Title: Effect of temporal predictability on exogenous attentional modulation of feedforward processing in the striate cortex

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Abstract

Non-informative peripheral visual cues facilitate extrastriate processing of targets [as indexed by enhanced amplitude of contralateral P1 event-related potential (ERP) component] presented at the cued location as opposed to those presented at uncued locations, at short cue-target stimulus onset asynchrony (SOA). Recently, two lines of research are emerging to suggest that the locus of attentional modulation is flexible and depends on 1) perceptual load and 2) temporal predictability of visual stimuli. We aimed to examine the effect of temporal predictability on attentional modulation of feed-forward activation of the striate cortex (as indexed by the C1 ERP component) by high-perceptual-load (HPL) stimuli. We conducted two ERP experiments where exogenously-cued HPL targets were presented under two temporal predictability conditions. In Experiment 1 [high-temporal-predictability (HTP) condition], 17 healthy subjects (age 18-26 years) performed a line-orientation discrimination task on HPL targets presented in the periphery of the left upper or diagonally opposite right lower visual field, validly or invalidly cued by peripheral cues. SOA was fixed at 160ms. In Experiment 2 [low-temporal-predictability (LTP) condition], (n=10, age 19-36 years) we retained HPL stimuli but randomly intermixed short-SOA trials with long-SOA (1000ms) trials in the task-blocks. In Experiment 1 and the short-SOA condition of the Experiment 2, validly-cued targets elicited significantly faster reaction times and larger contralateral P1, consistent with previous literature. A significant attentional enhancement of C1 amplitude was also observed in the HTP, but not LTP condition. The findings suggest that exogenous visual attention can facilitate the earliest stage of cortical processing under HTP conditions.

Keywords: visual attention, event-related potentials, C1, temporal predictability, perceptual load, striate cortex

1. Introduction

Abrupt changes in the periphery of the visual field attract attention even if they are non-informative and irrelevant to the behavioural goals at the time. Salient stimuli that immediately follow at this cued location elicit faster reaction times than those presented at uncued locations. This stimulus-driven attentional deployment is termed exogenous visuospatial attention (Posner, 1980). It is synonymous with the reflexive (Hopfinger and Mangun, 1998), automatic (Jonides, 1981) or involuntary (Fu et al., 2005) attention described elsewhere in the visual attention literature.

Hopfinger and Mangun (1998) showed that exogenous attention could facilitate early extrastriate processing, as indexed by enhancement of contralateral P1 (P1cl) event-related potential (ERP[1]) component over lateral parieto-occipital region over 90-140ms post-stimulus latency range. This effect has been replicated in many subsequent ERP studies (Fu et al., 2001; Fu et al., 2005; Hopfinger and Mangun, 2001; Hopfinger and West, 2006). However the C1 ERP component (a negative peak around 80ms post-stimulus) arising from initial feed-forward processing in the striate cortex (V1) was not modulated by attention in any of these studies, suggesting that initial striate processing is not amenable to attentional modulation. Single-cell recordings in monkeys (Motter, 1993) and fMRI studies in humans (Gandhi et al., 1999; Martinez et al., 1999) show that attention can modulate striate processing, but concurrent EEG recording indicates that that this differential activation represents feedback activation from V1 rather than modulation of initial afferent impulses (Martinez et al., 1999).

In contrast, some recent ERP and magnetoencephalography (MEG) studies indicate that the feedforward activation of the striate cortex could be facilitated by attention under certain experimental conditions. These include endogenous visuospatial attentional paradigms (Kelly

[1] Abbreviations: ERP, event-related potential; MEG, magnetoencephalopgraphy; HPL, high perceptual load; LPL, low perceptual load; UVF, upper visual field; LVF, lower visual field; SOA, cue-target stimulus onset asynchrony; EOG, electrooculogram; ANOVA, analysis of variance

et al., 2008), object-based exogenous attentional paradigms (Khoe et al., 2005), and exogenous visuospatial attention paradigms which directly (Fu et al., 2010a; Fu et al., 2009; Poghosyan and Ioannides, 2008) or indirectly (Rauss et al., 2009) manipulated attention to peripheral stimuli. In a recent review, Rauss et al. (2011) propose heterogeneity of experimental paradigms as a cause for presence or absence of C1 attentional effects in different studies (Rauss et al., 2011). The authors point out that when exploring the C1 attentional effects, the stimulus properties and the task demands should exploit the functional characteristics of V1 (e.g. contrast sensitivity and retinotopic organisation), so that V1 can substantially contribute to the task, thus enabling any top-down effects to modulate the degree of contribution by V1 in turn modulating C1 amplitude.

Perceptual load (Lavie, 1995) and *temporal predictability* (Nobre et al., 2007) seem to be two independent modulators of the magnitude and the locus of top-down attentional effects. Lavie's load theory of attention proposes that the stage of attentional filtering is flexible depending on the 'perceptual load' of visual stimuli (Lavie, 1995; Lavie et al., 2004; Lavie and Tsal, 1994): the higher the perceptual load, the earlier attention interacts with visual processing. The original concept of perceptual load has been refined in to two distinct operational definitions in more recent work: 1) *attentional load* which refers to 'the differences in processing demands in the absence of physical stimulus differences' (Ding et al., 2014; Fu et al., 2012; Rauss et al., 2012; Rauss et al., 2009) and 2) *perceptual load* which refers to 'the amount of stimulus information that need to be processed to perform a given task' (Fu et al., 2010a; Fu et al., 2009; Rauss et al., 2012). In the present study, we use the term 'perceptual load' in this latter sense. The effect of attentional load on C1 modulation shows inconsistent results. These contrasting findings are debated with regards to the optimal method of attentional-load manipulation that enables C1 modulation (Fu et al., 2012; Fu et al., 2010b; Rauss et al., 2012; Rauss et al., 2009) and the source localisation of the attentional effect (Ding

et al., 2014; Rauss et al., 2009). These last two studies indirectly manipulated the exogenous attentional load allocated to peripheral stimuli by modulating the attentional load allocated to an attentional task at fixation (Ding et al., 2014; Rauss et al., 2009). In contrast, perceptual-load studies by Fu *et al* employed a line-orientation choice-reaction task under direct exogenous cueing (Fu et al., 2010a; Fu et al., 2009). In these studies, the degree of target-distracter overlap and/or the relative orientation of target vs. distracter array determine the discriminability of the target from concurrent distracters, consequently modulating the perceptual load on a trial-by-trial basis. Their results in general, comply with Lavie's load theory, showing an attentional facilitation of C1 amplitude elicited by high-perceptual-load (HPL) but not low-perceptual-load (LPL) stimuli. Interestingly, the attentional facilitation of C1 for HPL stimuli was observed only when they were intermixed with LPL stimuli within a task block (Fu et al., 2009).

