, but their interaction. If frontal and parietal asymmetry reflect different aspects of attentional processing, they may well interact to predict attentional bias to threat. The circumplex model itself also predicts an interaction. According to the model, it is only those with rightward frontal asymmetry who are vulnerable to disorder; within that group, low right parietal activity predicts depression and high right parietal activity predicts anxiety. Our emerging understanding of frontal–parietal interactions in attention provides a more specific mechanism that predicts the same interaction. Specifically, leftward frontal asymmetry has been associated with good cognitive control in the face of emotional distraction (Banich et al., 2009; Bishop et al., 2004, 2007; Pérez-Edgar et al., 2013). We might therefore expect people with leftward frontal asymmetry to show no attentional bias on the dot-probe task. In the absence of good cognitive control (as reflected in a rightward pattern of frontal asymmetry), attentional biases that are related to parietal asymmetry can emerge. Although some studies on the correlates of cortical asymmetry report relationships with both frontal and parietal asymmetry, to our knowledge none have tested their interaction.

We examined attentional bias to both angry and happy faces, at short (300 ms) and long (1050 ms) SOAs. We used both emotions so we could determine whether any relationship was specific to threat, and used both SOAs so we could determine whether asymmetries were associated with early attentional orienting revealed at the short SOA, or with later strategic and controlled attentional processes revealed at the long SOA, or both. Previous studies have used a 500 ms SOA which does not clearly dissociate early from late attentional processes. Finally, because cognitive theories of vulnerability to depression and anxiety posit that negative cognitive biases are activated under conditions of emotional stress (Gotlib & Joormann, 2010; see Pérez-Edgar et al., 2013), we presented unpleasant images from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) before each block of experimental trials. The images were not included as an experimental manipulation; rather all participants saw them in order to maximise the likelihood of instituting a negative attentional bias in vulnerable individuals before the dot-probe task.

We also supplemented the traditional response time measures of attention bias by recording EEG during performance of the dot-probe task to obtain online measures of attentional processing. We focused on the N2pc, a useful component for tracking attentional selection of lateralised stimuli within multi-stimulus displays (Luck, 2012). The N2pc is defined as a voltage detected at posterior electrodes that is more negative at sites contralateral than ipsilateral to an attended stimulus. It is typically observed 200–300 ms after stimulus onset (Eimer, 1996; Luck & Hillyard, 1994). The N2pc likely reflects processes involved in spatially selective processing of an attended stimulus (Eimer, 1996; Mazza, Turatto, & Caramazza, 2009), and it has already proven useful in tracking attentional selection of emotional faces (Eimer & Kiss, 2007; Holmes, Bradley, Kragh Nielsen, & Mogg, 2009; Ikeda, Sugiura, & Hasegawa, 2013). In a dot-probe task similar to the one used here, Holmes et al. (2009) showed that angry and happy faces both show a selection advantage (as revealed in the N2pc) over neutral faces. The N2pc is also sensitive to individual differences in the attentional processing of emotional stimuli (Buodo, Sarlo, & Munafò, 2010; Fox, Derakshan, & Shoker, 2008; Weymar, Gerdes, Löw, Alpers, & Hamm, 2013). For example, Fox et al. (2008) found that the N2pc elicited by angry faces in a dot-probe task was modulated by trait anxiety; angry faces elicited an N2pc for participants high in trait anxiety but not for participants low in trait anxiety. Because it arises relatively soon after stimulus onset, the N2pc may be a good measure of early attentional selection of emotional faces. It thus complements the RT measure of attentional bias at short SOA. In contrast, attentional bias at long SOA (measured in RT) provides a measure of later and more strategic allocation of attention.

Participants were young women without clinical depression or anxiety, or history of disorder. We focused on healthy participants because our hypothesis is that trait asymmetries reflect biases in attentional processing that confer vulnerability to future disorder. Thus, relationships between trait asymmetry and attentional biases should be observed even in a non-clinical population with no history (past or present) of depression or anxiety. We limited our study to young women because depression is a heterogeneous disorder, with a different manifestation (and therefore likely different causes and vulnerability factors) in men and women (Hankin & Abramson, 2001; Nolen-Hoeksema, 2001). Furthermore, while the relationship between cortical asymmetry and vulnerability to depression is robust in women (Thibodeau et al., 2006), it is less clear that the same relationship holds in men (Stewart, Bismark, Towers, Coan, & Allen, 2010; Stewart, Towers, et al., 2011). Given that our goal was to target the brain asymmetry–attentional bias relationship, studying participants of only one sex allowed us to reduce heterogeneity and therefore maximise our power to detect the relationships of interest. Women in the 18–24 year age range (emerging adults) are at relatively high risk for first-episode depression (Kessler et al., 2005; Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013). Therefore, even though our young sample did not report current or historical depression or anxiety, it likely includes women who vary in cognitive vulnerability.

