Behavioral/Cognitive

Saccadic Suppression Is Embedded Within Extended Oscillatory Modulation of Sensitivity

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Action and perception are intimately coupled systems. One clear case is saccadic suppression, the reduced visibility around the time of saccades, which is important in mediating visual stability; another is the oscillatory modulation of visibility synchronized with hand action. To suppress effectively the spurious retinal motion generated by the eye movements, it is crucial that saccadic suppression and saccadic onset be temporally synchronous. However, the mechanisms that determine this temporal synchrony are unknown. We investigated the effect of saccades on contrast discrimination sensitivity over a long period stretching over >1 s before and after saccade execution. Human subjects made horizontal saccades at will to two stationary saccadic targets separated by 20°. At a random interval, a brief Gabor patch was displayed between the two fixations in either the upper or lower visual field and the subject had to detect its location. Strong saccadic suppression was measured between −50 and 50 ms from saccadic onset. However, the suppression was systematically embedded in a trough of oscillations of contrast sensitivity that fluctuated rhythmically in the delta range (at ~3 Hz), commencing ~1 s before saccade execution and lasting for up to 1 s after the saccade. The results show that saccadic preparation and visual sensitivity oscillations are coupled and the coupling might be instrumental in temporally aligning the initiation of the saccade with the visual suppression.

Key words: **action and perception; contrast sensitivity; eye movements; saccadic suppression; sensorimotor integration; visual oscillations**

Significance Statement

Saccades are known to produce a suppression of contrast sensitivity at saccadic onset and an enhancement after saccadic offset. Here, we show that these dynamics are systematically embedded in visual oscillations of contrast sensitivity that fluctuate rhythmically in the delta range (at ~3 Hz), commencing ~1 s before saccade execution and lasting for up to 1 s after the saccade. The results show that saccadic preparation and visual sensitivity oscillations are coupled and the coupling might be instrumental in aligning temporally the initiation of the saccade with the visual suppression.

Introduction

Action and perception are tightly coupled in everyday life. Although these sensorimotor integration mechanisms are pervasive in the brain, they are still poorly understood. Brain oscillations might be important in binding and integrating sensorimotor information (Engel et al., 2001) via a shared internal oscillator that coordinates the two systems. Recent experiments have shown that voluntary movements can synchronize oscillations of visual performance (Tomassini et al., 2015; Benedetto et al., 2016). Therefore, action not only interferes with perception through a single transient suppression at around movement time (a phenomenon called "motor-induced suppression"), but rhythmically interacting long before and after action execution. These rhythmical interferences may result from endogenous brain rhythms synchronized by the intention-to-move signal. In this view, the motor-induced suppression might be a stronger manifestation of a more general sensorimotor modulation.

The best-known example of motor-induced suppression is saccadic suppression. Visually driven saccadic eye movements are known to produce strong visual suppression at the time of saccades (Latour, 1962; Burr et al., 1982, 1994; Diamond et al., 2000). This suppression is transient, but highly precise in time, starting ~50 ms

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before saccadic onset and maximal at saccadic onset (Diamond et al., 2000). This contrasts with the highly variable saccadic reaction time, which can be >80 ms (Carpenter, 1988; Gezeck et al., 1997; Drewes and VanRullen, 2011). The suppression is present also in conditions of no visual references and with simulated saccades (for review, see Morrone, 2014), demonstrating that it does not arise from visual masking.

Physiological and psychophysical studies (Burr et al., 1994; Leopold and Logothetis, 1998; Diamond et al., 2000; Reppas et al., 2002; Royal et al., 2006; Ibbotson et al., 2008; Knöll et al., 2011) have demonstrated that suppression is followed by an enhancement 100–200 ms after saccades. Both suppression and enhancement are independent of stimulus eccentricity (Knöll et al., 2011) and thus are unlikely generated by spatial attention, which shifts from fixation to saccadic target very early, ~300 ms before saccadic onset (Kowler et al., 1995; Deubel and Schneider, 1996; Rolfs and Carrasco, 2012). The perisaccadic suppression and the subsequent enhancement form a cycle of an oscillation at ~3 Hz, suggesting that they might be part of a more prolonged oscillation linked to saccadic preparation, similarly to the visual oscillation demonstrated in preparation of an hand action (Tomassini et al., 2015; Benedetto et al., 2016).

How the brain ensures that the suppression occurs at saccadic onset is unknown. An active mechanism ("efference copy" or "corollary discharge") probably mediates the suppression (Diamond et al., 2000; Wurtz, 2008; Morrone and Burr, 2009), which is an anticipatory signal (Bremmer et al., 2009). However, we do not know whether it is a temporally punctual signal informing vision about the incoming saccades or if it is a sluggish signal that builds up during the preparation of the saccade. Many voluntary action onsets are preceded by a readiness potential that have a gradual buildup over 500 ms or more (Deecke et al., 1969; Libet et al., 1983; Ball et al., 1999; Toma et al., 2002; Bozzacchi et al., 2012). It is likely that, for saccades as well, there is a long buildup of the corollary discharge signal. In all cases, oscillations during the motor preparation phase might be a means to propagate in time the corollary discharge signal and to keep a precise representation of movement onset. Interestingly, it has been shown recently that spatial attention (Hogendoorn, 2016) and temporal integration or segregation (Wutz et al., 2016) oscillate rhythmically and in synchrony with saccades, reinforcing this suggestion.

To test whether saccadic suppression and postsaccadic facilitation are part of an ongoing oscillatory modulation of vision, we measured contrast discrimination over a long perisaccadic period. Our results show that perisaccadic contrast sensitivity is modulated in the delta range (2–3 Hz) and, crucially, saccadic suppression and enhancement are embedded in phase with these oscillations.

Materials and Methods

Participants. Eight volunteers (three women; mean age: 28 ± 4 years, including author A.B.) performed the experiment. All had normal or corrected-to-normal vision. Participants gave informed consent in accordance with the Declaration of Helsinki.

Apparatus. The experiment was performed in a quiet, dark room. Subjects sat in front of a monitor screen (40 × 30 cm) at a distance of 57 cm with their heads stabilized by a chin rest. Stimuli were generated with the ViSaGe (Cambridge Research System) in MATLAB r2010a (The MathWorks) and presented on a CRT monitor (Barco Calibrator) with a resolution of 800 × 600 pixels and a refresh rate of 100 Hz. The 2D position of the left eye was monitored at 1 kHz with an EyeLink 1000 system (SR Research) with an infrared camera mounted below the screen. Horizontal eye position recordings were linearized by means of a linear calibration performed at the beginning of each session.

