In Press:
Progress in Neurobiology

Towards building a more complex view of the lateral geniculate nucleus: recent advances in understanding its role

Masoud Ghodrati[1,2], Seyed-Mahdi Khaligh-Razavi[3,4], Sidney R. Lehky[5*]

[1] School of Cognitive Sciences
Institute for Research in Fundamental Sciences (IPM)
Niavaran, Tehran, Iran

[2] Department of Physiology, Monash University,
Melbourne, VIC, Australia

[3] Computer Science and Artificial Intelligence Laboratory,
Massachusetts Institute of Technology,
Cambridge, Massachusetts, USA.

[4] Royan Institute for Brain and Cognitive Sciences,
Tehran, Iran

[5] Computational Neurobiology Laboratory
The Salk Institute
La Jolla, California, USA

30,680 words

*Corresponding:

Sidney R. Lehky
Computational Neurobiology Laboratory
The Salk Institute
10010 N. Torrey Pines Road
La Jolla, CA 92037
USA
sidney@salk.edu

Contents

1 Abstract

The lateral geniculate nucleus (LGN) has often been treated in the past as a linear filter that adds little to retinal processing of visual inputs. Here we review anatomical, neurophysiological, brain imaging, and modeling studies that have in recent years built up a much more complex view of LGN. These include effects related to nonlinear dendritic processing, cortical feedback, synchrony and oscillations across LGN populations, as well as involvement of LGN in higher level cognitive processing. Although recent studies have provided valuable insights into early visual processing including the role of LGN, a unified model of LGN responses to real-world objects has not yet been developed. In the light of recent data, we suggest that the role of LGN deserves more careful consideration in developing models of high-level visual processing.

2 Introduction

We shall be reviewing the literature on the lateral geniculate nucleus (LGN), with a particular interest in its possible cognitive and computational role in higher vision, such as object recognition. Coming from computational backgrounds, we are interested in including a comprehensive summary of recent advances in LGN neurobiology that may assist those interested in developing more advanced and realistic modeling. As we are not proselytizing a specific theory of LGN function, the range of topics covered will be rather unfiltered and eclectic, with the hopes that this will foster a cross-fertilization in future experimental and theoretical work.

2.1 The lateral geniculate as a relay nucleus

The LGN, interposed between retina and primary visual cortex, has traditionally been characterized as a "relay nucleus" joining the two. What does it mean to be a "relay nucleus" in the brain? Perhaps the concept arose by analogy with 19th century telegraph relay stations that were placed at intervals along the line to boost signal strength attenuated by resistance in the wires. However, the demonstration by Adrian (1926) of the regenerative nature of neural action potentials, which did not attenuate with distance, showed that the brain did not need a "relay nucleus" in that sense. Despite that, the term has continued in use for decades, a vacuous placeholder for lack of a compelling theory of LGN function.

Another more recent concept of a relay, besides being an amplifier, is as a gating device in which a small signal controls a large signal, switching it on and off, as in an electromechanical relay or a transistor switch. We find this second concept of "relay" emphasized in ideas about LGN function developed several decades ago. Specifically,

modulatory inputs from brainstem nuclei to LGN are believed to behave as an on/off switch for transmission of signals from retina to cortex during sleep (Burke and Cole, 1978; McCormick and Bal, 1997; Singer, 1977; Steriade *et al.*, 1993).

While the idea of LGN as a gating relay associated with sleep/wakefulness certainly has merit, the elaborate anatomical organization of the LGN together with its known physiological response properties suggest that it also has a role in computationally *transforming* or *non-linearly filtering* visual signals that goes beyond simply *relaying* them. While the purpose of the LGN still remains a mystery, we hope that by emphasizing the literature related to visual functions in the awake state we shall be in a position to clarify and highlight the more central issues that need to be resolved in the future.

People used to think of the LGN as a simple, largely linear, early stage of visual processing whose details we do not have to worry about in the context of higher-level functions, like object-vision and attention. However, recent studies suggest that LGN plays a more active role in visual information processing (e.g. Andolina *et al.*, 2013; Briggs *et al.*, 2013; Cudeiro and Sillito, 2006; Kastner *et al.*, 2004; Kastner *et al.*, 2006; McAlonan *et al.*, 2008; O'Connor *et al.*, 2002; Saalmann and Kastner, 2009, 2011; Sillito *et al.*, 2006). This review links between neurophysiological, neuroimaging, and computational studies about the LGN. We also cover modeling studies of LGN neurons and networks, a topic that has not been extensively reviewed before. The modeling part in particular defines a roadmap to be considered for future modeling efforts. It has an impact not only on modeling the early visual processes but also on object-vision models of higher visual areas. We argue how considering more realistic models of LGN can help

to boost the performance of high-level object-vision models. In essence, we suggest a general framework in which a computational model of the LGN can be considered as an additional layer before the first layer of biologically inspired hierarchical models of object recognition (Dura-Bernal *et al.*, 2012; Ghodrati *et al.*, 2012; Rajaei *et al.*, 2012; Riesenhuber and Poggio, 1999; Serre *et al.*, 2007a; Wallis and Rolls, 1997).

2.2 Evolutionary constraints affecting LGN organization

Developmental constraints reflecting the evolutionary history of biological structures are likely to be a force for conservatism in their organization (Olson, 2012). Such constraints hinder radical reorganizations of those structures even when arrangements that are more efficient become possible as new contexts arise. The general topology of neural connections between retina, LGN, and cortex currently in mammals appears to have conserved patterns established at least three hundred million years earlier, amongst precursor structures that existed in the common ancestor to all amniotes (Butler, 1994a, b; Hofmann and Northcutt, 2012; Northcutt, 2011). Amniotes originated in the carboniferous era and comprise reptiles, birds, and mammals, all of which have a direct projection from retina to a dorsal thalamic nucleus, which in turn projected to the pallium (from which neocortex originated). The pallium has then sent a reciprocal projection back to the thalamic nucleus, with this feedback likely in existence since at least the common ancestor of tetrapods (amphibians, reptiles, birds, and mammals), and possibly earlier to include all jawed vertebrates, as data on feedback projections are incomplete. Thus, the general connectivity of the geniculocortical system has very ancient roots (Butler, 2008).

What is now mammalian LGN and striate cortex co-evolved over an extended period from something that originally had quite different functions [possibly with an olfactory dominated pallium (Hofmann and Northcutt, 2012)]. As the two structures are a co-evolved, coupled nonlinear dynamical system, functionality may have become distributed across them in a manner that makes it difficult to assign discrete, separable tasks to each one. Possibly a clean-slate design of the visual system, which ignores evolutionary history, might find it more efficient to have retina project directly to cortex without an interposed nucleus, with LGN functionality produced by a more elaborated cortical microcircuitry.

