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A retino-cortical model of anticipation

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Introduction

The integration of moving objects from the visual system takes about 100 ms [1]. With such a delay we wouldn't be able to catch moving objects. How does our visual system manage to compensate for this delay ?

We define **Anticipation** as a group of mechanisms in the visual system to compensate this lag.

Studies have shown the presence of two types of anticipation in the retina. The first, called "**adaptation**", corresponds to an earlier decay in peak activity, resulting in a shift in the peak and a lower amplitude [1]. The second type, called "**prediction**", shifts the entire peak without altering its amplitude [2].

There is also evidence of anticipation in the cortex. This anticipation, by "**latency**", leads to the propagation of a wave of activity transmitted by lateral connectivity of cortical fibers [3]. Cortical columns in contact with the bar activate those further away, especially those not sensing yet the object.

We reproduce these types of anticipation in a retino-cortical model. Here we wish to understand the retinal contribution to cortical anticipation. This enables us to gain a better understanding of the parameters essential to these mechanisms.

Anticipatory cortical wave in Monkey cortex V1

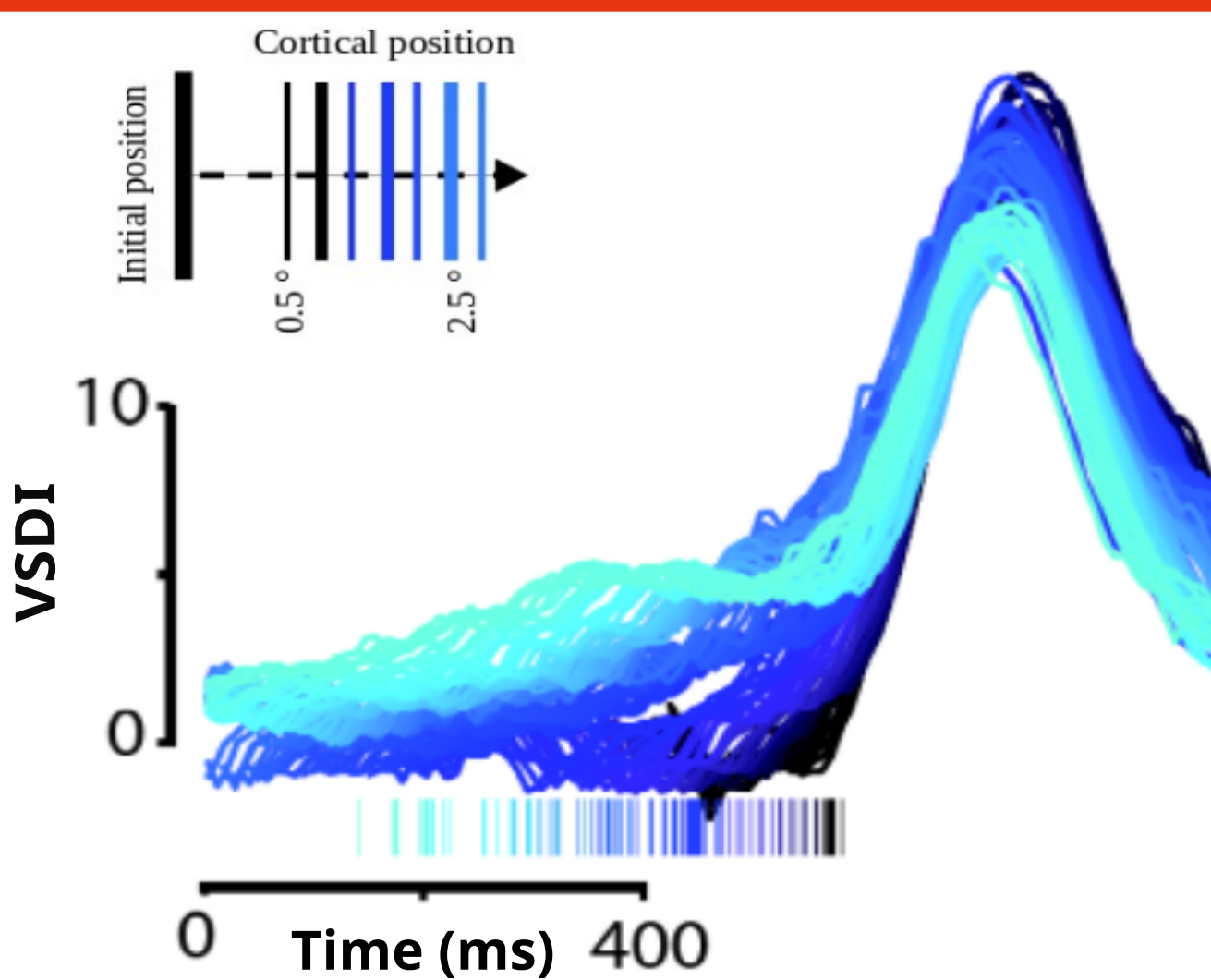


