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Microsaccadic Inhibition and P300 Enhancement in a Visual Oddball Task

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Abstract

It has recently been demonstrated that the presentation of a rare target in a visual oddball paradigm induces a prolonged inhibition of microsaccades. In the field of electrophysiology, the amplitude of the P300 component in event-related potentials (ERP) has been shown to be sensitive to the stimulus category (target vs. non target) of the eliciting stimulus, its overall probability, and the preceding stimulus sequence. In the present study we further specify the functional underpinnings of the prolonged microsaccadic inhibition in the visual oddball task, showing that the stimulus category, the frequency of a stimulus and the preceding stimulus sequence influence microsaccade rate. Furthermore, by co-recording ERPs and eye-movements, we were able to demonstrate that, despite being largely sensitive to the same experimental manipulation, the amplitude of P300 and the microsaccadic inhibition predict each other very weakly, and thus constitute two independent measures of the brain's response to rare targets in the visual oddball paradigm.

Abbreviations:

ERP Event-Related Potential

WOI Window of Interest

Introduction

During the last ten years, the interest for eye movements during fixations has increased considerably. Due to the introduction of video-oculographic methods for the recording of eye movements, it is now possible to reliably identify microsaccades, which are fast (up to 300°/s) mainly conjugate eye movements occurring about once per second (Møller et al., 2002). Many relevant results have emerged. For example, it has been shown that microsaccades modulate the firing of neurons in the visual system (Leopold & Logothetis, 1998; Martinez-Conde et al., 2000;

Martinez-Conde et al., 2002) by counteracting neural fatigue and the fading of peripherally presented stimuli during sustained fixation (Martinez-Conde et al., 2006). This hypothesis is consistent with the finding that microsaccades become more frequent when the retinal displacement produced by slower fixational eye movements (i.e. drifts) is reduced (Engbert & Mergenthaler 2006). Microsaccades have also been found to play a role in the maintenance of correct visual fixation (Engbert & Kliegl, 2004; Liang et al., 2005; Mergenthaler & Engbert, 2007). Finally, there is growing evidence that the orienting of spatial attention biases the direction of microsaccades (Engbert & Kliegl, 2003; Rolfs et al., 2004; Galfano et al., 2005; Rolfs et al., 2005, Laubrock et al., 2005, Betta et al., 2007; Turatto et al., 2007, Laubrock et al., 2007, but see Horowitz et al., 2007). Similar results have recently been confirmed on the subset of microsaccades known as saccadic intrusions (Gowen et al., 2007).

Microsaccades also seem to be influenced by higher-level cognitive factors other than spatial attention. In his seminal work on fixational eye movements, Barlow (1952) described a reduction in the rate of microsaccades when participants were required to perform a demanding cognitive task. This was based largely on non-systematic observations of the participants' behavior. However, recent works have confirmed that microsaccades are inhibited when participants encounter rare task-relevant visual stimuli, which have to be counted. Valsecchi et al. (2007) measured the rate of microsaccades in a visual oddball task, which consisted in the serial presentation of rare target stimuli (oddballs) and frequent non target stimuli in random order. The authors found that the probability of microsaccades following the presentation of standard stimuli showed a biphasic time course, with an early inhibition phase peaking at 100-150 ms post stimulus onset and a later rebound phase peaking at 300-350 ms post stimulus onset. This stereotypical response has been widely observed in response to visual (Engbert & Kliegl, 2003; Galfano et al. 2004) and acoustic (Rolfs et al. 2005) stimuli, and is considered a sub-cortical oculomotor reflex possibly occurring at the superior colliculus (SC) level, at least in its inhibitory component (Engbert, 2006). However, Valsecchi et al. (2007) found that the inhibitory phase of the

microsaccadic response was prolonged and the rebound almost abolished, after the presentation of an oddball stimulus. This effect was observed both with peripheral and central stimuli and for different stimulus onset asynchronies, but it was absent when the oddballs were not task-relevant. The authors suggested that the prolonged inhibition of microsaccades could be considered an index of the evaluation of task-relevant stimuli in the visual oddball paradigm. In a more recent study (Valsecchi & Turatto, 2007), the authors again found the prolonged inhibition of microsaccades in response to visual oddballs, while also showing, by using stimuli equiluminant with the background, that a cortical visual pathway can support the modulation of microsaccade rate in response to both oddball and standard stimuli.

Oddball paradigms have been extensively used in psychophysiological studies for four decades (e.g., Sutton et al., 1965; Näätänen, et al., 1978; Donchin & Coles, 1988; Johnson, 1988), and particularly with respect to the P300 component in the event-related brain potential (ERP). The P300 is a centro-parietal positivity peaking at around 300 ms post stimulus onset. This component has been considered an index of stimulus categorization (Kutas et al., 1977; Donchin & Coles, 1988; Verleger, 1988; Kok, 2001) and has been shown to be sensitive to both the stimulus category, that is, targets induce a higher-amplitude P300 than non-targets, and its overall, a priori frequency, that is, the less frequent a stimulus, the larger the elicited P300 (e.g., Duncan-Johnson & Donchin, 1977). P300 amplitude is also modulated by stimulus sequence (Squires et al., 1976; Jentzsch & Sommer, 2001), with disruptions of runs of stimulus repetitions or alternations eliciting larger P300 amplitudes than continuations of such runs. The sequence-based enhancement of P300 amplitude can be dissociated from the effect of overall stimulus frequency (Duncan-Johnson & Donchin, 1977).

Hence, microsaccadic inhibition and P300 enhancement are both observed in response to rare targets in visual oddball paradigms. However, it remains to be shown whether microsaccadic inhibition is also sensitive to target effects and stimulus sequence as is P300 amplitude. In order to answer these questions and to establish whether the similarity between the two measures goes

beyond the sensitivity to the same experimental manipulations, we conducted a visual oddball experiment, orthogonally manipulating stimulus frequency and stimulus category while simultaneously recording eye movements and ERPs. If a functional relationship exists between microsaccadic inhibition and P300 enhancement, we expected to find effects of stimulus category, stimulus frequency and stimulus sequence on both measures. If the two phenomena are directly related they should predict each other both at inter-trial and inter-participant levels.

Methods

Participants

Thirteen young adults took part in the experiment. One participant was discarded from analysis because of the presence of blinks in more than 50% of the epochs. The mean age of the remaining 12 participants was 25.6 years, 9 were females. All participants reported normal visual acuity, showed normal color vision according to the Ishihara Color Vision Test (Ishihara, 2003) and were right-handed according to the Edinburg Inventory (minimum score = 64; Oldfield, 1971). Two of the authors (M.V. and O.D.) took part in the experiment, whereas all other participants were naïve as to the purpose of the study. All participants gave informed consent and were remunerated either with 7 € per hour or course credits.

