Abstract

Trial-to-trial fluctuations in response strength are correlated between neurons with similar tuning properties. To evaluate whether this correlation is stimulus dependent, we recorded from pairs of layer 4 pyramidal neurons in the primary visual cortex of anesthetized paralyzed macaque monkeys. Neurons were generally separated by less than 300 microns and had overlapping receptive fields and similar orientation and spatial and temporal frequency preferences. We characterized the correlation between cells using multiple presentations (60-150) of drifting sine wave gratings of 3 orientations, each stimulus of different intensity, and each pair of drifting at least one of the two cells. We calculated total spike count correlation, Rsc, and cross-correlogram (CCG) at each stimulus condition. The Rsc values (>2) were on average independent of the orientation of the stimulus. Contrast peak values, however, was much larger for stimuli which evoked stronger responses in both cells than for suboptimal orientations. The dependence of CCG peak amplitude on stimulus orientation was not a simple function of response strength. When firing rate was manipulated across a similar range by lowering the stimulus contrast, CCG peak width broadened subordinately, or more little change in peak height. Similarly, CCGs obtained from spontaneous activity often had larger peaks than those obtained with stimuli of suboptimal orientation. These results indicate that while Rsc, is not stimulus dependent, correlation on a fine time scale is sensitive to several hundred microns, depending on stimulus orientation. Broad CCG peaks obtained with high contrast stimuli may reflect the lower temporal precision of spikes evoked by fine time scale events.

Effect of stimulus orientation on correlation

2 Spike count correlation

3 Example CCGs

Effect of stimulus contrast on correlation

6 Spike count correlation

7 Example CCGs

Methods

- Experiments performed in the primary visual cortex of anesthetized (sufentanil 4 mg/kg) macaque monkeys.
- Retinal input to the primary visual cortex was determined by stimulating the optical input to the visual thalamic nuclei.
- The visual thalamic nuclei of the two visual fields were individually imaged with 100, 20, 4, and 1.5 cycles per degree (c/°) stimulation.
- Stimulus strength was manipulated by lowering the stimulus contrast to 0.25.
- Orientations were individually imaged with 0.25 cycles/° stimulation.
- Orientation was measured at first zero crossing of the CCG. Orientations are ordered by firing rate (i.e. best orientation for each pair is that which evokes the highest mean firing rate).
- CCG peak height is strongly correlated with evoked firing rate. CCG peaks for suboptimal stimuli are typically smaller and less amenable to stimulus evoked correlation. This area shows the firing rate and peak height of the CCG against the stimulus evoking the strongest response for each pair.
- CCG peak height and base width is not correlated with stimulus orientation. Roughly 80% of pairs have CCG peak width of less than 100 ms. The mean is measured at first zero crossing of CCG. Orientations are ordered by firing rate (i.e. best orientation for each pair is that which evokes the highest mean firing rate).
- CCG peak base width is not correlated with stimulus orientation. Only stimulus conditions with firing rates greater than 0.1 p/s considered.

Effect of stimulus contrast on correlation

8 CCG peak height

9 CCG base width

Evoked vs spontaneous correlation

10 Evoked vs spontaneous correlation

11 CCGs

Summary & Conclusions

The reduction in CCG peak height with suboptimal orientations can not be due solely to a change in firing rate because CCG peak height is maintained as the contrast of an optimal stimulus is lowered. Rsc values increase and CCG base with broader suboptimally as contrast decreases. Collating the broadband CCGs at low contrast, there is a small increase in the mean Rsc and CCG base, and this increase is due to an increase in the width of the autocorrelation at low contrast. CCG peak height in spontaneous activity was often greater than for suboptimal, high contrast stimuli. In addition to the pairs evoked CCGs, peak for spontaneous activity was not evaluated activity.

These results suggest that neural correlation on a fine time scale is stimulus dependent, and the strength of the correlation is not a simple function of stimulus orientation, and that stimulus drive may disrupt ongoing correlation present during spontaneous activity (even if it does not change substantially with stimulus orientation).