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In the eye of the beholder: Individual differences in reward-drive modulate early frontocentral ERPs to angry faces

Benoit Bediou[a,c,d,e,*], Martin Eimer[b], Thierry d'Amato[c,d,e], Olaf Hauk[a], Andrew J. Calder[a,**]

[a] MRC Cognition and Brain Sciences Unit, 15 Chaucer Road, Cambridge CB2 7EF, UK
[b] School of Psychology, Birkbeck College, University of London, Malet Street, London WC1E 7HX, UK
[c] Université de Lyon, EA 4166, Lyon F-69003, France
[d] Centre Hospitalier « Le Vinatier », Bron F-69677, France
[e] IFR19, Bron F-69500, France

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ABSTRACT

Individual differences in reward-drive have been associated with increased attention toward facial signals of aggression, heightened experience of anger and vulnerability to display aggressive behaviour. Recent fMRI research suggests that these effects rely on reduced ventromedial prefrontal (and increased amygdala) response to aggressive facial displays compared with neutral and sad expressions in subjects scoring high on reward-drive. However, nothing is known about the timing of this modulation. Using event-related potentials (ERPs), we provide the first evidence that greater proneness to display hostile and aggressive behaviour (measured by high scores on the reward-drive) is associated with a reduced midline frontocentral response to aggressive faces within 200–300 ms. In addition to confirming a particular interaction between anger processing and aggression related personality traits in ventromedial prefrontal brain regions, our study brings a first indication of when their interaction occurs in the brain, strengthening results from previous classical as well as functional connectivity fMRI studies.

1. Introduction

Accumulating evidence suggests that there are major individual differences in the neural response to emotional stimuli (Canli, 2004; Hamann & Canli, 2004), including facial emotional expressions (Beaver, Lawrence, Passamonti, & Calder, 2008; Bishop, Duncan, Brett, & Lawrence, 2004), and that a significant proportion of the variance can be accounted for by variation in personality. Thus, understanding the neural underpinnings of the interaction between personality and facial expression processing is fundamental to our understanding of individual differences in social behaviour. The current study sought to examine the effects of individual differences in a personality trait linked to the drive to gain reward (reward-drive), on the neural response to aggressive facial displays.

The 'Behavioural Approach System' (BAS) has been primarily associated with sensitivity to reward and appetitive motivation (Carver & White, 1994; Corr, 2004). However, additional research shows that healthy individuals scoring high on this trait are also more likely to display hostile or aggressive behaviour (Harmon-Jones, 2003; Smits & Kuppens, 2005; Wingrove & Bond, 1998) and to experience heightened levels of anger (Carver, 2004; Harmon-Jones, 2003). These effects have been found most consistently using the BAS-drive scale (Beaver et al., 2008; Harmon-Jones, 2003; Putman, Hermans, & van Honk, 2004; Smits & Kuppens, 2005; Wingrove & Bond, 1998) [but see (Carver, 2004)] which measures trait differences in the drive or motivation to gain reward (reward-drive or appetitive motivation). Similarly, high BAS-drive individuals also show increased attention toward facial expressions of anger (Putman et al., 2004), which mirrors the same effect found in high trait anger participants (Van Honk, Tuiten, De Haan, van den Hout, & Stam, 2001); this has been attributed to the idea that anger-prone individuals are more likely to interpret facial displays of anger as signals of provocation (Beck, 1976; Putman et al., 2004; Van Honk et al., 2001).

In accordance with these findings, fMRI research has shown that individual differences in reward-drive correlate with neural activation in brain regions implicated in aggression and emotion regulation when healthy individuals view aggressive facial displays, compared to sad and neutral expressions (Beaver et al., 2008); more specifically, increased reward-drive scores correlated positively with amygdala activity (thought to reflect increased negative affect) and negatively with ventromedial prefrontal activity

* Corresponding author at: Swiss Centre for Affective Sciences, CISA, University of Geneva, Rue des Battoirs 7, CH-1205 Geneva, Switzerland. Tel.: +41 22 379 98 10.
** Corresponding author. Tel.: +44 1223 355 294; fax: +44 1223 359 062.
E-mail addresses: benoit.bediou@unige.ch (B. Bediou), andy.calder@mrc-cbu.cam.ac.uk (A.J. Calder).

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(thought to reflect decreased control). However, given the poor temporal resolution of fMRI, this study provides no information regarding the temporal characteristics of this effect. In the current study we addressed this using electroencephalographic (EEG) recordings.

Previous EEG studies have revealed responses sensitive to various emotional expressions at early processing stages (between 120 and 300 ms) over the frontal and frontocentral scalp regions (Ashley, Vuilleumier, & Swick, 2004; Eimer & Holmes, 2002; Eimer, Holmes, & McGlone, 2003; Esslen, Pascual-Marqui, Hell, Kochi, & Lehmann, 2004; Holmes, Vuilleumier, & Eimer, 2003). During these time intervals (130–200 and 200–300 ms), ERPs to emotional faces were generally more positive than ERPs to neutral faces. Although in principle, this differential early ERP effect of emotional expression could also be described as a 'reduced negativity' for emotional relative to neutral faces (*e.g.*, Schupp et al., 2004), we follow the terminology used in numerous previous studies and refer to this effect as an 'enhanced positivity' (Eimer & Holmes, 2002; Eimer et al., 2003; Holmes et al., 2003) for a review and more detailed discussion, see Eimer & Holmes (2007). It is important to note that this emotion-specific ERP effect cannot be easily described as an amplitude modulation of a specific ERP peak, since it typically overlaps with several successive peaks in ERP waveforms, such as the N1 and the P2. It is however also possible to describe differential ERP responses to emotional vs. neutral faces with respect to the specific ERP peaks that are affected. For example, Williams, Palmer, Liddell, Song, and Gordon (2006) found significant enhancements in positivity for P80, VPP and P300, together with a discrete reduction in negativity for N200 specific to fearful faces relative to happy and neutral faces over the medial frontocentral region. Selective responses to faces have also been recorded intracranially in the medial (Kawasaki et al., 2001; Rolls, Critchley, Browning, & Inoue, 2006) and lateral (Halgren et al., 1994; Marinkovic, Trebon, Chauvel, & Halgren, 2000) prefrontal cortices of humans; with the former showing a differential response to fearful compared to neutral faces (Kawasaki et al., 2001). Abolition of this early frontal positivity to fearful faces by orbitofrontal lesions (Ashley, Vuilleumier, & Swick, 2002) further supports a contribution of ventral prefrontal sources to these early frontal/frontocentral scalp ERPs. However, the impact of individual differences in personality on these early emotion-sensitive ERP responses is yet to be addressed.