Stimuli

Stimuli were red or green disks (2.04° in diameter), with a white fixation dot (0.48° in diameter) at the centre. In order to enhance the physical similarity between target and non target stimuli and to control for intensity effects, the luminance of the red and green colors was matched for each participant using 25 Hz flicker fusion (Ives, 1912). The background was black during the entire experiment. Stimuli were presented on a 19-inch LG Flatron 915FT CRT monitor at a refresh rate of 100 Hz and a viewing distance of 75 cm. Stimulus duration was 100 ms, inter-stimulus interval was 900 ms, and the fixation point remained visible during the inter-stimulus interval.

Stimulus presentation was controlled using Presentation software (Neurobehavioral Systems, Inc., San Francisco, CA).

Experimental Procedure

Participants sat in a dimly illuminated, acoustically and electrically shielded cabin. The experiment was divided into three conditions. In each condition, 500 stimuli were presented in random order. Participants had to silently count the stimuli matching the target color, which alternated between participants, and had to fixate the white dot while minimizing eye blinks during the experimental sessions. After each block of 100 stimuli, participants reported the number of stimuli and were allowed to rest. Forty additional stimuli were presented in a practice block before each experimental condition. In the first condition, 50% of the stimuli were targets, whilst in the following two conditions the frequency of targets could be either 20 or 80% (the order of the last two conditions was alternated across participants). The 50%-condition was always run first in order to avoid possible carry-over effects, that is, participants might otherwise have implicitly adopted a biased expectation about the global stimulus frequency from the previous condition.

Eye-movement recording and microsaccade detection

Eye movements were recorded monocularly, with an iView X Hi-Speed infrared eyetracker (SensoMotoric Instruments, Teltow, Germany). Movements of the head were limited by the eye tracker's built-in chin and forehead rest. Recording was from the right eye, though viewing was binocular. The system had a sampling frequency of 238 Hz, a tracking resolution of < 90 sec-arc and an absolute gaze position accuracy of up to 0.2°. A standard 9-point calibration was performed before the beginning of each block of 100 stimuli. Fixation was checked after every 10 trials. If the gaze was found outside of a 2.04° x 2.04° square centered around the fixation point the experiment was interrupted and the system recalibrated.

Microsaccades were detected using the algorithm introduced by Engbert and Kliegl (2003). The algorithm was applied to epochs ranging from 150 ms before stimulus presentation to 1050 ms

after stimulus presentation. Microsaccades were defined as parts of the eye position trace where velocity (calculated with a 5 point moving window) exceeded a combined threshold for the vertical and horizontal component equal to 6 times the standard deviation of the velocity profile within the epoch. Minimum allowed duration was 4 samples (16.8 ms) and maximum allowed peak velocity was 200°/s. Additionally, microsaccades starting less than 4 samples after the previous microsaccade were rejected. Epochs containing blinks or saccades with amplitudes greater than 1° were discarded from analyses.

Electrophysiological recording

The electroencephalogram (EEG) was recorded from 40 Ag/AgCl electrodes on the scalp and around the eyes. 34 of the electrodes were mounted in an elastic electrode cap (Electrocap International Inc., Eaton, USA) at positions Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, FT9, FC5, FC1, FC2, FC6, FT10, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, PO9, O1, Oz, O2, PO10, and Iz (American Electroencephalographic Society, 1994). Foam cushions were fitted to the participant's forehead in order to avoid direct pressure on the frontal electrodes. Six external electro-oculogram (EOG) electrodes were affixed at the outer canthi of the left and right eye, below each eye and on the left and right mastoid. An electrode at AFz was used as ground. All impedances were kept below 5 kΩ. A Brainamp DC amplifier (Brain Products GmbH, Munich, Germany) digitized the data at a sampling rate of 250 Hz, and a bandpass from DC to 70 Hz. Data was recorded with a PC running BrainVision Recorder Software (Brain Products GmbH). All channels were initially referenced to the left mastoid (A1) and converted to average reference off-line. Synchronization between EEG and eye tracker was achieved via TTL pulses sent from the stimulus presentation PC to both systems on every trial. The co-registration setup used in the present study has previously been applied and evaluated in several psycholinguistic experiments on reading (Dimigen et al., 2006).

Data analysis

The eye tracking data and the ERP data were first analyzed separately. In particular, we extracted three measures of microsaccadic inhibition, i.e. amplitude of the peak microsaccade rate, latency of the peak microsaccade rate and rate of microsaccades in a specific time window of interest (WOI), and one measure of P300 amplitude, that is, the average voltage at electrodes P3, Pz and P4 between 200 and 500 ms. Repeated-measures ANOVAs with Stimulus Category (Target vs. Non Target) and Stimulus Frequency (20%, 50% or 80%) as factors were performed on each of the different measures.

Subsequently, we looked for sequence effects on the microsaccade rate in the time WOI and on P300 amplitude. We identified continued and discontinued sequences of stimulus repetitions, which are known to generate different P300 amplitudes, in the 50% stimulus frequency condition, and we performed repeated-measures ANOVAs with Stimulus Category and Sequence as factors.

Finally, in order to explore the relationship between microsaccades and P300, we performed an across-participants analysis of the linear regression between P300 and microsaccadic effects of Stimulus Category, Stimulus Frequency and Sequence. Furthermore, within the 50% frequency target trials, we controlled whether the presence of a microsaccade in the time WOI and the P300 amplitude within a trial were predictive of each other.

Results

Counting task

The participants were highly accurate in counting the target stimuli. The mean absolute counting error was 1.19% in the experimental condition with 50% targets, 0.91% in the condition with 20% targets and 0.95% in the condition with a target frequency of 80%. No counting errors occurred in 77.09 % of the reports. The counting data were not statistically analyzed.

Microsaccade Rate: Stimulus Category and Frequency effects

On average, we were able to collect, per participant, 1353.1 epochs free of saccades larger than 1° and blinks. The minimum number of epochs for each cell of the experimental design (i.e. for each combination of participant, stimulus frequency and stimulus category) was 36.

The evolution of microsaccade rate in response to target and non target stimuli is depicted in Figure 1, separately for the three stimulus frequency levels (20%, 50% and 80%). The rate was calculated in a sliding time window of 100 ms, moving in steps of 4.2 ms (i.e. the maximum temporal resolution allowed by the sampling frequency of the eye-tracker). The plots were constructed for each participant, stimulus frequency and stimulus category, and subsequently averaged across participants.