Stimuli and procedure. Two red square saccadic targets (0.25°), aligned vertically and separated horizontally by 20°, appeared at the beginning of the experiment and persisted until the end of the session. The stimulus was a horizontal sinusoidal grating (1 cpd, pedestal contrast 10%, random phase) presented for 10 ms in a 5° circular window with Gaussian smoothed edge on the center of the screen at 10° distance from both fixation points (Fig. 1*A*). The contrast was incremented in a Gaussian window in the upper or lower half of the circular stimulus. The luminance $l(x, y)$ is given by the following:

$$l(x, y) = \sin(x\omega + \varphi)\left(K + \Delta K\, e^{-\left[\left(\frac{x}{\sigma_x}\right)^2 + \left(\frac{y \pm \mu_y}{\sigma_y}\right)^2\right]}\right) G(x, y)$$

Where x and y and are the spatial coordinates, K is the pedestal contrast (10%), ΔK is the contrast increment, $\sigma_x = 1.5°$ and $\sigma_y = 0.75°$ are the space constants, $\mu_y = 1.25°$ is the spatial vertical offset, $\omega = 1$ c/° is the spatial frequency, φ is the random phase, and the function $G(x, y)$ is a circular step function of diameter 5° convolved with a Gaussian function of constant equal to 0.5° to smooth the stimulus–background edges.

Individual thresholds for contrast increment were obtained during a training session with a QUEST procedure. The contrast increment value that elicited ~75% correct responses was selected and kept constant within each block. To balance perceptual learning improvement, the contrast increment was adjusted slightly from block to block to maintain 75% correct response. For the entire duration of each session, participants made 20° horizontal saccades at will from one stationary saccadic target to the other (Fig. 1*A*). After each saccade, they were instructed to maintain fixation for at least 3 s before performing a new saccade toward the opposite saccadic target. At a random interval, the stimulus was displayed, with a probability of ~1 presentation every 3 saccades (interstimulus interval, ISI: 12 ± 5 s; fixation duration: 4.3 ± 0.4 s). This was established to avoid an automatic allocation of attention at the center of the screen for every saccade. The ISI was random and controlled by the experimenter to maximize the amount of collectable data. Subjects were required to detect by two-alternative force choice procedure (2AFC) a threshold contrast increment in either the upper or lower field and report the response verbally to the experimenter after the execution of the next saccade. Each session lasted for 5 min. Single participants performed, on average, 3 h of eye movement recordings over different days (37 ± 10 sessions per participant). At the high spatial frequencies tested in our paradigm (i.e., 1 cpd), saccadic suppression is small (Burr, Morrone, and Ross, 1994) and, for all participants, the 10% contrast pedestal was always over threshold. In the rare occasion in which subjects did not see the pedestal, they did not respond and that trial was discarded.

Data analysis. In an offline analysis, eye position traces were examined and individual saccade modeled with a trapezoidal function. A positive slope segment with two abutting constant segments was used to fit the saccade trace and derive the saccadic onset and offset. We included in further analysis only saccades with intersaccadic separation >3 s and that were fit well by the trapezoidal model ($R^2 > 0.99$, ~80% of the saccades). To disentangle the contribution of saccadic preparation from the saccadic execution to the contrast sensitivity data, we restricted the analysis only to a temporal window of ±1.5 s from the saccadic onset and pooled together the data for the leftward and rightward saccades. The eye movement recording traces were also analyzed automatically to detect microsaccades on the basis of speed and amplitude criteria (events faster than 20°/s and shorter than 2°). Subsequently, individual microsaccades were validated via visual inspection.

To evaluate the presence of oscillations, we performed several analyses at a group level, where the individual subject data were first binned and then averaged across subjects, and also by pooling all data together in a single dataset (hereafter termed the "aggregate observer") and subsequently binned. For the aggregate observer data, we computed the percentage of correct responses in 80 ms independent bins. The variability was assessed via a bootstrap procedure performed before the binning (1000 iterations, with replacement and SD of the bootstrap reported as SEM).

Spectral analyses were conducted using the fast Fourier transform (FFT). We computed the spectral variability via a bootstrap procedure