Fig 1 : Response of macaque V1 neurons to the trajectory of a white bar moving at 6°/s. Each curve represents the VSDI signal from one region of interest. This signal was centred using the arrival time from the beginning trajectory of the bar center to the middle of the receptive field. The blue color gradient correspond to the distance (in degrees) between the cortical column and the initial position of the bar [3].

Methods

The **retinal model** reproduces and simplifies the biological retina structure [4] :

- **Photoreceptors** function is modeled by a convolution between stimulus and a spatio-temporal kernel with center/surround architecture.
- **Bipolars cells** set synapses with amacrine cells and have gain control.
- **Amacrine cells** inhibit the bipolars cells.
- **Ganglions cells** have gain control and receive input coming from multiple bipolar cells with a weight decreasing with distance.

The **cortical model** has been created to replicate **voltage sensitive dye imaging (VSDI)** by using cortical columns organization of the visual cortex [5, 6, 7]. The simulated cortical area is then summarized by a grid of **cortical columns** corresponding to mean field equations.

Each cortical column model emulates the mean voltage of 10 000 neurons split between excitatory and inhibitory population. The mean voltage of both populations is used to compute VSDI intensity.

Lateral connectivity in this model allows to obtain activity waves as observed on Monkeys [3].

Macular is a modular simulation software made at INRIA where retinal and cortical models are implemented. It allows to see activity of cell layers or specific cells.

