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MOTION CONTRAST IN PRIMARY VISUAL CORTEX: A DIRECT COMPARISON OF SINGLE NEURON AND POPULATION ENCODING

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ABSTRACT

Features from outside the classical receptive field (CRF) can modulate the stimulus-driven activity of single cells in the primary visual cortex. This modulation, mediated by horizontal and feedback networks, has been extensively described as a variation of firing rate and is considered the basis of processing features as, for example, motion contrast. However, surround influences have also been identified in pairwise spiking or local field coherence. Yet, evidence about co-existence and integration of different neural signatures remains elusive. To compare multiple signatures, we recorded spiking and LFP activity evoked by stimuli exhibiting a motion contrast in the CRFs surround in anesthetized cat primary visual cortex. We chose natural-like scenes over gratings to avoid predominance of simple visual features, which could be easily represented by a rate code. We analyzed firing rates and phase-locking to low-gamma frequency in single cells and neuronal assemblies. Motion contrast was reflected in all measures, but in semi-independent populations. Whereas activation of assemblies accompanied single neuron rates, their phase relations were modulated differently. Interestingly, only assembly phase relations mirrored the direction of movement of the surround and were selectively affected by thermal deactivation of visual inter-hemispheric connections. We argue that motion contrast can be reflected in complementary and superimposed neuronal signatures that can represent different surround features in independent neuronal populations.

INTRODUCTION

The firing rate of a neuron in primary visual cortex is driven mainly by simple stimulus features like orientation or movement direction of a contour within its classical receptive field (CRF). However, neuronal firing rates can also be modulated by the context in the CRF's surround (Angelucci & Bressloff, 2006; Blakemore & Tobin, 1972; Gilbert, 1992; Gilbert & Wiesel, 1990; Hubel & Wiesel, 1965; Nelson & Frost, 1978). Introducing a surround mostly provokes rate suppression (Cannon & Fullenkamp, 1991; Henry *et al*., 2013; Knierim & van Essen, 1992; Nelson & Frost, 1978; Schwabe *et al*., 2010; Shushruth *et al.*, 2013), but increases in firing rate are also observed (Hupé *et al*., 2001; Ichida *et al.*, 2007; Lamme, 1995; Marcus & Van Essen, 2002; Shushruth *et al*., 2012; Sillito *et al.*, 1995). In summary, magnitude and sign of firing modulation seem to be directly linked to specific combinations of features inside the CRF and near or far surround, which renders firing rate modulation a form of codifying context in early visual areas. Rate changes have been furthermore assumed to contribute to boundary detection defined by first-order features such as contour orientation, implemented by different connections at a different time point (Roelfsema *et al.*, 2002; Roelfsema & de Lange, 2016).

Most of the studies above, investigated firing rate as a function of highly regular and salient features in gratings or textures. Despite the advantage of easily being parameterized, this kind of stimulation likely introduces an activation bias. Already via the feedforward pathway, specific subpopulations of neurons are addressed separately and homogeneously, each of them densely interconnected by horizontal connections. Many of the first-order features represented in one of these subpopulations overlap, thus contextual grouping based on single cell firing rates is facilitated (Roelfsema, 2006).

In contrast to gratings and lines, natural stimuli exhibit multiple orientations at the same location influencing perception (e.g., Victor *et al*., 2013). Whereas modulation of firing rate of individual units might faithfully indicate the presence of borders respecting CRF limits, it may not be sufficient to represent the highly intricate spatial structure of a natural scene. Here, simultaneous stimulation and interaction of groups of neurons with diverse stimulus preferences already in early visual areas are more likely (Gallant *et al.*, 1998; Haslinger *et al*., 2012; Kayser *et al*., 2004; Vinje & Gallant, 2000;

Weliky *et al*., 2003). Features, such as the direction of motion, could be reflected in population activity resulting from the interaction of neurons with different simple response properties (Yuste, 2015). To extract representative variables of such population activity, it is necessary to use dimensionality reduction methods (Cunningham & Yu, 2014). Indeed, previous reports indicated that measures of population activity such as pairwise synchrony (Biederlack *et al*., 2006), sparseness (Vinje & Gallant, 2000) and sequentially active groups of neurons in time (Carrillo-Reid *et al*., 2015) could be meaningful about stimulus context not evident from simple features. In line, contrast structure and second-order statistics of natural images were observed to be correlated to the average response of a neuronal population with overlapping receptive fields and their local field potential (LFP) in the low-gamma range (Kayser *et al*., 2003; Weliky *et al*., 2003). Especially when challenged with a mixture of features in a natural context not respecting individual CRF limits, population activity could contribute to early visual codification, in parallel and complementarily, to single cell activity. To investigate this issue, we compared the two activity measures in the same neuronal population in response to different motion contrasts induced in natural-like scenes. To this end, we stimulated with a natural-like image configured in three motion contrast conditions. We recorded unit and LFPs activity from multiple parallel sites and computed both firing rate of single neurons and neuron assemblies - i.e., groups of neurons with the affinity to be co-activated (Hebb, 1949; Lopes-dos-Santos *et al.*, 2013). Subsequently, we analyzed the temporal synchrony of single cell and assembly activations with the population activity by estimating their phase-locking to the low-gamma rhythm (20Hz - 50Hz). As the processing of motion contrast requires the integration of spatial information mediated by horizontal and feedback cortical connectivity (Angelucci *et al*., 2002; Chen *et al*., 2014; Crick & Koch, 1998; Ramalingam *et al*., 2013; Roelfsema, 2006; Shushruth *et al*., 2012), we also manipulated visual inter-hemispheric connections (VIC) from isotopic contralateral areas as a model for horizontal connectivity (Schmidt, 2016).