2. Method

2.1. Participants

Forty-four right-handed women between the ages of 18 and 25 (M_{age} = 19.75 years, SD = 2.08) were recruited from an introductory

psychology course and from advertisements around campus. All participants had normal or corrected-to-normal vision and reported no history of depression, anxiety, or neurological disorder. The study was conducted with the approval of the Human Ethics Committee of the School of Psychology, Victoria University of Wellington (Wellington, New Zealand). All participants gave written informed consent prior to participation.

2.2. Procedure and apparatus

Testing took place in a dimly lit, electrically shielded chamber. Participants were fitted with a Lycra Quik-Cap cap embedded with 28 recording electrodes (Compumedics Neuromedical Supplies). All participants completed the tasks in the same order. First we recorded 4 min of resting EEG in four 1-min blocks. Participants had their eyes open (EO) for two blocks and closed (EC) for two blocks, and block order (EO–EC–EC–EO or EC–EO–EO–EC) was counterbalanced across participants. They then completed the dot-probe task followed by questionnaires to assess current depression and anxiety. They were seated in a comfortable armchair during recording of resting EEG. During the dot-probe task, a chinrest maintained head position and a constant viewing distance of 60 cm. The experiment was presented on a Dell Optiplex 760 computer with a Dell 1908FPb 19'' LCD monitor (1024×768 pixels, 60 Hz refresh rate). E-prime software (Schneider, Eschman, & Zuccolotto, 2002) was used to control stimulus presentation, record responses, and to synchronise stimulus presentation with electrophysiological recordings. EEG was amplified using standard BrainAmps and digitized using Brain-Vision Recorder software (Brain Products, Gilching, Germany).

2.3. Dot-probe task

2.3.1. Stimuli

All stimuli were presented on a black background. The face stimuli consisted of pairs of grey-scaled photographs of four male actors (M10, M11, M13, and M31) taken from the A series of the Karolinska Directed Emotional Faces database (Lundqvist, Flykt, & Öhman, 1998). These actors were selected based on the unbiased hit-rates[2] reported by Goeleven et al. (2008). Unbiased hit rates for the selected individuals were high (>80%) for angry, happy, and neutral expressions. One additional male actor was selected for presentation during the practice trials. The face stimuli were cropped to include only the face. These modified images subtended $6.9° \times 8.9°$ of visual angle. Each stimulus display consisted of two photographs of the same model; one portraying an emotional expression (either angry or happy) and one portraying a neutral expression. Faces were presented laterally and centred along the horizontal meridian, with the inner edge of each face appearing 2.4° of visual angle from fixation. The probe stimulus was either a white square subtending $0.6° \times 0.6°$ of visual angle or a white diamond (the same stimulus rotated 45°). The probe was centred 5.9° of visual angle to the left or right of the fixation cross (i.e., centred at the same location as one of the preceding face stimuli).

2.3.2. Procedure

Each trial began with a fixation cross (subtending $0.4° \times 0.4°$ of visual angle) which remained onscreen for the duration of the trial. After a random delay of between 800 and 1200 ms, a pair of face cues was presented. The faces were presented for either 250 ms or 1000 ms after which the screen was blank for 50 ms (except for the fixation cross). The probe was then presented in the visual field previously occupied by the emotional face (*valid cue*) or in the visual field previously occupied by the neutral face (*invalid cue*).[3] Participants were instructed to indicate the probe identity (square or diamond) as quickly and accurately as possible. Participants responded with the index and middle fingers of their right hand on the "1" and "2" keys of the numberpad on a standard keyboard. The response mapping was counterbalanced across participants. The probe remained onscreen until a response was made or until three seconds had elapsed. A feedback display was then presented for 500 ms, consisting of a blank screen after correct responses, "Incorrect" written in red text after incorrect responses, or "Please respond faster." written in red text when no response was made within three seconds of probe onset. Each trial was followed by a 500 ms inter-trial interval.

Participants completed 16 practice trials followed by two blocks of 256 trials (512 trials in total). The independent variables were SOA (300 or 1050 ms), emotion (angry-neutral or happy-neutral), and cue validity (valid or invalid), all of which were manipulated within-subjects. Each block of trials comprised two presentations of each unique combination of SOA, emotion, cue validity, location of emotional face (left visual field or right visual field), and probe identity for each of the four models ($2 \times 2 \times 2 \times 2 \times 2 \times 2 \times 4 = 256$ trials/block). Trial order within each block was randomised, and participants took a short break halfway through each block.

Participants viewed a series of images taken from the IAPS (Lang et al., 2008) before each block of trials. This was done to maximise the likelihood of instituting a negative attentional bias. Each series included six unpleasant and six neutral images[4]. Images were selected based on valence ratings from female college students obtained in a validation study of the IAPS (Lang et al., 2008). The mean valence rating was 1.42 ($SD = 0.20$) for unpleasant images and 4.95 ($SD = 0.32$) for neutral images on a 9-point scale (1 = unpleasant, 9 = pleasant). Each image was presented for six seconds, and the order of the images was randomised within each series. To ensure that participants attended to the images, they were required to indicate, using the number pad on a standard keyboard, the number of human bodies present in each image.