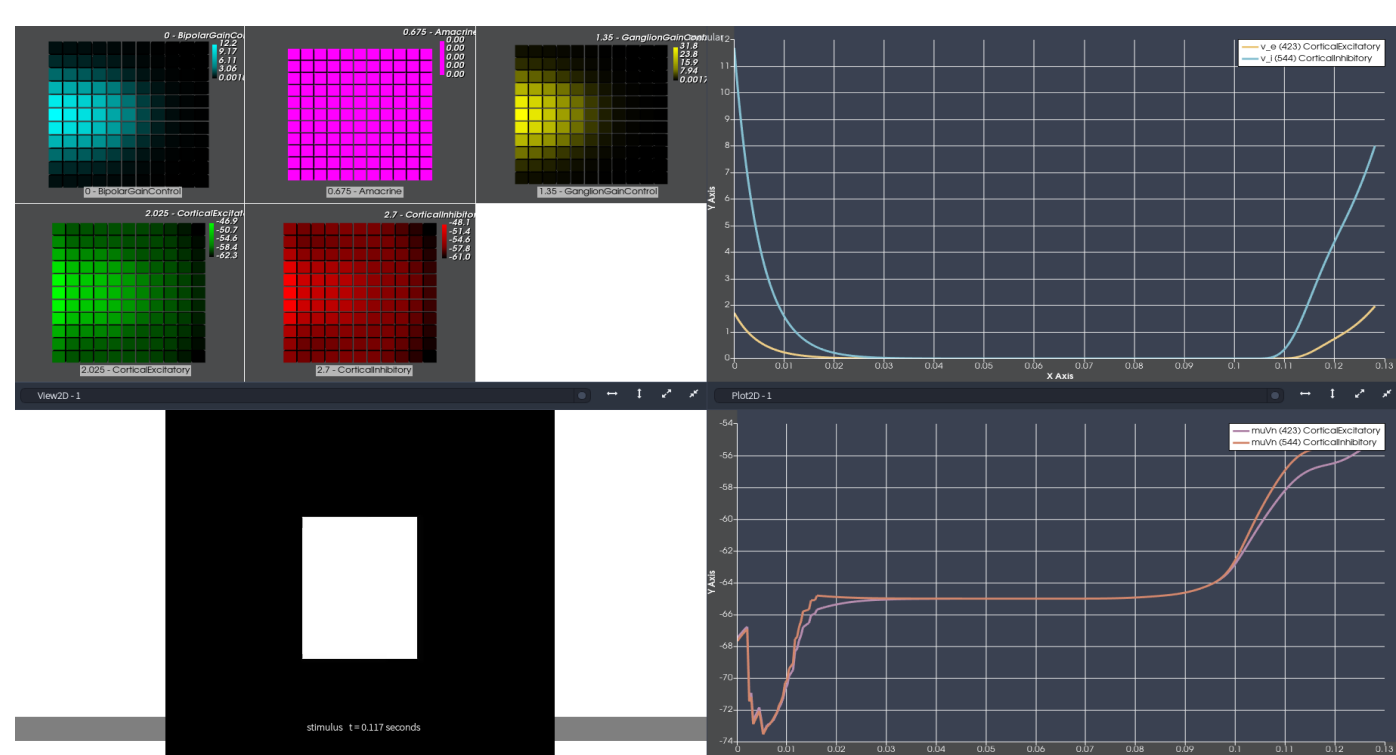


Fig 3 : Macular graphic interface. Top left are heatmaps of the five layers of our model : bipolar cells (cyan), amacrine cells (magenta), ganglion cells (yellow), excitatory cortical population (green) and inhibitory cortical population (red). At the bottom left is displayed the stimulus of the current simulation. On the right are graphs of specific cells. Stimulus, heatmaps and graphs are refreshed in real time with simulation progress.