MATERIALS AND METHODS

Preparation and surgical procedures

We performed experiments in five male adult cats bred at the Brain Institute's colony. All procedures were approved by the ethics committee of the Federal University of Rio Grande do Norte in Natal (UFRN) and are following the guidelines of the Society of Neuroscience. Initially, all animals were anesthetized by intramuscular injection of 10 mg/kg ketamine hydrochloride and 1 mg/kg xylazine hydrochloride. Subsequently, they were artificially ventilated with a mixture of 0.6/1.1 % halothane (for recording/surgery respectively) and N_2O/O_2 (70/30%). After completion of surgical procedures and throughout the experiment, animals were paralyzed by continuous intravenous infusion of pancuronium bromide (0.15 mg/kg/h). We continuously monitored the depth of anesthesia observing the electrocardiogram and expiration CO_2 levels. A heating pad stabilized body temperature. For additional details see (Wunderle *et al.*, 2013).

Electrode implantation

We recorded multi-unit activity and LFPs from up to 48 parallel microelectrodes. To this end, platin-iridium electrodes configured in 4 x 4 arrays with an inter-electrode spacing of 250 µm were implanted (MicroProbes, Gaithersburg, USA). All devices aimed at the transition zone between areas 17 and 18. For functional definition of the 17/18 border, optical imaging of intrinsic signals was performed as described in a previous report (Bonhoeffer *et al*., 1995). We were recorded from supragranular layers (recording depth between 200 µm – 700 µm controlled by Narishige microdrives). On the right hemisphere, we implanted a surface cryoloop (dimensions: 7 x 3.4 mm) on the topographically corresponding region.

Visual stimulation

Visual stimuli were presented on a 21'' monitor, configured with a refresh rate of 120Hz, and placed at a distance of 57 cm from the animal. Eyes were dilated with phenylephrine and atropine. Correction lenses were fitted to both eyes to refract at monitor distance using a Rodenstock refractometer. Eye

alignment was checked regularly throughout the experiment and corrected with an adjustable prism if necessary.

We presented a composite, grayscale natural image (mean gray value: 108.7 ± 37.32, out of 256) in three configurations: i) a no motion contrast (NoC) consisting of a full-field image moving in one of the four cardinal directions at 15 degrees per second (4 conditions), ii) an orthogonal motion contrast (ORT, 4 conditions) consisted of a circular patch of 10 degrees diameter, centered on the monitor, moving within its position orthogonally to the surround and iii) a motion contrast with the patch and surround moving in opposite directions (OPP, 4 conditions) (Figure 1A). The size of the patch was defined to be of sufficient size to elicit low gamma activity (Gieselmann & Thiele, 2008). For eliminating contours, which were not part of the natural image, the border between patch and surround was blurred using a 20-pixel moving average. Each condition was presented 20 or 40 times per recording stage (see cooling procedure).

Cooling procedure

Chilled methanol (-60°C) was pumped through the cryoloop. During the recording, the temperature of both methanol and the cortical surface was monitored to ensure the efficacy of the cooling. The surface temperature was maintained between 1°C and 3°C because this procedure efficiently silences the synaptic activity in all cortical layers in a region between the two loop shanks and up to 2 mm in its vicinity (Lomber *et al.*, 1999). A recording session consisted of three stages: Baseline, with the VIC intact, cooling, during thermal VIC deactivation, and recovery, starting after 30 minutes rewarming and thermal stabilization.

Data analysis

Receptive field mapping of multi-unit activity

Receptive fields were mapped throughout the experiment using an automatic back-projection method (Fiorani *et al*., 2014). In a first step, a PSTH of the spiking response to a bar drifting in 16 different directions was created (width = 1°, length = 30°, speed = 20°/s) for each direction separately using a Gaussian smoothing kernel with an SD of 12.7 ms. Then, each PSTH was normalized to its maximum

to weight each stimulus equally and then projected to visual space and summed across stimulus directions. The resulting response density maps were additionally low pass filtered (2D Gaussian smoothing with an SD of 5.88 deg) and the receptive field was defined as the area above 70% of the maximal response. Its center was determined as the center of mass within this region. We calculated the receptive field's position and area of all multi-units and positioned the monitor aiming to maximize the number of units stimulated by the centered patch during the MC configuration (Figure 1F and G).

Sorting

We used *WaveClus*; a spike sorting toolbox developed to determine the spiking times of single units within the multi-unit activity. The toolbox calculates a set of parameters based on wavelet decomposition of spike waveforms, followed by a superparamagnetic clustering (Quiroga *et al*., 2004).

Assembly detection

More details on the assembly detection algorithm are described elsewhere (Lopes-dos-Santos *et al.*, 2011, 2013). It is noteworthy that we obtained similar assemblies with different bin sizes (see Figure S1AB) and also when applying a different detection algorithm (Figure S1CD, Humphries, 2011).