2.4. Questionnaires

Depression was assessed with the Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21 item scale that assesses cognitive, emotional, and somatic symptoms of depression. Possible scores on the BDI-II range from 0 to 63, with scores greater than 29 indicating clinically-significant depression. One participant was excluded because her score exceeded this cut-off. Scores in the remaining sample ranged from 0 to 25 ($M = 8.51$, $SD = 6.13$). Anxious apprehension was assessed using the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), in which participants rate how well 16 statements characterise them on a 5-point scale (1 = not at all typical of me, 5 = extremely typical of me). Potential scores therefore range from 16 to 80. Scores in our sample ranged from 23 to 76 ($M = 50.27$, $SD = 15.79$). Anxious arousal was assessed with the Anxious Arousal subscale of the Mini Mood and Anxiety Symptoms Questionnaire (Mini MASQ; Clark & Watson, 1995), which consists of 10 items rated on a similar 5-point scale.

[2] Because there are a large number of possible emotions in an identification study, the unbiased hit rate has been corrected for the baserate of using any specific emotional category, see Goeleven, De Raedt, Leyman, and Verschuere (2008).

[3] The terms valid and invalid are commonly used when describing dot-probe studies to distinguish between targets that appear in the same location as the emotional stimulus vs. those that appear in the same location as the neutral stimulus. We use these terms here for consistency with that literature, but note that the emotion of the face does not predict the location of the target.

[4] The following IAPS images were used in the mood induction task: Unpleasant: 3001, 3015, 3053, 3261, 9075, 9140, 9410, 9412, 9413, 9570, 9635.1, and 9940; Neutral: 2190, 2840, 5130, 5740, 7000, 7006, 7020, 7031, 7041, 7060, 7080, and 7090.

Potential scores therefore range from a minimum of 10 to a maximum of 50. Scores on the Anxious Arousal scale of the Mini MASQ in our sample ranged from 10 to 25 ($M = 13.74$, $SD = 3.95$). Although population norms are not available for either the PSWQ or the Mini MASQ, these values are well within typical ranges for non-clinical student samples, and none of our participants were outliers on either measure (Meyer et al., 1990; Reidy & Keogh, 1997).

2.5. EEG data acquisition

The EEG was recorded from 28 Ag/AgCl electrodes (FP1, FP2, F7, F3, FZ, F4, F8, FT7, FC3, FC4, FT8, T7, C3, CZ, C4, T8, TP7, CP3, CP4, TP8, P7, P3, PZ, P4, P8, O1, OZ, and O2, according to the modified 10–20 system; American Electroencephalographic Society, 1994) referenced to the left mastoid. To detect eye movements and blinks, the electrooculogram (EOG) was recorded from electrodes positioned above and below the left eye and on the outer canthus of each eye. The EEG and EOG channels were filtered online with a highpass filter of 0.016 Hz, and digitally sampled at 500 Hz. Impedances at all critical electrodes (F7, F3, F4, F8, P7, P3, P4, P8, and the mastoids) were kept below 10 kΩ. Offline, the 28 scalp channels were re-referenced to the algebraic average of the left and right mastoids, were notch filtered at 50 Hz, and were digitally bandpass filtered from 0.1 to 30 Hz using a zero-phase-shift Butterworth filter (12 dB/oct). Vertical EOG (VEOG) was calculated as the difference between the electrodes positioned above and below the left eye, and horizontal EOG (HEOG) was calculated as the difference between electrodes positioned on the outer canthus of each eye.

2.5.1. Resting EEG analysis

EEG asymmetry measures were calculated following recommended procedures (Allen, Coan, et al., 2004; Hagemann, 2004). Each 1-min block was divided into epochs of 1024 ms with 50% overlap. Epochs with recording artefacts (voltage exceeding ±100 μV at any frontal or parietal electrodes) were removed. Epochs with blinks or saccades were rejected on the basis of visual inspection. Alpha power (8–13 Hz) in each epoch was extracted using a fast Fourier transform (Hamming window of 10%) and averaged across all epochs. Alpha power densities (power per unit bandwidth) were then averaged across the four 1-min blocks, because averaging across EO and EC conditions yields a more reliable estimate of frontal asymmetry than either condition alone (Hagemann, 2004; Tomarken, Davidson, Wheeler, & Kinney, 1992). Alpha power densities were averaged across lateral and medial electrode sites to form measures of left (F3/F7) and right (F4/F8) frontal power, and left (P3/P7) and right (P4/P8) parietal power (Nusslock et al., 2011). Frontal [ln(mean of F4/F8) − ln(mean of F3/F7)] and parietal [ln(mean of P4/P8) − ln(mean of P3/P7)] asymmetry indices were then calculated based on these combined regions. Split half reliabilities (with Spearman–Brown correction for test length) were .86 for frontal asymmetry and .80 for parietal asymmetry. For both asymmetry indices, positive values reflect relatively greater right alpha power and negative values reflect relatively greater left alpha power. Therefore, positive values of the asymmetry indices are taken to reflect greater relative left activity because alpha power is inversely related to cognitive activity (Allen, Coan, et al., 2004).