---------- Insert Figure 1 about here, please ----------

To ensure that the visual stimuli induced a reliable inhibition of microsaccades, we first identified the time point at which the minimum microsaccade rate was reached for each stimulus category and stimulus frequency. The average latency of the inhibition peak across stimulus category and stimulus frequency was 138.7 ms. The microsaccadic rates in two 100-ms bins, the first one centered on 0 ms latency (i.e. stimulus onset) and the second one centered on 138.7 ms latency (i.e. the inhibition peak), were analyzed in a three-way ANOVA with Bin (0 vs. 138.66 ms), Stimulus Category, (Target vs. Non Target) and Stimulus Frequency (20%, 50% or 80%) as factors. The main effect of Bin was significant ($F(1,11)=12.053$; $p<0.0052$), showing the overall presence of an inhibition effect. The Bin x Stimulus Category and the Bin x Stimulus Frequency interactions were not significant (both $ps>0.05$).

Of central interest for the present study, however, was the later rebound in microsaccade rate. A peak in microsaccade rate was reached in all conditions between 300 and 500 ms after stimulus onset. The magnitude of the peak rate and its latency seemed to be modulated by stimulus frequency and stimulus category, and this modulation was more evident in the case of target stimuli.

In particular, with increasing target frequency, the amplitude of the peak microsaccade rate seemed to increase, whereas the latency of the peak seemed to decrease.

This observation was confirmed by a statistical analysis. The magnitude of the peak microsaccade rate and its latency could be identified for each participant as the earliest point where the maximum value in microsaccade rate was reached in the single participant equivalent of the plots in Figure 1. Additionally, we calculated the microsaccade rate in a 100-ms time WOI centered on the latency of the peak microsaccade rate observed in response to the most frequent non target stimuli. The center of the WOI was set at 320 ms post stimulus onset according to the grand-averages in Figure 1, Panel B (also see Valsecchi et al. 2007). A separate 3 x 2 Analysis of Variance with Stimulus Frequency (20%, 50% or 80%) and Stimulus Category (Target vs. Non Target) as factors was applied to each of the three measures.

In the case of the magnitude of peak microsaccade rate (Figure 2, Panel A), we did not observe a significant effect of Stimulus Category and Stimulus Frequency (both $F<1$), but their interaction was significant ($F(2,22)=3.953$; $p<0.0341$). Post-hoc tests were performed separately for the two stimulus categories. In the case of target stimuli there was a non-significant trend for the magnitude of peak microsaccade rate to increase as a function of the stimulus frequency ($F(2,22)=3.048$; $p<0.0678$), whereas the peak microsaccade rate was unaffected by stimulus frequency in the case of non targets ($F(2,22)<1$).

The same analysis was applied to the latency of the peak microsaccade rate (Figure 2, Panel B) revealing significant effects of Stimulus Category ($F(1,11)=33.87$; $p<0.0001$) and Stimulus Frequency ($F(2,22)=12.104$; $p<0.0003$), whereas their interaction was only marginally significant ($F(2,22)=3.145$; $p=0.0629$). The latency of the peak microsaccade rate was shorter for non targets and decreased as the stimulus frequency increased. At least numerically, the effect of stimulus frequency seemed to be stronger in the case of targets as compared to non targets.

Stimulus Frequency had a significant effect also on the microsaccade rate in the time WOI ($F(2,22)=10.37$; $p<0.0007$) (Figure 2, Panel C), while the effect of Stimulus Category was not

significant ($F(1,11)=2.621$; $p=0.1337$). However, the interaction between the two factors was significant ($F(2,22)=21.617$; $p<0.0001$). Post-hoc tests were performed separately for the two stimulus categories. The rate of microsaccades in the time window centered at 320 ms post stimulus onset increased as a function of Stimulus Frequency in the case of target stimuli ($F(2,22)=21.202$; $p<0.0001$), whereas the effect was not significant in the case of non targets ($F(2,22)=1.491$; $p=0.247$).

---------- Insert Figure 2 about here, please ----------

P300: Stimulus Category and Frequency effects

EEG data were segmented into epochs extending from 100 ms before stimulus onset to 1000 ms after stimulus onset and baseline-corrected by subtracting for each channel the mean voltage in the 100 ms pre-stimulus interval. On average, we were able to collect 1319.4 epochs free of ocular artifacts (saccades longer than 1° or blinks) or drift artifacts (defined as absolute voltage values in the epoch exceeding 100 μV after baseline correction or a voltage difference between any two sampling points in the channel greater than 150 μV) for each participant. The minimum number of epochs for each cell of the experimental design (i.e. for each combination of participant, stimulus frequency and stimulus category) was 35. The grand average of the voltage amplitude at electrode Pz in response to target and non target stimuli is depicted in Figure 3, separately for the three stimulus frequency levels (20%, 50% and 80%). As in the case of microsaccade rate, there was a clear modulation of the waveform by stimulus frequency, and this seemed to be particularly evident in the case of target stimuli.

---------- Insert Figure 3 about here, please ----------

We chose to use the average voltage at electrodes P3, Pz, and P4 between 200 and 500 ms latency as a measure of P300 amplitude for statistical analysis. This choice was corroborated by the observation that in this time window the experimental manipulations most strongly affected the voltage at centroparietal electrodes (see Figure 4). A two-way ANOVA with Stimulus Frequency

and Stimulus Category as factors revealed significant main effects of Stimulus Category ($F(1,11)=61.942$; $p<0.0001$) and Stimulus Frequency ($F(2,22)=29.143$; $p<0.0001$) on P300 amplitude. The two factors also interacted significantly ($F(2,22)=7.872$; $p<0.0026$). The effect of Stimulus Frequency was more pronounced for target than for non-target stimuli. Nonetheless, post-hoc tests indicated that the frequency effect was significant for both targets ($F(2,22)=24.697$; $p<0.0001$) and non-targets ($F(2,22)=7.513$; $p<0.0033$). Notice that P300 amplitude decreased as a function of stimulus frequency, while the rate of microsaccades in the corresponding time window showed the opposite pattern, that is, it was higher for more frequent stimuli.