Neuroimaging (Coull and Nobre, 2008; Coull et al., 2000; Coull and Nobre, 1998) and EEG/ERP (Doherty et al., 2005; Miniussi et al., 1999; Praamstra et al., 2006; Rohenkohl and Nobre, 2011) evidence in humans and single cell recordings in monkeys (Ghose and Maunsell, 2002) show that attentional circuitry of the brain can be tuned to predict not only the location of visual stimuli but also their timing. Doherty et al. (2005) examined the combined effect of spatial and temporal predictability of on visual ERPs in an exogenous attention task. The target was a circle moving across the screen in steps and then disappearing beneath a band of occlusion. When it appeared on the other side the subjects had to respond only if the circle contained a dot in the middle. The researchers modulated the trajectory and regularity of initial movement of the circle thus modulating the spatial and temporal expectancy of its appearance beyond the occlusion. As expected, the targets that moved in a linear trajectory (i.e. high spatial orienting) and reappeared in predictable locations beyond the occlusion elicited larger P1 components. More importantly, this P1 facilitation was further augmented when the target

circle moved at regular time intervals (i.e. high temporal predictability). These results have been replicated by Rohenkhol and Nobre (2011). Collectively, the studies on temporal orienting suggest that high temporal predictability (HTP) of target stimuli help to pace top-down attentional systems to facilitate their processing.

Even though a large body of literature has addressed the significance of stimulus characteristics and perceptual/attentional load in attentional modulation of early visual processing, the role of temporal predictability of stimuli on C1 attentional effects has not been methodically examined. The aim of the present study was to determine the role of temporal predictability on attentional modulation of feedforward visual processing at V1. To maximize the contribution of V1 we administered an exogenously-cued, HPL, line-orientation discrimination task. We hypothesised that HPL and HTP should be the optimal combination to move the locus of attentional modulation as far downstream as possible and to facilitate initial stages of V1 processing of visual stimuli. If HPL is sufficient and HTP is not essential for attentional facilitation of V1 processing, we expected an attentional modulation of C1 to be present even when the SOA is variable. To examine these hypotheses, we examined the attentional effect on C1 ERP component by modulating the temporal predictability in two separate exogenous visuospatial attention experiments. In the first experiment we maintained a HTP of targets by employing a fixed SOA, whereas the second had a low temporal predictability (LTP) produced by a variable SOA.

2. Experiment 1

2.1. Materials and Methods

2.1.1. Participants

Twenty-three healthy university undergraduates participated in this study. All were right handed and had normal or corrected-to-normal vision. None of the participants had

significant neurological / psychiatric illnesses or used psychoactive drugs. The study was approved by the Human Research Ethics Committee of The University of Newcastle, Australia and was conducted in accordance with the standards laid down in the Declaration of Helsinki (1964). Informed written consent was obtained from all participants.

Data from six subjects were excluded: three due to low (<70%) accuracy rates, two due to excessive alpha activity in EEG recordings and one due to absence of an identifiable C1. Had we averaged more trials to generate ERP waveforms, exclusion due to last two causes could also have been mitigated, but at the cost of possible fatigue due to prolonged testing. Data are reported from the remaining 17 participants (15 women) aged between 18-26 years (mean=20.5, SD=2.6).

2.1.2. Stimuli and procedure

The task was a modified version of the Fu et al. 2009 experimental paradigm (Fu et al., 2009). Presentation (Neurobehavioral Systems, CA, USA) software was used to generate visual stimuli. The participants performed a visual stimulus discrimination task under an exogenous cuing paradigm. They maintained gaze on a 0.33^0 x 0.33^0 black central fixation cross in a white background from a distance of 70cm throughout each experimental block. The peripheral cue, a Kanizsa box (4.73^0 x 4.73^0) (figure 1a) flashed for a duration of 50ms, randomly in either the left upper visual field (UVF) or diagonally opposite right lower visual field (LVF) at an eccentricity of 7.4^0 from fixation. The centre of each cued location formed a 23.7^0 polar angle with the horizontal meridian. Each cue was followed by a target array (duration 100ms, 2.74^0 x 2.74^0) that randomly appeared at the cued location (in 50% of the trials viz. valid trials) or the diagonally opposite location (in other 50% of the trials viz. invalid trials). The target was either a forward diagonal ("/" requiring left index finger response, 50% probability) or backward diagonal ("\" requiring right index finger response, 50% probability)

embedded among distracters (Figure 1b). The inter-trial interval (stimulus offset to cue onset) varied randomly between 1200 and 1600ms. Each test block comprised 80 trials evenly but randomly distributed across the two visual fields and the two validity conditions, thus consisting of 20 trials per each validity-visual field combination. The cue-stimulus onset asynchrony (SOA) in test trials was 160ms. Participants completed 10 blocks of test trials thus completing 200 trials per each validity-visual field combination. Each participant underwent two practice trial blocks in the beginning of the experimental session.

We also administered five blocks of 80 'dummy' trials which were similar to test trials except that the SOA was 560ms. These blocks were incorporated as every third block in the experimental session. To correct for overlap of cue- and target-ERPs, the ERPs generated by these 'dummy' trials were cue-locked, and were subtracted from cue-locked ERPs elicited by test trials that had the cue in the same visual field[2]. All analyses were conducted on these difference waveforms.

2.1.3. Data acquisition and analysis

SCAN software Version 4.3 (Compumedics Neuroscan, North Carolina, USA) was used to acquire and process EEG data. Continuous EEG data were acquired and digitized at a rate of 1000Hz. Scalp EEG and electrooculogram (EOG) was recorded from a modified 32-channel Neuroscan Quickcap. A denser electrode array was used over posterior scalp sites in order to have a higher spatial resolution for C1 component. Recording scalp sites were FZ, CZ, C1, C2, C3, C4, CPZ, CP1, CP2, CP3, CP4, PZ, P1, P2, P3, P4, P7, P8, POZ, PO3, PO4, PO7, PO8, OZ, O1, O2, IZ, I3 and I4. EEG data were recorded using the left mastoid as a reference, and re-referenced offline to the average of the left and right mastoid. The ground electrode was

[2] We administered 'dummy' trials to keep the participants engaged and achieve a level of general motivation and arousal similar to test trials. The possible disadvantage of the method (compared to passive viewing of a cue-only condition) was the risk of generation of a preparation-related, CNV-like activity during the relatively long cue-target interval in the dummy trials but not in the short cue-target interval in the test trials, thus making the ERP waveforms of 'dummy' trials incompatible. We examined the pre-target interval in uncorrected averaged ERP waveforms of 'dummy' trials of each participant but there was no evidence of CNV like activity (uncorrected grand average waveforms for test trials and dummy trials are shown in Supplementary Figure 1).

placed at AFZ. The high-pass filter was set at 0.1Hz and the low-pass filter at 70Hz. Electrode impedance was maintained below 5kΩ in each channel during EEG acquisition.