We reasoned that the frontocentral ERPs to emotional faces that have been associated with the rapid encoding of affectively salient signals associated with threat or danger (Eimer & Holmes, 2002) might reflect the same source as the ventromedial prefrontal cortex activity observed in response to viewing angry faces in previous fMRI research (Beaver et al., 2008; Passamonti et al., 2008). To investigate the temporal properties of the interaction between reward-drive and the frontal response to angry faces, we recorded event-related brain potentials (ERPs) in subjects scoring high ($N = 12$) and low ($N = 12$) on the BAS reward-drive scale while they viewed angry, sad and neutral expressions. Based on previous fMRI findings showing reduced vmPFC activation to angry faces contrasted with sad or neutral faces as a function of increasing reward-drive (Beaver et al., 2008), we hypothesised a selective reduction of the frontal ERPs to angry relative to both sad and neutral faces in high compared to low reward-drive subjects.

ERP amplitude modulations by emotion have also been reported for the P1 (Campanella, Quinet, Bruyer, Crommelinck, & Guerit, 2002; Eger, Jedynak, Iwaki, & Skrandies, 2003; Pourtois, Dan, Grandjean, Sander, & Vuilleumier, 2005; Sprengelmeyer & Jentzsch, 2006) and N170 components (Batty & Taylor, 2003; Pizzagalli, Regard, & Lehmann, 1999; Streit et al., 2003) and early posterior negativity (EPN) (Sato, Kochiyama, Yoshikawa, & Matsumura, 2001; Schupp et al., 2004). Hence, we also addressed whether emotion and/or personality modulate ERP mean amplitudes recorded at O1/O2 within 80–130 ms post-stimulus for P1 and within 130–200 and 200–300 ms for the EPN, and at PO9 and PO10 within the 130–200 ms time window for the N170.

2. Methods

2.1. Subjects

Twenty-four paid healthy male volunteers (mean age 27 ± 6 years old) participated in this experiment. The study was approved by the Cambridge Psychology Research Ethics Committee and performed in compliance with their guidelines and with the 1964 Declaration of Helsinki. Written informed consent was obtained from all participants. Individuals with symptoms or history of psychiatric care, neurological disease or head injury were excluded.

Prior to the EEG recording, participants completed the BIS/BAS questionnaire (Carver & White, 1994), a self-report measure of personality based on Gray's bidimensional personality theory (Gray, 1973), to assess participants' trait sensitivity to reward (BAS) and punishment (BIS). We focused on BAS-drive (reward-drive)—a measure of goal-directed drive to pursue reward (*e.g.*, *When I see something I want I go all out to get it*)—because this subscale has been more consistently associated with aggression. The other two BAS scales, BAS-Reward Responsiveness and BAS-Fun Seeking, assess positive affect/excitability in the context of reward (*e.g.*, *When good things happen to me, it affects me strongly*) and the tendency to seek out new rewarding situations (*e.g.*, *I'm always willing to try something new if I think it will be fun*), respectively. The Behavioural Inhibition System (BIS) scale reflects sensitivity to punishment and has been related to anxiety (*e.g.*, *I feel pretty worried or upset when I think or know somebody is angry at me*). Participants also completed standard questionnaires measuring state and trait anxiety (Spielberger, 1983) and depression (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Age, personality measures and performance on the experimental task are summarised in Table 1.

Subjects were divided into a group of high BAS-drive score ($N = 12$) and a group of low BAS-drive score ($N = 12$) according to the median split of BAS-drive scores (median = 10.5). The high and low BAS-drive groups were matched for age, other subscales of the BIS/BAS (reward responsiveness, fun seeking, and behavioural inhibition), and other personality measures—state and trait anxiety (Spielberger, 1983) and depression (Beck, Steer, & Brown, 1996).

2.2. Stimuli and task

Subjects sat in a dimly lit sound-attenuated cabin, in front of a computer screen placed at a viewing distance of 120 cm. The stimuli were pictures of angry, sad and neutral facial expressions posed by 20 different individuals selected from the NimStim Face Stimulus Set (www.macbrain.org) and the Karolinska Directed Emotional Faces (KDEF) (Lundqvist, Flykt, & Ohman, 1998); 60 faces in total. Stimuli were presented at the centre of a computer screen, subtending a visual angle of approximately 4.5° (horizontal) $\times$ 7.5° (vertical). The experiment consisted of eight experimental blocks of 69 trials each. On 60 of the trials, single angry, sad or neutral faces were presented in random order, with equal probability. On nine randomly interspersed trials per block, the face presented on the preceding trial was immediately repeated. Subjects were instructed to respond with a right- or left-hand button press to these immediate repetitions of physically identical faces across successive trials, and to refrain from responding on all other trials. Each block contained three immediate repetitions of angry, sad, and neutral faces, respectively. Response hand was counterbalanced across subjects. To avoid different numbers of presentations of unrepeated faces, the faces used in the repeated trials were never presented in unrepeated trials. Stimuli were presented for 300 ms each, and were separated by a fixed interstimulus interval of 1300 ms. Stimulus delivery and response collection was controlled by the E-Prime software (Psychology Software Tools, Pittsburgh, PA).

2.3. EEG recordings and analysis

The electroencephalogram (EEG) was continuously recorded in an electrically and acoustically shielded EEG booth. Data were recorded from 64 Ag/AgCl electrodes mounted on an electrode cap (Easycap, Falk Minow Services, Herrsching-Breitbrunn, Germany) using SynAmps amplifiers (NeuroScan Labs, Sterling, VA), arranged according to the extended 10/20 system with Cz reference and re-referenced offline to mastoids (TP9, TP10). The impedance for electrodes was kept below 5 kΩ. Data were acquired with a sampling rate of 500 Hz. The electrooculogram (EOG) was recorded bipolarly through electrodes placed above and below the right eye (vertical) and at the outer canthi (horizontal). Amplifier bandpass was 0.1–100 Hz and additional 0.1–40 Hz filter was applied to the averaged data. ERP analyses were conducted relative to a 100 ms pre-stimulus baseline, and were restricted to non-repetition (non-target) trials only, to avoid contamination by key-press responses. Trials with lateral eye movements (HEOG exceeding ± 50 μV) and eye blinks (vEOG exceeding ± 50 μV), or other artefacts (a voltage exceeding ± 100 μV at any electrode) measured after target onset were excluded from analysis.