We concatenated single unit activity from baseline, cooling and recovery recording stage and computed a rastergram binned in 5 ms non-overlapping time windows (Figure 3A). This procedure allowed us to track the activity of each assembly throughout the same stages and guaranteed the assembly identity. Each neuron's activity was z-scored. Based on the z-scored rastergram (Z: B x N matrix where B is the number of bins and N the number of neurons) we identified neuronal assemblies: First, the eigenvalues, lambda, of the autocorrelation matrix of Z are computed. If Z is a random matrix (i.e., without assembly activity), these eigenvalues will follow the Marcenko-Pastur distribution defined by:

$$p(\lambda) = \frac{q}{2\pi\sigma}\frac{\sqrt{(\lambda_{max} - \lambda)(\lambda_{min} - \lambda)}}{\lambda} \quad (1)$$

Where $q = B/N$, and σ is the standard deviation of Z, which in this case is equal to 1; λ_{max} and λ_{min} are the upper and lower limit of the distribution and are defined by:

$$\lambda_{min}^{max} = \sigma^2\left(1 \pm \sqrt{1/q}\right)^2 \quad (2)$$

If Z is not random (i.e., contains assembly activity), some eigenvalues lie outside the theoretical boundaries determined by equation 2, with the number of eigenvalues above the upper boundary (N_{as}) indicating the number of assemblies in Z. The result of this assembly detection is a set of N_{as} assembly patterns of N values where N_i is the weight of the i_{th} neuron in a given pattern (Figure 3A, stemplot). The larger the weight, the more often that neuron takes part in the assembly. We sorted the weights of each pattern in descending order and considered the set of neurons that account for 70% of the cumulative sum of all weights as neurons forming that particular assembly. As the last step, each pattern was projected along the rastergram to quantify the assembly's activation in each time bin (Figure 3A, bottom black trace). Activation times were defined as the center of the time bins with projection values above the 99th percentile (Figure 3B, black dotted line). We considered only assemblies formed by units with well-defined CRFs. The CRFs of each unit participating in a certain assembly were superimposed, and the external contour was defined as the population CRF of that assembly (Figure 3C).

Computation of mean firing rate of single cells and number of assembly activations

Both mean firing rate of single units and number of assembly activations followed the formula:

$$FR_{c,k} = \frac{\sum_N fr_i}{N} \quad (3)$$

Where $FR_{c,k}$ stands for: the mean firing rate of cell k or the mean number of activations of assembly k, evoked by stimulus condition c. N is the number of repetitions of stimulus condition c and fr_i stands

for the firing rate of a cell *k*, or the number of threshold crossings of the activity of assembly *k* evoked during the i^{th} presentation of the same condition.

Phase-locking

All local field potentials were filtered in the low-gamma range (LG: 20Hz - 50Hz) using a two-ways least-squares FIR filter available in the *eeglab* toolbox (Delorme & Makeig, 2004). Then, for every single cell, we extracted the LG phase of the LFP, recorded from the same electrode, at the time a spike was fired. A uniform distribution of spike-phases indicates no spike-LFP phase locking. In the case of assemblies, we extracted the LG phase, at the time of each assembly's activation. For evaluating uniformity of the resulting phase distributions, a circular Rayleigh test of non-uniformity was performed. When this test indicated non-uniformity, we computed the strength of phase-locking by fitting the phase distribution with a Von Mises function. From this fit, we extracted the dispersion parameter kappa, with a high kappa indicating a high phase-locking (Aydore *et al.*, 2013). Since units recorded from more than one electrode form an assembly, and thus, more than one kappa was calculated, we defined the highest of those kappa values as the assembly's kappa. To avoid kappa bias generated by a low number of spikes or assembly activations we considered only single cells with a mean firing rate of higher than three spikes per trial and assemblies, with at least one significant activation. In both cases, we considered the responses to the preferred stimulus.

Contrast Modulation Index (MCI)

To quantify the response modulation by motion contrast we defined a contrast modulation index as follows:

$$MCI = \frac{R_{MC} - R_{NoC}}{R_{MC} + R_{NoC}} \quad (5)$$

Where MCI is the context modulation index, R_{MC} denotes the magnitude of the neuronal response (e.g., single unit firing rate, assembly rate or kappa) to a given motion contrast (MC) configuration, and R_{NoC} denotes the magnitude of the response to the corresponding NoC configuration. We excluded units with zero firing rates during either baseline or cooling stages.

RESULTS

We recorded single cell and population activity while stimulating with an image with natural spatial structure presented in three possible configurations displaying either orthogonal (ORT), opposite (OPP) or no motion contrast (NoC). In the first two, the motion contrast was defined between a central rounded area (patch) and its surround (see Methods for details). Since we are interested in the modulation of neuronal activity associated with the motion contrast, we compared the responses only to preferred stimulus pairs, formed by the preferred NoC condition (Figure 1A, left) and its corresponding motion contrast conditions. The preferred NoC condition is that which evoked the highest firing rate or number of assembly activations. Its corresponding condition is either the ORT or OPP condition with the same direction of movement inside the patch area (see Figure 1A, right). We quantified the difference between responses to each condition in the preferred pair by computing the motion contrast index (MCI, see Methods) indicating stronger responses towards motion contrast conditions (positive values until 1) or NoC conditions (negative values until -1) respectively.