2.3 General anatomy and physiology

LGN is a structure in the dorsal thalamus that carries visual information from retina to the primary visual cortex (striate cortex, or V1) – see Figures 1 and 2. "Geniculate", derived from Latin, means bent like a knee. Activity in the LGN is driven by input from retinal ganglion cells, which synapse directly onto LGN thalamocortical cells. The thalamocortical cells are the principal output neurons from LGN, projecting to primary visual cortex. Thus, there is a disynaptic pathway from retina to cortex, RGC→ thalamocortical cell → V1. The retina is the sole driver of thalamocortical cell activity. All other LGN inputs modulate that activity but cannot drive the thalamocortical cells (though recent data report exceptions, as described in Section 5). Those modulatory inputs include inhibitory interneurons located within the LGN, as well as feedback from striate cortex, feedback from the thalamic reticular nucleus (TRN), and inputs from various brain stem structures. Early work in LGN has been comprehensively reviewed by Singer (1977). The lateral geniculate nucleus is considered a *first-order* thalamic

nucleus, as it receives its major input from an ascending subcortical pathway (in this case from retina), as opposed to *higher-order* thalamic nuclei that receive their driving inputs from descending pathways originating from cortex (Guillery and Sherman, 2002a, b).

There is considerable species variability in thalamocortical organization of mammals. Here we are most strongly interested in various monkeys as they most closely resemble humans, as well as the small but growing human fMRI literature, while also drawing on the massive amount of data from carnivores (cats). Rats and mice, the center of rapidly increasing research efforts in recent years, will be given less coverage, primarily at the level of biophysical properties of individual neurons rather than at the system level. Occasional reference will be made to non-visual thalamocortical interactions when they seem likely to generalize.

The significance of the various differences between species is not clear at the computational level. Referring to Marr's three levels of analysis (Marr, 1982), such differences may be significant at the computational level, such that the visual system is computing different things depending on the relevant features of each species' ecological niche. Alternatively, species differences may be significant at the algorithmic level, such that the visual system retains the same computational goal, but has found different algorithmic solutions to that same goal. Finally, species differences may be significant at the implementation level, so that the same algorithm is being implemented in different ways. Undoubtedly, all three of these come into play, depending on how large the evolutionary divergence is between species.

Three particularly noteworthy features of the LGN from our perspective are: 1. A laminated structure in primates and carnivores; 2. Spatial receptive fields that are very

similar to those of retinal inputs, but temporal properties that are substantially different from retina; 3. Massive feedback from cortex, in which the number of feedback synapses far exceeds the feedforward input from retina. Early observations of strong similarities between LGN and retinal cells caused puzzlement over the purpose of LGN, and motivated many lines of research trying to establish interesting differences between the two.

2.3.1 Laminations

The LGN in catarrhine primates (Old World monkeys, apes, humans) has six layers, except for gibbons (de Sousa *et al.*, 2013; Kaas *et al.*, 1978) (Figure 1). These six layers are divided into four dorsal parvocellular layers and two ventral magnocellular layers (de Sousa *et al.*, 2013; Hickey and Guillery, 1979; Kaas *et al.*, 1978; Livingstone and Hubel, 1988; Malpeli *et al.*, 1996). In addition to the laminar segregation of magnocellular and parvocellular neurons, there is also laminar segregation of on-center and off-center neurons. Cells in the two more dorsally located parvocellular layers in macaques are mostly on-center, while those in the two more ventrally located parvocellular layers are mostly off-center (Schiller and Malpeli, 1978). The two classes of cells are mixed in the magnocellular layers.

Each layer in the LGN is monocularly driven from inputs of one hemiretina, either from the ipsilateral or contralateral eye, covering the contralateral visual field. For example, right LGN receives inputs from the nasal hemiretina of the right eye, covering the left visual field. These ipsilateral inputs innervate layers 1, 4, and 6 of the LGN. The other layers in the right LGN (2, 3, and 5) receive contralateral inputs from the temporal hemiretina of the left eye, also covering the left visual field; see Figure 2a. All layers are

retinotopically aligned; so, a line drawn perpendicularly to the layers passes through receptive fields covering the same spot of the visual field.

Representation of the central visual field encompasses a disproportionately large fraction of LGN tissue volume. The volume of tissue per solid visual angle rapidly declines as eccentricity increases. That volume can be quantified by the *magnification factor* (mm^3/steradian), measured both by neurophysiological mapping in monkeys (Malpeli and Baker, 1975; Malpeli *et al.*, 1996; Sanderson, 1971b; Schein and de Monasterio, 1987) and by human fMRI (Schneider *et al.*, 2004).

There is little in the way of direct connections between LGN layers (Guillery, 1966; Wong-Riley, 1972), with just a few dendrites from cells near the laminar borders extending into the adjacent lamina (Guillery, 1966; Saini and Garey, 1981). That is in sharp contrast with cortex, where there are extensive axonal and dendritic interlaminar connections (Callaway, 1998). However, coordination between layers of a sort is provided by axons from cortical feedback neurons (as well as axons from TRN (Bragg *et al.*, 2017)), which can traverse straight across several layers (Ichida and Casagrande, 2002; Kaas *et al.*, 1972; Wang *et al.*, 2001), like "toothpicks in a club sandwich" in the memorable phrase of Walls (1953).

Laminar organization varies greatly across mammalian orders (Campbell, 1972; Striedter, 2005). For example, small rodents often have poorly demarcated layers (Reese, 1988), while laminations are quite distinct not only in primates, but carnivores (such as cats) and a few marsupials as well. Where laminations exist, it is variable whether magno/parvo analogues are segregated or mixed, and whether on- and off-center cells are segregated or mixed (Conley, 1988). The scattered occurrence of well-defined LGN

lamination in different mammalian orders indicates that such layering evolved independently at least three times (Striedter, 2005). Repeated independent evolution of the same structural organization suggests that a particular problem is being addressed by LGN laminations, although we don't know what that problem is. Campbell (1972) has suggested that laminations occur in mammals specialized for rapid movements of various sorts, facing the challenge of rapid evaluation of spatial relations including depth perception. Along similar lines McIlwain (1995) has suggested that LGN laminations are involved in stereoscopic vision and Kaas *et al.* (1972) also suggests laminations are connected to binocular vision. The role of laminations in LGN functionality has largely been ignored by LGN theoreticians.