Separate averages were computed for angry, sad and neutral faces, resulting in three average waveforms for each electrode and participant. The electrodes of interest were derived from literature (Bediou et al., 2007; Eimer et al., 2003) and consisted of frontal (F3/z/4) and frontocentral (FC3/z/4) electrodes. Given that the early

Table 1
Groups description and behavioural results.

		Age (years)	BAS-drive	BASr	BASfs	BIS	STAI-state	STAI-trait	BDI	Hits anger (%)	Hits sadness (%)	Hits neutral (%)	RT anger (ms)	RT sadness (ms)	RT neutral (ms)
All subjects ($N = 24$)	Mean	27.00	10.42	12.08	17.38	20.58	34.38	41.00	8.5	88.79	84.78	81.68	314.79	331.91	315.02
	S.D.	5.74	2.12	1.95	2.06	3.91	10.01	11.61	6.65	10.28	13.98	14.99	87.08	93.24	84.96
Low BAS-drive ($N = 12$)	Mean	28.9	8.67	11.75	17.00	20.58	32.25	37.83	7.50	91.40	89.64	85.84	305.67	328.84	321.22
	S.D.	6.0	1.37	2.45	1.71	4.29	11.17	11.98	5.95	8.36	9.80	9.31	80.49	94.01	93.76
High BAS-drive ($N = 12$)	Mean	25.0	12.17	12.42	17.75	20.58	36.50	44.17	9.50	86.19	79.93	77.51	323.91	334.99	308.81
	S.D.	4.9	0.94	1.31	2.38	3.68	8.66	10.79	7.40	11.66	16.17	18.59	95.88	96.54	78.85
Group difference[a] (p)		–	<0.001	–	–	–	–	–	–	–	–	–	–	–	–

S.D. = standard deviation. *Note*: BAS-drive = Behavioural Approach-Drive, BASr = Behavioural Approach-reward responsiveness, BASf = Behavioural Approach-Fun seeking, BIS = Behavioural Inhibition System. STAI = State and Trait Anxiety Inventory (Spielberger, 1983). BAS-drive, BASr, BASF and BIS are subscales of the BIS/BAS system (Carver & White, 1994).
[a] Two-tailed independent samples t-tests.

'enhanced positivity' for emotional vs. neutral faces overlaps with several successive visual evoked brain potentials, such as the N1 and the P2, it is not equated with a specific ERP peak. Hence, mean ERP amplitudes for angry, sad and neutral faces were analysed by repeated measures ANOVAs within two successive post-stimulus time intervals (130–200 and 200–300 ms) that were chosen in line with previous research (Eimer & Holmes, 2002; Eimer et al., 2003). The factors were Group (high BAS-drive, low BAS-drive; between-subjects), Emotion (angry, neutral and sadness; repeated measure), Electrode site (frontal, frontocentral; repeated measure), and Laterality (midline, left, right; repeated measure). In addition, in order to investigate possible emotional expression and/or personality effects on the visual P1 component, we also analysed mean amplitudes at lateral occipital electrodes O1 and O2 obtained in a time window that was centred on P1 peak amplitude (80–130 ms post-stimulus). Similar analyses were performed for the mean amplitudes at electrodes PO9 and PO10 in a time window centred on the face-sensitive N170 peak amplitude (130–200 ms post-stimulus). Finally, possible modulation of the EPN was investigated by analysing the mean ERP amplitudes at electrodes O1 and O2 during the same time windows as the frontocentral positivity 130–200 and 200–300 ms. Greenhouse-Geisser adjustments to the degrees of freedom were performed when appropriate, and the corrected p-values are reported. Estimates of effect sizes are provided as eta squared (η^2) for the ANOVAs and Cohen's d for Student's t-tests (Cohen, 1988).

3. Results

3.1. Behavioural results

Behavioural performance in the face repetition detection task is summarised in Table 1. Mean correct responses for angry, sad and neutral faces (detection of direct face repetition in the one-back task) were submitted to a repeated measure ANOVA examining the factors Emotion (anger, neutral, sadness; repeated measure) and Group (high, low BAS-drive; between subjects). There was a main effect of Emotion ($F(1.7, 37.8) = 3.74$, $p < 0.05$) that did not interact with Group ($F < 1$); subjects were more accurate at detecting repetitions of angry faces compared to both sad ($t(23) = 2.09$, $p < 0.05$) and neutral faces ($t(23) = 2.41$, $p < 0.05$), whereas no difference in the accuracy of repetition detection was found between sad and neutral faces ($t(23) = 1.09$, $p > 0.2$). Average response time was 321 ± 88 ms overall; no significant effect or interaction emerged from the repeated measure ANOVA examining the factors Emotion and Group (Fs < 1).

3.2. ERP results

Fig. 1 shows grand average ERPs recorded at the electrodes included in our analyses (i.e., O1 and O2, PO9 and PO10 and F3, FC3, Fz, FCz, F4, FC4) for all participants (i.e., collapsed across low and high BAS-drive group). These ERPs show the P1 and N170 components as well as early frontal/frontocentral positivity and early posterior negativity to emotional compared with neutral faces. Topographic voltage distribution maps for the amplitude of the angry–neutral difference ERP are also shown to illustrate the general distribution of the early emotion effect in the whole group, within the time windows analysed, i.e., 130–200 and 200–300 ms; while there was no systematic effect of Emotion on the P1 component, the N170 component and the early frontal/frontocentral positivity were enhanced for emotional (angry or sad) as compared to neutral faces. In contrast, the EPN was enhanced for angry compared to sad and neutral faces but only within 130–200 ms and not between 200 and 300 ms.