Motion contrast modulates firing rates and phase-locking in different neuronal populations

We considered the activity of 505 units, which had more than 50% of their CRF area residing in the central patch (see Methods for details about patch definition). All of those neurons had their CRF center inside the patch, 49% were entirely inside, and the rest of the CRFs included the motion contrast border from inside the patch area. This means that stimulus change occurred either at the outer margin of the CRF or its immediate surround. We found that the firing rate of only 22.7% of the 505 units meeting the above criterion was significantly modulated by changes in the context (paired

T-test, $p<0.05$ for 115 units, Figures 1B and 1C). These modulations consisted mainly in rate decreases when introducing a motion contrast.

Separately, we also evaluated if significant variations of phase-locking to the low gamma rhythm (LG) reflected the stimulus change. We included in this new analysis only units, which, in addition to having more than 50% CRF area residing inside the patch, were also phase-locked to both conditions forming the preferred pair. Thus, for each unit, phase-locking was assessed by testing its spike-phase distribution for uniformity. Only those showing non-uniform distributions were considered as phase-locked (Rayleigh test, $p<0.05$ for 168 units). We quantified the strength of the phase-locking by the dispersion parameter kappa of a Von Mises function fitting (see Methods for details). To establish which units indicated a significant variation of their coupling with LG, we computed the absolute value of all kappa-based MCIs and defined the median of the resulting MCIs distribution as a threshold. According to this definition, 84 units showed significant phase-locking modulation (Figure 1D-G). Subsequently, we divided the significant responses into two different groups indicating either phase-locking enhancement (Figure 1G, color-coded dots above the identity line) or phase-locking decrease (Figure 1G, color-coded dots below the identity line) during MC stimulation.

Remarkably, we identified only a small group of single units (n=27) having their rate (out of 115), and phase-locking (out of 84 units) modulated simultaneously by motion contrast (Gray dots in Figure 1C and 1G). This result indicates that changes in motion contrast can modulate different neuronal signatures in independent populations (purple and green dots in Figure 1C and 1G; see also Figure S2).

Modulation of rates and phase-locking of single cells are similar for orthogonal and opposite motion contrast

In the following, we evaluated whether distinct motion contrasts (Figure 2A) were reflected differently in the modulation of single cells activity. The strength of responses to orthogonal (ORT)

and opposite (OPP) motion contrast conditions was indistinguishable as expressed by their distribution of MCI values (Figure 2B right, Wilcoxon test, p=0.27). Regarding the modulation of their coupling with LG, kappa values indicated that changes in the context modulated the phase preferences towards either an increase or decrease of phase-locking (Figure 2C, left). This variation of kappa was considerable as reflected by MCI values ranging from -0.42 to 0.45. However, separate distributions of MCI values derived from ORT (n_{resp}=115) and OPP (n_{resp}=116) stimulation did not indicate differences in phase-locking modulation related to the relative direction of movement of the surround (Wilcoxon test, p=0.58, also see Figure 2C, right).

Motion contrast modulates assembly and single cells rates

Subsequently, we analyzed whether synchronous firing of groups of neurons (i.e. assemblies) was modulated, and also, whether the motion-associated changes in this neuronal signature differed from those in single cell activity. We identified 76 neuronal assemblies in our dataset and characterized their activity by computing the number and time of occurrence of their activations per stimulus condition (Figure 3A-B, see Methods for details). In order to determine which part of the visual field drove these activations, we computed the centroid of all CRF centers of each unit forming the assembly (Figure 3C). For further analyses, we considered only assemblies with their CRF centroid inside the patch area (n_{assem}=57).

We found that 22.8% (n_{assem}=13) of the assemblies showed significant differences in the number of activations (assembly rate) when evoked by the preferred NoC condition as opposed to at least one of its correspondent contrast motion conditions. In all these cases, we encountered lower assembly activity when driven by stimulus conditions with a motion contrast between the patch and surround (Wilcoxon signed-rank test $p<0.05$ in 14 responses, Figures 3F and 3G, left). The strength of such modulation, expressed by the MCI, did not reveal differences between surrounds moving in opposite or orthogonal directions (Wilcoxon test p=0.6, Figure 3G, right).

These results are quantitatively and qualitatively equivalent to those found for the rate modulation of single cells. Therefore, we compared the cells with significant rate modulation (Figure 2B) and the cells forming the assemblies with significant modulation of activations. It turned out that only 37% of all neurons forming the 13 assemblies also showed significant differences in their individual responses. At first glance, this relatively low percentage suggests that introducing a motion contrast in the scene could modulate the synchronous firing of groups of neurons without affecting their firing rates. However, neurons that were modulated in their rates had also a higher weight in the respective assembly than neurons that were not rate-modulated (T-test, $p=0.23x10^{6}$). Moreover, these highly weighted neurons preferred the same stimulus conditions as the assembly. This observation is compatible with the interpretation that the activations of single cells and assemblies are similarly influenced by the stimulus. Accordingly, modulation of assembly rate could be a co-variation of the activity of the contributing neurons, rather than an independent process of firing synchronization.