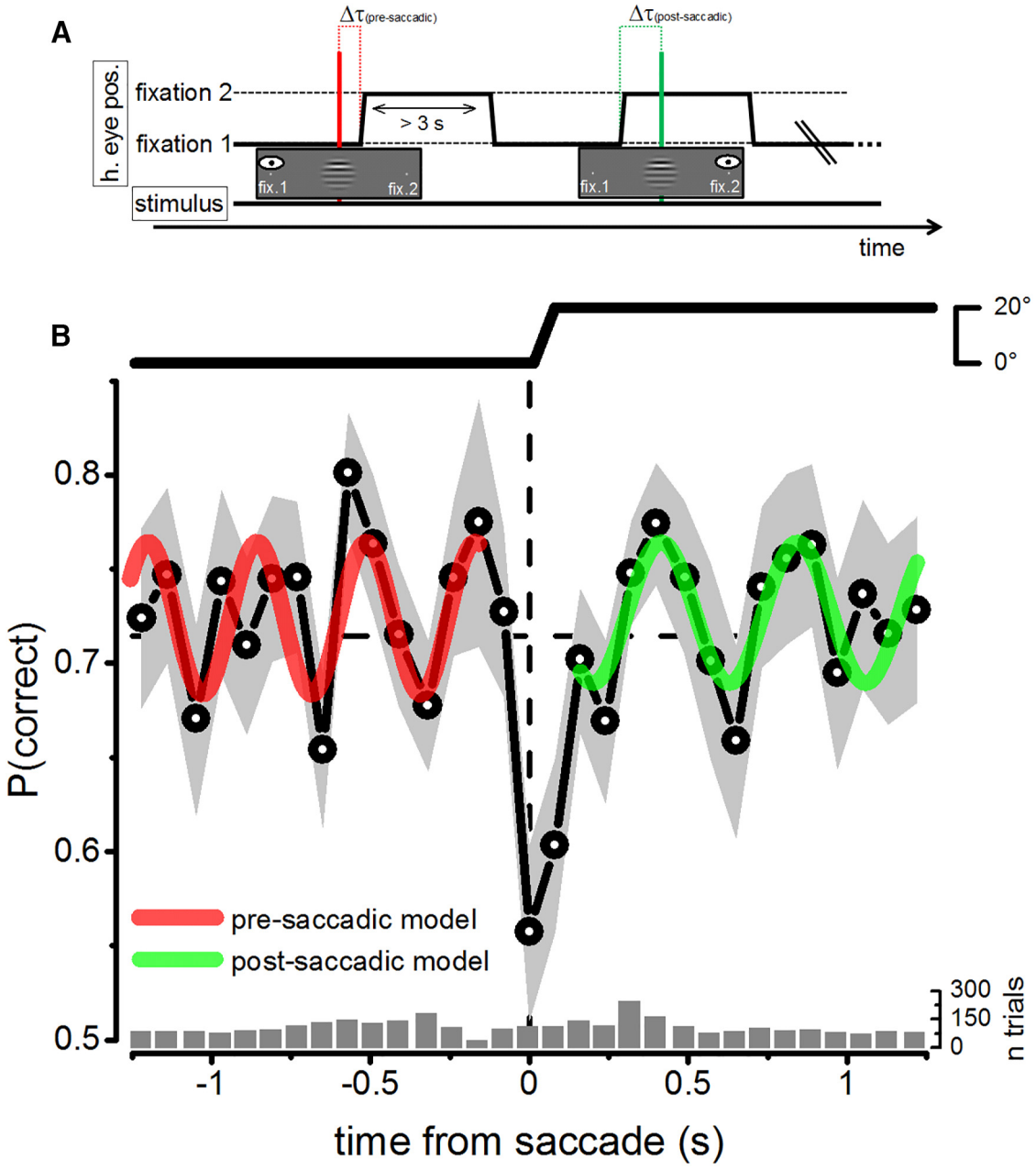

Figure 1. ***A***, Illustration of the experimental procedure. Participants performed saccades at their own pace to stationary saccadic targets (fixation 1 and fixation 2). At random delay from the saccadic onset (Δt), a brief Gabor stimulus with a contrast increment was presented in its upper or lower side and participants were asked to report the location of the increment. ***B***, Presaccadic and postsaccadic contrast discrimination performance as a function of time from saccadic onset (aggregate observer, pooling together the single trial data of eight individual subjects). The bar plot shows the number of observations for each bin (106 ± 38). The gray area represents ±1 SEM from bootstrapping; thick lines represent the best sinusoidal fit to the data for presaccadic responses (in red at around 3 Hz) and for postsaccadic responses (in green at around 2 Hz). Dashed vertical and horizontal lines report the time from saccadic onset and the median probability of correct response, respectively. Top trace shows the mean horizontal eye position.

for the aggregate observer (1000 iterations, with replacement). A 2D statistical significance test was run on the real and imaginary components for each frequency. A nonparametric two-tailed sign test was run to determine whether the distribution of data points was different from zero in at least one of the two components, implying that the 2D cloud of bootstrapped data was not centered at the origin. These analyses were conducted separately for the presaccadic response (−1.46 to 0.08 s), for the postsaccadic response (0.08–1.13 s) and for the whole signal (−1.46 to 1.13 s). For the whole signal, the relative *p*-values were corrected for multiple comparison using the false discovery rate (FDR) method (Benjamini and Hochberg, 1995). Note that both presaccadic and postsaccadic responses FFT excluded perisaccadic data within 80 ms from saccadic onset (perisaccadic gap of 80 ms).

In addition to the FFT, we used a different approach that requires that the oscillations are stationary in time. The presaccadic (from −1.38 s) and postsaccadic (to 1.22 s) time series were fitted separately with two independent sinusoidal functions. The best-fit statistical significance was evaluated using a bootstrap procedure on surrogate data obtained by randomly shuffling the time stamps (−1.38 to 1.22 s) of the single trial and then performing the standard binning procedure. The surrogate data were fit with a sinusoidal waveform of the same frequency as the original data, with amplitude and phase as free parameters. A one-tailed nonparametric bootstrap *t* test was run to assess whether the R^2 of the best fit of the data was statistically higher than the 95% of the R^2 distribution obtained from the bootstrapped surrogate data. To evaluate the effect of saccadic suppression on the oscillatory performance, we ran the same

analysis on both presaccadic and postsaccadic responses by extending the perisaccadic gap from 80 to 400 ms in steps of 80 ms. For the gap of 160 ms, we also ran a more stringent statistical test: the surrogate data were fit with a sinusoidal waveform with all free parameters (frequency varying between 1.5 and 6 Hz) and the real data best fit was compared against the best fit of the noise distribution independently of the frequency. Finally, we tested the statistical significance of all the possible sinusoidal models from 1.5 to 6 Hz (in steps of 0.1 Hz) using the same procedure described above that take into account the correction for multiple comparison. For both FFT and best-fitting analysis, phase angles are calculated with respect to the origin set at 0 ms and are relative to a cosine function.

To evaluate the oscillatory effect on single subjects, we computed the z-score performance in bins of 160 ms, overlapped by 90% with the adjoining one. The group mean z-score was fit by sinusoidal waveforms with the same procedure described above for the presaccadic and postsaccadic intervals. A one-tailed nonparametric bootstrap *t* test was run to assess whether the R^2 of the best fit of the data was statistically higher than the 95% of the R^2 distribution obtained from the bootstrapped surrogate data for each individual subject.

Results

Subjects made saccades at their own pace to stationary saccadic targets. At random times, we sporadically presented a brief Gabor stimulus with a contrast increment that subjects had to localize in a 2AFC (Fig. 1*A*). We measured how contrast discrimination accuracy varied as a function of stimulus presentation from the saccadic onset. Figure 1*B* shows the result obtained by pooling together the data from all subjects (aggregate observer). A strong perisaccadic suppression is evident, being maximal at saccadic onset, similar to that commonly observed for visually driven saccades. Subjects performed nearly at chance level for perisaccadic stimuli (±40 ms) and ~75% away from the saccade. However, for times long before and long after the saccade, performance was not constant, but rather oscillated with ~10% of modulation. To assess quantitatively the nature of these oscillations, we best-fitted the performance time course with sinusoidal waveforms. To avoid possible biases in the frequency and phase estimation introduced by the strong minima of saccadic suppression, we fitted independently the presaccadic and postsaccadic responses, excluding the ±160 ms around the saccadic onset (as detailed in the Materials and Methods). The best sinusoidal model was obtained at a frequency ~3 Hz for the presaccadic performance (2.9 ± 0.4 Hz, mean and 95% confidence bounds; Fig. 1*B*, red curve) and ~2 Hz (2.3 ± 0.4 Hz, mean and 95% confidence bounds; Fig. 1*B*, green curve) for the postsaccadic performance.

To evaluate the significance of both of these models, we compared the R^2 values of these fits with the distribution of the R^2 obtained by fitting a sinusoidal waveform of the same frequency to surrogate data (obtained by shuffling the timestamps of each trial). Figure 2*A* shows the results of this analysis for the presaccadic model (Fig. 2*A*, left) and the postsaccadic model (Fig. 2*A*, right). For both models, the goodness of fit was statistically higher than that expected from a noise distribution (presaccadic model: $R^2 = 0.49$, $p = 0.007$; postsaccadic model: $R^2 = 0.62$, $p = 0.005$).