2.4 Response properties of LGN cells

There are three major classes of thalamocortical cells in monkey LGN: the parvocellular (P), the magnocellular (M) and the koniocellular (K) cells (Casagrande, 1994; Kaas *et al.*, 1978; Xu *et al.*, 2001). In cats there are also three classes, X, Y, and W (So and Shapley, 1979; Sur and Sherman, 1982). The relationship between the monkey and cat classes are not entirely clear and will be discussed further later on.

In the sections below we shall first outline general spatial and temporal properties of LGN cells that seems general to the three classes, then look specifically at differences between the classes, and then finally describe various other major characteristics of LGN responses.

2.4.1 Spatial properties of thalamocortical cell receptive fields

The receptive fields of LGN thalamocortical cells in all three classes are approximately circular and have a concentric center/surround organization for luminance

(Hubel and Wiesel, 1961; Irvin *et al.*, 1993). Receptive fields of intrinsic inhibitory interneurons are similarly organized (Dubin and Cleland, 1977; Wang *et al.*, 2011b; Wang *et al.*, 2007). On-center cells are maximally excited by a bright spot in the center and a dark annular surround. Reversing the contrast polarities (dark center, bright surround) will often inhibit the on-center cells relative to spontaneous activity, so that there is a push-pull pattern in the responses (Wang *et al.*, 2011a). Off-center cells behave analogously to on-center cells when contrast polarities are reversed (i.e. maximally stimulated by a dark center and bright surround, and so forth).

The center-surround receptive field organization of LGN cells is commonly described in the experimental literature by a Difference of Gaussians (DoG) function, introduced by Rodieck (1965) and Enroth-Cugell and Robson (1966). Various alternative mathematical descriptions of the curve are found in the modeling literature, notably the Laplacian (second derivative) of a Gaussian (e.g. Lindeberg, 2013; Marr and Hildreth, 1980), which closely resembles a DoG, as well as others, as will be outlined in Section 8.2.

The DoG function is a linear description of the spatial receptive field, as is the Laplacian. Although spatial receptive fields in LGN are frequently described as linear in textbook descriptions, there are cells that show nonlinear spatial summation. In carnivores (predominantly cat studies), while X cells have a linear spatial response, Y cells have a nonlinear response (Kaplan and Shapley, 1982; Price and Morgan, 1987; Shapley and Hochstein, 1975; So and Shapley, 1979). The Y cell spatial nonlinearity is inherited from the retina (Hochstein and Shapley, 1976), which has cells with similar properties. The situation is less clear in monkeys. Everyone agrees all P cells are linear.

However, some studies report that a minority of M cells are nonlinear (Blakemore and Vital-Durand, 1986; Levitt *et al.*, 2001), while others report that all M cells are linear (Derrington and Lennie, 1984; Usrey and Reid, 2000; Xu *et al.*, 2001). Therefore, here we see that the analogy made earlier between cat X and Y cells with monkey M and P cells may break down with regard to the spatial linearity of responses. The function of intrinsic spatial nonlinearities that LGN thalamocortical cells may inherit from retina has not attracted much theoretical interest. Models generally treat the thalamocortical cells as linear spatial filters, with any nonlinearities arising within LGN through network effects.

The spatial center-surround organization in the receptive fields of LGN output (thalamocortical cells) is very similar to that of LGN input (retinal ganglion cells). The main difference is that LGN cells have a stronger and broader surround modulation than retinal ganglion cells (Bullier and Norton, 1979; Cleland *et al.*, 1971b; Hubel and Wiesel, 1961; Ruksenas *et al.*, 2000; Sillito and Kemp, 1983; Singer and Creutzfeldt, 1970; Usrey *et al.*, 1999). The surround modulation is mediated through enhanced inhibitory interactions upon thalamocortical cells (Kimura *et al.*, 2013; Ruksenas *et al.*, 2000; Sillito and Kemp, 1983; Singer *et al.*, 1972) which may include contributions from both feedforward inhibition within LGN and feedback inhibition from the TRN (Lindström and Wróbel, 2011). The stronger surround inhibition has the effect of converting spatial low pass filtering in retina to moderate band-pass filtering in LGN (Cheng *et al.*, 1995; Maffei and Fiorentini, 1973; Shapley *et al.*, 1981).

We said LGN receptive fields are “approximately circular”. There actually appears to be weak orientation bias in the receptive fields of M and P cells in monkeys as well as X and Y cells in cats (Ahmed and Hammond, 1991; Ichida and Casagrande, 2002;

Kremers and Weiss, 1997; Naito *et al.*, 2013; Podvigin *et al.*, 2001; Shou *et al.*, 1986; Shou and Leventhal, 1989; Soodak *et al.*, 1987; Suematsu *et al.*, 2012; Thompson *et al.*, 1994a; Thompson *et al.*, 1994b; Vidyasagar, 1984; Vidyasagar *et al.*, 2015; Vidyasagar and Urbas, 1982; Viswanathan *et al.*, 2011, 2015; Xu *et al.*, 2002b). Possible sources of orientation bias may be inheritance from the retina (Podvigin *et al.*, 1992; Soodak *et al.*, 1987) together with sharpening by intrageniculate inhibition (Podvigin *et al.*, 1992; Vidyasagar, 1984; Vidyasagar and Urbas, 1982). (Although keep in mind that a circular receptive field will show weak orientation selectivity if the stimulus is not exactly centered (Yakimova and Chizhov, 2015).) A subset of LGN K cells shows strong orientation selectivity (Cheong *et al.*, 2013).

Thus, in contrast to the standard textbook view that LGN receptive fields are circular, we see extensive evidence of at least moderate orientation selectivity. The question is whether this orientation selectivity is functional, or whether it represents developmental noise (as you wouldn't expect receptive fields that are *perfectly* circular). One possibility is that the weak LGN orientation tuning may serve as the foundation of strong orientation tuning in striate cortex, through being sharpened by striate intracortical inhibition (e.g., Kuhlmann and Vidyasagar, 2011; Xu *et al.*, 2002b). We shall take up the issue of LGN orientation tuning again looking at models in Section 8.2.

2.4.2 Temporal properties of thalamocortical cell receptive fields

LGN cells have a biphasic temporal impulse response function, which causes spatial organization to reverse polarity over time. For example, in an ON center cell the initial phase of the response to a spot of light is excitatory, followed by a weaker inhibitory phase (Cai *et al.*, 1997; Reid *et al.*, 1997). Thinking in terms of a unitary

spatiotemporal receptive field, the temporal aspect of the response causes the shape of the spatial receptive field to evolve over time (DeAngelis *et al.*, 1995). Also, the LGN temporal response itself can vary dynamically over time (Mukherjee and Kaplan, 1995), so that LGN may function as a tunable temporal filter.