Motion contrast modulation of assembly phase-locking differs for the two surround conditions

For the analysis of assembly phase-locking, we applied the same criteria as for phase-locking of single cells. We found that the activity of 20.3% (n_{assem}=20) of the assemblies was phase-locked to low-gamma during NoC and at least one of its correspondent MC conditions. 17 responses to a preferred pair revealed significant kappa modulation when introducing a motion contrast. Both enhancement and decrease of phase-locking were observed (Figure 3H and 3I, left). It turned out that only 22.5% of the single units forming assemblies with significant kappa modulation, also significantly modulated their phase-locking values. In this case, we observed no relationship between the weights of single cells forming the assemblies and the individual kappa modulations (T-test, p=0.12). Most interestingly, the sign of the MCI value for assembly phase-locking indicated the type of motion contrast introduced. The opposite motion contrast tended to enhance the phase-locking as opposed to uniform whole-field stimulation (i.e., positive MCI values). In contrast, responses to

orthogonal motion context tended to be less phase-locked than those to the corresponding NoC (i.e., mainly negative MCI values) (Figure 3I right, Wilcoxon test, p=0.0056).

In the above MCI analysis, we compared responses for each MC with its NoC condition. Direct comparison of the two motion contrast conditions revealed also a significant difference between them (p=0.011; see Figure S3).

Deactivation of VIC affects context-associated modulation of single cell and assembly rates only weakly

Deactivating visual interhemispheric input reduced significantly the number of spikes evoked by both NoC (mean reduction: 43.8%) and MC (mean reduction: 42.2%) configurations (T-test, NoC: $p=5.1x10^{-9}$, OPP: $p=3.1x10^{-7}$, ORT: $p=7.2x10^{-6}$) (Peiker *et al*., 2013; Wunderle *et al*., 2013). The motion-associated modulation of firing rates vanished in the absence of VIC in about half of the neurons (44.3%, $n_{units}=51$), which had been modulated during baseline (Figure 4A; cross marks). However, we noted that this specific sub-population had been only weakly modulated during baseline. The affected units had a lower baseline MCI (mean MCI: -0.13 ± 0.1) than those which were not affected by inactivating VIC (Figure 4A; dot markers; mean MCI:-0.21 ± 0.1) (Wilcoxon test, $p=3.3x10^{-8}$). This result suggests that VIC input, although it participates, may not be driving the motion-modulation of those firing rates. Accordingly, the separate analysis of the two MC configurations confirmed that the lack of VIC did not significantly modify the firing rate MCI observed with either OPP or ORT stimulation (Figure 4B, Wilcoxon signed-rank test, OPP: p=0.94, ORT: p=0.27).

Similar to firing rates, assembly rates also decreased in the absence of visual callosal input, regardless of the stimulus conditions (mean reduction with NoC stimulus: 54.6%; with OPP: 59%; with ORT: 59%; T-test; NoC: p=0.001, OPP: p=0.0052, ORT: p=0.021). Thereby, about half ($n_{resp}=8$) of the preferred pairs that previously indicated significant suppression induced by MC conditions lost this modulation (Figure 5A; cross marks). These response pairs had already low absolute MCI values

during baseline, and within the population of assemblies, the reduction of the context-associated modulation of assembly rates by cooling was not significant (Figure 5B).

Deactivation of VIC redistributes the context-associated modulation of single cell phase-locking

The absence of VIC input caused a redistribution of MCI values for single cell phase-locking. In fact, 67.5% (n_{resp}=81) of the response pairs that surpassed the threshold during baseline maintained their significance during cooling; yet, response pairs previously indicating phase-locking enhancement when introducing the motion contrast (i.e., positive MCI) could either maintain, lose or significantly diminish phase-locking (i.e., negative MCI) after VIC deactivation. The analog also occurred for response pairs with negative baseline MCI (Figure 6A). Therefore, the new distribution of MCI generated during cooling had its median close to zero, though with a large range of positive and negative values. These results suggest a high sensitivity of phase-locking to callosal input for both OPP and ORT protocols (Figure 6B, Wilcoxon signed-rank test; Increased kappa populations: p_{ORT}=0.017 p_{OPP}= 0.0064, decreased kappa populations: p_{ORT}=0.035 p_{OPP}= $8.5x10^{-4}$).

Deactivation of VIC affects assembly phase-locking in a stimulus-dependent manner

Interestingly, the absence of VIC predominantly diminished the advantage in phase-locking with OPP over NoC stimulus configuration observed in baseline recordings (Figure 7A) whereas modulation with the ORT stimulus remained unchanged. This particular effect was reflected in the significant reduction of MCI values found only for those stimulus pairs formed by NoC and its correspondent opposite moving surround stimulus (Figure 7B and 7C, *Wilcoxon signed-rank test p=0.036, **Wilcoxon test p=0.0085). Changes of assembly phase-locking MCIs induced by cooling recovered to baseline (Figure S4).