Similar results were obtained for a range of perisaccadic gaps between 80 and 400 ms in five steps of 80 ms (Fig. 2*C*). All of the best sinusoidal fits were statistically significant, with the exception of the presaccadic model with 400 ms gap that was marginally significant (*p*-values for different perisaccadic gaps for presaccadic and postsaccadic responses, respectively: gap = 0.08 s, $p = [0.009\ 0.032]$; gap = 0.16 s, $p = [0.007\ 0.005]$; gap = 0.24 s, $p = [0.028\ 0.007]$; gap = 0.32 s, $p = [0.044\ 0.02]$; gap = 0.4 s, $p = [0.07\ 0.026]$). We also evaluated the significance of the oscillation with a more stringent test comparing the R^2 of the best fitting sinusoidal model against the R^2 distribution of the best fitting of the surrogate data across all possible frequencies. Figure 2*B* shows that, for both models, the R^2 obtained from the aggregate observer data was statistically higher than the R^2 surrogated distribution (presaccadic response: $p = 0.041$; postsaccadic response: $p = 0.019$) for the best fit across all frequencies. For the presaccadic data, no other frequency of the model in the range between 1.5 and 6 Hz (in steps of 0.1 Hz) survived statistical significance with this stringent test (Fig. 2*B*, left), whereas for the postsaccadic data, oscillations were significant ($p < 0.05$) in the range between 2.2 and 2.5 Hz (with a peak of significance at 2.3 Hz). No single frequency model fitted both periods significantly, indicating the presence of long-lasting delta oscillatory modulation of contrast discrimination of different frequencies for the presaccadic and the postsaccadic range.

Subjects saccaded at their own pace. In principle, the oscillations observed before the saccadic onset might have been related to the execution of the previous saccade or even to the spurious retinal motion generated by the previous saccadic execution. To control for this possible confound, we aligned the responses to the previous saccade excluding the response to stimuli that were closer than ~100 ms to the following saccade onset. Figure 3 shows that the performance after the first 1.5 s from saccadic onset was modulated randomly around the average value of 75%. The best fitting of the data of Figure 3 (between 0.08 and 3 s) with a single sinusoidal function was not statistically significant ($R^2 = 0.07$, $p = 0.31$) for any frequency in the range 1.5 and 6 Hz, whereas the modulation of the first second was qualitatively well captured by the best fit of the postsaccadic modulation of Figure 1 (Fig. 3, green curve). This suggests that presaccadic oscillations were not related to the previous saccade, but were genuinely phase locked to the preparation of the upcoming saccade. Interestingly, we never observed a linear increase in the overall performance with time, suggesting no change in attentional allocation to the time of stimulus appearance (hazard rate; Nobre et al., 2007).

Having confirmed that a separation of 3 s is sufficient to disentangle the effect of the previous saccade from that of the following one, we performed a spectral analysis on the whole signal of Figure 1*B* (without perisaccadic gap). Two main frequency peaks were detected at ~2 and 3 Hz in the FFT (Fig. 4), confirming the fitting results of Figure 1*B*. We ran the 2D spectral statistical analysis for each frequency (see Materials and Methods) and the obtained *p*-values were corrected for multiple comparisons using an FDR procedure. A two-tailed sign test showed that only these two components were statistically significant (1.9 Hz: $p = 0.006$ after FDR correction: $p = 0.048$; 3 Hz: $p = 0.004$ after FDR correction: $p = 0.048$).

Figure 5 shows a similar spectral analysis performed separately for the presaccadic responses (Fig. 5*A*, red curve, interval −1.46 to −0.08 s) and the postsaccadic responses (Fig. 5*A*, green curve, interval 0.08 and 1.13 s), with gap of 80 ms. Amplitude peaks were present at 2.8 and 1.8 Hz for the two intervals, respectively. The bootstrapped amplitude and phase evaluations clustered away from the zero amplitude, indicating that the oscillations were statistically significant (2.8 Hz: $p = 0.014$, Fig. 5*B*; 1.8 Hz: $p = 0.012$; Fig. 5*C*) and different from random noise. The average phase, computed with respect to the origin at 0 ms, of these significant frequencies (Fig. 5*B*,*C*, black vectors) were 2.81 ± 0.4 and 2.23 ± 0.45 rad, respectively.

An important question is whether the period of saccadic suppression in embedded in phase with the oscillation. Saccadic suppression is usually reported to be maximally between 0 and 30 ms

