# Layer 4C in Monkey V1 May Improve the Information Content of Simple Cell Responses: A Hypothesis

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**Abstract**

We hypothesize that the enormous number of cells in layer 4C of monkey primary visual cortex provides a "hidden layer" that inverts the saturating nonlinearity of LGN inputs, improving the match between visual signals and the dynamic range of simple cells in cortical layers 2-3. This is important to do at low spatial frequencies where the pooling of large numbers of LGN inputs results in a high signal to noise ratio. This only works if the noise added by the hidden layer is significantly smaller than that present at the low spatial frequencies, which is achieved by using a large number of neurons.

Reasonable functional interpretations have been given for the physiological properties of several cell populations in the primate early visual pathway. However, in layer 4C of the primary visual cortex, between the LGN afferents and the simple cells in layers 2-3, there is a massive population of tiny, densely-packed "spiny stellate" cells whose reason for existence has thus far remained a puzzle.

LGN afferents to layer 4C exhibit a striking fanout, each LGN fiber synapsing onto 30-100 of the spiny stellate neurons [1]. Histologically, layer 4C is characterized by a precise patterning of spiny stellate dendritic arbors and LGN termination sites, which is highly sensitive to afferent activity during development [4]. These facts underscore an enormous investment of resources, and strongly suggest some important functional role for visual cortical layer 4C in processing sensory information on its way to simple cells.

At other stages in the visual hierarchy, new computations are reflected in a progressively more complex selectivity of cellular responses for features of the visual input. However, layer 4C cells appear to simply inherit the receptive fields of their LGN drivers. This fact, coupled with difficulties in collecting detailed physiological data due to the small size of spiny stellate cells, has rendered giving a computational explanation for layer 4C difficult. There have been two main previous attempts. First, based on the large fanout of incoming LGN fibers, Horace Barlow suggested that layer 4C might serve as an "interpolating grid" [1]. Image samples conveyed by LGN afferents would be interpolated by

the more numerous,densely-packed spiny stellate cells, and this higher resolution neural image representation would then serve as the readout underlying visual hyperacuity. In another hypthesis, Olshausen, Anderson and Van Essen proposed that layer 4C might serve as a grid on which rescaling and rerouting of visual information takes place, as part of the operation of a "shifter circuit" [3]. Unfortunately, subsequent experimental work has failed to validate key predictions of either proposal (c.f. [1]).

In this paper we outline a new proposal for the function of layer 4C. To motivate our proposal, we first discuss in detail an often overlooked problem encountered in early visual processing, namely, that visual signals undergo *nonlinear distortion* during transmission from retina to cortex. This fact at first appears inconsistent with the well-known observation that simple cells show an essentially *linear* dependence on visual inputs. By what mechanism is linearity achieved, and why? *We propose that layer 4C functions to reverse the distortion, and by doing so, prevents the loss of visual information.* We provide illustrations from computer simulations to demonstrate the principles of our proposal, and highlight ways in which our hypothesis may help to explain the experimentally observed linearity of simple cell physiology.

The early stages of visual processing admit an essentially linear description. That is, in response to images projected on the retina, the firing rates of ganglion-, lateral geniculate-, spiny stellate-, and simple cells are well predicted using models which compute the inner product (spatial integral of the product) of a linear filter with the incoming image, and pass the result through a static nonlinear function. We use the notation $\mathcal{I}, \mathcal{F}$, and $g$ for the image, linear filter, and static nonlinearity, respectively. Then we express a neural firing rate in in terms of the input image as

$$r(\mathcal{I}) = g[< \mathcal{I}, \mathcal{F} > -\theta]$$

where $\theta$ is a threshold, and $<,>$ denotes the inner product. Here we have neglected the effects of noise, but will discuss these later. For simple cells, the underlying linear filter is usually described as a Gaussian-weighted sine wave, i.e. a Gabor function. Current evidence suggests that these receptive fields arise basically in a feedforward fashion from alternating stripes of ON and OFF centers of LGN receptive fields, as originally proposed by Hubel and Wiesel [9]. However, comletely explaining the linear behavior of simple cells with the Hubel and Wiesel model is complicated by the presence of the static nonlinearity in the retinal ganglion and LGN cells, $g$. Separate studies by Troyer *et al [6]* and Wielard *et al [7]* have addressed some aspects of this issue in detail. These authors took $g$ for the LGN afferents to be a simple rectification, i.e.