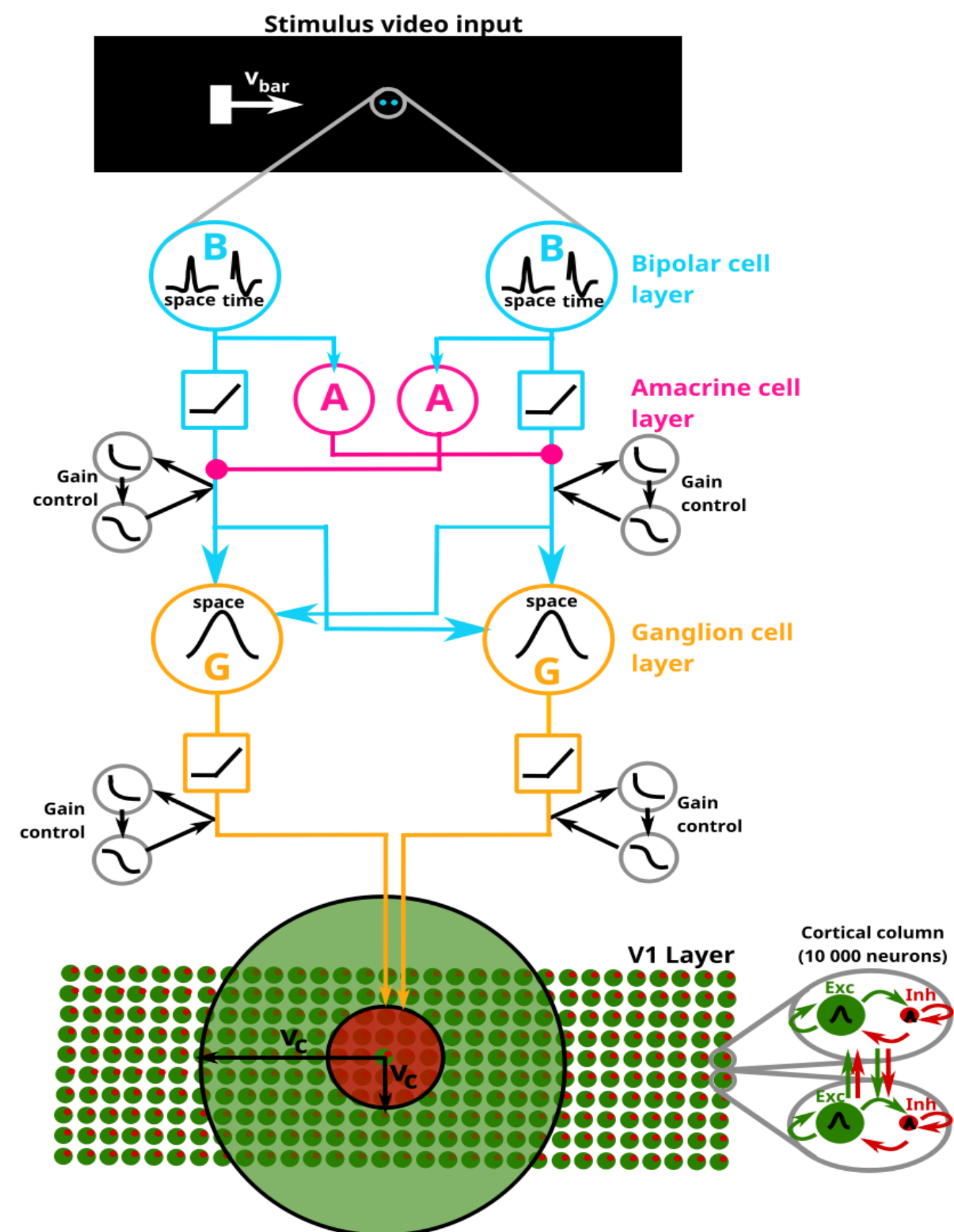


Fig 2 : Structure of the retinal model each receiving a pixel of the video stimulus (top) and connected to a cortical model (bottom).