Differences between retina and LGN responses are more pronounced in the temporal domain than in the spatial domain. Relative to the biphasic temporal impulse responses in retina, the second (rebound) phase of LGN biphasic responses have a larger amplitude (Stevens and Gerstein, 1976; Usrey *et al.*, 1999), analogous to stronger inhibitory surround in the spatial domain. The increased transience of LGN impulse responses is also seen in step responses (Cleland and Lee, 1985; Mukherjee and Kaplan, 1995). As expected, greater transience of LGN responses in the time domain is reflected in attenuated responses to low temporal frequency stimuli, causing a shift from essentially low-pass filtering in retina to a more band-pass filtering in LGN (Hamamoto *et al.*, 1994; Kaplan *et al.*, 1987; Levine and Troy, 1986). Increased transience of LGN responses relative to retina may be due to inhibitory interactions from intrinsic neurons within the LGN. Another contributing factor may be enhancement in synaptic transmission efficacy from retina to LGN when interspike intervals are short (Section 3.1). This biophysical synaptic mechanism acts as a high pass temporal filter.

Interestingly, while LGN temporal responses are high-pass filtered relative to retina, responses are then severely low-pass filtered going from LGN to cortex, as seen in both physiological (Hawken *et al.*, 1996) and fMRI (Bayram *et al.*, 2016) studies. Such low-pass filtering has been modeled in terms of biophysical properties of the thalamocortical synapse (Henke *et al.*, 2014; Krukowski and Miller, 2001). While the

retinothalamic high-pass filtering might be understood in terms of efficient coding (see below, Dong and Atick (1995)) it seems less likely that an efficient coding argument could be made for the thalamocortical low-pass filtering. Placing low-pass and high-pass filters in series creates a bandpass filter. It may be that the purpose of this arrangement in LGN is to band-pass filter stimuli to accentuate the range of temporal frequencies having greatest ecological relevance for the behavior of the animal.

The surround responses of LGN receptive field have longer latencies than the centers by several milliseconds (Cai *et al.*, 1997; Dawis *et al.*, 1984), due to extra synaptic delay in the inhibitory contribution to the surround. A weaker surround during the early part of the LGN response affects spatial tuning of the cells, so that they respond better to low spatial frequencies. The lag in the development of surround inhibition may therefore contribute to a coarse-to-fine temporal sequence in early visual processing of spatial patterns (Allen and Freeman, 2006; Moore *et al.*, 2014; Purushothaman *et al.*, 2014).

Motion selectivity, similar to orientation selectivity (Section 2.4.1), is another property found in some LGN cells that is not part of the conventional picture of LGN processing. Cells selective for the direction of motion have been reported in both cat and monkey (Hu *et al.*, 2000; Thompson *et al.*, 1994a; Thompson *et al.*, 1994b; Xu *et al.*, 2002b). They are more common in mice, as will be described in Section 2.4.7. The occurrence of these LGN cells raises the possibility that motion selectivity in striate cortex might represent to some extent the sharpening of tuning inherited from LGN rather than being created entirely *de novo* in cortex, or it might reflect feedback from cortex.

A significant feature of LGN temporal activity not present in the retinal input is the occurrence of lagged and non-lagged responses, first reported by Mastronarde (1987). The start of stimulus responses for lagged cells are delayed 40-80 ms relative to non-lagged cells, indicative of a short leading suppression at the start the response (Humphrey and Weller, 1988; Mastronarde, 1987; Saul, 2008a; Saul and Humphrey, 1990; Wolfe and Palmer, 1998). In response to a sinusoidal stimulus, the responses of lagged cells are phase-lagged relative to the stimulus while those on non-lagged cells are phase-advanced. Very roughly, lagged and non-lagged cells can be thought of as analogous in the temporal domain to on- and off-center cells in the spatial domain.

Almost all lagged cells in cats are X cells, forming about a third of the X cell population (Humphrey and Weller, 1988; Mastronarde, 1987). Lagged Y cells do occur rarely (Mastronarde *et al.*, 1991). Lagged cells also have been reported in monkeys (Saul, 2008b). Although generally viewed as two classes of cells (e.g., Humphrey and Saul, 1992), Uhlrich *et al.* (1990) have alternatively suggested that lagged and non-lagged are two modes of response within a single class, switched under control of the parabrachial nucleus.

The mechanism for generating lagged cells is believed to be feedforward inhibition mediated by interneurons participating in synaptic triads within glomeruli (Vigeland *et al.*, 2013) (triads and glomeruli are discussed in Section 3.1). Within this triadic circuitry, the lagged LGN cell would receive both excitation and inhibition from the same retinal ganglion cell, with a slight delay in the arrival of inhibition because that pathway is disynaptic.

Regarding functional significance, modeling indicates that having lagged and non-lagged cells is critical for efficient coding in the temporal domain through temporal decorrelation of LGN population responses (Dong and Atick, 1995). Another function suggested for lagged and non-lagged cells is in producing directionally selectivity for motion in cortical cells (Saul and Humphrey, 1990). The direction selectivity idea is based on the model of Adelson and Bergen (1985) that constructs directionally selective cells from pairs of neurons whose spatiotemporal receptive fields are in quadrature phase (shifted by 90^o relative to each other). Although the idea that lagged and non-lagged cells combine to produce directionally selective cells is widely mentioned, it has been criticized by Wolfe and Palmer (1998) as well as Peterson *et al.* (2004) as being unworkable on the grounds that the temporal phase shift observed between lagged and non-lagged cells is too small to meet the needs of the quadrature pair model.

Looking further at the characteristics of a unitary spatiotemporal receptive field (Cai *et al.*, 1997; DeAngelis *et al.*, 1995; Eckhorn *et al.*, 1993; Ghazanfar and Nicolelis, 2001; Golomb *et al.*, 1994; Stevens and Gerstein, 1976), we briefly examine here the mathematical notion of spatiotemporal separability. The basic question is to what extent the spatial and temporal properties are linearly separable, as the occurrence of linear separability simplifies the interpretation of experimental data and the modeling of that data. Linearly separable means that the spatiotemporal receptive field R(s,t) can be expressed by multiplying a spatial response R(s) with a temporal response R(t); R(s,t) = R(s)*R(t). If a receptive field is linearly separable then the spatial receptive field does not change shape as a function of time, although the amplitude of the spatial receptive field response is modulated by the temporal response function.