EEG data were epoched time-locked to target onset. Artefact rejection thresholds were ±30μV for EOG channels and ±80μV for other channels. Accepted epochs were averaged separately for UVF-valid, UVF-invalid, LVF-valid and LVF-invalid trials.

Automatic peak detection was performed to measure amplitudes and latencies of C1, posterior-midline P1 (P1m), ipsilateral P1 (P1il) and N1 (N1il) and contralateral P1 (P1cl) and N1 (N1cl) components in averaged ERP waveforms. The latency ranges for peak-detection for C1, P1, and N1 were 50-100ms, 80-170ms and 130-230ms, respectively. C1 and P1 elicited by LVF stimuli had the same polarity and considerable component overlap affecting the reliability of their peak amplitude and latency measurements. Therefore, the present analysis was conducted on amplitudes and latencies of the ERP components elicited only by UVF targets.

Reaction time (RT) latencies and accuracy rates for valid and invalid trials were compared with paired sample t tests. The amplitude and latency data for midline ERP components were analysed in a two-way validity x site within-subject ANOVA model with subsequent pairwise comparisons at each site. The lateral P1 and N1 data were initially analysed in a three-way validity x hemisphere x site within-subject ANOVA model and simple effects were calculated subsequently. When pairwise comparisons of a given ERP component were carried out at multiple scalp sites, the significance of the results were interpreted after Bonferroni correction for the number of scalp sites ($p = 0.05/3 = 0.017$ for C1 analysed at OZ, POZ and PZ; $p = 0.025$ for lateral P1 and N1 analysed at two lateral parieto-occipital sites). As measures of effect size, partial eta square (η^2) value was calculated for ANOVA tests, and Cohen's d (d) for Student's t tests. All statistical analyses were conducted using Statistical Package of Social Sciences (SPSS^{TM}) version 16.0. (SPSS Inc. Chicago, IL).

We examined the target-locked averaged horizontal EOG (HEOG) waveforms for 160ms SOA trials to detect any deviation of eyes towards the cued location at the target onset. In the grand average waveforms, the mean HEOG amplitude during 0-20ms post-target was less than 1μV under each validity x visual field condition. We statistically examined the effect of HEOG on ERP components by including the 0-20ms post-target mean HEOG amplitude as a covariate into the above ANOVA models. However, HEOG amplitude did not emerge as a significant covariate in any of the models, so that HEOG amplitude was dropped as a covariate from the final set of models which are reported in this paper.

2.3. Results and Discussion

2.3.1. Behavioural measures

Response accuracy rates and RT latencies for valid and invalid trials for each visual field are summarised in Table 1. The accuracy rate for valid trials was higher than that for invalid trials for UVF targets [$t(16) = 3.626$, $p = 0.002$, $d = 0.879$] and LVF targets [$t(16) = 3.285$, $p = 0.005$, $d = 1.020$]. As expected, valid trials also elicited faster RTs than invalid trials for UVF targets [$t(16) = 6.884$, $p < 0.0001$, $d = 1.67$] and LVF targets [$t(16) = 7.108$, $p < 0.0001$, $d = 1.72$].

Behavioural data thus confirm the occurrence of exogenous attentional facilitation. The average accuracy rate is lower and the mean RT observed in the present study is longer than those in the Fu et al. (2009) experiment. This is not surprising particularly because their participants were trained on the task until they reached an accuracy rate above 90%.

2.3.2. ERP measures

ERP amplitude data for UVF stimuli are summarised in Table 2.

2.3.2.1. C1 and P1m

Grand averaged waveforms in posterior midline electrodes are shown in Figure 2. The observed C1 changed polarity across upper and lower visual fields confirming striate cortical origin of the component (Clark et al., 1995; Jeffreys and Axford, 1972). Conforming to our hypothesis, valid targets elicited larger C1 amplitudes at posterior midline sites [$F(1,16) = 6.142$, $p = 0.025$, $\eta 2 = 0.0.277$]. C1 for valid targets also had longer mean peak latencies than those for invalid targets in the midline sites [$F(1,16) = 6.411$, $p = 0.022$, $\eta 2 = 0.286$].

In contrast to C1, invalid trials elicited significantly larger P1m amplitudes: the validity main effect was highly significant [$F(1,16) = 20.169$, $p = 0.0004$, $\eta 2 = 0.0.558$]. There was a significant validity x site interaction with the amplitude difference becoming more marked from OZ to POZ to PZ [$F(1,16) = 30.583$, $p < 0.0001$, $\eta 2 = 0.657$]. Pairwise post-hoc comparisons showed that invalidly-cued targets eliciting significantly larger P1m amplitudes at POZ [$t(16) = 4.275$, $p = 0.001$, $d = 1.04$] and PZ [$t(16) = 6.302$, $p < 0.0001$, $d = 1.52$]. The difference was not significant at OZ [$t(16) = 1.860$, $p = 0.081$, $d = 0.451$]. Target validity did not significantly change P1m latency.

Even though we did not intermix HPL stimuli with LPL stimuli, these attentional effects on C1 and P1m (Figure 2) are similar to those observed by Fu et al. (Figure 3, (Fu et al., 2010a) and Figure 3, (Fu et al., 2009)) who intermixed high- and low-load stimuli. Enhanced C1 amplitude, prolonged C1 peak and attenuated P1m could also be interpreted as a more generalised negative deflection that spans late C1 to P1 latency range. To explore this, we conducted running t-tests between ERP waveforms for valid and invalid conditions over C1-P1 latency window. Valid trials exhibited a significant negative deflection ($p < 0.05$) over 78-203ms latency range at POZ and over 85-198ms at PZ.

2.3.2.3. Lateral P1 and N1

Grand average waveforms of contralateral and ipsilateral P1 and N1 elicited by UVF stimuli are depicted in Figure 3. For left UVF stimuli, P1cl and N1cl were measured at P8 and PO8, whereas P1il and N1il were measured at P7 and PO7. There was significant validity x hemisphere interaction for P1 amplitude at lateral parietal (P7/P8) sites [$F(1,16) = 32.61$, $p < 0.0001$, $\eta 2 = 0.671$] and lateral parieto-occipital (PO7/PO8) sites [$F(1,16) = 26.18$, $p = 0.0001$, $\eta 2 = 0.621$]. Further exploration showed larger P1cl for valid targets at P8 [$t(16) = 3.084$, $p = 0.007$, $d = 0.748$], and trend towards smaller P1il for valid targets at P7 [$t(16) = 2.343$, $p = 0.032$, $d = 0.568$]. No significant attentional effect was observed at PO7 or PO8.