3.2.1. P1 component (Fig. 1)

There was no main effect of emotion on mean ERP amplitudes recorded at O1 and O2 in the P1 time window (80–130 ms post-stimulus). A significant Hemisphere × Emotion interaction was observed ($F(1.8, 39.1) = 3.40$, $p < 0.05$, $\eta^2 = 0.03$), reflecting reduced P1 amplitude for angry compared to neutral faces at O1 ($F(1, 23) = 4.27$, $p = 0.05$) but not O2 ($F < 1$) and a non-significant trend

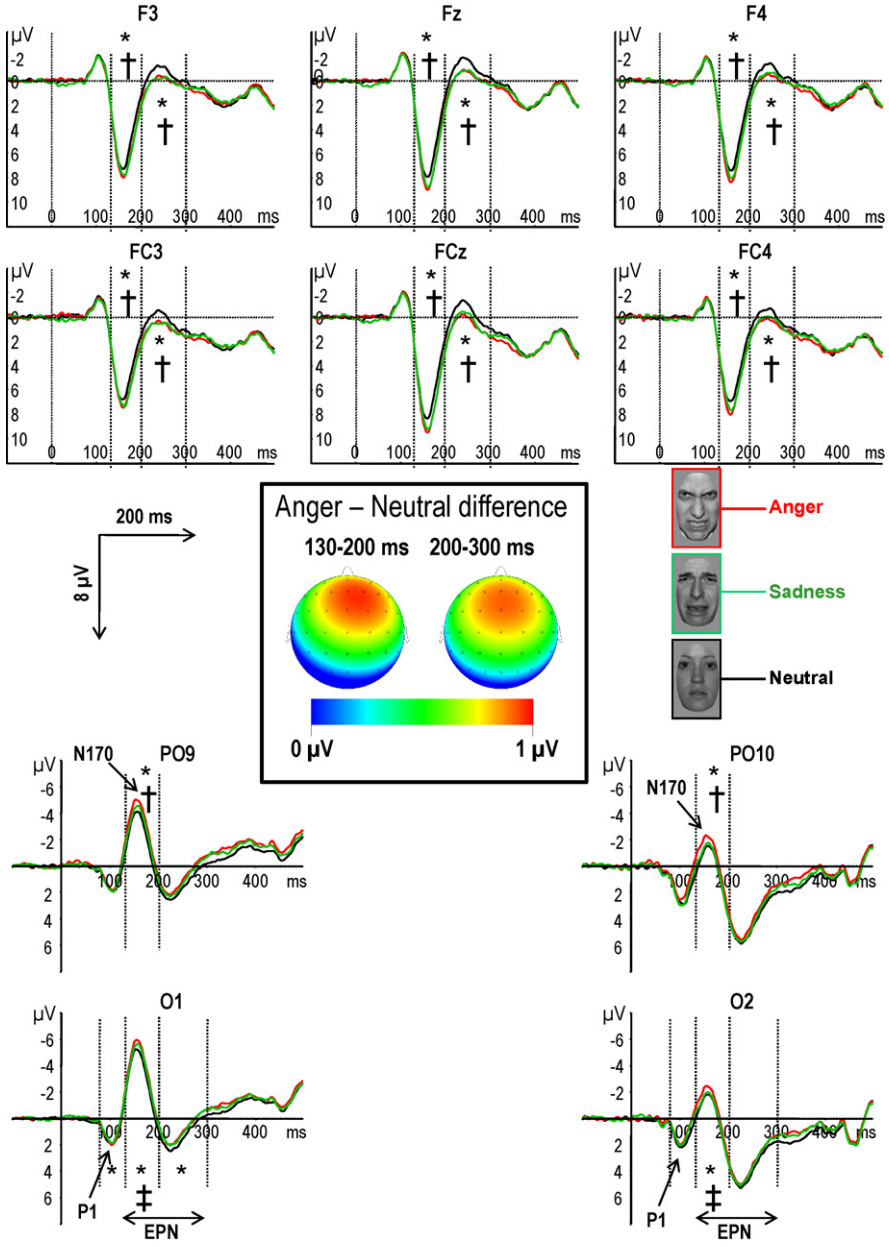

Fig. 1. Grand average ERP responses to angry (red), sad (green) and neutral (black) faces in the whole group ($N = 24$). Selected electrodes show the P1, the N170 as well as the early posterior negativity (EPN) and early frontal/frontocentral positivity at F3/Fz/F4 and FC3/FCz/FC4. The voltage distribution map is also shown for the two time windows of interest (130–200 and 200–300 ms) for the angry–neutral difference ERP to illustrate the general distribution of emotional expression effects. Voltage map scale is 0–1 μV. Vertical bars delineate respective time windows of analyses, i.e., 80–130 ms for the P1 (O1, O2), 130–200 ms for the N170 (PO9, PO10) as well as 130–200 and 200–300 ms for the early posterior negativity (O1, O2) and early frontal/frontocentral positivity (F3, FC3, Fz, FCz, F4, FC4). Significant differences: *, anger vs. neutral; †, sadness vs. neutral; ‡, anger vs. sadness. All subjects ($N = 24$). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

toward reduced P1 amplitude for angry relative to sad faces at O1 ($F(1, 23) = 4.10$, $p = 0.055$) and O2 ($F(1, 23) = 4.01$, $p = 0.057$). P1 amplitude did not differ significantly for sad compared to neutral faces (F's < 1.2, p's > 0.3). There was no main effect or interaction involving the factor Group.

3.2.2. N170 component (Fig. 1)

A main effect of Emotion ($F(1.9, 42) = 17.60$, $p < 0.001$, $\eta^2 = 0.44$) was observed for mean ERP amplitudes recorded at PO9 and PO10 in the N170 time window (130–200 ms post-stimulus), reflecting enhanced N170 amplitudes for emotional faces compared to neutral faces (angry vs. neutral $F(1, 23) = 35.86$, $p < 0.001$; sad vs. neutral $F(1, 23) = 15.70$, $p < 0.001$), and a non-significant trend toward enhanced N170 amplitude for angry faces relative to sad faces ($F(1, 23) = 4.13$, $p < 0.06$). There was no main effect or interaction involving the factor Group.

3.2.3. Early posterior negativity (Fig. 1)

The analysis of mean ERP amplitudes recorded at O1 and O2 within the 130–200 ms post-stimulus time window revealed a main effect of Emotion ($F(1.6, 35.4) = 11.12$, $p < 0.001$, $\eta^2 = 0.34$). ERPs were more negative to angry faces relative to neutral and sad faces (angry vs. neutral $F(1, 23) = 24.81$, $p < 0.001$; angry vs. sad $F(1, 23) = 4.89$, $p < 0.05$), whereas ERPs to sad and neutral faces did not significantly differ ($F(1, 23) = 1.61$, $p > 0.2$). There was no main effect or interaction involving Group.