DISCUSSION

In this study, we analyzed how changes in the visual context modulate four different neuronal signatures: firing rates and phase-locking of single cells and neuronal assemblies. We also investigated the role of lateral connectivity in such modulation. To do so, we disrupted part of the horizontal network using thermal deactivation of visual callosal input. We observed that changes in the context were not only mirrored in rates but also in phase relationships to the LFP. All motion-associated modulations could be simultaneously present in independent neuronal subpopulations. Therefore, signatures of different complexity levels could hold features of different complexity levels. Whereas single and population firing rates indicated the mere presence of a motion contrast close to the border of their CRF - either inside or outside -, only the phase-locking of cell assemblies to the low gamma rhythm reflected its directions of movement. We further found that also the deactivation of visual callosal input selectively affected the assembly phase-locking in responses to opposing, in contrast to orthogonal directions of motion.

Overall, all four neuronal signatures that we considered as possible coding strategies reflected the motion contrast. However, assembly activations could be deduced from the rates of single units contributing mostly to the respective assembly. Both single cell firing and assembly activation were mainly suppressed by the introduction of the non-homogenous background, independent of its exact direction of motion. Interestingly, the phase-locking analysis uncovered a subpopulation of single cells that did not exhibit a significant variation of firing rate but of the synchrony between their spike times and the ongoing low-gamma fluctuation.

Our results are partially in line with earlier investigations of temporal coupling using whole field grating stimuli and separating a figure from a background by introducing an orientation or phase offset (Biederlack *et al*., 2006; Gail *et al*., 2000). In anesthetized cats, the out of phase but iso-oriented surround enhanced spike synchrony between neurons responding to the figure whereas an orthogonally oriented background decreased it (Biederlack *et al*., 2006). This finding is homologous to our result of assembly phase locking increasing with non-collinear and decreasing with orthogonal

background motion. Many other studies using textures or lines argue against a systematic relationship of spiking synchrony with the perceptual organization of the stimulus but instead draw a link to the functional connectivity of V1 (e.g., Lamme *et al*., 1998; Roelfsema *et al*., 2004). Although we aimed to minimize the systematic influence of the functional connectivity by choosing natural stimuli, assembly activations to the composite CRF, reflecting the synchronized activity to the foreground, systematically decreased with the introduction of a second background object (Figure3D), as also did the spike count (Figure 2B). Noteworthy, however, the phase locking of those rare assembly events to low gamma was differentially modulated by different backgrounds (Figure 3E).

It is well known that low gamma variations are informative about synaptic activity within a range of several millimeters (Bringuier *et al*., 1999; Kajikawa & Schroeder 2011; Buzsáki *et al.,* 2012) reflecting the coordination of excitatory and inhibitory inputs (Buzsáki & Wang, 2012). They have also been attributed to coordinate spiking activity (Fries, 2009; Gray & Singer, 1989; Jia *et al*., 2013). Recently, the power of low gamma activity has been proposed to directly reflect the extent of the integration between responses to the classical receptive field and its surround (Vinck & Bosman, 2016). Here, we directly support that the phase relation between unit or assembly firing and low gamma-frequency range can reflect the previously proposed integration.

In V1, oscillatory activity in the gamma band is commonly related to high-level visual processing involving attention (Fries *et al*., 2001) or feature binding (Singer, 1999). However, the functional role of gamma oscillations in stimulus encoding is an ongoing debate. One of the arguments against their encoding potential is their direct relation to low-level stimulus features being well described by simple models of excitation-inhibition interactions (Ray & Maunsell, 2015). Indeed, gamma band power of the membrane potential has been reported to be highly selective for contour orientation in anesthetized cats (Azouz & Gray, 2003). In natural scenes, changes in specific frequency bands of the LFP signal, including low-gamma, have been correlated with changes in motion, but not with the scenes' spatial structure (Kayser *et al*., 2003; Siegel *et al*., 2007). These observations motivated the choice of our stimuli, and agree with our finding on assembly phase-locking.

Of note, only a small number of neurons in our study modulated at the same time rate and phase - locking to motion contrast. We conclude that the phase-locking modulation has its origin in low-gamma fluctuations generated by the receptive field's surround, not in the rate modulation produced concurrently by the CRF center in response to low-level features. More importantly, our results suggest that rate and spatiotemporal coding strategies exist in parallel and can be carried out by different neuronal subpopulations in V1. Along with this line, psychophysical studies also indicate that stimulus features of different statistic orders could be processed separately (e.g., Victor *et al*., 2013).

We propose that independent neuronal subpopulations can adopt variable coding strategies within the same area thus enhancing the feature space, which can be represented simultaneously.

There is evidence of independent population coding from imaging studies in that spatiotemporal population activity to superimposed gratings is not necessarily predicted by the sum of the activity evoked by the individual component gratings (MacEvoy *et al*., 2009; Schmidt *et al*., 2006). In addition, this type of population activity captured by imaging is high during epochs of gamma oscillations but is not linearly linked to the underlying spiking activity (Niessing *et al*., 2003).

Further, almost 57% of the recorded neurons and 50% of the assemblies responded well to both NoC and MC stimuli but showed no significant context modulation, in neither rate nor phase-locking. This observation lets us assume that there might exist more possible codes for contextual changes than those addressed here. In accordance, parallel components of coding based on firing rate variations arising from different neural circuits, have been proposed to differentiate far- and near-surround modulation (Nurminen & Angelucci, 2014).