2.5.2. ERP analysis

Analysis of ERPs was confined to occipital–temporal electrodes P7 and P8, where the N2pc has been reported to be maximal in a similar dot-probe task (Holmes et al., 2009). EEG was segmented into 600 ms epochs (including a 100 ms pre-stimulus baseline), time-locked to onset of the faces. Segments were baseline corrected by subtracting the average signal recorded during the pre-stimulus baseline. Segments containing lateral eye movements (a change in voltage in the HEOG channel exceeding 50 μV within any 200 ms period), eyeblinks[5] (a change in the VEOG channel exceeding 100 μV within any 200 ms period), or other artefacts (a voltage exceeding ±100 μV at electrodes P7/P8) were rejected. One participant was excluded from the ERP analyses because high incidence of recording artefacts prevented identification of lateral eye movements. In the remaining 42 participants, recording artefacts or eye movements led to the rejection of an average of 11.6% of trials per participant.

To ensure that the N2pc was not contaminated by systematic eye movements, we inspected a recalculated HEOG channel for evidence of these artefacts. This new HEOG channel was calculated as the contralateral–ipsilateral difference (relative to the location of the emotional face). Lateral eye movements towards the emotional face would generate a negative shift in the recalculated HEOG channel. Averaged HEOG was inspected for angry and happy cues separately. Averaged HEOG activity did not exceed ±5 μV for any participant for either cue type. Furthermore, grand average HEOG activity did not exceed 3.2 μV for either cue type, ensuring that systematic eye-movements did not exceed 0.2° and that propagated voltage at posterior sites did not exceed 0.1 μV (Lins, Picton, Berg, & Scherg, 1993).

3. Results

3.1. Behavioural analyses

Trials with RTs less than 200 ms or greater than 1000 ms were discarded as outliers, leading to the rejection of an average of 6.1% of trials per participant. Mean RTs for correct trials were calculated for each participant in each condition (see Table 1). No participant's mean RT was based on fewer than 42 correct responses per cell. RTs were analysed in a 2 (SOA: 300 or 1050 ms) × 2 (emotion: angry-neutral or happy-neutral) × 2 (cue validity: valid or invalid) repeated measures analysis of variance (ANOVA). RTs were significantly faster at the shorter SOA, $F(1,42) = 12.249$, $p = .001$, $\eta_p^2 = .226$. The main effects of cue validity and emotion, and all interactions, were non-significant. Thus, in the sample as a whole, there was no systematic attentional bias either toward or away from emotional faces, at either SOA.

3.2. N2pc

The N2pc is defined as a more negative voltage at posterior electrodes in the contralateral waveform than in the ipsilateral waveform, typically beginning around 200 ms post-stimulus. Average waveforms observed at electrode sites P7/P8 were used to test for an N2pc component related to facial expression. Fig. 1A shows the average waveforms at P7/P8 that were contralateral or ipsilateral to the emotional face, collapsed across the short and long SOA conditions. For both angry and happy cues, a clear N2pc started approximately 200 ms after cue onset, seen as a more negative contralateral waveform compared to the ipsilateral waveform (see Fig. 1B). We analysed the N2pc amplitude in a 150-ms window running from 200 to 350 ms post-stimulus. Although the probe in the short SOA conditions was presented within this N2pc time window (300 ms after onset of the faces), it was not expected to produce any difference in the waveform until after the end of the window. This was confirmed by including SOA as a variable in a

[5] Due to technical difficulties, 10 participants had a high incidence of recording artefacts at the electrode placed above the left eye, preventing identification of eyeblinks. For these participants, eyeblinks were rejected based on voltages at electrodes FP1 and FP2; segments containing a change in either of these channels exceeding 100 μV within any 200 ms period were rejected.

Table 1
Mean RTs and standard deviations on dot probe task.

SOA	Trials			
	Valid		Invalid	
	M	*SD*	*M*	*SD*
Short				
Angry	631	47	630	45
Happy	635	47	631	45
Long				
Angry	640	55	643	52
Happy	644	53	646	52

2 (laterality: contralateral, ipsilateral) × 2 (emotion: angry-neutral, happy-neutral) × 2 (SOA: 300 or 1050 ms) repeated measures ANOVA. The main effect of laterality, $F(1,41) = 54.334$, $p < .001$, $\eta_p^2 = .570$, indicated an N2pc that further interacted with emotion, $F(1,41) = 4.269$, $p < .05$, $\eta_p^2 = .094$. Although the N2pc was significant for both, it was larger for angry, $F(1,41) = 42.459$, $p < .001$, $\eta_p^2 = .509$, than for happy faces, $F(1,41) = 21.140$, $p < .001$, $\eta_p^2 = .340$. Critically, there were no main effects or interactions involving SOA ($Fs < 2$), indicating that the N2pc effects did not differ in the short and long SOA conditions. We therefore collapsed across short and long SOA to calculate N2pc amplitudes for angry and happy faces for subsequent analyses.