---------- Insert Figure 4 about here, please ----------

Sequence effects

Sequence effects on microsaccade rate and P300 amplitude were analyzed only in the case of the 50% stimulus frequency condition, where all sequences of a given order were equiprobable. We analyzed the two fourth-order sequences (i.e. based on the type of the current stimulus and of the preceding three, see Squires et al., 1976), which are expected to generate the lowest and the highest P300, respectively. "Continued" sequences, that is, sequences constituted by four repetitions of the same stimulus, induce the lowest P300 in response to the current stimulus, whereas "discontinued" sequences, that is, sequences where the current stimulus is preceded by three stimuli of the other type, induce the highest P300 (Squires et al., 1976; Jentzsch & Sommer, 2001). We excluded epochs which contained artifacts (ocular or other) or an interruption of the stimulus sequence, that is, a pause between blocks of trials or a recalibration of the eye-tracker. The average number of epochs for each participant, stimulus type and sequence in this sub-set of data was 25.5 and the minimum was 14. This number of trials was not sufficient to precisely identify peaks of microsaccade rate; consequently, we used the presence or absence of at least one microsaccade in the time WOI between 270 and 370 ms post stimulus onset as an index of microsaccadic inhibition. The mean values of the two measures for these two sequences are plotted in Figure 5. The pattern of

modulation appeared to be opposite in the two measures and the modulation was apparently stronger for the P300 measure.

---------- Insert Figure 5 about here, please ----------

A two-way ANOVA of microsaccade rates with Stimulus Category (Target vs. Non Target) and Sequence (Continued vs. Discontinued) as factors and the probability of occurrence of at least one microsaccade in the WOI as dependent variable yielded a significant effect of the factor Sequence ($F(1,11)=6.004$; $p<0.0322$), whilst the effect of the factor Stimulus Category and the interaction were not significant (both $Fs<1$). The same analysis was applied on the P300 measure and yielded a significant effect of the factor Sequence ($F(1,11)=10.149$; $p<0.0086$) and Stimulus Category ($F(1,11)=30.56$; $p<0.0002$), whilst their interaction was not significant ($F<1$). Once again, when the P300 was higher in amplitude for the discontinued sequences, the probability of occurrence of a microsaccade in the time WOI was lower. In addition, the P300 amplitude for target stimuli was higher as compared to non targets, whereas no significant difference was found for the measure of microsaccadic inhibition.

Relation between microsaccadic and P300 effects

In order to investigate whether our measures of microsaccadic inhibition and P300 are functionally related, we addressed whether a between-participants linear relation existed between the effects of Stimulus Category or Stimulus Frequency on the two measures. An across-participant linear regression analysis showed no sign of relation ($R^2= 0.0941$) between the microsaccadic and P300 Stimulus Category effects (targets minus non targets; see Figure 6, Panel A). This is not surprising, given that the effect of Stimulus Category on the measure of microsaccadic inhibition was not significant in the first place. In contrast, the factor Stimulus Frequency had shown significant effects on both the P300 and the microsaccadic inhibition measures. Nonetheless, the effect measures (differences between levels 20% and 80%) showed only a rather weak ($R^2=0.2181$) and nonsignificant ($F(1,10)=2.789$; $p=0.1258$) negative linear relation across participants (Figure 6,

Panel B). We also calculated the linear regression of the effect of Sequence on P300 amplitude as a function of the effect of Sequence on microsaccadic inhibition (difference between discontinued and continued sequences), as in the case of Stimulus Frequency, although both effects were significant in the first place, they were not related (R^2=0.0283; F(1,10)=0.291; p=0.6011) (see Figure 6, Panel C).

---------- Insert Figure 6 about here, please ----------

Of course, the absence of a significant correlation may also reflect lack of statistical power for this analysis. Thus, we conducted a further analysis to investigate the relationship between P300 and microsaccadic inhibition, i.e. we checked whether the observation of at least one microsaccade in the time WOI was predictive of the P300 amplitude. In particular, for each participant and among the subset of target trials from the 50% frequency condition, we identified the epochs in which at least one microsaccade was observed in the time WOI, and epochs in which no microsaccade was observed in the same time window. This subset was chosen because we had enough trials, the amplitude of P300 to targets was large enough to allow its detection on single trials and because all stimulus sequences occurred with the same probability. The average number of saccade-present epochs for each participant was 162.08 (minimum=79), the average number of saccade-absent epochs for each participant was 61.5 (minimum=12). A paired t-test showed that the amplitude of P300 was not significantly higher in saccade-absent epochs than in saccade-present epochs (t(11)=1.502; p=0.1610), see Figure 7, Panel A.

We also conducted the converse analysis, i.e., in the same epochs (Target and 50% Stimulus Frequency) we checked whether the measure of P300 was predictive for the execution of a microsaccade independently of stimulus frequency and stimulus category. We performed a median split of the subset of trials based on the amplitude of P300 (average voltage at Pz, P3 and P4 between 200 and 500 ms post stimulus onset). This analysis yielded an average number of 111.5 epochs per participant and P300 amplitude bin (high vs. low), the minimum number of epochs per cell was 90. A paired *t*-test showed that the probability of observing at least one microsaccade in the

time WOI was not significantly lower in the high-P300 epochs than in the low-P300 epochs ($t(11)=0.688$; $p=0.505$), see Figure 7, Panel B.

---------- Insert Figure 7 about here, please ----------

Discussion

Several studies in the last decade have shown that microsaccades can be used as a tool to investigate the state of the cognitive system (see Engbert, 2006). In particular, Valsecchi et al. (2007) showed that the rate of microsaccades presents a prolonged inhibition when a rare target is encountered in a visual oddball task. In the same paradigm, an enhancement of P300 amplitude is also commonly observed in response to visual oddballs (Hermann & Knight, 2001). The amplitude of the P300 component is sensitive to the sequence of the stimuli preceding the upcoming one (Squires et al., 1976; Duncan-Johnson & Donchin, 1977; Jentzsch & Sommer, 2001), so that stimuli discontinuing the preceding sequence elicit a higher P300.

Category / Frequency effects on microsaccades

The first aim of the present study was to establish whether the prolonged inhibition of microsaccades which was observed by Valsecchi et al. (2007) was due to target effects, to frequency effects, or to a combination of both. To answer this question, we conducted a visual oddball experiment varying the frequency of targets, which was 20, 50 and 80% in different conditions. Given the fact that the rebound in the rate of microsaccades, which normally follows the inhibition peak after the presentation of a visual stimulus (e.g. Engbert & Kliegl, 2003; Galfano et al., 2004), was clearly recognizable in the single-participant plots, we were able to individuate three measures of microsaccadic inhibition. The first measure was the rate of microsaccades in the time window where the rebound in response to the most frequent non targets was observed. The second measure was the latency of the rebound peak and the third was its amplitude. The three measures were not equally sensitive to the experimental manipulations, but in general we observed a more pronounced inhibition of microsaccades in response to less frequent stimuli and this effect was

stronger for targets. Pure target effects were only observed for the latency of the rebound peak, whilst they were not significant for the other two measures.