There was a significant validity x hemisphere interaction for lateral N1 amplitudes at P7/P8 [$F(1,16) = 37.96$, $p < 0.0001$, $\eta 2 = 0.703$] and PO7/PO8 [$F(1,16) = 16.00$, $p < 0.0001$, $\eta 2 = 0.644$]. The direction of the interaction was the opposite of that for P1, with valid targets eliciting significantly smaller N1cl at P8 [$t(16) = 4.547$, $p = 0.0003$, $d = 1.102$] and PO8 [$t(16) = 3.925$, $p = 0.001$, $d = 0.952$] and larger N1il at P7 [$t(16) = 4.529$, $p = 0.0003$, $d = 1.098$] and PO7 [$t(16) = 3.93$, $p = 0.001$, $d = 0.953$]. Lateral P1 or N1 latencies were not significantly different between valid and invalid trials.

The reversal of the attentional modulation of lateral P1-N1 complex across hemispheres is similar to the observations of Fu et al. (2005, Figure 3a) who administered a very similar exogenous cuing paradigm, albeit with LPL stimuli. P1cl amplitude difference in valid and invalid trials signifies an attentional facilitation of early extrastriate processing. This effect has been widely documented in previous studies that employed fixed SOA paradigms (Fu et al., 2005) as well as those with jittered SOA (Hopfinger and Mangun, 1998, 2001; Hopfinger and West, 2006) paradigms. Rather unexpectedly, such attentional facilitation of P1cl was not observed in the other two recent studies that reported exogenous attentional facilitation of C1 (Fu et al., 2010a; Fu et al., 2009).

Attentional facilitation of ipsilateral components of N1 and inhibition of contralateral component is noteworthy. This pattern has also been observed previously in an exogenous cueing paradigm (Fu et al., 2001).

3. Experiment 2

Similar to the Experiment 1, this was an exogenously-cued, HPL, line-orientation discrimination task, but with low temporal predictability. To diminish the temporal predictability we inter-mixed a short-SOA condition (160ms) with an additional long-SOA (1000ms) condition within the same test block. If the temporal predictability is an essential condition for attentional facilitation of feedforward striate processing, we expected attentional modulation of C1 to be absent in this experiment, whereas if HPL is sufficient and a temporal expectancy is not essential, we expected short-SOA (i.e. 160ms) trials to show attentional facilitation of C1.

In addition, the long-SOA condition was expected to elicit an inhibition of return (IOR) effect with the valid targets generate slower RT (Posner and Cohen, 1984). To this end, we examined whether exogenous attention enhances striate and extrastriate ERP components in the facilitatory phase of attention (i.e. at short SOAs) and inhibits the components in the inhibitory phase (i.e. at long SOAs), notwithstanding diminished temporal predictability in the paradigm.

3.1. Materials and Methods

3.1.1. Participants

Twelve healthy female university undergraduates (age 18-36 years) participated in this study. All were right handed and had normal or corrected-to-normal vision. None of the participants had significant neurological / psychiatric illnesses or regularly used psychoactive

drugs. Informed written consent was obtained from all participants. Data from two subjects were excluded: one due to excessive alpha activity in EEG recordings and one due to absence of an identifiable C1. Data are reported from the remaining 10 participants aged between 18-36 years (mean = 23.1, SD = 5.2).

3.1.2. Stimuli and procedure

The fixation, cue and the target array, their locations on the visual display and inter-trial interval were the same as those of the first experiment. However, we reduced temporal predictability by inserting long-SOA (1000ms) trials within the test blocks. Similar to Experiment 1, we also used 'dummy' trials with an SOA of 1300ms and these were also inserted in to the test blocks. Thus there were trials with three different SOAs – 160ms, 1000ms and 1300ms – presented within the same trial block with a probability of 40%, 40% and 20%, respectively. Trials of each type were randomly distributed within each block. A total of 2000 trials were administered in 10 task blocks of 200 trials each. Similar to the Experiment 1, half of the target arrays in each validity and SOA condition were presented in the left UVF, and the other half in the diagonally opposite right LVF. The total testing time of the task was about 90 minutes.

3.1.3. Data acquisition and analysis

EEG recording parameters, artifact rejection, epoching and peak detection criteria were the same as those of the Experiment 1. EEG epochs were averaged separately for valid and invalid trials for each of the three SOAs (viz. 160ms, 1000ms and 1300ms) conditions. To correct for overlap of cue- and target-ERPs, the ERPs generated by 1300ms-SOA trials were cue-locked, and were subtracted from cue-locked ERPs elicited by 160ms-SOA trials and 1000ms-SOA trials that had the cue in the same visual field. Further analyses were conducted

on these difference waveforms. Similar to the Experiment 1, ERP data analysis was conducted on amplitudes and latencies of the components elicited only by UVF targets.

Behavioural data were analysed in a within-subject two-way validity x SOA ANOVA design for targets presented in each visual field. Planned simple effects were compared only between the valid and invalid trials in each SOA condition for each visual field. The data for ERP components were analysed in a two-way validity x SOA within-subject ANOVA model in each site to examine effect of SOA on attentional modulation on ERP components. Subsequent pairwise comparisons were done on each ERP component between valid and invalid trials at each site in each SOA condition. Results were interpreted at a significance level (p) of 0.05 for behavioural data and C1 and P1m data. Similar to Experiment 1, when pairwise comparisons of a given ERP component were carried out at multiple scalp sites, results were interpreted after Bonferroni correction.

3.2. Results and Discussion

3.2.1. Behavioural Measures

Behavioural measures of the Experiment 2 are summarised in Table 3. No significant validity x SOA interaction observed in accuracy for the targets presented either in the UVF [$F(1,9) = 1.765$, $p = 0.217$, $\eta^2 = 0.164$] or the LVF [$F(1,9) = 0.072$, $p = 0.795$, $\eta^2 = 0.008$]. The mean response accuracy did not differ significantly between different validity or SOA conditions in either visual field. As expected, validly-cued targets elicited significantly faster means reaction times than invalidly-cued targets presented at short SOAs in both UVF [$t(9) = 7.683$, $p < 0.0001$, $d = 2.43$] and LVF [$t(9) = 3.086$, $p = 0.013$, $d = 0.976$]. However, there was no difference in RTs for valid and invalid trials at the Long SOAs, indicating that we failed to elicit an IOR effect in this experiment.