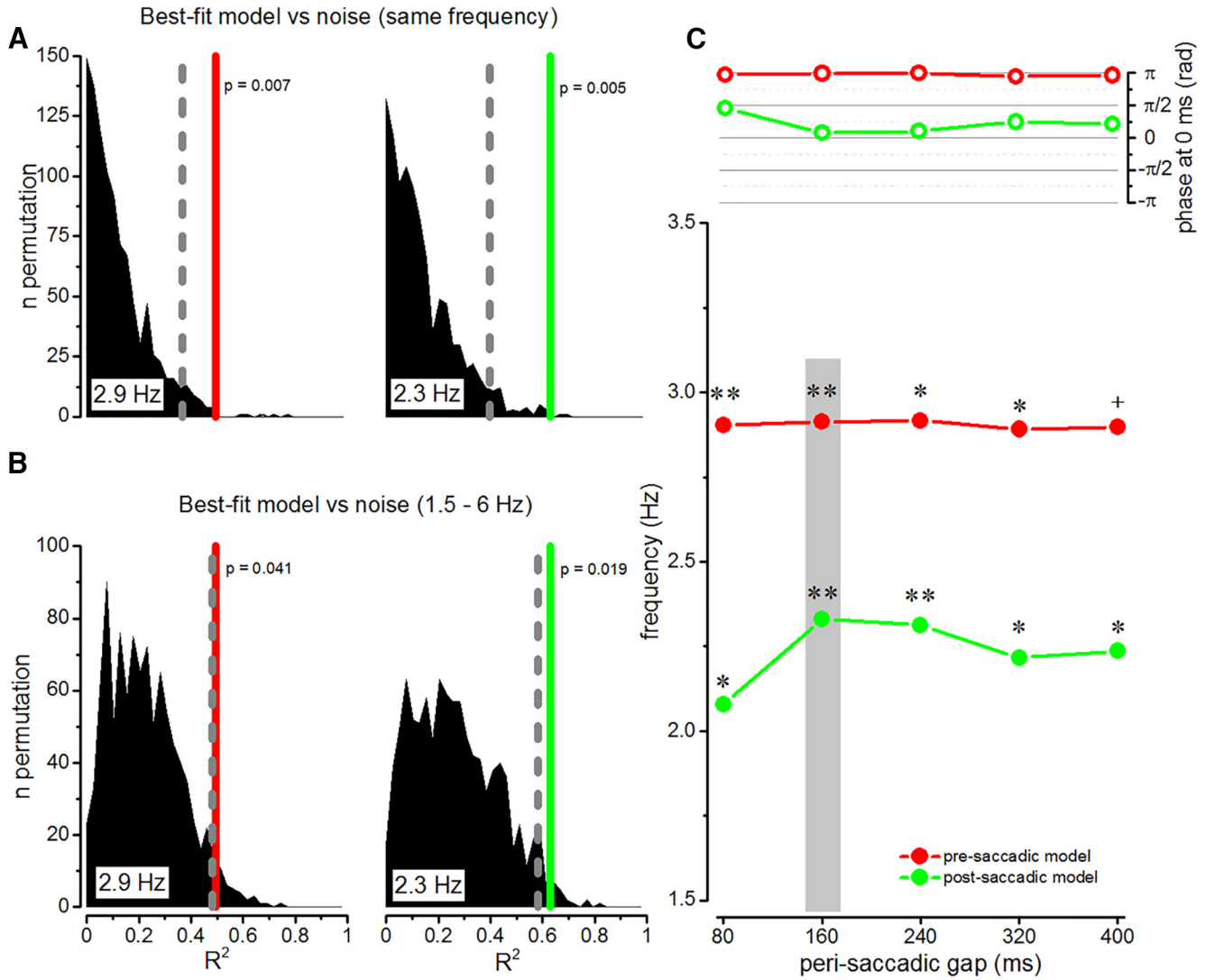

Figure 2. ***A***, R^2 distribution obtained by fitting the random shuffled data with the sinusoidal functions from Figure 1 with amplitude and phases as free parameters (perisaccadic gap set to 160 ms); thick lines mark the R^2 for the presaccadic model (red, 2.9 Hz; $p = 0.007$) and the postsaccadic model (green, 2.3 Hz; $p = 0.005$). Dashed lines mark 0.95 probability; ***B***, Same analysis as reported in ***A***, but with an R^2 permuted distribution obtained by best fitting the random shuffled data with frequency as a free parameter. The best fit was statistically higher than noise level for both presaccadic response (red, $p = 0.041$) and the postsaccadic response (green, $p = 0.019$). ***C***, Best fitting frequency and phase of the aggregate data as function of different perisaccadic gaps Phase is calculated with respect to a 0 ms origin and is reported for a cosine function. Asterisks indicate significant points following the procedure in ***A*** ($0.1 > + > 0.05 > * > 0.01 > ** > 0.001$).

from saccadic onset (Diamond et al., 2000; Michels and Lappe, 2004; Knöll et al., 2011). This time is very close to the estimated time by the oscillation for the FFT analysis: the arrival time of the minimum of the 2.8 Hz presaccadic oscillation falls around the time of saccadic onset (19 ± 23 ms). Similarly, the arrival time of the minimum of the postsaccadic 1.8 Hz oscillation is delayed by ~81 ± 40 ms from saccadic onset. Interestingly, this time corresponds to the bin including saccadic offset (mean saccadic duration was 62 ± 6 ms), suggesting that saccadic suppression is embedded in phase with the presaccadic oscillations and that presaccadic and postsaccadic oscillation minima straddle the onset and offset of the saccades.

Oscillations might result from periodic microsaccades, that affect vision in a similar way to normal saccades, producing visual suppression and enhancement (for review, see Rucci and Poletti, 2015). It is known that fixational eye movements have little effect on low spatial frequencies (Rucci et al., 2007; Rucci and Poletti, 2015) and may not contribute to the visibility of our stimulus. However, to confirm that microsaccades were not relevant to the oscillation of sensitivity, we measured the average frequency of microsaccades across subjects in bins of 20 ms (Fig. 6). The temporal distribution of microsaccades showed a peak at ~120 ms after saccadic execution, followed by a nearly constant rate with negligible (<0.5%) modulation.

Analysis of aggregate observer data is generally robust and relatively unaffected by differences in the amount of data for different time bins and participants. However, it conceals individual differences, so the results could be driven by a few subjects with strong oscillations. To rule out this possibility, we calculated the group mean performance across the individual subjects (Fig. 7*A*). The group average analysis gave very similar results to the aggregate observer data, with significant oscillations at 3.1 and 2 Hz for presaccadic and postsaccadic responses, respectively (presaccadic: 3.1 ± 0.12 Hz, $p = 0.008$; postsaccadic: 2 ± 0.11 Hz, $p = 0.01$; Fig. 7*B*). Two individual subjects (Fig. 7*C*) also showed oscillations at frequencies similar to those measured in the aggregate observer data.

Discussion

The visual effects of saccades are traditionally analyzed within a narrow temporal window of a few hundred milliseconds around saccadic onset. Here, we analyzed the temporal dynamics of contrast sensitivity in a 3 s window centered at saccadic onset. Our data replicated the well known effects of saccadic suppression and saccadic facilitation. Crucially, they show strong contrast sensitivity oscillations in the delta-range from ~1 s before to 1 s after saccadic execution, with saccadic suppression and saccadic enhancement embedded in the phase with these oscillations. The presaccadic oscillation was slightly faster than the postsaccadic one (2.9 vs 2.3 Hz, respectively) and slower than the oscillations measured for hand movements (reported at ~6 Hz; Tomassini et al., 2015; Benedetto et al., 2016) for the same visual task.

Visual contrast sensitivity oscillations began at least 1 s before saccadic onset, lasting for up to 1 s after. Given that the saccades were not visually driven (subjects saccaded freely between two stationary small targets), we can exclude that oscillations were initiated by a transient appearance of the saccadic target. It is also unlikely that oscillations were generated by the transient retinal motion produced by the eye movement (Campbell and Wurtz, 1978; Diamond et al., 2000; Knöll et al., 2011). Aligning all the responses with the previous saccadic onset, we observed oscillations only around the first second. Thereafter, the oscillations disappeared gradually, confirming a decay with time from saccade execution (Wutz et al., 2016). This pattern of results indicates that the oscillations are synchronized with motor preparation, not to the transient appearance of perceptual stimuli, a phenomenon demonstrated by previous work (Landau and Fries, 2012; Romei et al., 2012). It is also unlikely that oscillations are related to the spatial attentional allocation toward the stimulus position because more than half of saccades were made without the presentation of the stimulus and we did not detect any hazard rate (Nobre et al., 2007). We can also dismiss a role of microsaccades. In agreement with the evidence that fixational eye movements have little effect on the visibility of the low spatial frequency stimuli (Rucci et al., 2007; Rucci and Poletti, 2015) used here, we show that their rate is constant over time with the exception of microsaccades that correct the physiological saccadic overshooting (Kapoula et al., 1986).