Category / Frequency effects on P300

Simultaneously to microsaccades, we also recorded ERPs. We found P300 amplitude to be larger for less frequent stimuli and for targets as compared to non targets. Moreover, the P300 amplitude modulation by stimulus frequency was stronger for targets than for non targets, a pattern of results that has been reported previously (e.g. Duncan-Johnson & Donchin, 1977; Potts et al., 2004) and that has been interpreted as a sign of attentional effects on stimulus processing (Kok, 2001). In other words, the rare targets would capture attention more than frequent and irrelevant stimuli. In the present study, microsaccadic inhibition and P300 were modulated in a coherent way by the task-relevance and by the overall frequency of the stimuli, except for the fact that the target effect was less reliable for microsaccades, being significant only when the latency of the rebound peak was taken as a measure of microsaccadic inhibition.

Sequence effects

We further addressed whether microsaccadic inhibition is influenced by the stimulus sequence. It has long been known that stimuli interrupting a series of identical stimuli induce a higher P300, irrespective of the a priori stimulus probability (Squires et al., 1976; Duncan-Johnson & Donchin, 1977; Jentzsch & Sommer, 2001). We replicated this observation in our P300 measure. Even when target and non target stimuli were equally probable overall, after three identical stimuli in a row, a stimulus alternation elicited a higher P300 than another repetition. This was true independently of whether the final stimulus was a target or not. Interestingly, the same pattern emerged in microsaccadic inhibition; the rate of microsaccades was more inhibited for discontinued than for continued repetition runs. As in the case of P300, the sequence effect on microsaccadic inhibition was observed both for targets and non targets.

We can thus conclude that the overall probability and the preceding stimulus sequence determine both the amplitude of P300 and the extent of microsaccadic inhibition elicited by task-

relevant stimuli. The somewhat weaker impact of task relevance on microsaccades as compared to stimulus frequency suggests that stimuli with an extremely low subjective probability could induce a microsaccadic inhibition even when task-irrelevant. This, for example, could be the case of stimuli which are task-irrelevant and are presented only once in the experiment, and which are known to elicit the so-called "novelty" P300 (Courchesne et al., 1975; see Friedman et al., 2001) Moreover, we can not exclude the possibility that sequence effects on microsaccadic inhibition are observed even when all stimuli are task-irrelevant. However, this seems unlikely, since we have shown that, when participants passively view the stimuli, even overall rare stimuli have little effect on microsaccadic behavior (Valsecchi et al., 2007).

Comparison with previous studies

In the current study we confirmed that the flashing of a visual stimulus induces an inhibition of microsaccade rate with a latency between 100 and 150 ms, which is then followed by a rebound. It is interesting that the peak rate of microsaccades in response to frequent non-target stimuli in the present study was much higher than in the study of Valsecchi et al. (2007). This might depend on the different eye-tracking systems used in the two studies. In particular, the system used in the present study would only have supported binocular recording with a lower image quality, and had a lower sampling frequency, being thus more noise-sensitive. Nonetheless, we basically replicated the finding that standard stimuli induce a double-phase inhibition-rebound modulation in the absolute microsaccade rate, and that the inhibition phase was longer and the rebound delayed in response to oddballs. In the study by Valsecchi et al. (2007) the rebound phase was almost abolished in response to oddball stimuli, whilst in the present study it was still clearly identifiable. We suspect that this might depend on two differences between the experimental procedures. First, the frequency of rare stimuli was raised from less than 10% in the Valsecchi et al. (2007) study to 20% in the present one, thus reducing the frequency effect. Second, in the present study the stimulus series were fully randomized, whereas Valsecchi et al. (2007) used pseudo-randomized series, in which at least 6 standard stimuli were presented between two oddballs. Hence, in the latter case the sequence

effect should be much stronger than in the present case, leading to a more pronounced inhibition of microsaccades.

Relationship between microsaccadic inhibition and P300 enhancement

To summarize, we showed that stimulus category, stimulus frequency and the previous stimulus sequence modulated the amplitude of P300, and that stimulus frequency and category interacted synergically. The same effects were also observed as far as microsaccadic inhibition is concerned, with the following difference: a stimulus category effect was significant in the latency of the peak microsaccade rate, whereas the other measures of microsaccadic inhibition only showed a reliable stimulus frequency effect. As stated above, in all of the previous studies on microsaccadic inhibition in the oddball task, it was not possible to isolate the peak microsaccade rate in response to targets. Therefore, it is still an open question whether or to what extent the different measures of microsaccadic inhibition we used in this paradigm indexed different aspects of stimulus processing in the oddball task.

A final question we addressed was whether microsaccadic inhibition and the P300 shared more than the sensitivity to the same experimental manipulations. Our data indicate that the functional relationship between the two phenomena presumably does not go beyond the common sensitivity to target probability. We were able to show that the across-participant correlation of stimulus category, frequency and sequence effects on P300 and microsaccadic inhibition was, if anything, weak. In general, P300 was enhanced and microsaccade rate was inhibited when an improbable target was encountered, but those participants who show a strong P300 enhancement may show a limited microsaccadic modulation and vice-versa. A similarly weak correlation between these two measures of brain activity comes from the analysis showing that, within a specified cell of our experimental design, the microsaccadic behavior was not predictive of the P300 amplitude and vice-versa. That means that the microsaccadic behavior was neither predictive of the P300 results at the trial level nor at the participant level. We can thus suggest that P300 and

microsaccadic inhibition could be used as two independent measures of the brain's response to rare targets in a visual oddball task.

The debate over the functional meaning of P300 has continued for four decades since this component was first reported (Sutton et al., 1965). In general, the most accepted view is the one that P300 is an index of context updating (Donchin, 1981; Donchin & Coles, 1988). This theoretical perspective considers P300 as a sign of the attentive restructuring of the stimulus representation in working memory when a new stimulus is encountered. Over the years, extensive evidence regarding the neural processes which could support the attention and memory operations related to P300 generation has been collected. Overall, the data seem compatible with the hypothesis that P300 reflects the neural inhibition which is functional to the focusing of activity on the processing of target stimuli (Polich, 2007). As far as the current evidence is concerned, we can propose that a similar mechanism also subtends the inhibition of microsaccades elicited by oddball stimuli, it is in fact clear that the inhibition of microsaccades is observed when a stimulus which requires a deeper restructuring of the task-related representation is encountered.