3.2.2. ERPs

Figure 4 shows the grand average ERP waveforms elicited by short-SOA and long-SOA trials and Table 4 summarises the C1 and P1m amplitudes at the posterior midline sites viz. OZ, POZ and PZ. No significant validity main effect [$F(1,9) = 0.035$, $p = 0.855$] or SOA x validity interaction [$F(1,9) = 0.327$, $p = 0.581$) was observed in C1 amplitude. C1 latency also did not show a significant validity main effect [$F(1,9) = 0.106$, $p = 0.756$], but there was a significant validity x SOA interaction [$F(1,9) = 16.99$, $p = 0.006$] suggesting validly-cued targets elicit shorter C1 latencies in the short-SOA condition whereas the invalidly-cued targets to generate shorter C1 latencies in the long-SOA condition. However, subsequent pairwise comparisons showed no significant validity effect on C1 marginal mean latencies across-sites or C1 mean latency in any of the sites at any of the SOA conditions (Table 5). P1m component amplitudes did not show a significant validity main effect [$F(1,9) = 0.077$, $p = 0.077$] or SOA x validity interaction [$F(1,9) = 0.240$, $p = 0.636$].

The validity main effect for P1cl was significant at P8 [$F(1,9) = 7.559$, $p = 0.023$, $\eta^2 = 0.456$] and approached significance at PO8 [$F(1,9) = 5.01$, $p = 0.052$, $\eta^2 = 0.358$]. Similar to the observations of the Experiment 1, there was a trend for the valid trials to elicit larger P1cl at the short-SOA [P8: $t(9) = 2.539$, $p = 0.032$, PO8: $t(9) = 2.069$, $p = 0.06$] but not at the long SOA condition [P8: $t(9) = 0.855$, $p = 0.42$, PO8: $t(9) = 0.725$, $p = 0.49$]. N1cl amplitudes did not show significant validity main effects, SOA x validity interaction, or simple effects.

Overall the above ERP results are consistent with the findings of the previous exogenous attention studies that applied jittered SOA paradigms (Hopfinger and Mangun, 1998, 2001).

4. Conclusions

The polarity reversal of the first ERP component elicited by UVF and LVF stimuli in this study confirms that component is the C1, which is generated by feedforward visual

processing in the striate cortex (Clark et al., 1995). We found that exogenous attention enhances C1 elicited by HPL stimuli under HTP, but not LTP conditions. Modulation of temporal orienting only (Miniussi et al., 1999) – by presenting stimuli at visual fixation and thus eliminating any spatial orienting effect – has shown that HTP facilitates P300, a late stages of visual processing. Although, more recent studies that modulated spatial and temporal orienting has shown a synergistic effect of the two factors on early extrastriate processing (Doherty et al., 2005; Rohenkohl and Nobre, 2011), they – because they employed LPL targets and ERP averaging across UVF and LVF – were not intended to examine the attentional effects on C1 component. Nevertheless, our results together with the above findings highlight the importance of the temporal dimension in attentional modulation of visual processing.

The difference in C1 between valid and invalid targets observed in this study is unlikely to be due to overlap of cue- and target-evoked ERPs, because we eliminated cue-related effects by a subtraction procedure. Previous exogenous cueing studies with fixed SOA paradigms have adjusted for this with the same method (Fu et al., 2009) or, in case of jittered SOA (Fu et al., 2010a), with ADJAR algorithm (Woldorff, 1993). The subtraction procedure however does not negate other forms of cue-target sensory interactions. Refractoriness is a form of interaction that occurs at cellular level. However, cue-induced refractoriness would diminish the amplitude of C1 evoked by targets presented in the same location (i.e. the valid targets) rather than increasing its amplitude. Cues could also trigger oscillatory activity in V1 (predominantly alpha rhythms). The amplitude of C1 evoked by subsequent target depends on the oscillatory phase in which the target enters the V1. Targets reaching V1 in the alpha peaks then would evoke larger C1s compared to those reaching V1 during the troughs. This would introduce a systematic bias only if the oscillations are triggered at a relatively constant interval after cue-onset and those oscillations have a relatively constant wavelength. If this is the case the oscillatory activity should show up in averaged ERP recordings especially during the cue-

target interval in the 560-ms SOA trials, but we did not observe such systematic oscillations. It should also be noted that we excluded two subjects with excessive alpha activity.

Based on two exogenous attention studies, Fu et al. (Fu et al., 2010a; Fu et al., 2009) suggest that attentional enhancement of C1 by HPL stimuli occurs only when they are intermixed with LPL stimuli. It is noteworthy that our findings indicate that intermixing of HPL and LPL stimuli is not essential for this attentional enhancement: initial striate processing of HPL stimuli – even when presented in isolation – can be facilitated by exogenous attention if the temporal predictability is high.

Interestingly, temporal-predictability manipulation in our study also led to attention mediated 'blunting' of the adjacent P1m component which has the same topography as the C1. The running t-tests on ERP amplitudes indicate that the attentional modulation begins within the C1 time window extending to the P1m time window and can be characterised as a protracted negativity superimposed on the C1 and P1m peaks. Very similar ERP patterns had been observed in the C1-P1m range in both previous exogenous attention experiments that showed enhancement of C1 (Fu et al., 2010a Figure 3; Fu et al., 2009 Figure 3). Further, disappearance of attentional enhancement of C1 in our second experiment was also accompanied with the disappearance of attentional inhibition P1m. While it is possible that attentional manipulation affects C1 and P1m components independently, a more parsimonious explanation for the above observations would be attentional enhancement of a third negative component (that spans C1-P1m time range) under HTP conditions.

In conclusion, we propose that exogenous cueing with HTP paces the attentional systems to deploy maximum top-down signals at the predicted time of feed-forward activation of V1 by target stimuli. Although the latency and topography of ERP changes are consistent with initial V1 processing, the attentional effect does not seem to be an isolated enhancement of the C1 component as we initially hypothesised or as reported in previous studies (Fu et al.,

2010a; Fu et al., 2009). Further studies with source localization and component analysis will help to elucidate the underlying spatio-temporal dynamics of this attentional modulation.

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Figure Captions:

Figure 1: Stimuli. (a) peripheral cue (4.73^0 x 4.73^0), (b) target array (2.74^0 x 2.74^0), (c) stimulus presentation sequence in a test trial (ITI: inter-trial interval)

Figure 2: Experiment 1. Grand average waveforms for targets presented in upper visual field. POZ tracing also displays the waveforms elicited by lower visual field targets (thin lines) to illustrate polarity reversal of C1 (* $p < 0.05$).

Figure 3: Experiment 1. Grand average waveforms of valid and invalid trials depicting of contralateral (P8 and PO8) and ipsilateral (P7 and PO7) P1 and N1 components (*$p<0.05$, **$p<0.01$).

Figure 4: Grand average waveforms for targets presented in upper visual field at short- and long-cue-stimulus asynchrony (SOA) conditions. POZ tracing also displays the waveforms elicited by lower visual field targets (thin lines) to illustrate polarity reversal of C1.