In the subsequent 200–300 ms time window, the only significant effect was a Hemisphere × Emotion interaction ($F(2.0, 43.5) = 3.45$, $p < 0.05$, $\eta^2 = 0.02$), reflecting more negative ERPs for angry faces than for neutral faces on the left ($F(1, 23) = 6.30$, $p < 0.05$) but not on the right ($F(1, 23) = 2.71$, $p > 0.1$). No main effect or interaction involving Group was found.

3.2.4. Frontal and frontocentral activity (Fig. 2)

Fig. 2 shows grand average ERP recorded at frontal and frontocentral electrodes (i.e., F3, FC3, Fz, FCz, F4 and FC4) as well as voltage distribution maps for the angry–neutral difference ERP amplitude within 200–300 ms time window separately for low and high BAS-drive subjects ($N = 12$ per group). Note that the scale for the voltage distribution maps is three times larger in low BAS-drive compared

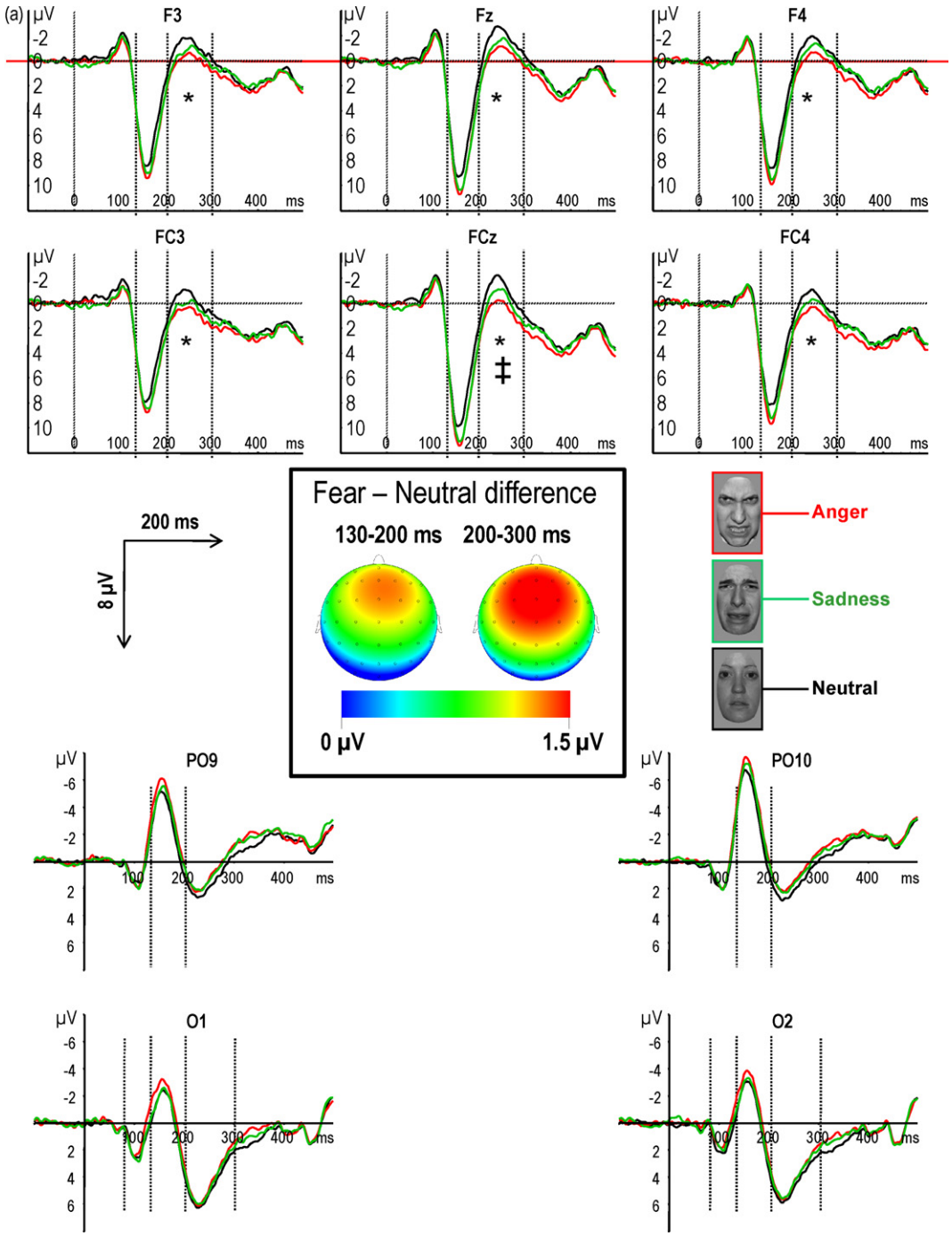

Fig. 2. Grand average ERP responses to angry (red), sad (green) and neutral (black) faces separately for the low BAS-drive group ((a) $N = 12$, top panel) and the high BAS-drive group ((b) $N = 12$, bottom panel). Vertical bars show time windows of analyses, i.e., 130–200 and 200–300 ms and the corresponding voltage distribution maps are shown for the angry–neutral difference ERP in the 130–200 and 200–300 ms time windows. Note that the scale for the topographic voltage maps is three times larger for the low BAS-drive group (0–1.5 μV) compared to the high BAS-drive (0–0.5 μV). Significant differences: *, anger vs. neutral; †, sadness vs. neutral; ‡, anger vs. sadness. Note that posterior electrodes (O1/O2, PO9/PO10 are provided for illustrative purposes only. These electrodes were not analysed separately for each BAS-drive group as there was no effect or interaction involving Group in the initial between-group ANOVA. (a) Low BAS-drive group ($N = 12$). (b) High BAS-drive group ($N = 12$). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