To a rough first approximation, LGN receptive fields may be treated as linearly separable. The primary deviation arises from the previously mentioned fact that the surround activity lags behind the center activity, causing the receptive field shape to change with time during early phases of the response. Spatiotemporal separability has also been examined in the frequency domain, showing temporal frequency effects on the shape of the spatial frequency tuning curve that can be understood in terms of a nonlinear interaction between the spatial and temporal responses caused by a lag in development of the surround (Dawis *et al.*, 1984; Derrington and Lennie, 1984). Golomb *et al.* (1994) provide a mathematical method for quantifying spatiotemporal separability based on singular value decompositions of the LGN spatiotemporal impulse responses.

Moving to a broader perspective of temporal processing in LGN, Funke and Wörgötter (1997) have suggested that a major function of the LGN is to transform a rate code into a temporal code. Evidence supporting this idea comes from Richmond and colleagues (Gawne *et al.*, 1991; McClurkin *et al.*, 1991a; McClurkin *et al.*, 1991b), whose data showed that information about stimulus shape in LGN could be carried by the temporal waveform of the response, in addition to mean firing rate for the stimulus. Contributing to different shapes producing different temporal responses may be the longer latencies of the classical inhibitory surround as well as the broader non-classical suppressive field (Section 2.4.4) compared to the receptive field center. The degree to which spatial patterns overlap center and surround regions of the receptive field would then lead to different temporal patterns in the response. The proposal that stimulus shape in LGN can be coded by a small number of discrete temporal basis functions has been

criticized by Golomb *et al.* (1994) on the grounds that such temporal coding would not produce a unique identification of shape.

The general idea that a major function of the LGN is to transform signals in the temporal domain in some manner remains attractive, given that the differences in temporal properties between LGN and retina is substantially larger than the differences in spatial properties. Consistent with that are the observations of Wang *et al.* (2010b) that thalamic spikes encode novel, emergent, temporal features not conveyed by retinal spikes, but do not code novel spatial features. The appearance of novel temporal properties going from retina to LGN has also been emphasized in the review by Victor (1999).

2.4.3 Properties of magnocellular, parvocellular, and koniocellular neurons

As we said earlier, there are three classes of thalamocortical cells in monkey LGN: the parvocellular (P), the magnocellular (M) and the koniocellular (K) cells (Casagrande, 1994; Kaas *et al.*, 1978; Xu *et al.*, 2001). “Magno” and “parvo” are derived from the Latin words for big and small respectively, referring to the cell body sizes, while “konio” comes from the classical Greek word for dust, because those neurons are so tiny. The largest category of LGN thalamocortical cells is formed by the P cells, which comprise around 80% of the total, with M and K cells each contributing about 10% (Casagrande, 1994; Connolly and Van Essen, 1984). The three monkey LGN classes inherit their properties from distinct types of retinal ganglion cells (RGCs) (Schiller, 2010). P cells receive inputs from midget RGCs and M cells from parasol RGCs (Leventhal *et al.*, 1981; Perry *et al.*, 1984, using older nomenclature). Retinal inputs to K cells are diverse,

but many appear to come from small bistratified RGCs (Dacey and Lee, 1994; Szmajda *et al.*, 2008).

The three classes can be neurophysiologically distinguished based on a variety of spatiotemporal and chromatic characteristics. Focusing on M and P cells, those characteristics include differences in both spatial and temporal properties, with M cells having lower spatial resolution (tuned to lower spatial frequencies) and higher temporal resolution (tuned to higher temporal frequencies) than P cells (Derrington and Lennie, 1984; O'Keefe *et al.*, 1998; So and Shapley, 1981; Usrey and Reid, 2000). They also have different contrast sensitivities, with M cells being more sensitive than P cells to luminance (achromatic) contrast (Derrington *et al.*, 1984; Hubel and Livingstone, 1990; Kaplan and Shapley, 1982; Lee *et al.*, 1983a; Shapley *et al.*, 1981). M cells also show greater gain for luminance contrast (Derrington and Lennie, 1984; Kaplan *et al.*, 1987; Shapley *et al.*, 1981; Usrey and Reid, 2000). Another distinguishing characteristic is latency. P cells have longer latencies to visual stimuli than M cells (Cheong and Pietersen, 2014; Dreher *et al.*, 1976; Kaplan and Shapley, 1982; Maunsell *et al.*, 1999; Schmolesky *et al.*, 1998; Sestokas and Lehmkuhle, 1986; Usrey and Reid, 2000). The longer latency for P cells is likely because they are innervated by retinal ganglion cells with smaller diameter axons than those innervating M cells (Weng *et al.*, 2005). Smaller diameter axons have slower conduction velocities under the Hodgkin-Huxley equations.

Because of its sensitivity to high temporal frequencies, the M stream has been suggested as being particularly important for motion perception, while P cells might be important for fine spatial details (DeYoe and Van Essen, 1988; Livingstone and Hubel, 1988). However, this should not be regarded as a strict separation of functions as it

appears that M and P inputs from LGN are more extensively mixed within striate cortex than previously thought (Callaway and Wiser, 1996; Lachica *et al.*, 1992; Sawatari and Callaway, 1996; Sincich and Horton, 2005; Yabuta and Callaway, 1998; Yoshioka *et al.*, 1994). Furthermore, mixed contributions from both streams can be detected in both dorsal and ventral extrastriate areas (Ferrera *et al.*, 1992; Nassi *et al.*, 2006).

Moving to color, in addition to an antagonistic center surround organization for luminance, P cells also show red/green center/surround color opponency, excited by one color in the center and inhibited by a different color in the surround (De Valois *et al.*, 1966; De Valois *et al.*, 2000; Reid and Shapley, 2002; Wiesel and Hubel, 1966). M cells on the other hand have spectrally broadband response characteristics reflecting mixed red/green inputs, and do not show strong color opponency. A consequence of this is that P cells are far more sensitive to red/green color contrast than M cells (Hubel and Livingstone, 1990), while, as mentioned earlier, M cells are more sensitive to luminance contrast. However, the separation of red/green and achromatic signals between P and M channels is not absolute. FMRI data shows adaptation of red/green responses to both red/green and achromatic stimuli, suggesting a dual role for the P channel in transmitting both types of stimuli (Chang *et al.*, 2016). K cells handle yellow/blue color signals, as discussed below. Color properties of LGN have been reviewed by Martin (2004).