Supplementary Figure 1: Grand-averaged target-locked ERP waveforms (POZ) for test trials [cue-target stimulus onset asynchrony (SOA) = 160ms] and dummy (SOA = 560ms) trials. Cue- and target-evoked ERP overlap is seen in test trials, but not in dummy trials. No contingent negative variation-like activity is seen during the pre-target interval in the latter condition.

Table(s)

Table 1: Experiment 1. Behavioural data (UVF: upper visual field, LVF: lower visual field)

	Accuracy (% correct) Mean (SD)		Reaction time (ms): Mean (SD)	
	Valid	Invalid	Valid	Invalid
Upper visual field	85.6 (5.5)	82.0 (7.4)	600.2 (75.3)	637.9 (65.6)
Lower Visual field	86.6 (8.5)	83.0 (10.4)	590.9 (76.3)	627.4 (73.3)

Table 2: Experiment 1. ERP amplitude comparison for valid and invalid upper visual field targets.

Component	Site	Mean Amplitude (SD)	
		Valid	Invalid
C1	OZ	-2.66 (0.96)	-2.36 (1.27)
	POZ**	-3.63 (1.25)	-2.79 (1.42)
	PZ	-3.40 (1.57)	-2.46 (1.59)
P1m	OZ	3.37 (1.74)	4.66 (2.93)
	POZ***	2.48 (2.15)	6.08 (3.73)
	PZ***	0.58 (1.52)	5.44 (3.53)
Contralateral P1	P8**	4.12 (1.79)	2.84 (1.41)
	PO8	5.21 (1.98)	4.36 (2.80)
Ipsilateral P1	P7	1.96 (1.56)	3.15 (1.79)
	PO7	3.06 (1.63)	4.07 (2.46)
Contralateral N1	P8***	-2.71 (3.10)	-4.63 (3.41)
	PO8***	-3.22 (3.43)	-5.46 (3.94)
Ipsilateral N1	P7***	-3.32 (1.36)	-1.51 (1.96)
	PO7***	-3.57 (1.99)	-1.53 (2.38)

*$p < 0.025$, **$p < 0.017$, ***$p < 0.001$

Table 3: Experiment 2 - reaction time data (SOA: cue-target stimulus onset asynchrony, UVF: upper visual field, LVF: lower visual field).

	Visual field	SOA	Mean (SD)		Significance (p)
			Valid trials	Invalid trials	
Accuracy (% correct)	Upper	Short	84.2 (6.9)	83.0 (6.4)	NS
		Long	83.1 (8.9)	84.7 (7.2)	NS
	Lower	Short	88.0 (7.5)	84.9 (7.1)	0.033
		Long	89.7 (6.0)	87.1 (6.9)	0.031
Reaction time (ms)	Upper	Short	617.4 (71.5)	663.0 (79.4)	<0.0001
		Long	648.3 (74.2)	651.6 (84.3)	NS
	Lower	Short	606.2 (80.7)	625.8 (88.2)	0.013
		Long	626.4 (84.0)	627.7 (89.5)	NS

Table 4: Experiment 2 – ERP amplitudes for valid and invalid upper visual field targets.

Component	Site	Mean amplitude (SD), uV			
		Short SOA		Long SOA	
		Valid	Invalid	Valid	Invalid
C1	OZ	-1.99 (1.42)	-2.06 (1.32)	-2.35 (1.15)	-2.40 (1.29)
	POZ	-3.48 (1.77)	-3.59 (2.15)	-3.81 (2.55)	-3.60 (2.96)
	PZ	-3.14 (1.66)	3.50 (1.69)	-3.39 (2.42)	-3.34 (2.47)
P1m	OZ	3.43 (2.04)	2.93 (1.68)	4.63 (1/96)	4.37 (2.31)
	POZ	2.98 (2.65)	3.30 (2.11)	4.98 (2.21)	5.76 (2.38)
	PZ	2.17 (2.76)	3.58 (2.01)	4.07 (2.61)	5.06 (2.96)

Table 5: Experiment 2 – C1 ERP latencies for valid and invalid upper visual field targets.

Site	Mean latency (SD), ms			
	Short SOA		Long SOA	
	Valid	Invalid	Valid	Invalid
OZ	79.6 (7.8)	83.2 (8.0)	83.2 (6.9)	79.8 (6.2)
POZ	80.0 (3.2)	81.1 (4.1)	81.6 (6.4)	79.9 (2.5)
PZ	79.5 (3.4)	80.6 (4.1)	83.6 (5.9)	79.4 (3.0)

Figure 1

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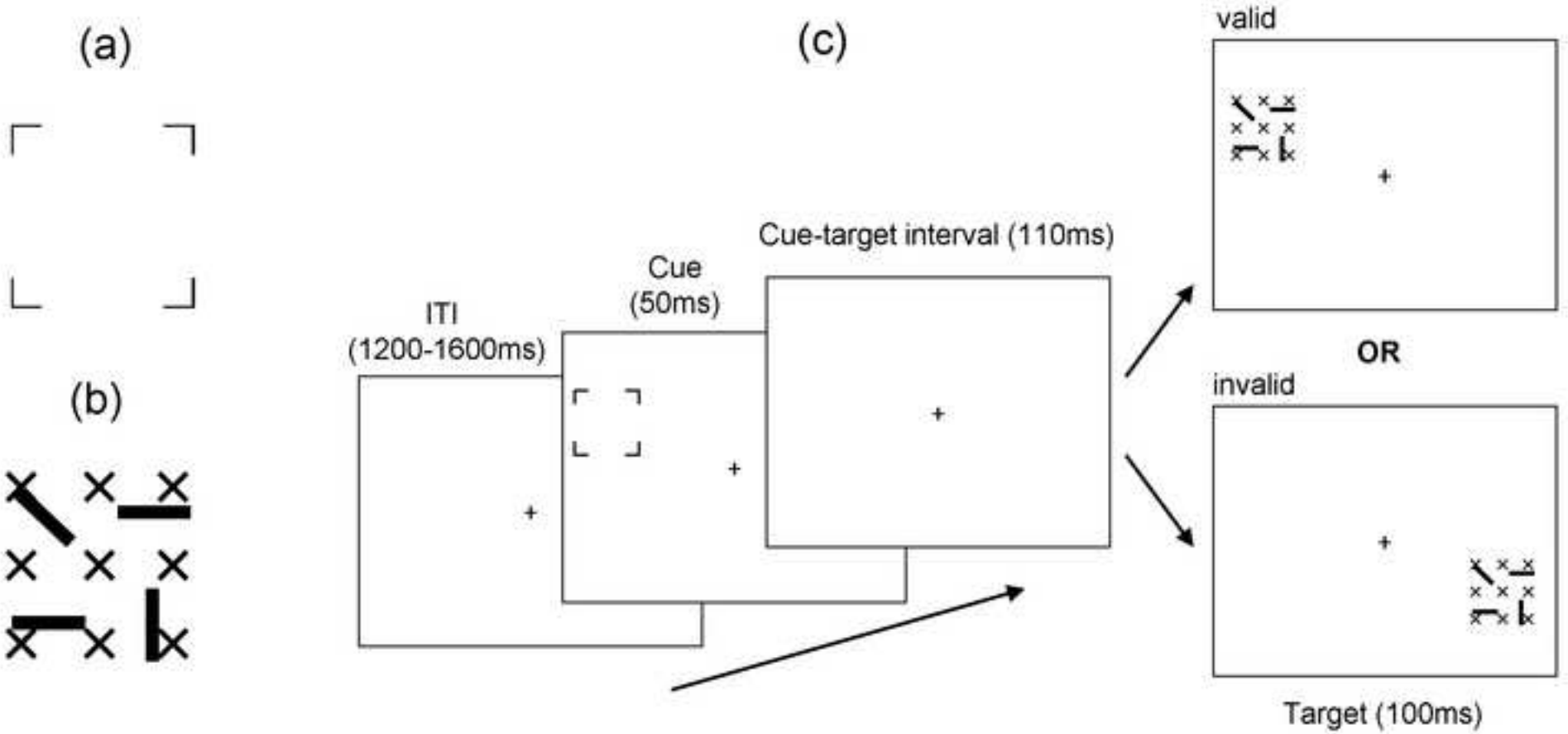