Results

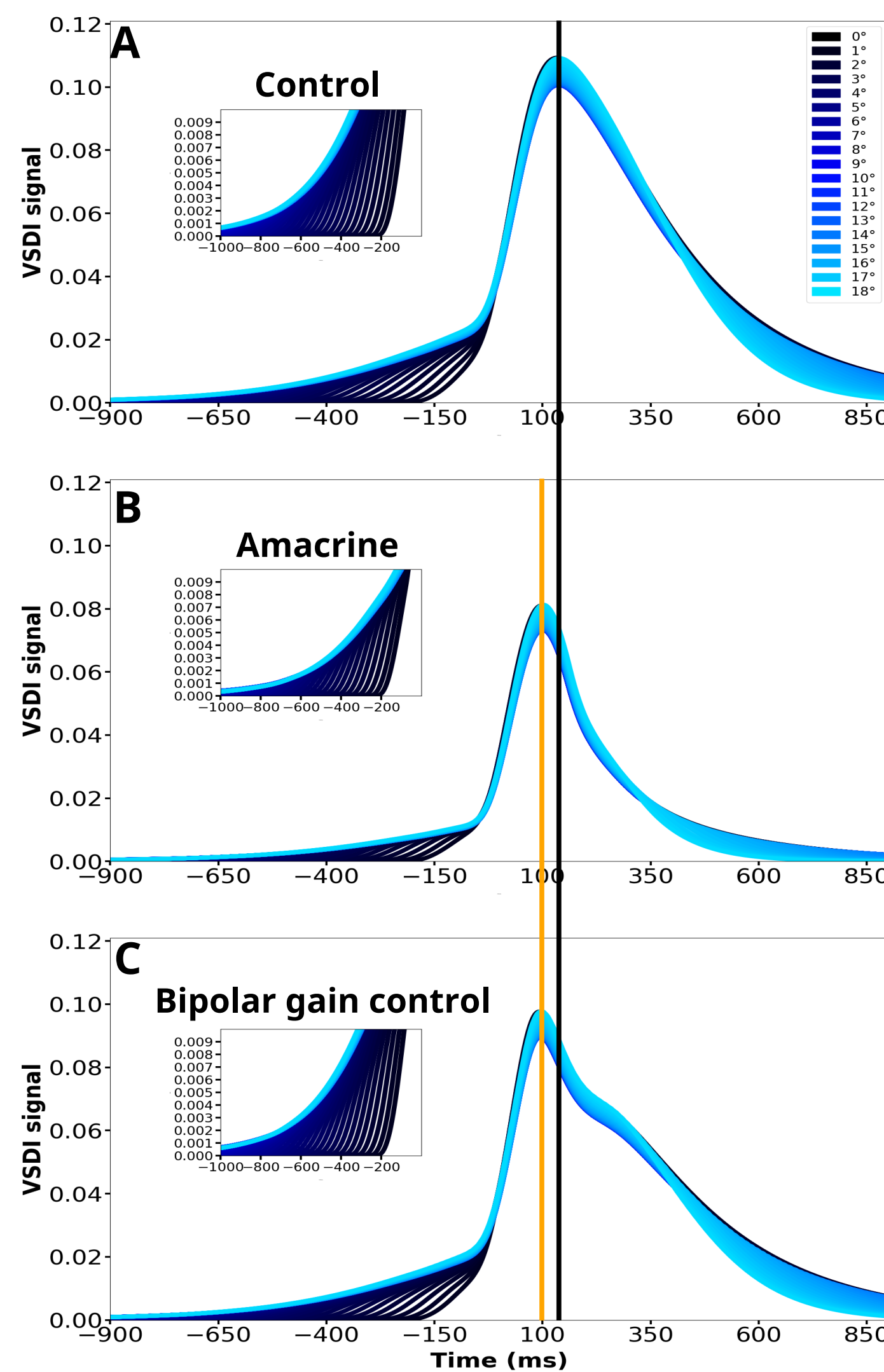


Fig 4 : Effect of amacrine and bipolar gain control on cortical response. Each curve represents the VSDI signal from one simulated cortical column. This signal was centred using the arrival time from the centre of the bar to the middle of the receptive field [3]. The blue color gradient correspond to the distance (in degrees) between the cortical column and the initial position of the bar. Black and orange vertical line are the VSDI peak with default parameters and bipolar gain control. The inset zooms in on the proximal part of the curve where latency anticipation can be seen.

Anticipatory cortical waves

Figure 4A shows a good reproduction of the experimental results of the cortical anticipation wave [3] in Figure 1. The further the bar start to move from the cortical column, the earlier they activate. After a certain distance, the curves start to overlap. Hence anticipation saturates. This is the range of anticipation.

Anticipatory cortical waves with amacrine to bipolar connectivity

In the presence of bipolar-inhibiting amacrine cells (Fig. 4B), the VSDI signal peak is lower and 36ms earlier. The latency slopes are more compressed and begin to increase

at a shorter delay. The curves at which the effect saturates are also closer to the origin of the bar. This corresponds a reduction in the maximum latency and the range of anticipation.

Anticipatory cortical waves with bipolar gain control

Activation of the gain control in the bipolars (Fig. 4C) reveals a peak in the VSDI signal that is advanced and slightly less reduced than in the presence of amacrine (Fig. 4B). In contrast, the maximum latency at which the curves begin to increase and the distance at which they saturate are equal to the control condition.

Amacrine and bipolar gain control effect on spatio-temporal profiles :

Temporal (5A, B, C) and spatial (5D, E, F) profiles are both weaker and narrower with gain control and even more with amacrine. In addition of the advance in the temporal peaks, we can also see spatial peaks closer to the position of the bar. The latency slope can be seen on the early part of the temporal profile and the distal part of the spatial profile. This slope is reduced with amacrine.