A deeper knowledge of the neural system involved in the generation of microsaccades and in their inhibition could also probably be helpful in disentangling the differences between this oculomotor effect and the enhancement of P300 observed in response to infrequent targets in the oddball task. The neural generators of P300 have been studied using intracranial recordings (Halgren et al., 1995a, 1995b, 1998; Roman et al., 2005). A widespread network of cortical areas in the parietal, frontal and temporal lobe, and subcortical areas such as the hippocampus and the amygdala were identified as generators of P3-related ERPs. These findings have been confirmed by fMRI studies (Clark et al., 2000; Stevens et al., 2000; Ardekani et al., 2002; Bledowski et al., 2004a, 2004b). As far as microsaccades are concerned, there is indirect evidence that they are triggered by fixational activity within the SC, mainly derived from the observation that saccades, which are known to be elicited by stimulation of the SC (Robinson, 1972), feature a kinematic

profile similar to the one of microsaccades (Zuber et al., 1965). Furthermore, it has been demonstrated that the inhibition of microsaccades in response to visual stimuli can be mediated by a cortical visual pathway sending afferences to the SC (Valsecchi & Turatto, 2007). Therefore we can not exclude that some of the cortical generators of P300 could also be responsible for the prolonged inhibition of microsaccades. There is currently no evidence of a complete anatomical segregation between microsaccade- and P300-generating structures, which could partially explain the lack of correlation between these two measures. On the contrary, it is well possible that the prolonged microsaccadic inhibition reflects inhibitory processes within cortical oculomotor areas, a mechanism similar to the one which has been proposed for P300 (Polich, 2007).

A number of factors are known to underlie individual differences in P300 amplitude. It is quite clear that P300 amplitude is partly genetically determined (e.g. Katsanis et al., 1987). Some of the biological determinants of P300 amplitude might be the size of specific cortical areas (Ford et al., 1994), the activity of catecholaminergic systems and the acute or chronic intake of common drugs (see Polich & Criado, 2006). Moreover, it has been suggested that P300 amplitude might correlate with personality attributes (e.g. Sternberg, 1994). Some of those factors, in particular those directly related to the anatomy of cortical areas, might influence P300 amplitude without being directly related to its antecedents, such as stimulus processing or neural inhibition. On the other hand, some characteristics specific to the oculomotor system might selectively influence microsaccadic inhibition. Nonetheless, it is still possible that both measures index the same processes.

To conclude, we propose that P300 enhancement and prolonged microsaccadic inhibition are two independent measures of the brain's processing of subjectively rare relevant stimuli. Further research is needed to clarify the extent to which these two measures are functionally related and the reasons why some participants show a pronounced P300 effect without showing a prolonged microsaccadic inhibition or vice versa.

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Figure Legends

Figure 1. Evolution of microsaccade rate in response to target (Panel A) and non target (Panel B) stimuli for the three levels of stimulus frequency (20%, 50% and 80%). The rate has been calculated in a 100 ms wide time window moving in 4.2 ms steps

Figure 2. Mean magnitude of peak microsaccade rate (Panel A), mean latency of peak microsaccade rate (Panel B), and mean microsaccade rate in the time WOI between 270 and 370 ms post stimulus onset (Panel C). Error bars are between-participant standard errors of the mean.

Figure 3. Evolution of ERP amplitude at Pz in response to target (Panel A) and non target (Panel B) stimuli for the three levels of stimulus frequency (20%, 50% and 80%).

Figure 4. Topographic maps of the ERP averaged over the time WOI (between 200 and 500 ms post stimulus onset) and over participants. Light gray indexes positive voltages. The centroparietal scalp distribution typical for the P300 component can be seen in all conditions. The location of electrode Pz (see Figure 3) is highlighted with an arrow.

Figure 5. Sequence effects on the probability of execution of a microsaccade in the time WOI (between 270 and 370 ms post stimulus onset) and on the amplitude of the P300 (average ERP amplitude at P3,Pz and P4 between 200 and 500 ms post stimulus onset). The plots refer to the stimuli presented in the 50% frequency condition. “Continued” indicates a sequence of 4 identical stimuli while “Discontinued” indicates a sequence with three identical stimuli followed by a different one (the current stimulus). Error bars are between-participants standard errors of the mean.

Figure 6. Between-participant correlation of microsaccadic and P300 effects of Stimulus Category (target-non target, Panel A), Stimulus Frequency (20%-80%, Panel B) and Sequence (Discontinued-Continued, Panel C). The measure for the microsaccadic effect is the probability of observing at least one microsaccade in the time WOI, the measure of P300 is the average ERP amplitude at P3, Pz and P4 between 200 and 500 ms post stimulus onset. Each point represents a participant. The dashed lines represent the linear regression of the microsaccadic effect sizes on the respective P300 effect sizes

Figure 7. Panel A: average P300 amplitude in 50% frequency Target epochs as a function of the presence of at least one microsaccade in the time WOI between 270 and 370 ms post stimulus

onset. Panel B: average probability of observing at least one microsaccade in the time WOI between 270 and 370 ms post stimulus onset in 50% frequency Target epochs as a function of P300 amplitude (median split classification). Error bars are between-participants standard errors of the mean.

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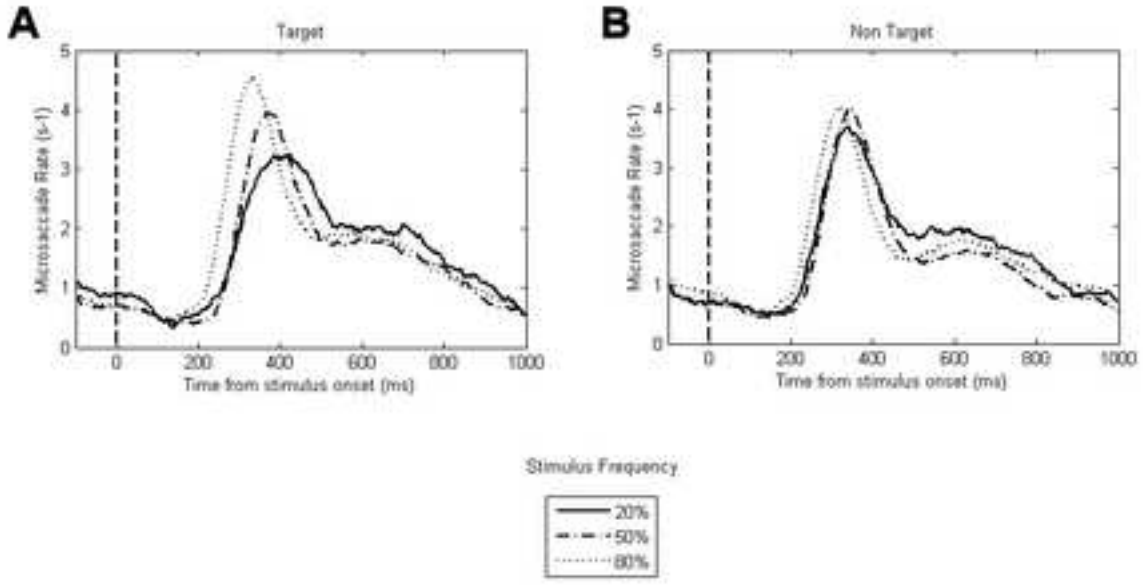