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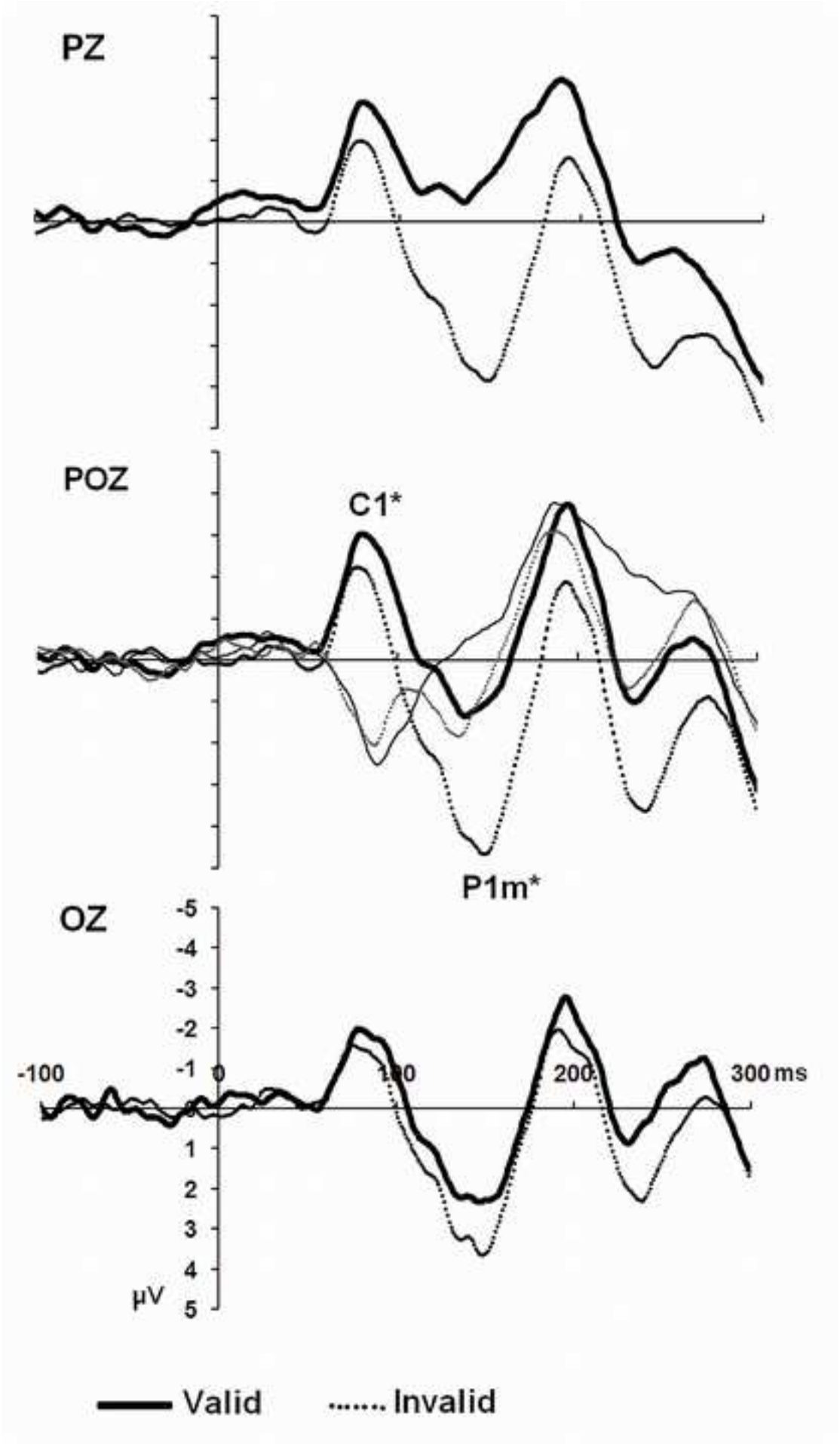

