Angry faces bias the spatial allocatoin of attention at early stages of processing

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1. ABSTRACT and BACKGROUND

There is plentiful evidence from behavioural studies to support the theoretical view that threat related material are processed faster than neutral material. The early P1 component of the ERP has been found to be modulated by threat-related expressions of fear (Pourtis et al. 2004) and by threat related negative scenes (Li et al. 2005). Using a covert orienting task (Posner, 1980) these authors found that the P1 amplitude to targets replacing the fearful and negative threat related scenes was greater compared to neutral faces or scenes. Li et al (2005) found that this effect was modulated by trait anxiety levels. The aim here was to demonstrate that the P1 amplitude to validly cued targets is greater for *angry* facial expressions. Results found support for an enhanced P1 amplitude for validly cued angry targets at short cue-target-onset-intervals but one that was not affected by levels of trait anxiety. These findings indicate that angry facial expressions bias the spatial allocation of attention at very early stages of processing.

2. METHOD and EEG recording

Low and high trait anxious participants performed a covert spatial cueing task. Trials consisted of a pair of faces: one emotional (either angry or happy) and one neutral followed by a probe presented in the location of one of the faces. There were two CTOAs (short and long). Participants were asked to judge the orientation of the probe. The P1 was measured for valid and invalidly cued angry and happy targets on the no-go trials where the orientation probe and thicker portion of cross were mismatched.

EEG was recorded (Brain Products GmbH, Germany) from 26 Ag-AgCl scalp electrodes mounted in a lycra electrode cap (EASYCAP GmbH, Germany) according to the International 10 – 20 System. P1 to target was analysed within the time window of 100 ms – 150 ms from target onset and was maximal over occipito-parietal electrodes PO7/8. The P1 for trials in which the target replaced the emotional face (*valid trial*) were compared with when it replaced the neutral face (*invalid trial*) for both CTOA durations. This yielded the following within subject variables; **Emotional expression (2), CTOA (2), Validity (2), Visual field of target (2), and Hemisphere (2).**

Experimental task and predictions
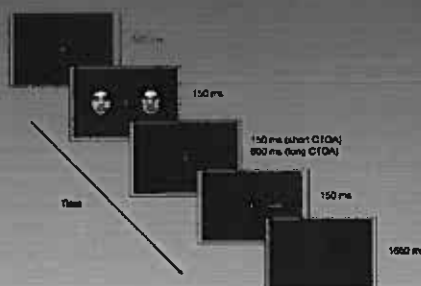
We predicted that angry faces would elicit greater P1 amplitudes to validly cued targets and that this effect would be modulated by trait anxiety levels.

(Fox et al. 2005; Bishop, 2007; Eysenck, Derakshan, et al. 2007).

3. RESULTS

Analysis revealed a significant two-way interaction of Visual Field X Hemisphere, $F(1, 26) = 11.60$, $p<.003$, a three way interaction of Visual Field X Hemisphere X CTOA, $F(1, 26) = 30.51$, $p<.001$, and a four-way interaction of Emotional expression X Visual Field X Validity X Hemisphere, $F(1, 26) = 5.19$, $p<.032$.

Analysis on contralateral P1 showed Emotional Expression X Validity X CTOA, $F(1,26) = 5.46$, $p = .02$. The P1 amplitude was greater for *valid* compared with *invalid* targets that were preceded by angry faces at the *short* CTOA ($1.81\mu V$ vs. $1.56\mu V$, $t(27) = 2.45$, $p = .02$). See Figs1 and 2. No significant effects were found for happy faces. No significant interactions were found with trait anxiety.

Figure 1. Grand averages to valid and invalidly cued angry and happy targets at short and long CTOAs.
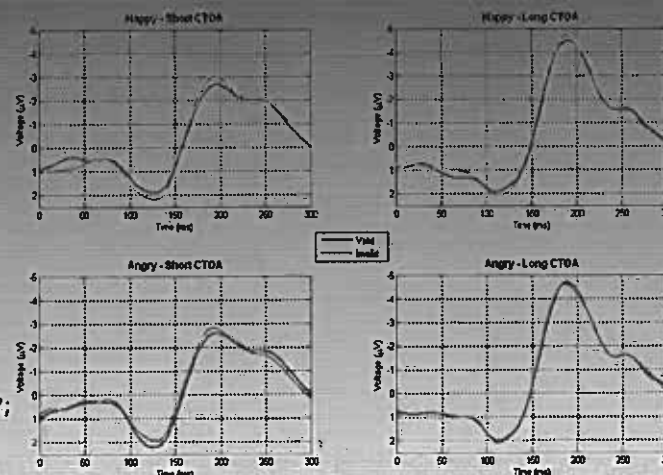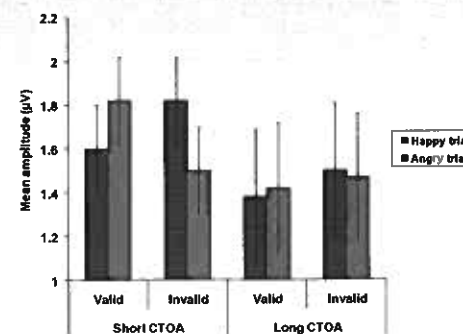

Figure 2. P1 mean amplitude to valid and invalidly cued angry and happy targets at short and long CTOAs.

4. CONCLUSIONS

Enhanced P1 amplitudes for validly cued angry targets were found at short but not long CTOA, indicating an early and rapid attentional shift towards threat. Our results replicate and extend previous work to show that unattended angry facial expressions bias the spatial location of attention at very early stages of processing (Vuilleumier & Pourtois, 2006). The methodological differences here, such as short cue exposure duration and a relatively longer cue-target-interval in our study, could be responsible for the lack of a P1 modulation by trait anxiety.