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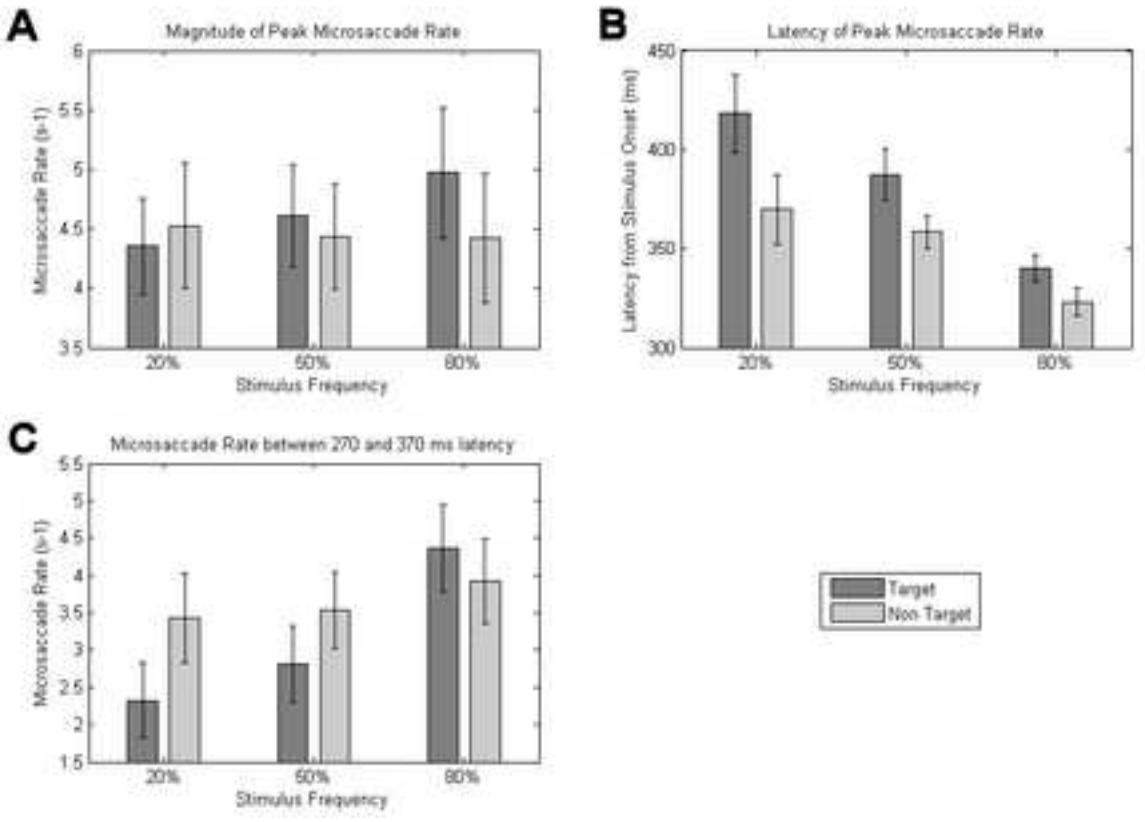

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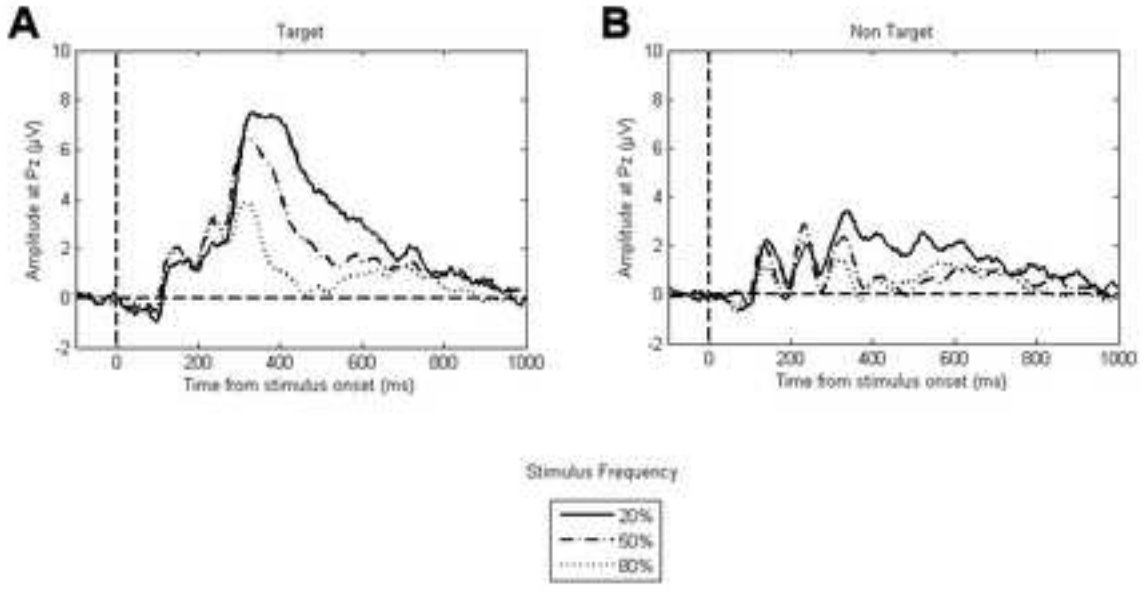