Figure 3

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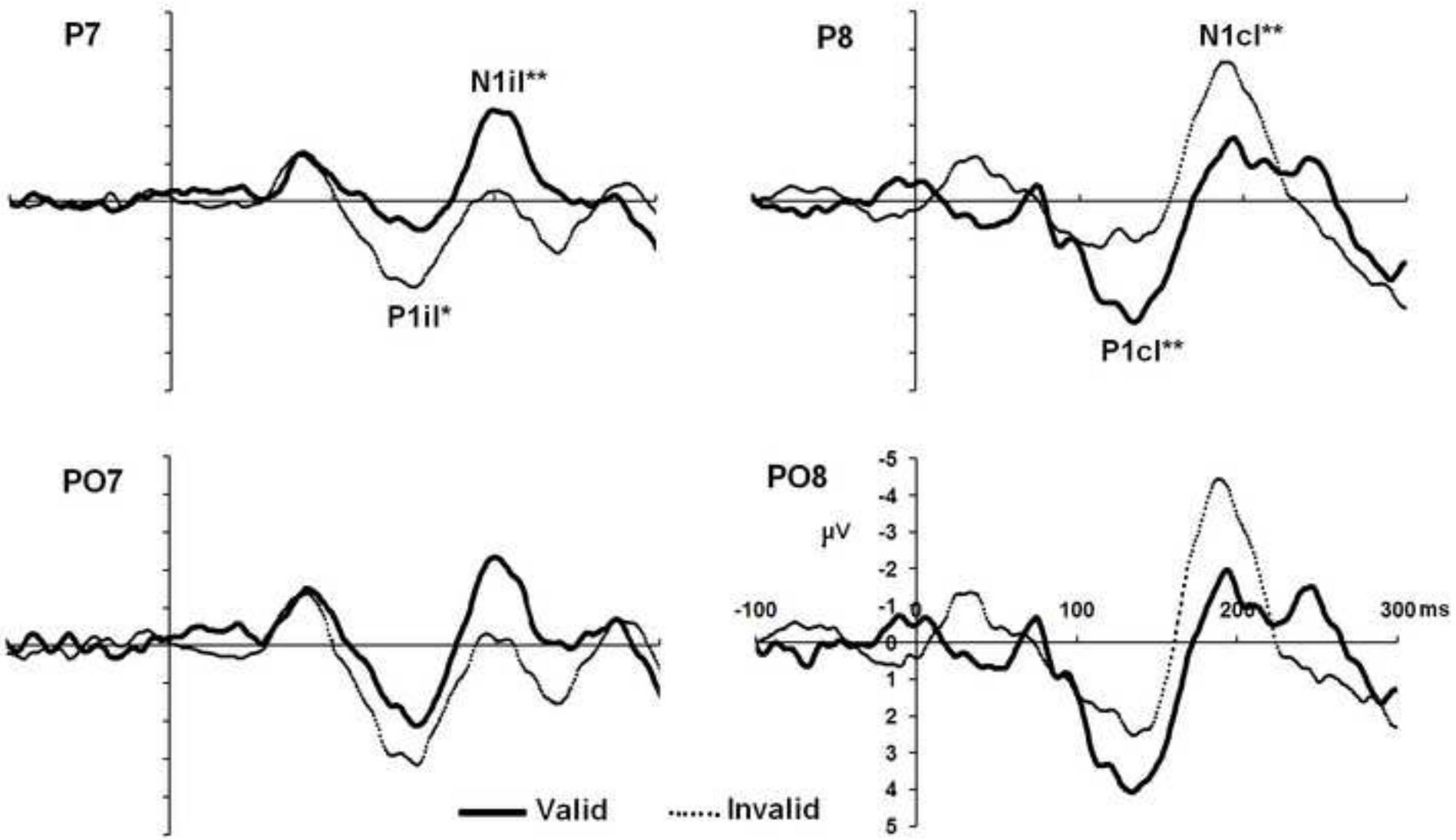

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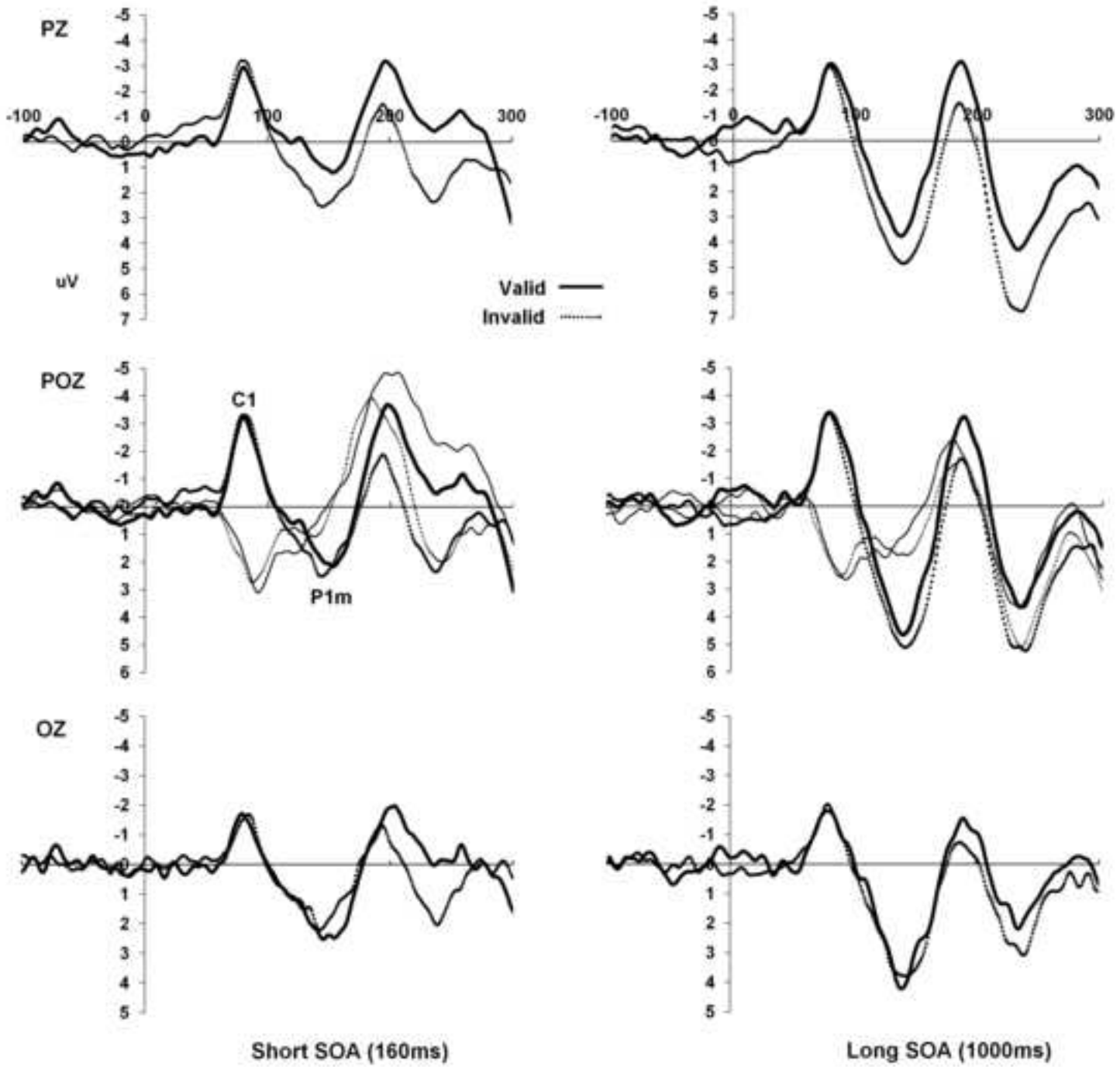

Supplementary Figure

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