$$g(x) = [x]_+ = \begin{cases} x & x \geq 0 \\ 0 & x < 0 \end{cases} ,$$

reflecting the constraint that neural firing rates are nonnegative, and developed detailed mechanistic models to explain how simple cell linearity can arise in spite of the rectifying nonlinearity of the LGN input. However, there is evidence for

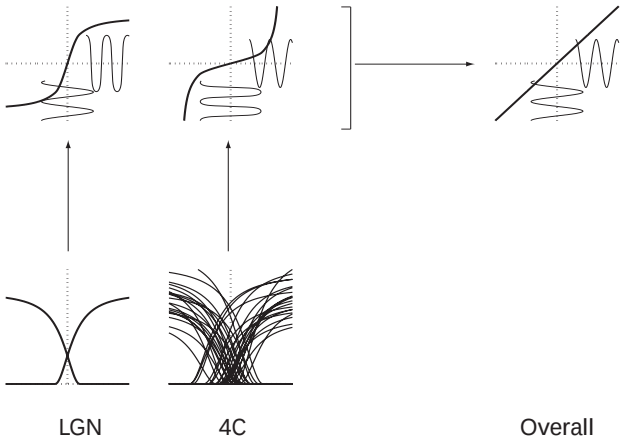


Figure 1: Bottom: Response curves for an ON/OFF pair of LGN relay neurons (left) and their postsynaptic layer 4C targets. Top: Amplitude transfer function for visual signals using responses of the ON/OFF pair (left), and using layer 4C as a distortion inverter (middle). Combining these in tandem yields an overall linear transfer (right).

an additional nonlinearity of LGN responses: saturation. Thus, a more accurate model for $g$ is the thresholded sigmoid (see fig. 1). For convenience, our curves our plotted using the model $g(x) = kx^2/(\sigma + x^2)$, where $k$ and $\sigma$ are constants determining the maximum firing rate and half saturation level.

The essential problem introduced by saturation is illustrated in fig (1). Consider a time-varying sinusoidal signal transmitted down the retinogeniculate pathway by pairs of ON and OFF RGCs and LGN RCs. As shown on the left in figure 1, the ON and OFF responses jointly capture both the positive and negative components of the visual signal. Saturation at high signal amplitudes produces a compressed sinewave as the output of RGCs, and the effect is cascaded through the LGN to produce more pronounced distortion in the output of the ON/OFF pair of LGN cells. In the following paragraphs we first explain how layer 4C can "linearize" the overall transfer of signals from the retina to simple cells by reversing the effects of saturation. We postpone addressing the *benefits* of linearization to allow us to frame the discussion in the context of the hypothetical question, "what if layer 4C did not exist?"

The implementation of our hypothesis is simple. As stated above, each LGN cell arborizes to contact 30-100 layer 4C cells. Due to this direct feedforward connection, each spiny stellate cell will inherit the basic ON or OFF, difference-of-Gaussians-shaped receptive field of its presynaptic LGN afferent. Suppose, however, that there is moderate diversity amongst either the form of the static nonlinearities $g$ for the cells, e.g. different maximum firing rates, or some scatter among the thresholds $\theta_i$. Then by taking an appropriate synaptically weighted sum of ON and OFF spiny stellate cell inputs to a simple cell, the overall

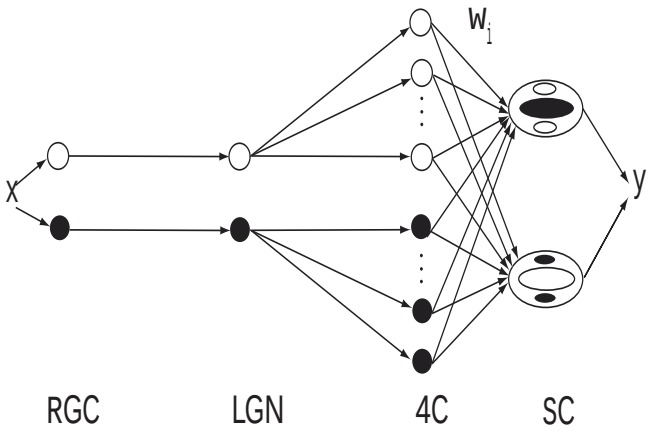


Figure 2: Schematic of the feedforward neuronal implementation of our proposal. Pairs of neurons are used to represent both positive and negative signal components.