Consistent with Tomassini et al. (2015), the cyclic modulation of visual contrast sensitivity observed here is phase locked with action planning (or the intention to move), corroborating the hypothesis that oscillations play a key role in binding action and perception (Engel et al., 2001; Wood et al., 2015; Gupta and Chen, 2016). This sensorimotor synchronization may be mediated by a time-keeping mechanism shared between visual and motor processes. It is well known that humans are extremely good at producing repetitive movements (Stevens, 1886), including saccadic eye movements (Joiner and Shelhamer, 2006), and we can perform saccades with precise timing also for intervals over seconds. The close link between time mechanisms and sac-

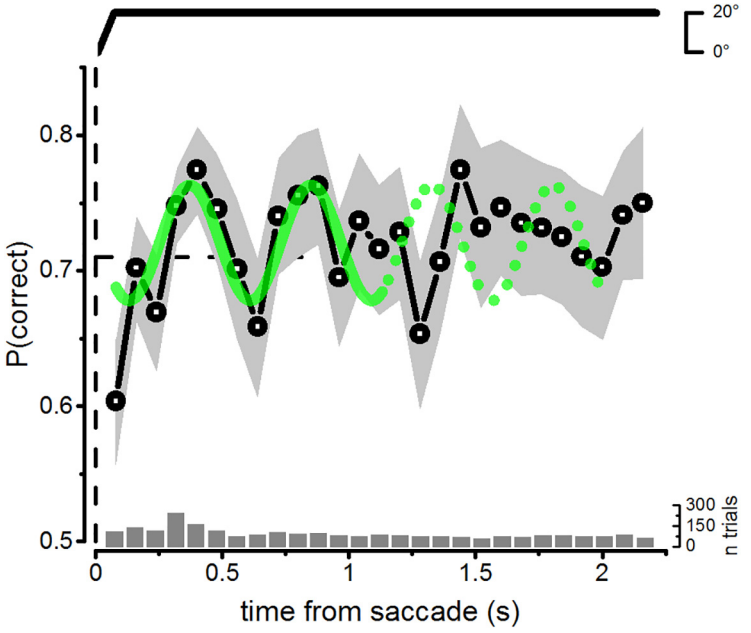

Figure 3. Postsaccadic contrast discrimination performance as function of delay from the onset of the previous saccade. The gray area represents ±1 SEM from bootstrapping; thick line represents the best sinusoidal fit of Figure 1*B* (green curves); dotted line shows that, after about the first second, the model does not fit the dataset well. Note that the first 1.5 s corresponds to the postsaccadic data of Figure 1. Data from saccades with latency <3 s are not included. Dashed vertical and horizontal lines report the time from saccadic onset and the median probability of correct response, respectively. Top trace is the mean horizontal eye position.

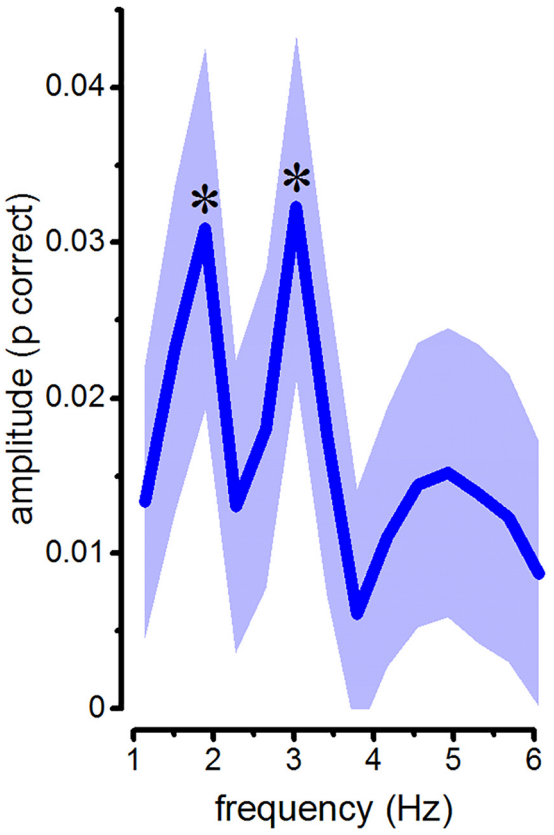

Figure 4. FFT spectral analysis for the aggregate observer. Amplitude spectra of the signal ±1 SEM are shown. The local maxima at 1.9 and 3 Hz are the only to reach statistical significance (1.9 Hz: $p = 0.006$; 3 Hz: $p = 0.004$). Asterisks indicate the significance after FDR correction ($0.1 > + > 0.05 > * > 0.01$).