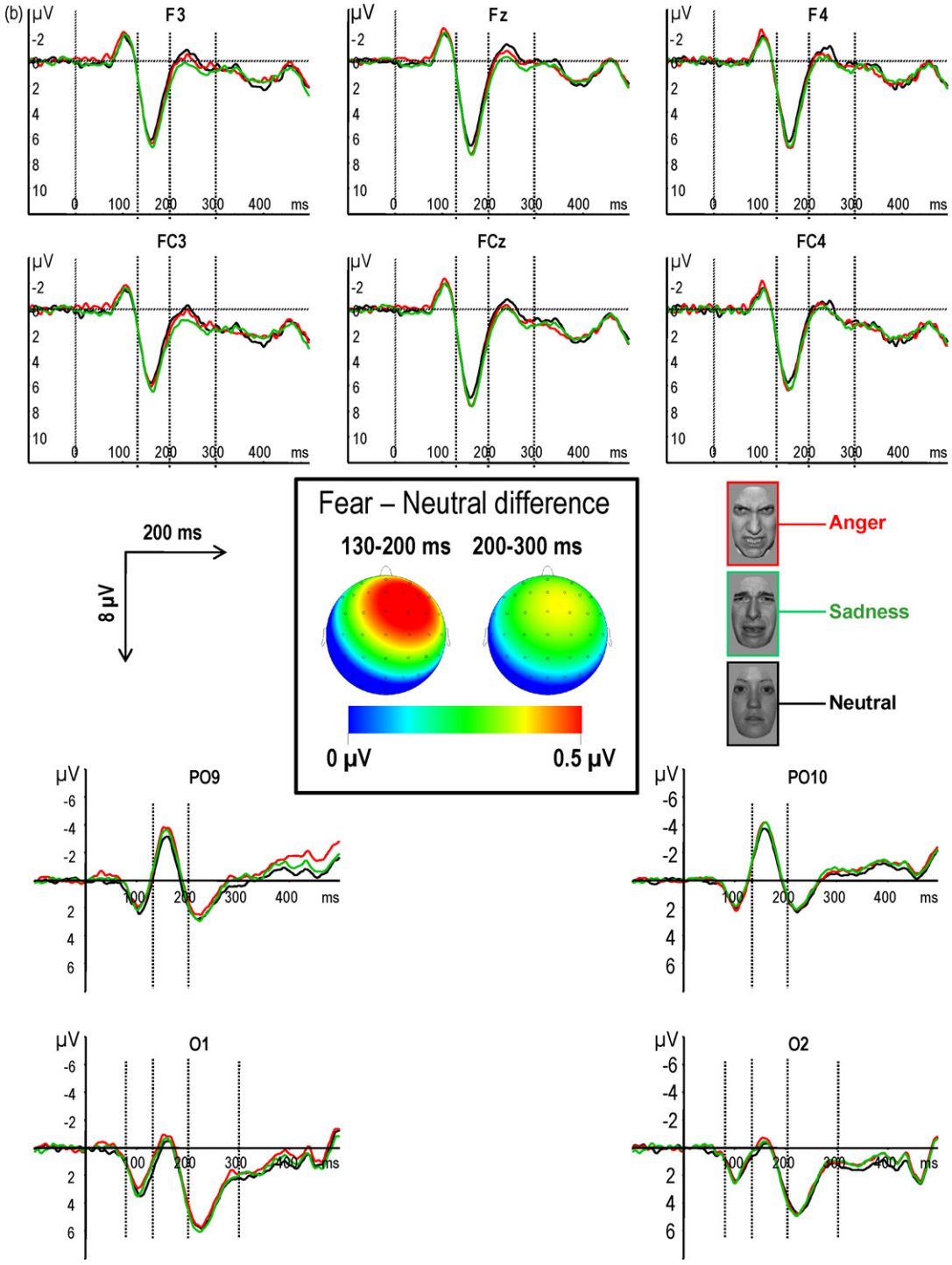

Fig. 2. (*Continued*).

to high BAS-drive group and that the colours in the high group reflect a non-significant difference.

In the 130–200 ms time window, there was a main effect of Emotion ($F(1.5, 33.5) = 12.98$, $p < 0.001$, $\eta^2 = 0.37$) reflecting enhanced positive ERPs for emotional faces compared to neutral faces in the whole group (angry vs. neutral $F(1, 19) = 30.05$, $p < 0.001$; sad vs. neutral $F(1, 23) = 11.95$, $p < 0.01$; angry vs. sad $F < 1$). Although the ERPs differences between emotional and neutral faces were numerically larger for the low BAS-drive group than for the high BAS-drive group (see Fig. 2b), there was no significant interaction involving the factors Emotion and Group.

In the 200–300 ms time window, there was a significant main effect of Emotion over frontal/frontocentral electrodes ($F(2.0, 43.9) = 10.02$, $p < 0.001$, $\eta^2 = 0.31$), reflecting enhanced positive ERPs to angry and sad faces relative to neutral (angry vs. neutral $F(1, 23) = 12.48$, $p < 0.01$; sad vs. neutral $F(1, 23) = 14.22$, $p < 0.01$), whereas ERP to angry and sad faces did not differ significantly ($F < 1$). Critically, there was also a significant Emotion × Group interaction ($F(2, 36) = 4.14$, $p < 0.03$, $\eta^2 = 0.11$), demonstrating that

emotional expression effects differed systematically between high and low BAS-drive groups.

Given previous fMRI research showing that BAS-drive modulates frontal response to angry faces relative to both neutral and sad faces (Beaver et al., 2008), the Emotion × Group interaction was examined further by submitting ERP data to repeated measures ANOVAs conducted separately for each pair of expressions (angry/neutral, angry/sad and sad/neutral). Crucially, significant Emotion × Group interactions were found in the angry/neutral ANOVA ($F(1, 22) = 6.61$, $p < 0.02$, $\eta^2 = 0.15$) and in the angry/sad ANOVA ($F(1, 22) = 5.76$, $p < 0.03$, $\eta^2 = 0.21$) but not in the sad/neutral ANOVA ($F < 1$).

To address these interactions further, repeated measures ANOVAs were conducted separately in each BAS-drive group, for angry vs. neutral and angry vs. sad facial expressions, respectively. In the low BAS-drive group, there was a main effect of Emotion in the angry/neutral ANOVA ($F(1, 11) = 21.94$, $p < 0.01$) that was accompanied by an Electrode × Laterality × Emotion interaction ($F(1.9, 21.1) = 6.97$, $p < 0.01$). Low BAS-drive subjects exhibited enhanced positive ERPs for angry compared to neutral faces over all frontal and frontocentral electrodes (all electrodes, F's > 15.30, p's < 0.01, maximal at FCz). In the angry/sad ANOVA an Electrode × Laterality × Emotion interaction was present ($F(1.7, 39.8) = 11.11$, $p < 0.001$), reflecting an increased positive ERP amplitude for angry compared to sad faces that was significant at FCz ($F(1, 11) = 5.17$, $p < 0.05$). In marked contrast, and importantly, no effect or interaction involving the factor Emotion emerged from the angry/neutral and angry/sad ANOVAs in the high BAS-drive group (F's < 2.5, p's > 0.14), see Fig. 2, topographic voltage maps with different scales).