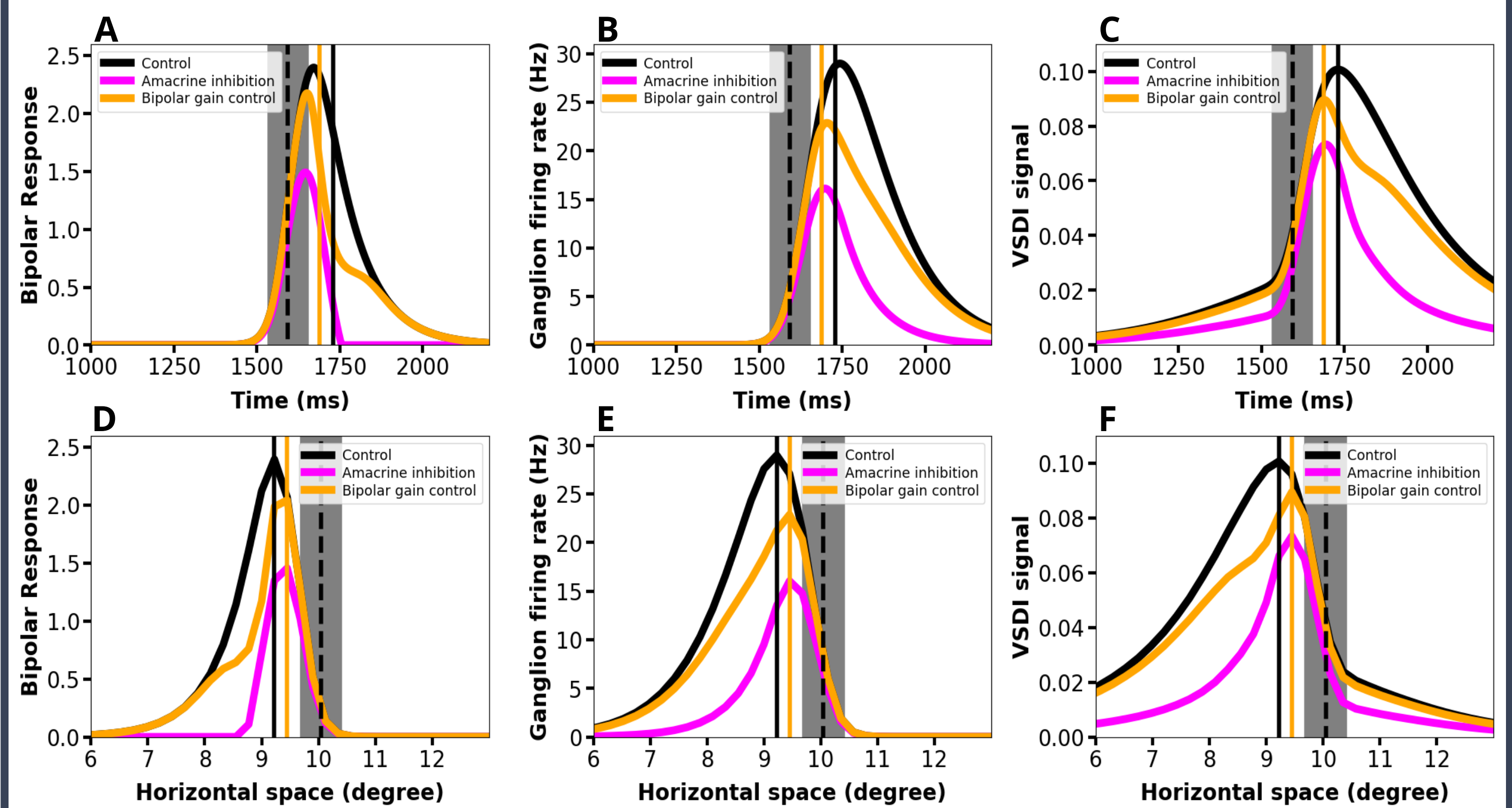


Fig 5 : Effect of amacrine and bipolar gain control on the spatio-temporal profile of retino-cortical responses. Line A, B, C is the temporal profile and line D, E, F is the horizontal spatial profile. The first column (A, D) is the bipolar response, the second column (B, E) is the ganglion firing rate and the third column (C, F) is the VSDI signal. Each sub-figure contains a control curve (black), with amacrine connected to bipolars (magenta) and with bipolar gain control (orange). The grey rectangle with its central dotted black bar in A, B and C corresponds to the time at which the bar is in contact with the centre of the receiving field. Those in D, E and F correspond to the position of the bar at the current time.

Conclusions & Perspectives

In this poster, we proposed a retino-cortical model implemented in our simulator software. With them, we are capable of acting on both retinal and cortical parameters with simple, changeable stimuli. We can consider more elaborate retinal models or models from different species.

This model reproduces Benvenuti's optical imaging experiments [3]. It also describes the influence of retinal anticipation on cortical anticipation. In the presence of amacrine-bipolar connectivity or bipolar gain control, retinal adaptive anticipation is transferred to the cortex. In addition, this amacrine connectivity to the nearest neighbour reduces cortical anticipation by latency.

We would now like to continue our experiments with the reproduction of anticipation by prediction in the retina. To do this, we need to obtain symmetrical biphasic amacrine cells [2]. Unlike anticipation by adaptation, we think that the effect of anticipation by prediction could reinforce cortical anticipation by latency.

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