K cells were not identified as a separate stream until late (Casagrande, 1994), with much of their characterization having been done recently (Hendry and Reid, 2000; Martin and Lee, 2014). K cells are located in six thin layers, each associated with one of the six main M and P layers, and located below its associated main layer (Hendry and Reid, 2000; Hendry and Yoshioka, 1994). Thus, the K layers are intercalated between the main

LGN layers, except for the bottom most K layer, which is between the bottom M layer and the optic tract. K cells have diverse properties and it is often difficult to generalize their characteristics. They tend to have poor spatial resolution, with larger receptive fields than P or M cells (Tailby *et al.*, 2008; White *et al.*, 2001), despite having smaller cell bodies. Contrast sensitivity and temporal frequency response are intermediate between P and M cells (Solomon *et al.*, 1999). Many K cells have blue/yellow color opponent responses (Chatterjee and Callaway, 2003; Martin *et al.*, 1997; Szmajda *et al.*, 2006), with blue-ON cells (center excited by blue, inhibited by yellow) far outnumbering blue-OFF cells (excited by yellow, inhibited by blue). Compared to M and P cells, K cells have far longer latencies (Pietersen *et al.*, 2014). As mentioned earlier, a small fraction of K cells are strongly orientation selective (Cheong *et al.*, 2013). K cells project to the superficial layers of striate cortex, unlike M and P cells that project to layers 4 and 6, as will outlined in Section 3.2.

Two distinguishing features of K cells are that they are the only LGN cells to receive input from the superior colliculus (Harting *et al.*, 1991a; May, 2006), and they are the only LGN cells to receive signals originating from blue (S) cones (Roy *et al.*, 2009; Tailby *et al.*, 2008). Collicular inputs and blue cone inputs may go to different subpopulations of K cells. Those with superior colliculus input occur preferentially in the ventral K layers, associated with the M layers (Harting *et al.*, 1991a). On the other hand, those with blue cone inputs tend to be located in the more dorsal K layers, associated with the P layers, particularly the deeper P layers (Hendry and Reid, 2000; Martin *et al.*, 1997; Schiller and Malpeli, 1978; Szmajda *et al.*, 2006).

K cells may represent the primordial color system in mammals, with color selectivity in P cells being a relatively recent development specific to primates (Mollon, 1989) (This is consistent with the speculation of Bishop (1959) that smaller diameter sensory afferent fibers are in general phylogenetically older than larger diameter ones). Most vertebrates have four cones classes (e.g., fish, birds, reptiles), though amphibians have three. Mammals generally stand out as having impoverished color vision, with only two cone pigments, a short-wavelength sensitive (S) pigment and a medium/long sensitive (M/L) pigment, so that mammals are typically red/green color blind (Jacobs, 2009). Early mammals are thought to have been nocturnal animals in which the color system degenerated. The loss was retained as many mammals moved into diurnal lifestyles (an example of brain organization being constrained by evolutionary history rather than being set by optimal design). The typical dichromatic mammalian organization can be seen in color responses in cat LGN, in which both X (magno) and Y (parvo) cells are achromatic, and only W (konio) cells are color selective, showing blue/yellow opponency (Buzás *et al.*, 2013). Amongst mammals, primates are the only ones to have trichromatic vision, with M (green) and L (red) pigments diverging from a common ancestor 9-35 million years ago (Shyue *et al.*, 1995; Yokoyama and Yokoyama, 1989). Trichromatic vision allowed development of a red/green color opponent representation, which apparently piggybacked itself onto a pre-existing high-acuity P system (Martin *et al.*, 2011).

In summary, monkey LGN can be viewed as having two spatiotemporal channels, the P cells (high spatial resolution/low temporal resolution), and the M cells (low spatial

resolution/high temporal resolution), as well as two color channels, the P cells (red/green) and K cells (blue/yellow), with the M cells being achromatic.

What is the relation between the monkey M, P, K cells with the cat X, Y, W cells? The P and M cells in macaque monkey may correspond to cat X and Y cells respectively, when one looks only at their spatial and temporal tuning characteristics. P cells are tuned to higher spatial frequencies and have more transient temporal properties than M cells (Derrington and Lennie, 1984), and the same relationship holds between X and Y cells (Derrington and Fuchs, 1979). The analogy breaks down, however, when one examines other characteristics, such as spatial linearity, contrast sensitivity and color responses (Shapley and Perry, 1986). M cells have linear spatial summation while Y cells have nonlinear summation. P cells show red/green color selectivity, while X cells do not. P cells have lower contrast sensitivity than M cells, while there is not a great difference in contrast sensitivity between X and Y cells. Possibly M and P cells are homologous to X and Y cells, as suggested in the similarity in basic spatiotemporal properties, but acquired various other divergent properties as adaptations to nocturnal and diurnal life in cats and monkeys respectively. Monkey K cells may correspond to cat W cells, as both have very small cell sizes, selectivity for blue/yellow chromaticity, and project to the superficial layer of cortex rather than layer 4.

Under the conventional perspective that we have summarized here, three independent functional streams form the essence of the organization that LGN inherits from retina. In reality, however, the retinal input to LGN is far more complex. Although the three streams are a prominent aspect of retinal organization, beyond that there are around 15-20 classes of retinal ganglion cells whose functions are poorly understood

(Gollisch and Meister, 2010; Masland, 2012). Such retinal diversity strongly suggests that the visual signals arriving in LGN from retina are more highly processed than current accounts indicate. With this more complex but undefined input, it's possible the LGN may itself engage in more complex signal processing that goes beyond being a spatiotemporal filter with "smart gating". Thus, one road to future progress in understanding the LGN is better characterization of its multitude of ganglion cell input types.

2.4.4 The extraclassical suppressive field and contrast gain control

The world presents an environment in which stimulus strength can vary greatly (Tadmor and Tolhurst, 2000), yet it must be encoded by neurons with a limited dynamic range. The extra-classical suppressive field in LGN contributes to efficient use of limited neural dynamic range in by normalizing stimuli intensity through the implementation of a contrast gain control. The suppressive field in LGN has been reviewed by Jeffries *et al*. (2014), and contrast gain normalization by Carandini and Heeger (2012).