This modulatory and anger-specific effect of the factor Group was further investigated in direct between-group comparisons for the angry–neutral, angry–sad and sad–neutral difference ERP obtained in the 200–300 ms time interval at FCz, where emotional expression effects were maximal (see Fig. 2). These comparisons revealed that ERP differences between angry and neutral and between angry and sad facial expressions were significantly smaller in high BAS-drive compared to low BAS-drive subjects (angry–neutral $t(21.7) = 2.70$, $p < 0.02$, Cohen's $d = 1.10$; angry–sad $t(22.0) = 2.35$, $p < 0.03$, Cohen's $d = 0.96$). Importantly however, the groups did not differ with respect to the size of the sad–neutral difference ($t < 1$). To add further support that the reduced frontocentral response to angry faces is related to individual differences in BAS-drive, we calculated the Pearson's coefficient of correlation between BAS-drive scores and the difference ERP amplitudes at FCz within 200–300 ms. Consistent with the breakdown of the interaction between Emotion and Group, there was a significant negative correlation between BAS-drive and the amplitude of the anger–neutral ($R = -0.37$, $p < 0.05$, one-tailed, Cohen's $d = 0.85$) and anger–sadness differences ($R = -0.39$, $p < 0.05$, one-tailed, Cohen's $d = 0.80$); whereas the correlation between BAS-drive and the sadness–neutral difference amplitude was not significant ($R = -0.039$, $p > 0.4$, one-tailed).

4. Discussion

The study investigated whether individual differences in BAS-drive modulate the early frontal response to angry faces compared with neutral and sad faces. Consistent with previous research, frontal and frontocentral ERPs were more positive for angry and sad faces relative to neutral faces, starting 130 ms after stimulus onset. Effects of BAS-drive on these electrodes occurred subsequently within 200–300 ms following stimulus onset; low BAS-drive subjects showed enhanced positive ERPs in response to angry faces relative to both neutral and sad faces, whereas no significant differences were found in high BAS-drive subjects. Critically, this effect was confirmed by a significant interaction between Group and Emotional expression. ERP results also revealed enhanced early posterior negativity in response to angry faces compared with neutral and sad faces, as well as enhanced N170 to angry and sad compared with neutral faces. However, these early emotion-specific ERP modulations at posterior sites were not significantly affected by differences in BAS-drive.

Our study shows for the first time that individual differences in a personality trait related to aggression are associated with different early (200–300 ms) frontal responses to facial signals of aggression. Moreover, the present ERP results accord with previous fMRI research showing that the magnitude of ventromedial prefrontal cortex (vmPFC) response to facial expressions of anger (relative to both sad and neutral expressions) is influenced by individual differences in BAS-drive (Beaver et al., 2008; Passamonti et al., 2008).

Increased early positive frontal ERPs were originally demonstrated for fearful compared with neutral faces (Eimer & Holmes, 2002) and interpreted as reflecting a rapid categorisation of signals related to threat or danger. Subsequent work has demonstrated that increased frontocentral positivities can also be found in response to other negative or positive facial expressions (*e.g.*, sadness, disgust, anger, happiness (Eimer et al., 2003; Holmes, Kiss, & Eimer, 2006; Holmes et al., 2003; Holmes, Winston, & Eimer, 2005)). Consistent with these earlier results, we found enhanced positive ERP amplitudes over frontal and frontocentral regions for both angry and sad expressions compared to neutral expressions that started at about 130 ms after stimulus onset.

Notably, the early phase of this frontocentral emotional expression effect (130–200 ms) was not significantly modulated by participants' BAS-drive scores. In contrast, the effects of facial expression in the 200–300 ms time window differed significantly between high and low BAS-drive groups. In the low BAS-drive group, ERPs to angry faces were more positive than ERPs to either neutral or sad faces, whereas no significant difference was found between ERPs to the three face categories in the high BAS-drive group. It should be noted that the ventral prefrontal source for ERP activity recorded in the 130–200 and 200–300 ms time windows has not been firmly established. However, supportive evidence comes from elsewhere. Intracranial recordings in human ventromedial prefrontal cortex (vmPFC) have shown increased electrophysiological response to fearful compared with happy facial expressions (and also to threatening compared to non-threatening emotional scenes) starting around 120–160 ms (Kawasaki et al., 2001). A frontal source for the early phase (130–200 ms) of these ERPs is also supported by patient work showing that enhanced frontal positivities to fearful faces are absent in patients with orbitofrontal damage (Ashley et al., 2002). Despite the non-uniqueness of the EEG inverse problem (Hauk, 2004), source localisation results are consistent with a participation of ventral prefrontal sources to these early frontal emotion-sensitive responses (Carretie, Hinojosa, Mercado, & Tapia, 2005; Esslen et al., 2004). Though, more posterior sources have also been suggested (Williams et al., 2006). For example, Williams et al. (2006) found that the prefrontal focus of fear-specific ERP responses is strongly present up to 220 ms after stimulus onset, and beyond 280 ms post-stimulus, while a more posterior source was active during the 150–280 ms time interval. Hence, while the exact time course of anterior and posterior sources that are responsible for emotion-specific modulations of ERPs needs to be established in future research, the fact that our findings mirror the frontal effects in previous fMRI studies—i.e., reduced differences between anger and neutral and between anger and sadness in high compared to low BAS-drive subjects—is at least consistent with a ventromedial prefrontal source of this effect.

As discussed, our previous fMRI research showed very similar effects of BAS-drive on vmPFC activity to angry faces (Beaver

et al., 2008). Higher BAS-drive scores were associated with an increase in amygdala activity as well as in a reduction of ventromedial prefrontal activity triggered by angry faces, suggesting that high scores are linked to decreased top-down control of negative affect. This is further supported by an fMRI connectivity study showing that coupling from the vmPFC to the amygdala (but not vice versa) is modulated by BAS-drive (Passamonti et al., 2008). Given the similarity of these findings with the anger-specific interaction with BAS-drive found in the current study—i.e., reduced difference between angry and neutral and between angry and sad but not between sad and neutral faces in high compared to low BAS-drive subjects—the vmPFC activation observed by Beaver et al. (2008), and the frontocentrally distributed emotional expression effect observed in the present study between 200 and 300 ms after stimulus onset may reflect the same underlying brain processes. If this assumption is correct, our current ERP findings add important new information regarding the timing of these top-down emotional control processes and their modulation by individual differences that could not have been obtained on the basis of fMRI measures alone.