It is intuitive that adding coding strategies based on temporal patterns of population activity, like neuronal co-activation and phase-locking to LFP, could facilitate stimulus encoding. An increase of degrees of freedom for representation of features was previously suggested (Samonds *et al*., 2004) showing that neuronal assemblies formed by the coordinated activity of single neurons can be more efficient and can carry more information than individual units. Miller et al. (2014) proposed that to

represent changing visual attributes intrinsically generated ensembles of coherently and spontaneously active flexible groups of neurons are recruited. Evidence for the coding capabilities of population activity within natural scenes was also presented for the ferret's visual cortex (Weliky *et al*., 2003).

In the current work, we led the concept of population coding a step further, namely to the coordinated activity between populations, the locking of the assembly's activation to the phase of the low gamma LFP fluctuation. This measure did not only indicate the presence of a non-homogenous surround but also carried information about its direction of motion, orthogonal versus opposite to the foreground, that was absent in the activity of single cells. Such information could be derived from differences in the generation of the LFP with the two stimuli. The LFP results from the integrated activity of separated neuronal populations stimulated by the motion contrast (foreground and surround). With opposite stimulation (OPP), the populations which process foreground and surround are localized within iso-oriented domains and would be equally excited and sustained by long-range excitatory connections between them. If we consider the gamma phase as representative of the state of the network excitability (Fries *et al*., 2007), the similarity in the level of excitation within these populations would be reflected in the reliability of gamma phase. Coordination is less expected with ORT stimulation because excitatory connections between orthogonal orientation domains are less frequent.

Further, and in contrast to single cell activity, the phase-locked activation of neuronal assemblies could increase the system's robustness through signal redundancy (Takiyama & Okada, 2012) since it is not necessary that all individual neurons forming the assembly discharge to produce an assembly activation. This apparent redundancy was also observed in cortical sub-networks simultaneously activated by natural scene stimulation in the mouse visual cortex (Kampa *et al*., 2011).

When interrupting the lateral network at the visual field's midline, some coding strategies proved to be more robust than others. As observed previously we noted an overall loss of excitatory input (on average, rate decreases, data not shown) (Peiker *et al*., 2013; Schmidt *et al*., 2011; Wunderle *et al*., 2013). This loss did not change the relationship between rates or assembly activations during uniform

versus motion contrast stimulation as expressed by the MCI values. One could assume that fewer assembly activations naturally follow a reduction of single cell firing rates. Nevertheless, although we only considered activations that did not occur as a co-variation of single cell rates by chance, assemblies almost replicated the effects on units.

In contrast, the single cell phase relations between uniform and motion contrast stimulations changed dramatically, sometimes even reversed. If we consider baseline phase relations as informative about context, these highly unspecific changes (ups and downs of different magnitude) could be attributed to a high sensitivity of this coding strategy to the excitatory/inhibitory balance also expressed in intra-hemispheric LFP desynchronization (Carmeli *et al*., 2007) during removal of VIC input. Interestingly, the cooling effect on assembly phase-locking was specific, selectively reducing the MCI with opposite motion contrasts but not with orthogonal ones. This effect is in line with the above discussed baseline difference in phase-locking for the two motion contrasts and the role of excitatory connections because cooling interrupts the interhemispheric connections (VIC) between iso-orientation domains across the two hemispheres (Makarov *et al*., 2008; Schmidt, 2013). It points towards a role of the VIC in the modulation by opposite motion contrast like, for example, one that crosses the visual field's midline (Peiker *et al*., 2013).

In summary, our results illustrate that context could act through different coding strategies related to different levels of feature complexity. Levels of coding ranged from rate modulation within one iso-tuned population, to synchronization within (co-activations) and between (phase-locked) populations. Along with this line, an orientation contrast could be represented in varying rates of differently tuned sub-populations. However, to process a motion contrast in a stimulus without orientation bias might require the recruitment of additional coding strategies like the phase relation between assemblies and LFP.

The notion of different levels of coding complexity does not imply the prevalence of one coding strategy over the other. Instead, it enriches the scenario where multiple codes, even used by different sub-populations, could co-exist.

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Author contribution: S.A.C-O and K.E.S conceived the experiments, analyzed the results and wrote the manuscript; S.A.C-O processed the data; all authors collected the data.

Data accessibility: Data are available from corresponding author on request.

Abbreviations: CRF, classical receptive field; LFP, local field potential; LG, low-gamma; MC, motion contrast; MCI, motion contrast index; NoC, No contrast; OPP, opposite motion contrast; ORT, orthogonal motion contrast; PSTH, peri-stimulus time histogram; SD, standard deviation; VIC, visual inter-hemispheric connections;

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FIGURE CAPTIONS

Figure 1. Motion contrast modulates different neuronal signatures in independent populations. (A) No motion contrast (NoC) condition, and its corresponding motion contrast (MC) conditions moving either opposite (OPP, white dashed) or orthogonal (ORT) to the patch's direction of movement. (B) Example of a peri-stimulus time histogram (PSTH) of a single neuron in response to the NoC and ORT conditions. Black arrow, stimulus onset. (C) Each dot represents a pair of responses of one single unit to the preferred NoC and its corresponding MC conditions. Only pairs showing significant modulation of firing rate are depicted. (D) Filtered LFP at low gamma range (upper trace) and spiking activity of a single neuron (lower trace) during one trial. Note that this neuron tends to fire in the same phase of the oscillation (inset). (E) Circular histogram of the relation between LFP phase and spiking activity during the same trial as in (D). (F) Spiking probability of an example single unit, computed for all trials of NoC and OPP conditions. (G) Same as (C) for units showing significant phase – locking variation. Units with both firing rate and kappa modulated for the same stimulus pair (overlapping population) are represented in gray in (C) and (G), the population with exclusively rate or kappa modulation is displayed in red and green, respectively.