It is always possible that systematic sensory imbalances may have accounted for the N2pc effect. If so, the difference between the angry and neutral faces on lateralised ERP waveforms should be greatest at short latencies, because sensory differences should predominantly influence early stages of processing (Mazza, Turatto, Umiltà, & Eimer, 2007). To discount this possibility, we examined lateralised ERP activity at electrodes P7/P8 in the P1 window (110–150 ms). These data were analysed in a 2 (laterality: contralateral, ipsilateral) × 2 (emotion: angry-neutral, happy-neutral) × 2 (SOA: 300 or 1050 ms) repeated measures ANOVA. Importantly, there was no main effect of laterality, and laterality did not

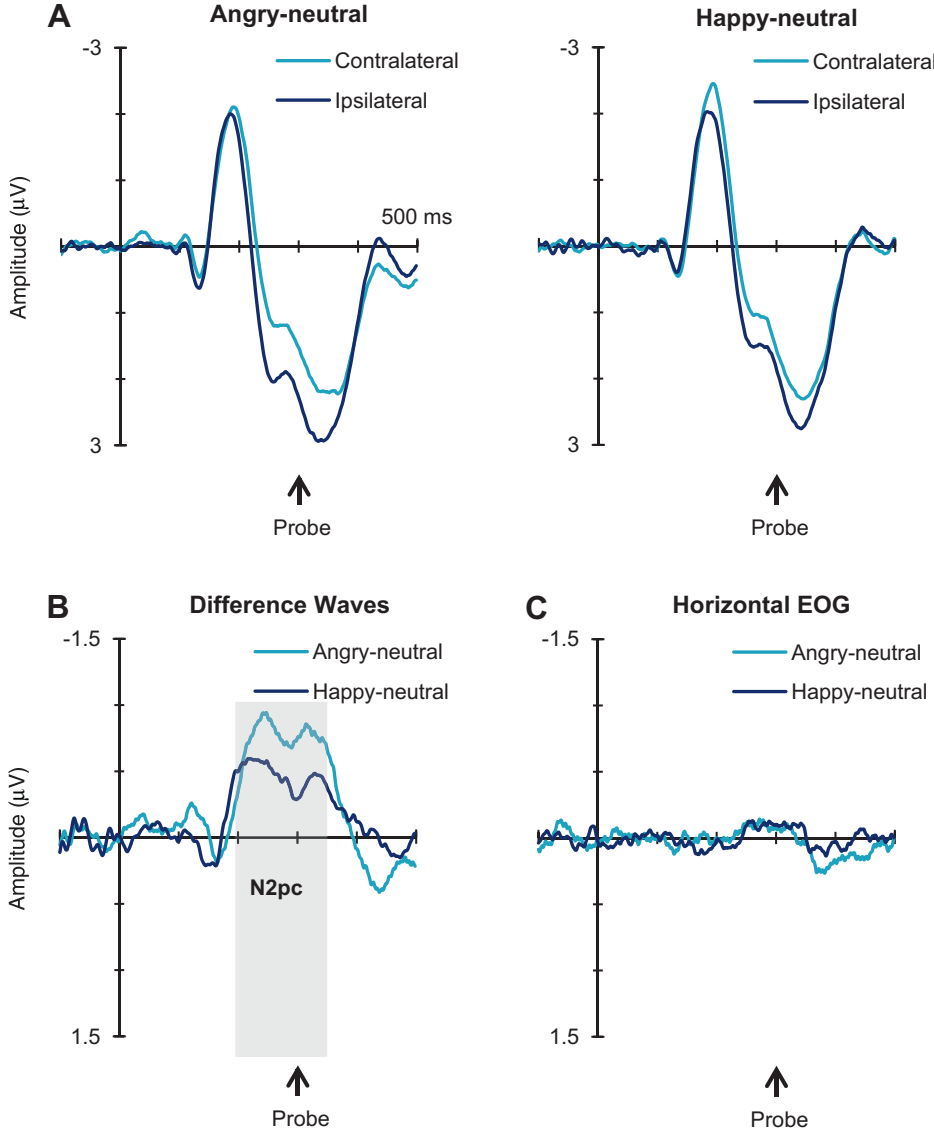

Fig. 1. (A) Grand average ERPs time-locked to the onset of angry-neutral (left) and happy-neutral (right) face cues at electrodes P7/P8 contralateral and ipsilateral to the emotional face in each pair. Waveforms are collapsed across short and long SOA trials. The arrow indicates onset of the probe at 300 ms on the short SOA trials. (B) Difference waveforms (contralateral–ipsilateral) at electrodes P7/P8 for angry-neutral and happy-neutral face cues. The N2pc time window (200–350 ms) is marked with a grey rectangle. (C) Horizontal EOG was recalculated such that negative voltages indicate eye movements towards the emotional face. EOG shows no systematic deflection in the N2pc time window.

interact with either of the other factors (Fs < 3), indicating that sensory differences between the emotional and neutral faces did not affect early lateralised ERP activity. Thus the N2pc effects cannot be attributed to sensory imbalances.