Although emotional facial expression modulated anterior ERPs throughout the whole time interval considered (130–300 ms), the effects of BAS-drive on the response to angry compared to sad and neutral faces only emerged during the later phase of this interval (200–300 ms). This accords with Passamonti et al.'s (2008) proposal that there are at least two successive stages of emotional processing in the vmPFC. *A first stage* (up to about 200 ms) may reflect frontal encoding of stimulus significance, resulting in a rapid categorisation of the stimulus as emotional or not. This stage may rely on interactions of the vmPFC with the amygdala and posterior face-sensitive areas that show similar modulation by emotional expression, unaffected by personality. During *a second phase* (starting around 200 ms), interactive effects of emotion and personality take place, and differences in the subjective relevance or salience of affective stimuli are computed to assist the guidance of adaptive ongoing behaviour. During this stage, the selective reduction of the midline frontocentral response to angry faces in high BAS-drive subjects may be indicative of a reduced ability of vmPFC to down-regulate amygdala activity in response to angry faces, consistent with what is described in aggressive states (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Davidson, Putnam, & Larson, 2000; Dougherty et al., 2004; Raine, Buchsbaum, & LaCasse, 1997). The negative correlation between BAS-drive scores and the amplitude of the angry–neutral and angry–sad but not for the sad–neutral difference ERPs observed in the present study further supports the hypothesis of a relationship between the midline frontocentral response to facial signals of aggression and individual differences in aggressive behaviour in the healthy population.

In addition to the emotional expression effects at anterior electrodes described above, we also observed effects of facial expression on early posterior ERP components in the N1 time range. Effects of facial expression on the face-sensitive N170 are disputed. While some studies report no modulation by emotional expression (Bediou et al., 2007; Halgren, Raij, Marinkovic, Jousmaki, & Hari, 2000; Herrmann et al., 2002; Krolak-Salmon, Fischer, Vighetto, & Mauguiere, 2001) almost as many recent reports document a modulation (Ashley et al., 2004; Batty & Taylor, 2003; Eger et al., 2003; Miyoshi, Katayama, & Morotomi, 2004; Sprengelmeyer & Jentzsch, 2006). In our current study, we found N170 enhancements for angry and sad compared to neutral faces, in line with the latter set of studies. However, this effect of emotion on the N170 was not expected to be affected by personality; accordingly no effect of BAS-drive was found.

Modulations of posterior brain potentials by emotional facial expression have also been documented at similar or later latencies as those reported here (Krolak-Salmon et al., 2001; Sato et al., 2001; Schupp et al., 2004). These ERP effects are usually referred to as early posterior negativity, maximal at occipitotemporal areas within 130–300 ms after stimulus onset) and as the late positive potential (LPP, maximal within 300–600 ms post-stimulus over centroparietal regions). Here, we found increased negative response to angry faces compared to both neutral and sad faces over occipital electrodes (within 130–200 ms but not within 200–300 ms), consistent with the idea that the EPN reflects encoding of stimulus arousal. However, the fact that no enhancement was found in response to sad compared with neutral faces is only partly consistent with previous studies reporting enhanced EPN for fearful (Schupp et al., 2004) and happy (Sato et al., 2001) faces. Recent studies showing that diverted attention disrupts this effect (Schupp et al., 2007a) and that similar effects can be observed for non-emotional but attended (task-relevant) stimuli (Schupp et al., 2007b) have led to the alternative interpretation that the EPN reflects the processing of stimulus salience rather than greater arousal or evolutionary significance (*e.g.*, threat) associated with this emotion. Whatever this component reflects, unlike the frontocentral ERPs, we found no clear evidence that the EPN was significantly modulated by personality. The fact that the difference ERP amplitude between angry and neutral faces on the one hand, and between angry and sad faces on the other hand, both correlate with BAS-drive is consistent with a role for these frontal positivities in the evaluation of the subjective relevance of a stimulus. Indeed, the subjective relevance of an angry expression may depend on the level of aggressiveness of the individual observing this expression.

Our study shows for the first time that increased risk for aggressive behaviour is associated with reduced early frontal responses to facial signals of aggression within 200–300 ms post-stimulus. More specifically, midline frontocentral ERPs differed significantly between angry and neutral faces and between angry and sad faces in the low BAS-drive group, but not the high BAS-drive group; this was further confirmed by correlations with whole group analysis showing that the same difference ERPs correlated with BAS-drive scores. This pattern of result is consistent with previous research showing that the magnitude of ventromedial prefrontal cortex (vmPFC) response to facial expressions of anger relative to both sad and neutral expressions decreases with increasing BAS-drive (Beaver et al., 2008; Passamonti et al., 2008).

5. Conclusion

Consistent with our hypothesis, this study shows that an increased frontocentral response to angry compared to neutral and sad faces elicited between 200 and 300 ms post-stimulus is absent in individuals scoring high on BAS-drive. The midline frontocentral location of this effect is consistent with sources in the ventromedial prefrontal cortex. Moreover, the direction of the effect of personality on this ERP component—reduced angry–neutral and angry–sad differential response in high compared to low BAS-drive subjects—is in line with neuroimaging studies of clinically and non-clinically aggressive populations, showing reduced recruitment of prefrontal cortex to facial signals of aggression in subjects with heightened levels of aggressive behaviour (Amen, Stubblefield, Carmichael, & Thisted, 1996; Blair, 2003; Raine et al., 1997; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000; Volkow & Tancredi, 1987). Heightened aggression, or predisposition toward heightened aggression, is also associated with increased amygdala response and reduced coupling from the ventromedial prefrontal cortex to the amygdala in response to aggressive facial displays (Coccaro et al., 2007; Passamonti et al., 2008). Although evidence for a ventral prefrontal source is based essentially on the similarity with fMRI findings and therefore indirect, the excellent temporal resolution of EEG provides further important information regard-

ing the temporal properties of the interaction between personality and neural processing of emotional expressions, consistent with an early effect.

The adaptive and evolutionary functions of such an interaction between emotion processing and personality are obvious. While a fast automatic detection of danger is important to rapidly prepare a corresponding 'fight, flight or freeze' reaction and optimize survival, the rapid conscious and attention-dependent activation of prefrontal cortical structures is likely to reflect the control exerted by frontal regions upon this automatic processing in order to prevent emotional stimuli from interfering with ongoing behaviour by capturing or distracting attention and consuming resources. Our results suggest that this second stage is particularly sensitive to individual differences in personality.

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