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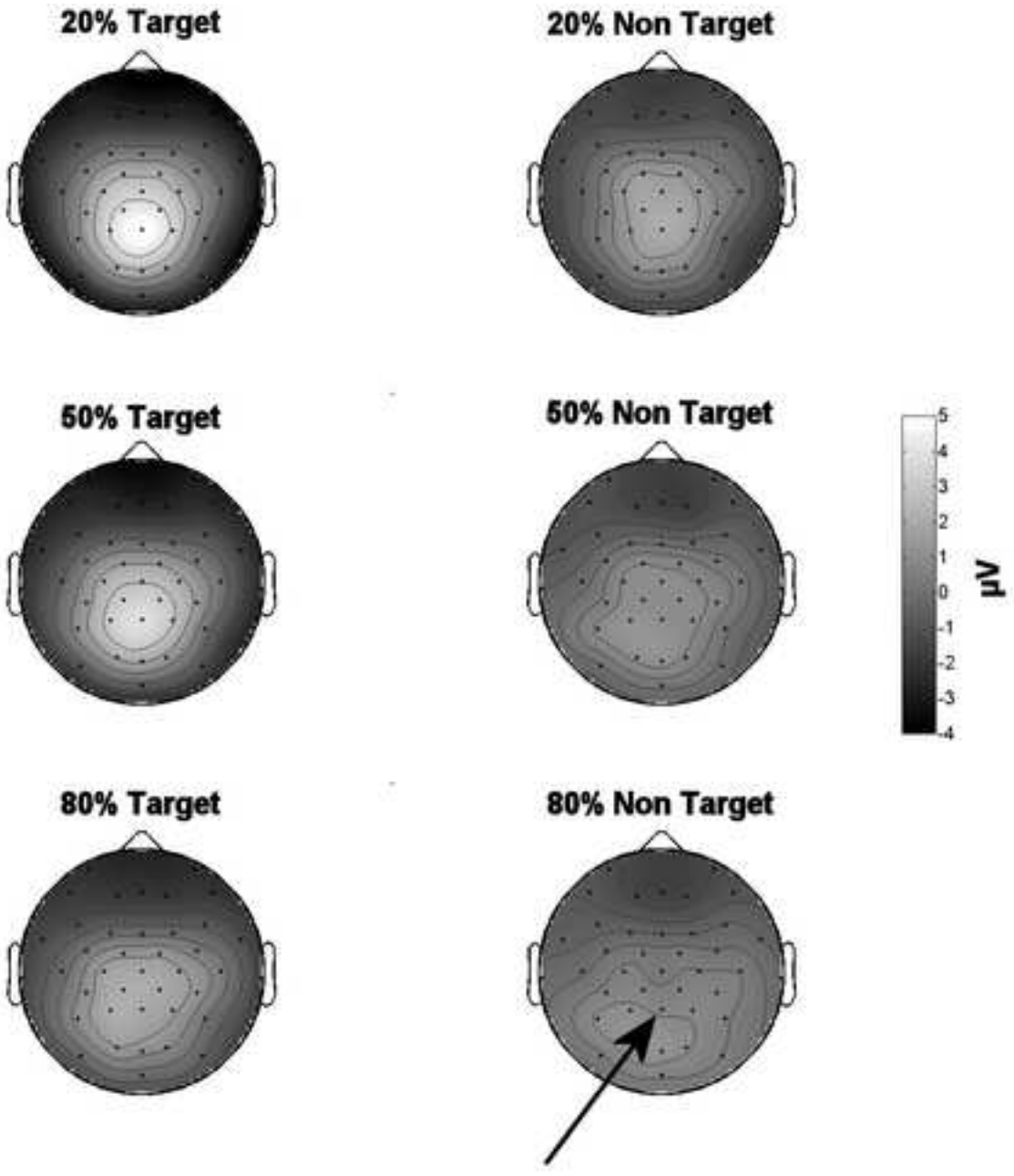

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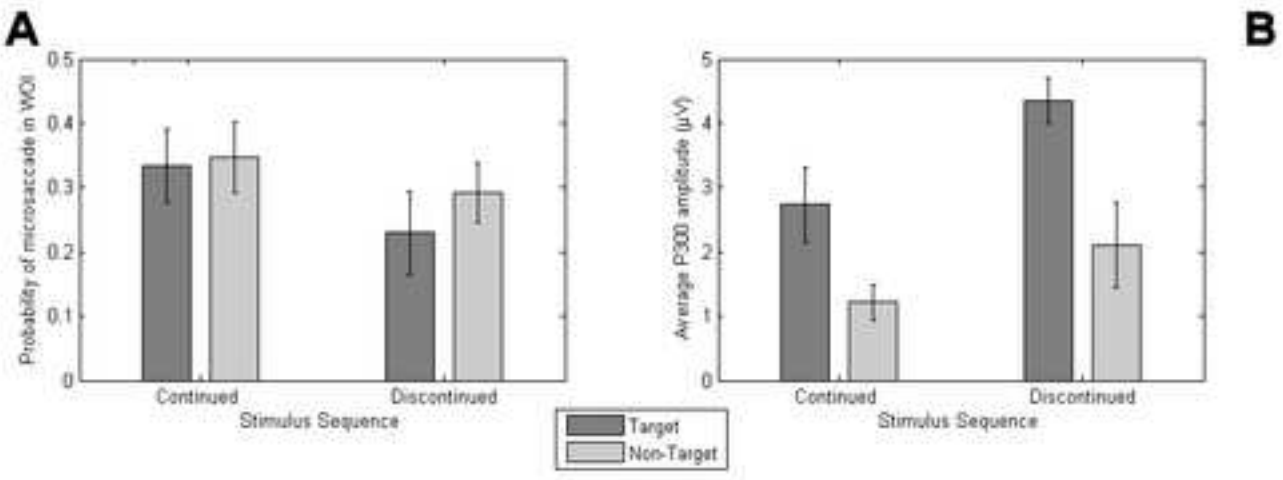

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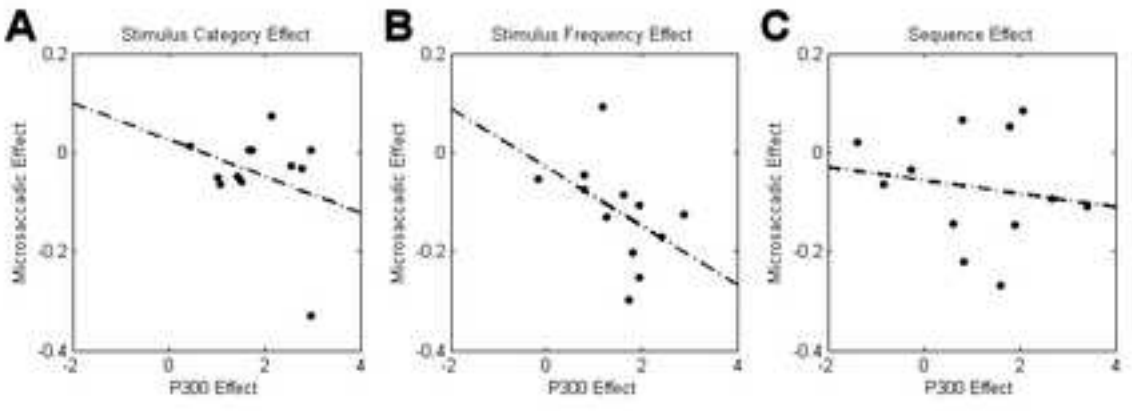

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