3.3. Predicting attentional biases

Although our dot-probe task did not reveal a consistent attentional bias across participants, there was considerable variability in individual participants' performance, with some showing bias toward, and some away from, emotional faces. We therefore used regression analyses to determine whether individual differences in frontal and/or parietal asymmetry (or their interaction) predicted individual differences in attentional bias. For each participant we calculated an attentional bias score based on their behavioural data for each combination of emotion and SOA by subtracting RTs for valid trials from RTs for invalid trials. Positive values therefore indicate attentional bias toward emotion, and negative values indicate attentional bias away from emotion. We also calculated the amplitude of the N2pc (i.e., the magnitude of the contralateral–ipsilateral difference at electrodes P7/P8) for each participant, as an index of early attentional selection of the emotional face. We therefore had six outcome variables; four behavioural measures (2 SOAs × 2 emotions) and two ERP measures (N2pc amplitude for angry and happy faces). In each regression we entered frontal and parietal asymmetry (centred) as predictors in the first step, followed by their interaction in the second step. We included questionnaire measures of depression and/or anxiety as predictors if their zero-order correlations with outcome variables were significant.

The zero-order correlations amongst all predictor and outcome variables appear in Table 2. As expected, questionnaire measures of depression and anxiety correlated with each other, even in this non-clinical sample. However, neither depression nor anxiety predicted behavioural or N2pc measures of attentional bias, with the exception of a negative correlation between attentional bias to happy faces at the long SOA and scores on the PSWQ, $r(42) = -.37$, $p = .004$.

The regressions to predict the ERP measures of attentional selection appear in Table 3 and the regressions to predict the behavioural measures of bias appear in Table 4 (for angry faces) and Table 5 (for happy faces). Notably, only one of these regressions produced a model that accounted for significant variance in attentional bias. For attentional bias to angry faces at long SOA, the first step of the regression (including frontal and parietal asymmetry as predictors) was significant, $F(2,40) = 3.473$, $p = .041$, and the inclusion of their interaction in the second step significantly improved the model, $F(1,39) = 4.980$, $p = .031$, yielding a significant complete model, $F(3,39) = 4.206$, $p = .011$, that accounted for 25% of the variance in attentional bias. Examination of the beta weights in Table 4 indicates that both parietal asymmetry and the frontal × parietal interaction were unique predictors of attentional bias. Regression diagnostics confirmed that there were no outliers and no influential data points in the regression (maximum Cook's distance = 0.13).

Table 3
Multiple regressions predicting N2pc.

	Predictor	R^2	ΔR^2	β	t	p
Angry faces						
Model 1		.048				.386
	FA			.195	1.204	.236
	PA			−.058	−.360	.721
Model 2		.102	.054			.246
	FA			.189	1.182	.245
	PA			−.130	−.783	.438
	FA × PA			.244	1.518	.137
Happy faces						
Model 1		.063				.281
	FA			.226	1.403	.168
	PA			−.065	−.403	.689
Model 2		.066	.003			.450
	FA			.227	1.397	.170
	PA			−.047	−.276	.784
	FA × PA			−.061	−.371	.712

Note. FA = frontal asymmetry; PA = parietal asymmetry; β = standardised beta.

Table 4
Multiple regressions predicting attentional bias to angry faces.

	Predictor	R^2	ΔR^2	β	t	p
Short SOA						
Model 1		.017				.712
	FA			−.132	−.818	.418
	PA			−.049	−.302	.764
Model 2		.022	.005			.833
	FA			−.142	−.865	.392
	PA			−.073	−.427	.672
	FA × PA			.075	.446	.658
Long SOA						
Model 1		.148				.041
	FA			−.181	−1.211	.233
	PA			.301*	2.006	.050
Model 2		.222*	.096*			.011
	FA			−.136	−.940	.353
	PA			.410*	2.713	.010
	FA × PA			−.329*	−2.232	.031

Note. FA = frontal asymmetry; PA = parietal asymmetry; β = standardised beta.
* $p < .05$.

Table 2
Correlations among study variables.

Variable	1	2	3	4	5	6	7	8	9	10	11
1. FA	–										
2. PA	−.23	–									
3. BDI-II	.10	.12	–								
4. PSWQ	.20	.06	.35*	–							
5. MASQ-AA	−.07	.02	.44**	.27	–						
6. Angry bias short SOA	−.05	.01	.10	.08	−.16	–					
7. Angry bias long SOA	−.17	.35*	.22	−.17	−.14	−.02	–				
8. Happy bias short SOA	−.06	.22	−.16	−.17	.14	.26	−.10	–			
9. Happy bias long SOA	.06	.04	−.16	−.31*	.01	.02	.15	.46**	–		
10. Angry N2pc	.19	−.09	−.19	−.03	−.13	−.07	.03	−.13	.09	–	
11. Happy N2pc	.07	.02	.23	.23	.24	.09	−.26	−.23	−.19	.08	–

Note. FA = frontal asymmetry; PA = parietal asymmetry; BDI-II = Beck Depression Inventory II; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptoms Questionnaire [Anxious Arousal].
* $p < .05$.
** $p < .01$.

Table 5
Multiple regressions predicting attentional bias to happy faces.