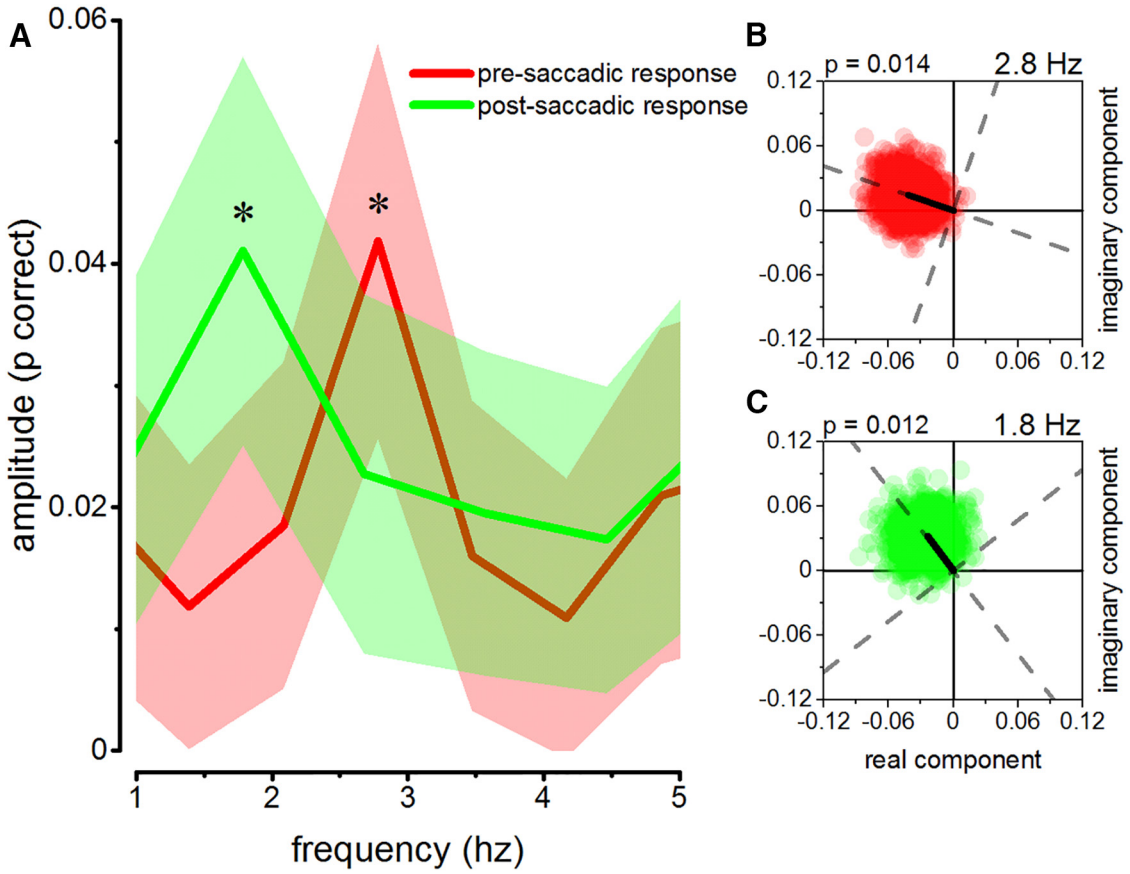

Figure 5. **A**. FFT mean amplitude spectra ±1 SEM for presaccadic responses (red curves) and postsaccadic responses (green curve). ***B***, ***C***, 2D bootstrap analysis performed for the presaccadic response at 2.8 Hz (***B***, $p = 0.014$) and the postsaccadic response at 1.8 Hz (***C***, $p = 0.012$). The black vectors show the average amplitudes and phases at 2.8 and 1.8 Hz. The phases are calculated respect to 0 ms origin and reported for a cosine function. Asterisks indicate significance ($0.1 > + > 0.05 > * > 0.01 > ** > 0.001$).

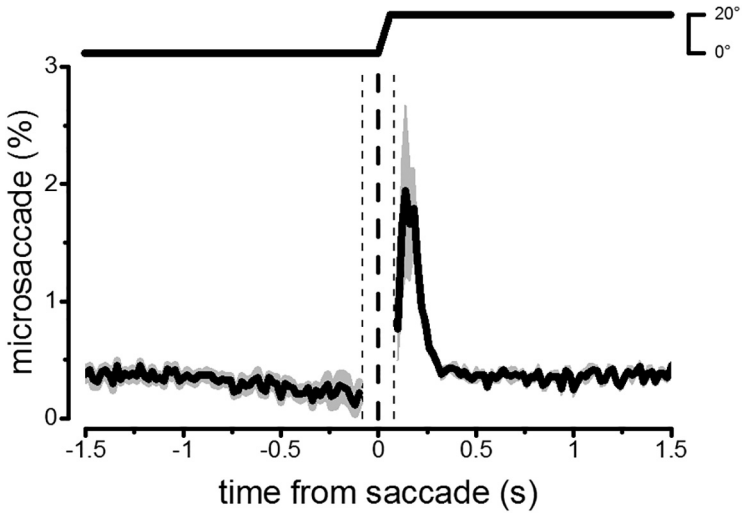

Figure 6. Horizontal microsaccadic frequency and SEM as a function of time from saccadic onset for group-level data ($n = 8$) calculated in bin of 20 ms. The thick vertical line represents the saccadic onset, thin vertical lines delimit perisaccadic boundaries. The microsaccadic rate decays rapidly in the first 120 ms after saccadic onset, being nearly constant before and after saccadic execution. The top trace shows a mean horizontal eye position.

cades is demonstrated by the profound alteration of time perception for perisaccadic stimuli (Yarrow et al., 2001; Morrone et al., 2005; Binda et al., 2009). Interestingly, saccadic reaction times to abrupt visual stimuli are highly variable and can be predicted by the phase of ongoing brain oscillations, as has been observed for many other visual functions (VanRullen and Koch, 2003; Busch et al., 2009; Dugué et al., 2011; Milton and Pleydell-Pearce, 2016). Therefore, a system based on endogenous oscillations synchronized by an internal clock could produce the close temporal alignment of perceptual and motor events at the face of the erratic saccadic reaction time.

Here, we show that saccades are synchronous with long-lasting visual delta oscillations at ~3 Hz. We scan the world at a similar rate of ~3 saccades/s, thought to be optimal timing given the temporal dynamics of visual perception (Findlay and Gilchrist, 2003; Morrone and Burr, 2009). Interestingly, hand-onset actions are also synchronous with oscillations of visual contrast thresholds (Tomassini et al., 2015; Benedetto et al., 2016). However, for hand action, visual oscillations are at higher frequencies (~6 Hz) and these frequencies correspond to the maximum hand movement rate while maintaining accurate timing (Repp, 2005). All of these results suggest that the motor and sensory circuitry oscillates in synchrony in the brain and that these periodicities may be orchestrated by effector-specific clocks. Our results are consistent with the hypothesis of a shared internal clock