A suppressive surround can be demonstrated in a broad region beyond the classical receptive field in LGN cells. A stimulus presented in a peripheral region suppresses responses to another stimulus within the classical field, but by itself the peripheral stimulus has little or no effect on the firing of the cell (Alitto and Usrey, 2008, 2015b; Felisberti and Derrington, 1999; Felisberti and Derrington, 2001; Girardin *et al.*, 2002; Krüger, 1977; Levick *et al.*, 1972; Seim and Valberg, 2013; Valberg *et al.*, 1985; Webb *et al.*, 2002; Webb *et al.*, 2005). In addition to this stimulus masking, the suppressive surround may manifest itself by producing a size tuning effect, in which response of the cell drops as stimulus diameter increases beyond a certain point (Alitto

and Usrey, 2008; Bonin *et al.*, 2005; Fisher *et al.*, 2016; Sceniak *et al.*, 2006; Solomon *et al.*, 2006). Although often demonstrated using peripheral stimuli beyond the classical receptive field, the suppressive surround is generally described as a Gaussian *suppressive field* overlapping and aligned with the classical receptive field (Bonin *et al.*, 2005; Carandini and Heeger, 2012; Solomon *et al.*, 2006). Just as the suppressive field may spatially extend beyond the borders of the classical receptive field, sensitivity of the suppressive field to other stimulus parameters may also extend beyond the response range of the classical receptive field. Specifically, a masking stimulus can have a suppressive effect at spatial or temporal frequencies beyond the range that the classical receptive field responds to (Bonin *et al.*, 2005; Nolt *et al.*, 2007). The suppressive field is more prominent in M cells than P or K cells in monkey (or in Y cells rather than X cells in cats) (Alitto and Usrey, 2008; Camp *et al.*, 2009; Derrington and Felisberti, 1998; Krüger, 1977; Solomon *et al.*, 2006; Solomon *et al.*, 2002; Webb *et al.*, 2002). The presence of the suppressive field adds a strong nonlinear component to the responses of LGN cells (Solomon *et al.*, 2010), so that they cannot be treated merely as linear filters.

Extraclassical suppression is present throughout early visual processing, occurring in retina (Shapley and Victor, 1978; Victor and Shapley, 1979)(see review by Demb (2008)), LGN (as described in the previous paragraph), and striate cortex (Gulyás *et al.*, 1987; Ozeki *et al.*, 2004; Sengpiel *et al.*, 1997; Walker *et al.*, 2000). The question arises whether suppressive effects in LGN are inherited from the retina, generated *de novo* within LGN, or the product of feedback from cortex. The data suggests that extra-classical suppressive fields are largely inherited from retinal inputs (Alitto and Usrey, 2008; Bonin *et al.*, 2005; Camp *et al.*, 2009; Nolt *et al.*, 2004; Seim and Valberg, 2013),

just as the classical receptive field is inherited from retina. However, the suppressive field is enhanced in LGN compared to retina (Fisher *et al.*, 2016). Striate cortex contributes as well (Murphy and Sillito, 1987; Nolt *et al.*, 2007; Przybyszewski *et al.*, 2000; Webb *et al.*, 2002), though based on latency considerations any cortical effects would be expected to arrive late in the LGN surround responses.

LGN responses show a compressive nonlinearity as a function of contrast (with a stronger nonlinearity for M cells) (Usrey and Reid, 2000). Increasing the contrast of a masking stimulus in the suppressive field causes the contrast response curve to shift (Solomon *et al.*, 2002). It also shifts as stimulus diameter increases beyond a certain point and the stimulus covers more of the suppressive field (Bonin *et al.*, 2005). Because the suppressive field overlaps the receptive field, any stimulus will cause some degree of self-masking. Thus the observed contrast response curve of the receptive field + suppressive field is more nonlinear than the contrast response of the receptive field would be in isolation.

Overall, for any given stimulus contrast, anything that stimulates the suppressive field reduces the response to that contrast. Thus, the suppressive field can act as a contrast gain control. Modeling indicates that the effect of the suppressive field is to implement contrast gain control through divisive normalization, rather than having a subtractive effect (Ayaz and Chance, 2009; Bonin *et al.*, 2005; Carandini and Heeger, 2012; Cope *et al.*, 2013, 2014; Mante *et al.*, 2008). Under divisive normalization, the response of each LGN thalamocortical cell is divided by a measure of average contrast in its neighborhood (with contrast is defined as the standard deviation of luminance divided by mean luminance (Bonin *et al.*, 2006)).

As with the suppressive surround, the contrast adaptation described above starts in retina (Baccus and Meister, 2002; Demb, 2008; Scholl *et al.*, 2012; Shapley and Victor, 1978) and is enhanced in LGN (Kaplan *et al.*, 1987; Rathbun *et al.*, 2016; Scholl *et al.*, 2012). Studies of contrast adaptation in LGN using natural images have been done by (Lesica *et al.*, 2007; Mante *et al.*, 2005) producing results consistent with simpler stimuli.

In addition to fast contrast adaptation (milliseconds) that we have been focusing on here, there is also persistent contrast adaptation in LGN that operates on a slow time scale (seconds or minutes) (Chang *et al.*, 2016; Solomon *et al.*, 2004). Possibly this slow contrast adaptation may involve synaptic depression (Chen *et al.*, 2002) rather than the suppressive surround. Luminance adaptation is another important way that the early visual system compresses the broad dynamic range of natural stimuli, but that mechanism appears to be confined to retina (Fain *et al.*, 2001; Meister and Berry II, 1999; Shapley and Enroth-Cugell, 1984) without additional contributions from LGN.

The characteristics of spatiotemporal filters that maximize the flow of information through noisy channels with limited dynamic range has been studied theoretically by Van Hateren (1993). That model produced filters in which both spatial and temporal properties depended on stimulus intensity. In LGN, we do see changes in spatial and temporal properties of neurons that appear to be associated with the nonlinear contrast gain control mechanism. In the temporal domain, increased contrast leads to a shorter integration time, an upward shift in peak frequency tuning, and an advance in the response phase (Alitto and Usrey, 2004; Lesica *et al.*, 2007; Mante *et al.*, 2008; Rathbun *et al.*, 2016; Usrey and Reid, 2000). Increased contrast also leads to an upward shift in the peak of the spatial frequency tuning curve (Nolt *et al.*, 2004).

While we have emphasized the role of the LGN suppressive field in contrast gain control, other functions are possible. For example, the suppressive field may aid in segmenting stimulus shapes by allowing cells to modulate their responses based on long-range contextual information (Albright and Stoner, 2002). It may also be involved in producing saccadic suppression (Felisberti and Derrington, 1999; Felisberti and Derrington, 2001) (see Section 2.4.6), as well as improve coding efficiency by reducing spatial redundancy among neurons (Schwartz and Simoncelli, 2001).