amplitude transfer function characterizing the relay LGN to the simple cell input can be made to approximate the inverse of the preceeding distortion function, achieving an overall linear transfer of the visual signal to the target simple cell. In other words, we propose that layer 4C serves as a "hidden layer," in which the response curves of its neurons serve as basis functions with which the inverse of the RGC/LGN amplitude transfer function is computed. For our example, we have used 30 ON and 30 OFF neurons, and have computed weights $w_i$ by the method of least squares, i.e. by minimizing

$$E(\{w_i\}) < ||g^{-1}(x) - \sum_i w_i a_i(x)||^2 >_x,$$

where $a_i$ are the response functions ("$g$'s") for layer 4 cells, $x =< \mathcal{I}, \mathcal{F} >$, and $<,>_x$ indicates an average over the ensemble of possible scalar inputs x to the cells, which for simplicity we assumed as uniform. In this case, the simple cell response will not show evidence of the distortion, but will have a linear response, consistent with experimental observations. It should noted that a fully plausible implementation of the circuit cannot be purely feedforward. In particular, the inhibitory interneurons present in cortex (about 25% of cortical neurons) are required to implement negative-valued weights used in the model. Also, our model is not incompatible with a role for intracortical interactions between simple cells in contributing to simple cell linearity, as proposed in [6] and [7] to deal with LGN rectification, mentioned above. For clarity, we supress these details in most of the discussion, as they are not needed to convey the basic principle of our hypothesis.

We now discuss the benefits of removing distortions, which we refer to as "linearization." The most immediate benefit is that linearization reformats LGN

signals in such a way that the responses of simple cells satruate less. The consequence is that simple cell responses provide more information about the initial retinal input than if they were directly fed the LGN output. To demonstrate this point, we performed the following numerical experiments: Suppose that the visual input to the static nonlinearity $g$ of a single ON/OFF pair of RGC is a scalar valued, zero-mean random variable $x$, with variance $\sigma^2$; i.e., $x \sim \mathcal{N}(0, \sigma^2)$. The variance is chosen so that the RGC and LGN responses occasionally saturate. Also, assume that at each synapse, the signal is corrupted by additive Gaussian noise, where the noise sources at each stage are independent. (The same qualitative findings also hold for multiplicative noise, not shown). The firing rate of an "antiphase pair" of simple cells (obtained by taking the difference of the responses of the simple cells in fig. 2) is then a (non-Gaussian) random variable $y$.

We produced $1,000,000$ random sample input/output pairs $(x, y)$ and used these to form Monte-Carlo histogram estimates of the probability densities $p(y)$ and $p(y|x)$. We used these histograms and the assumed pdf for $x$, $p(x)$, to compute the Shannon information (mutual information) between the visual signals and the firing rates of the model simple cell pair, given by

$$I(y; x) = \sum_{x,y} p(x)p(y|x) \log \frac{p(y|x)}{p(y)}.$$

We repeated these experiments for two cases: with and without the layer 4C "inverter," for several different noise levels. We find generally that so long as the noise involved in the inversion process is small compared to the noise already present in the incoming signals, linearization by layer 4C produces substantial improvements in the overall information transfer, $I(y; x)$. We find increases of between $30 - 100\%$ for various noise levels. Notice that the benefits will be greatest for simple cells responding to low spatial frequency information. This is because these simple cells presumably pool signals from a larger number of layer 4C spiny stellate cells, so that computing the inverse becomes an almost noiseless computation.

More speculatively, we suppose that distortion inversion for low spatial frequency information is important for visual perception. Most of the information in natural images resides in the low spatial frequency bands; this high signal to noise level makes fine discrimination possible, so that residual distortions would be noticeable if not inverted.