	Predictor	R^2	ΔR^2	β	t	p
Short SOA						
Model 1		.060				.292
	FA			.080	.510	.613
	PA			.250	1.587	.120
Model 2		.095	.035			.268
	FA			.053	.333	.741
	PA			.184	1.111	.273
	FA × PA			.199	1.234	.225
Long SOA						
Model 1		.153				.087
	FA			.004	.027	.979
	PA			.236	1.551	.129
	PSWQ			−.324*	−2.144	.038
Model 2		.177	.024			.107
	FA			−.209	.836	.441
	PA			.178	1.103	.277
	PSWQ			−.299	−1.953	.058
	FA × PA			.165	1.047	.301

Note. FA = frontal asymmetry; PA = parietal asymmetry; β = standardised beta; PSWQ = Penn State Worry Questionnaire.
* $p < .05$.

To further explore the nature of this interaction, we plotted it in modgraph (Jose, 2008). This graph (see Fig. 2) shows the relationship between parietal asymmetry and attentional bias at three different levels of frontal asymmetry; in those with asymmetry scores 1 *SD* or more below the mean (i.e., most rightward), those with asymmetry scores 1 *SD* or more above the mean (i.e., most leftward), and the middle group who fall within 1 *SD* of the mean. As Fig. 2 shows, individuals with the most leftward frontal asymmetry scores showed no attentional bias. In women with rightward or no asymmetry, a parietal effect was observed; those with the most rightward scores on frontal asymmetry had the strongest relationship between parietal asymmetry and attentional bias. Those with relatively leftward parietal asymmetry (that is, relatively low right parietal activity) showed an attentional bias toward angry faces. However, those with a relatively rightward parietal asymmetry (that is, relatively high right parietal activity) showed an attentional bias away from angry faces.

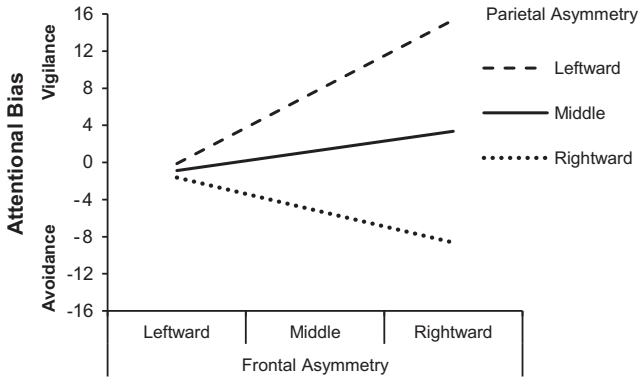

Fig. 2. Modgraph showing the interaction between frontal and parietal asymmetry in predicting attentional bias to angry faces at the long SOA. Frontal asymmetry appears along the *x*-axis and is reverse-plotted, so that values to the left end of the axis reflect leftward asymmetry and values to the right end of the axis reflect rightward asymmetry. Relative leftward and rightward groups score one standard deviation above and below the mean, respectively. The three lines reflect three levels of parietal asymmetry, also scoring one standard deviation above and below the mean. The top line reflects those with the most leftward parietal asymmetry (relatively low right parietal activity) and the bottom line reflects those with the most rightward parietal asymmetry (relatively high right parietal activity). Attentional bias is plotted on the *y*-axis (in ms); positive values reflect vigilance and negative values reflect avoidance.

4. Discussion

We examined the relationship between cortical EEG asymmetries and attentional bias to emotional faces. The most important finding was a relationship between attentional bias to angry faces and parietal asymmetry that further interacted with frontal asymmetry. Consistent with our predictions, women with the most leftward frontal asymmetry showed no attentional bias at all, a pattern typical of healthy (i.e., non-depressed and non-anxious) individuals (Bar-Haim et al., 2007). In women with balanced or rightward frontal asymmetry, attentional biases emerged that were predicted by parietal asymmetry. Those with low levels of right parietal activity showed the vigilance to threat that is commonly observed in depression, and those with high levels of right parietal activity showed the avoidance pattern that is sometimes seen in anxiety. The correlation between resting parietal asymmetry and attentional bias to angry faces replicates that reported by Pérez-Edgar et al. (2013). However, they did not test the frontal × parietal interaction, and so it is unclear whether their parietal effects might have been similarly moderated by frontal asymmetry. To our knowledge, ours is the first study to specifically test for frontal × parietal interactions in the prediction of any aspect of cognitive or affective processing. It remains to be seen whether similar interactions are observed in other domains or whether they are specific to top-down attentional processes which are known to rely on the function of a frontal–parietal network (Corbetta & Shulman, 2002; Shomstein, 2012; Shomstein et al., 2012). Fortunately there is a great deal of data already available on this question. Most studies that have examined EEG correlates of cognitive or affective processing will have recorded from both frontal and parietal regions. Therefore, reanalysis of extant data to include the frontal × parietal interaction has the potential to delineate the conditions under which such interactions are observed.