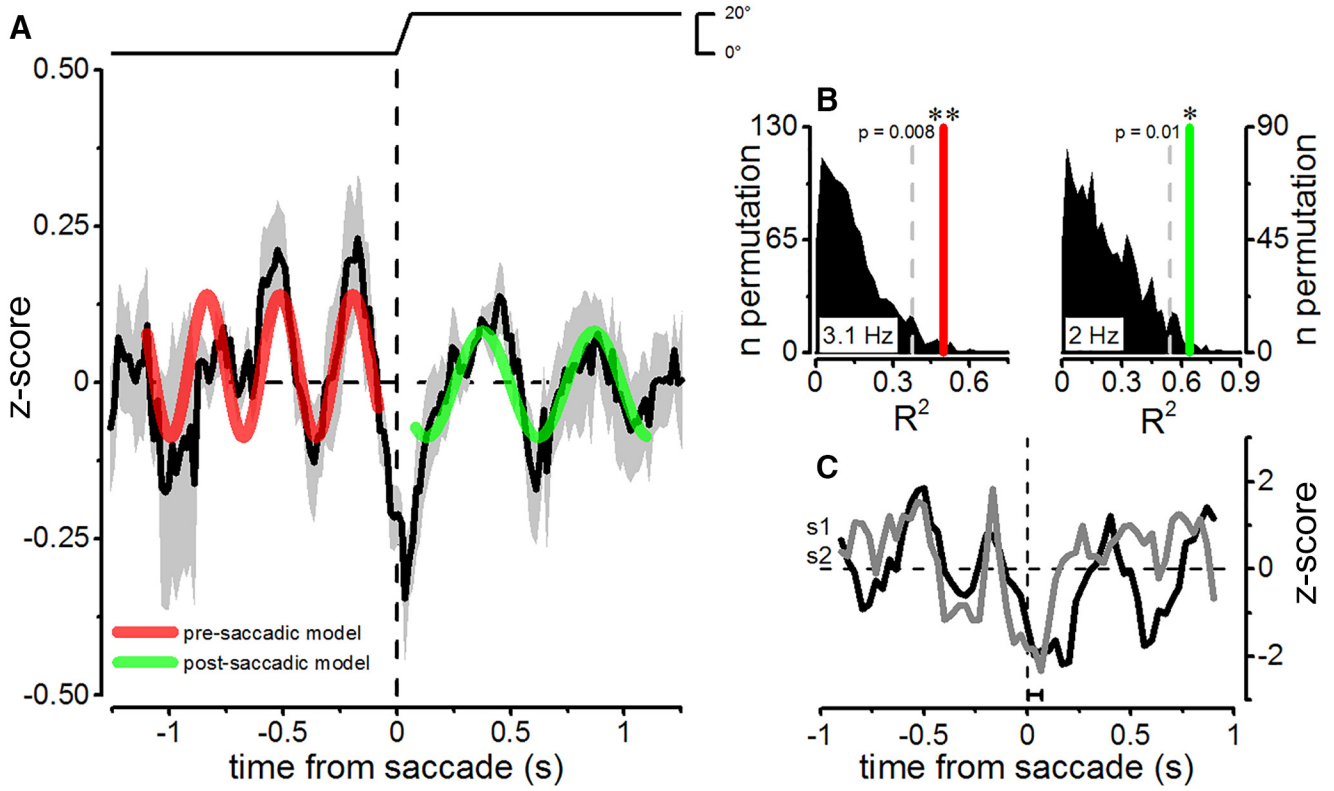

Figure 7. ***A***, z-scores averaged across subjects ($n = 8$) as a function of delay from saccadic onset. The gray area represents ±1 SEM; thick lines represent the best sinusoidal fit to the data for presaccadic responses (in red at 3.1 Hz) and for postsaccadic responses (in green at 2 Hz). Dashed vertical and horizontal lines indicate the time from saccadic onset and the median probability of correct response, respectively. The top trace shows mean horizontal eye position. ***B***, R^2 distribution obtained by fitting the random shuffled data with the sinusoidal functions from ***A*** with amplitude and phases as free parameters. Dashed lines mark 0.95 probability; thick lines mark the R^2 for the presaccadic model (left, 3.1 Hz, $p = 0.008$) and the postsaccadic model (right, 2 Hz, $p = 0.01$). Asterisks indicate significant points ($0.05 > * > 0.01 > ** > 0.001$). ***C***, z-scores as a function of time from saccadic onset for two representative subjects.

between action and perception, which helps to maintain visual stability and coordination between these two systems. This sensorimotor hypothesis is also corroborated by the fact that both saccadic suppression and saccadic enhancement are embedded in phase with visual oscillations: oscillations might play a key role in precisely inhibiting/enhancing vision according to the motor state of the subject.

The major motor mechanism that informs the visual brain about the upcoming eye movement is a corollary discharge signal (Sommer and Wurtz, 2008). Electrophysiological evidence indicates that corollary discharge signals takes time to emerge and can reverberate for several hundreds of milliseconds (Hanes and Schall, 1996; Sommer and Wurtz, 2006). It is still unknown whether this motor signal is short and punctate in time or long and rhythmically modulated. A punctate corollary discharge may lock the ongoing visual oscillation directly or, conversely, the corollary signal itself may be oscillatory, producing the modulatory effect on visual performance observed in our data. Both models can explain the oscillation observed here. However, both models imply that the corollary discharge is active 1 s before saccade, not just 200 ms as is commonly assumed by current research on eye movements (Wurtz, 2008; Morrone, 2014). An anticipatory corollary discharge signal has been already proposed as a mechanism to explain the complex changes in oscillatory activity during eye movements. In monkeys, an increase of high-frequency power and phase reset of low-frequency oscillations have been observed after the execution of eye movements (Rajkai et al., 2008; Bosman et al., 2009) and was suggested to be responsible for the transient perceptual enhancement measured psychophysically at the new fixation onset (Dorr and Bex, 2013). The corollary discharge signal, generated at an early stage during motor preparation, could thus keep the ongoing activity in visual areas phase locked.

The early emergence of corollary discharge is similar to the readiness potential observed in other voluntary actions (Deecke et al., 1969; Libet et al., 1983; Ball et al., 1999; Toma et al., 2002; Bozzacchi et al., 2012). A long-lasting active sensing process that starts ~1 s before saccadic onset might be important to prepare and organize the visual system for spatial and temporal patterns of visual inputs linked directly to oculomotor events (Schroeder et al., 2010; Wutz et al., 2016). Consistent with this interpretation, a recent study (Wutz et al., 2016) has shown that saccadic onset locks the phases of 3 Hz oscillations for temporal integration or segregation of visual information. Here, we observed similar frequencies for a different, but equally important property: saccadic suppression. Attention oscillates rhythmically in synchrony with saccades, but at a higher frequency than that observed here (at 4 Hz; Hogendoorn, 2016), for a period much closer to the saccadic onset (~500 ms), and with a strong hazard rate. Inter alia, that narrower time window is consistent with the shift of the allocation of spatial attention to saccadic target that it known to take place ~300 ms before the saccadic onset. None of these phenomena were observed in the present data, suggesting that our results are linked to early visual processing mechanisms. Spurious retinal motion induced by the eye movement can modulate sensitivity, particularly postsaccadically (Knöll et al., 2011). It is reassuring that our data and those of Hogendoorn (2016), who used very different visual references (minimal for

the present study and very strong for Hogendoorn, 2016), show similar postsaccadic oscillation, although at different frequencies and with different temporal decay. This reinforces the suggestion that the postsaccadic oscillation are not synchronized only by perceptual signals, as demonstrated in previous studies (Landau and Fries, 2012; Fiebelkorn et al., 2013).

In conclusion, our data are consistent with the idea of a supramodal neuronal timing mechanism that synchronizes visual and motor oscillations. Motor oscillations determine the time of the saccade and visual oscillations determine the time of saccadic suppression or enhancement. Oscillations may have the crucial role of coordinating visuomotor information, helping, not only in maintaining visual stability, but also in defining our sense of agency. This may result in actions being constrained to start around particular phases of endogenous oscillations. This would imply that we are not free to move the eyes when we want: the possible onset times may be predetermined by internal mechanisms long time before the actual movement, as proposed previously by Libet et al. (1983). However, further experiments are necessary to verify the fascinating idea of an oscillatory free will.

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