Figure 2. Modulation of single unit activity during baseline. Only cases with significant differences between conditions with (MC) and without (NoC) motion contrast are depicted. (A) Responses in all figure panels are color-coded according to the preferred stimulus pair, ORT (orange) and OPP (blue). (B, left) Mean firing rate of single units during NoC and MC stimulation. (B, right) Boxplots representing the median context modulation index for both MC conditions. (C) Same as (B) for single cell phase-locking. For both ORT and OPP conditions, preferred pair responses demonstrated enhancement and decrease of phase-locking (left), and were divided into positive and negative MCI distributions respectively (right).

Figure 3. Modulation of assembly activity during baseline. (A) Rastergram of a complete recording session (baseline, cooling, and recovery) containing 48 sorted single units. One typical assembly pattern on the right, its normalized activity (N. Act) during the entire recording session on the bottom black trace. Neurons forming the assembly are denoted in black and their individual weight indicated by the bars' length. Neurons in gray are not part of the assembly. (B) Detail (250ms red window) of (A). Neurons forming the assembly (black) are re-organized following the descending order of weights in the assembly. Red squares indicate time bins where assembly activity (bottom black trace) surpasses statistical threshold (dashed black line). The z - score of individual firing rates is indicated by the bins' gray level with darker levels codifying higher firing rate. (C) Example of an assembly's receptive field, defined by the external contour (white) of individual receptive fields of the neurons forming the assembly (black). The centroid (white cross) is computed from individual receptive field centers (black crosses). (D) LFP filtered at low gamma range (upper trace) and example assembly activation (lower trace) during one trial. This assembly had 9 activations. Dotted line, activation threshold; black arrow, stimulus onset. (E) Circular histogram of the relation between LFP phase and assembly activation during the same trial as in (D). (F) Example of an assembly activation PSTH in response to NoC and OPP (upper) or ORT (lower) conditions. (G, left) Mean number of activations for preferred stimulus pairs (n=14, formed by 13 assemblies) to OPP (blue) and ORT (orange) conditions. Only cases significantly different from each other are depicted. (G, right) Boxplots

representing the median MCI value distribution. Note that the MC stimuli always evoke less assembly activity than the NoC. (E) Probability of activation of an example assembly computed from all trials of NoC and OPP (up) or ORT (down) conditions. (I) Same as (G) for assembly phase-locking variation (n=17 pairs, formed by 15 assemblies; ** Wilcoxon test, $p<0.01$).

Figure 4. Effects of VIC deactivation on firing rates of single cells. (A) MCI indicating a significant modulation of firing rates evoked by preferred stimulus pairs (n=115) to OPP (blue) and ORT (orange) conditions. Note that single cells, whose MCI dropped below significance during cooling (crosses, n=39) had a low MCI during baseline. Units maintaining significant modulation during cooling (solid dots, n=76) also tended to have higher MCIs during baseline. (B) MCI distributions from the data in (A) show no significant change during VIC deactivation for both motion contrast conditions.

Figure 5. Effects of VIC deactivation on assembly activations. (A) MCI for all neurons showing a significant surround modulation of assembly activations during baseline (solid dots). For some neurons, this surround modulation disappeared during cooling (crosses). Note that these assemblies had already a low MCI in the baseline. (B) Boxplots representing the median MCIs of all responses in (A) and showing no significant change during VIC deactivation for both OPP (dark blue) and ORT (orange) stimulation.

Figure 6. Effects of VIC deactivation on phase-locking of single cells to low-gamma. (A) MCI variation during VIC deactivation. Crosses indicate responses losing modulation in the absence of VIC. Note that baseline values from both increase and decrease response groups redistributed into positive and negative MCI values during VIC deactivation. (B) Accordingly, the median MCIs of the

Kappa values in A for phase-locking approximated zero for both OPP (blue) and ORT (orange) stimulation (Wilcoxon signed-rank test *$p<0.05$, ** $p<0.001$).

Figure 7. Effects of VIC deactivation on phase-locking of assemblies to low-gamma. (A) Probability distributions of phase angles for the activation of two different assemblies: The example demonstrates both stronger phase-locking for OPP (top row, dark blue) and weaker for ORT (bottom row, orange) as compared to the corresponding NoC (gray). During VIC deactivation (right panels), the phase-locking difference disappears for OPP but not ORT responses (B) MCI modulation during VIC deactivation. Crosses indicate responses, which lost that modulation during VIC deactivation. Note that responses of preferred pairs formed by OPP (blue) are more affected by cooling than pairs formed by ORT stimulation (orange) (C) Accordingly, the median MCI dropped significantly for responses to OPP (blue, *Wilcoxon signed-rank test $p<0.05$) but not ORT stimulation (orange). **Wilcoxon test $p<0.01$