The relationship between cortical asymmetries and attentional bias was observed for angry faces at a long SOA, suggesting that asymmetries reflect top-down attentional control in response to threat. This finding is consistent with work that links resting alpha to activity in the frontal–parietal network (Laufs et al., 2003; Mantini et al., 2007) that is critical for the control of attention, particularly in the face of emotional distraction (Banich et al., 2009; Bishop, 2008; Bishop et al., 2007; Braver, 2012; Pessoa, 2008; Vanderhasselt, De Raedt, Baeken, Leyman, & D'Haenen, 2006). Furthermore, asymmetries did not predict attentional biases at short SOA, nor the N2pc to emotional stimuli – both indices of early attentional selection processes. We offer a caveat here though, as it is not possible with the current research design to determine whether the predictive value of the frontal × parietal interaction differed significantly for different bias measures. Such an analysis would require manipulation of emotion and SOA between subjects in a very large sample. In future research it will be important to determine the specificity of the relationships between asymmetries and attentional biases in order better identify the mechanisms that support them.

Although the N2pc was not related to either frontal or parietal asymmetry, it was sensitive to emotion and indicated early attentional selection of both happy angry faces. Our finding of an emotion-related N2pc adds to a growing body of research using it to show that emotional faces compete for selection, even when they are task-irrelevant (Holmes et al., 2009; Ikeda et al., 2013). Interestingly, biases in attentional selection (as indicated by the N2pc) did not translate into attentional bias as indicated in RT, even at the short SOA. In the dot-probe paradigm, the N2pc indexes

selection of a face, but RT indexes time to respond to the probe. It is possible that attentional selection of the face (as reflected by the N2pc) does not necessarily orient spatial attention to its location (as reflected by attentional bias in RT). Similar dissociations between the N2pc and RT have been observed in other studies in which participants respond to a different stimulus than the faces (Fenker et al., 2010; Ikeda et al., 2013).

Participants in this study viewed negative IAPS images before each block of dot-probe trials. This was not an experimental manipulation, rather it was used with all participants to maximise the likelihood of instituting a negative attentional bias in vulnerable individuals (see Joormann et al., 2007, who used a similar procedure to show a relationship between familial vulnerability to depression and attentional bias to angry faces). Because our research focused on the relationship between trait asymmetries and attentional bias, we assessed asymmetry before, but not after, presentation of the images. Pérez-Edgar et al. (2013) found that change in frontal asymmetry in response to a stressor predicted attentional bias to angry faces. Recent studies of emotion regulation also suggest that the ability to recruit left frontal control mechanisms under conditions of emotional challenge may be more important than trait asymmetry in predicting successful emotion regulation (Coan, Allen, & McKnight, 2006; Goodman, Rietschel, Lo, Costanzo, & Hatfield, 2013; Parvaz, MacNamara, Goldstein, & Hajcak, 2012). It will therefore be useful in future to consider the contributions of both resting (trait-based) asymmetries and the asymmetric response to emotional challenge in predicting attentional bias.

Note that our participants reported no history of depression or anxiety, and were not clinically depressed or anxious. Furthermore, their cortical asymmetries were not related to current sub-clinical levels of either depression or anxiety as assessed by questionnaires. This was not unexpected given that we purposefully selected a healthy sample without current, or past, depression or anxiety. The two previous studies (Pérez-Edgar et al., 2013 and Schutter et al., 2001) that examined relations between asymmetries and attentional bias in healthy participants similarly report relationships in the absence of depression- or anxiety-related effects. The fact that the relationship between asymmetry and attentional bias is independent of current depression or anxiety is consistent with cognitive theories that suggest that attentional biases are vulnerability factors that exist in healthy individuals. This finding also suggests that the relationship between asymmetry and attention may be more fundamental than the relationship between asymmetry and psychopathology; it is only in the context of significant life stress that such vulnerability is expected to give rise to emotional disorder. Longitudinal research will be necessary to determine whether the cortical asymmetries and attentional biases reported here predict future depression and/or anxiety, and whether the relationship between asymmetry and disorder is mediated by attentional bias.

5. Conclusions

Our findings extend understanding of the attentional correlates of trait asymmetries in several ways. First, we show that frontal and parietal asymmetries interact to predict attentional bias to threat, and that this relationship is independent of depression or anxiety. Second, our SOA manipulation suggest that asymmetries selectively predict top-down attentional control processes. We further demonstrate the value of using both behavioural and electrophysiological measures to target specific stages of attentional processing and so yield a more nuanced picture of brain–behaviour relationships. Taken together with the few other studies that have specifically studied relationships between asymmetries and attention (e.g., Miskovic & Schmidt, 2010; Pérez-Edgar et al., 2013), our findings suggest that a focus on the cognitive correlates of asymmetry (and attentional control processes more specifically) may extend our understanding of how trait asymmetries could give rise to a broad range of individual differences in personality and emotional processing (see also Grimshaw & Carmel, 2014).

Of course, there are limitations in our approach; the most notable is that a correlational study cannot be used to establish a causal relationship between asymmetries and cognitive function. However, the correlational approach can be used to generate hypotheses which can then be tested through direct manipulation of cortical asymmetry using repetitive transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS). The use of complementary methodologies in future research will help us to pinpoint the specific neurocognitive mechanisms that confer vulnerability to depression and anxiety and could guide the development of behavioural and neurocognitive strategies for their prevention and treatment.

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