We have outlined a novel computational hypothesis for the function of layer 4C in primate visual cortex. Details we have not discussed include the role of interneurons; these in fact play an important role in the fully detailed implentation the layer 4C "hidden layer," since some of the needed weights are negative. Also, the mechanism we propose for making linear simple cells is not exclusive; the mechanisms proposed in [6] and [7] may cooperate with layer 4C distortion inversion to produce linear simple cell responses. Some explicit testable predictions of our hypothesis are that there exists a diversity of neural response curves in layer 4C. To our knowledge, the data to confirm of refute this pre-

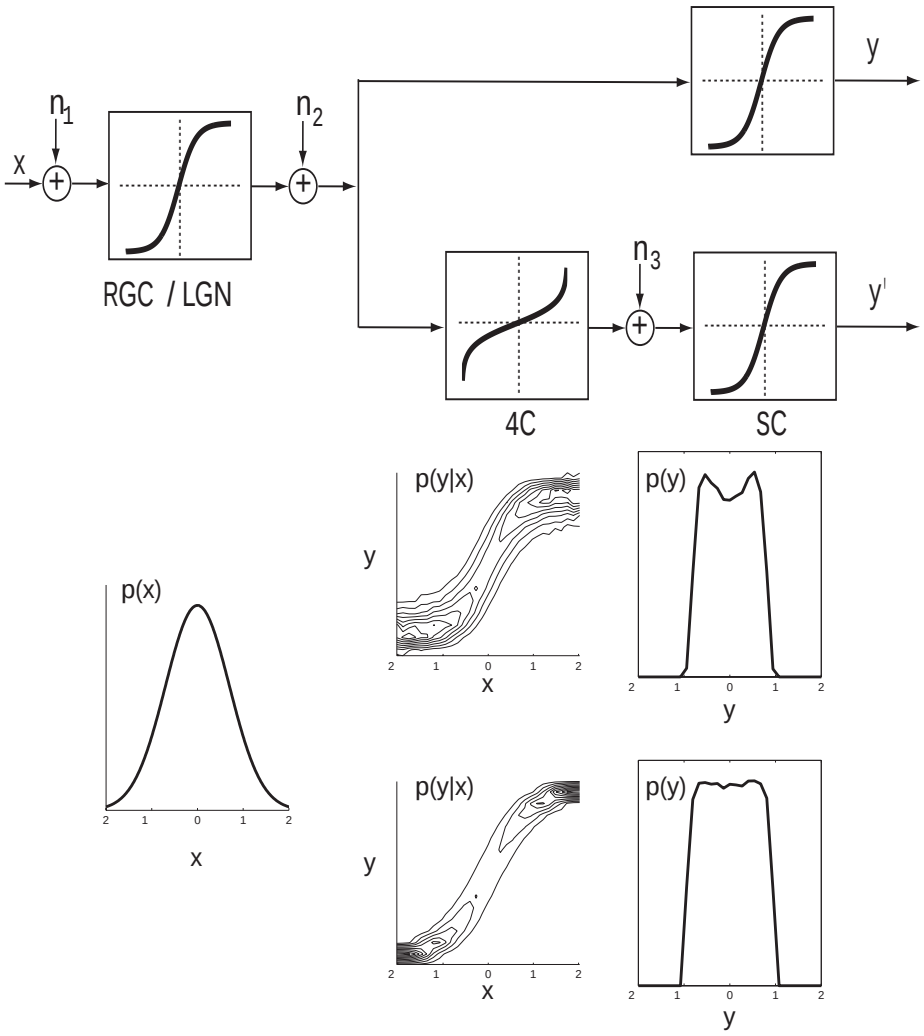


Figure 3: Top: Block diagram for the numerical experiments described in the text. Top pathway, without layer 4C; bottom pathway, layer 4C acting as distortion inverter. Bottom: Plots and contour plots for estimated pdf's. See text.

diction does not yet exist. A second predicition is that saturation of responses of RGCs and LGN cells will be a significant problem under natural operating conditions, providing the need for linearization. While there is evidence that staturation does occur, there has been no systematic study of its severity under normal visual conditions. A third is that the information delivered to individual simple cells sensitive to low spatial frequency information will have lower noise levels and lower distortion than simple cells responding to high frequencies, due to pooling over more layer 4C inputs. Finally, the wiring details of the primary visual cortex are not completely known; our hypothesis assumes that simple cells receive the majority of their connections via spiny stellate cells from layer 4C, rather than monosynaptically from the LGN, which remains to be tested.

A final interesting related question for further study is whether a similar operation takes place in cats, which lack the densely packed layer 4C found in primates [1]. Instead, a similar though less dramatic fanout ($1 \rightarrow 4-10$ in cats vs $1 \rightarrow 30-100$ in primates) occurs in cat LGN. We hypothesize that this fanout in cat LGN may serve the same purpose of distortion inversion we have hypothesized for layer 4C in primates.

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