2.4.5 Binocular properties of thalamocortical cell receptive fields

There are numerous reports in the early cat literature of weak binocular interactions in the LGN, primarily inhibition by the non-dominant eye of activity driven by the dominant eye, but occasionally excitatory input from the non-dominant eye as well (Lindsley *et al.*, 1967; Marchiafava, 1966; Marrocco and McClurkin, 1979; Murphy and Sillito, 1989; Noda *et al.*, 1972; Pape and Eysel, 1986; Sanderson *et al.*, 1971; Sanderson *et al.*, 1969; Singer, 1970; Suzuki and Kato, 1966; Suzuki and Takahashi, 1970; Vastola, 1960). Similar binocular interactions occur in monkey (Rodieck and Dreher, 1979; Schroeder *et al.*, 1990), most prominently in magnocellular neurons. Lindström and Wróbel (1990b) suggest the small percentage of LGN cells showing binocular excitation may be displaced TRN cells reflecting developmental errors. Data from fMRI showing reduced LGN activity in adult amblyopes (Hess *et al.*, 2009) is evidence that binocular inhibition in LGN has functional significance, at least during development.

LGN binocular interactions with short latencies may be due to thalamocortical cell dendrites crossing laminar borders (Schroeder *et al.*, 1990; Singer, 1970) , which is known to occur anatomically (Guillery, 1971; Saini and Garey, 1981). Another source of

binocular interactions in LGN thalamocortical cells may be the TRN (or perigeniculate nucleus in cat) (Singer, 1977; Zhou *et al.*, 2003). Although cortical feedback to LGN is binocular (Casagrande and Boyd, 1996; Ichida and Casagrande, 2002; Schmielau and Singer, 1977), it does not appear to be the primary source of the short latency binocular interactions. However, under binocular rivalry conditions (for example, orthogonal gratings presented to the two eyes), cortical feedback might contribute to longer latency binocular effects in LGN (Varela and Singer, 1987).

LGN binocular responses are not disparity tuned (Guido *et al.*, 1989; Xue *et al.*, 1987). Nevertheless, that does not preclude an LGN involvement in modulating stereopsis properties in striate cortex. As mentioned previously (Section 2.3), both McIlwain (1995) and Schmielau and Singer (1977) suggest that LGN may be involved in dynamically modulating disparity tuning of striate cells.

While M and P cells (or X and Y cells in cats) exhibit weak binocular interactions, K cells in marmoset monkeys can be strongly driven by both eyes (Wallace *et al.*, 2016; Zeater *et al.*, 2015). These strong binocular effects in the K cells appear not to be involved in perceptual stereopsis, but may be inherited from midbrain structures such as the superior colliculus.

Recent LGN research has not focused on binocular properties as much as the earlier work, and there remains a rich potential for further binocular studies, both experimentally and through modeling.

2.4.6 Saccadic modulation

Despite the constant occurrence of saccades, we see the world as stable. Human psychophysical evidence indicates reduced contrast sensitivity during saccades (reviewed

by Ross *et al.* (2001)), which would mitigate motion blur during eye motion. The psychophysics indicates that visual masking caused by stimulus shift across the retina is one aspect of saccadic suppression (perhaps involving the broad non-classical suppressive field described in Section 2.4.4). In addition, psychophysics suggests that there is a non-retinal component to the saccadic effects as well.

The site of saccadic suppression is likely to be the LGN (McFarland *et al.*, 2015; Thilo *et al.*, 2004). The visual masking component of saccadic responses is shown by suppression in LGN caused by abrupt stimulus shifts, as would occur during saccades (Derrington and Felisberti, 1998; Sylvester *et al.*, 2005). The non-retinal component of saccadic responses can be isolated by measuring LGN responses to saccades in the dark or when using non-patterned luminance stimuli. Under those conditions, saccades lead to a biphasic modulation of the LGN, with weak early suppression followed by strong late facilitation (Adey and Noda, 1973; Bartlett *et al.*, 1976; Lee and Malpeli, 1998; Reppas *et al.*, 2002; Royal *et al.*, 2006). The suppression starts 200-300 ms before the start of the saccade and peaks 100 ms before saccade start, smoothly transitioning to the late facilitation which peaks around 100 ms after saccade end (Lee and Malpeli, 1998). A similar biphasic response to saccades occurs in striate cortex, probably inherited from LGN (McFarland *et al.*, 2015). Human fMRI measurement can pick up the late facilitatory component of responses to saccades in the dark (Sylvester *et al.*, 2005; Sylvester and Rees, 2006). In addition to saccades, visual responses in LGN are modulated by changes in static eye position (gaze angle) (Lal and Friedlander, 1989, 1990).

One source of extra-retinal saccadic modulation of LGN (and TRN) is proprioception from the extraocular muscles (Donaldson and Dixon, 1980; Lal and Friedlander, 1990; Molotchnikoff and Casanova, 1985). Also, the pretectum modulates LGN during saccades, in particular the nucleus of the optic tract and the posterior pretectal nucleus (Fischer *et al.*, 1998; Schmidt, 1996). Efference copy of eye movement motor signals also contributes to stable vision during saccades (Cavanaugh *et al.*, 2016), but this as yet has not been connected with LGN modulations.

Because saccadic suppression starts more than 100 milliseconds before the saccade, Royal *et al.* (2006) suggest it is a motor planning signal, and therefore cognitive in nature. The function of the late facilitatory phase of the non-retinal component of saccadic responses remains a puzzle. Lee and Malpeli (1998) interpret it as promoting rapid central registration and analysis of the new image after each saccade. Saccadic modulations of LGN activity have been reviewed by Casagrande *et al.* (2005).

2.4.7 The mouse LGN

Interest in studying the mouse visual system has increased greatly in recent years (Huberman and Niell, 2011), in part because of the relative ease in performing genetic manipulations on mice compared to monkeys. We are currently at a stage where previous characterizations of LGN properties in cats and monkeys are being repeated in mice, with the intention that more advanced experimental techniques available in mice, notably genetic knockout methods, will lead to progress.

The mouse (and rat) LGN has cells that are regionally clustered by morphology and ocular dominance, though without overt laminations (Krahe *et al.*, 2011; Reese, 1988). The major division is between a core region and a dorsal shell region. The core

region sends projections to layer 4 of striate cortex, while the dorsal shell projects to the superficial layers of striate cortex (Cruz-Martín *et al.*, 2014). In addition to receiving retinal inputs, the dorsal shell receives input from the superior colliculus (Grubb and Thompson, 2004; Reese, 1984).

Within the core region there are cells morphologically analogous to X and Y classes of cells (Krahe *et al.*, 2011). However, despite these anatomical distinctions, it has proved difficult, based on neurophysiology, to establish distinct functional classes of thalamocortical cells, as their response properties appear rather homogenous (Denman and Contreras, 2016; Grubb and Thompson, 2003; Grubb and Thompson, 2005; Krahe *et al.*, 2011). The cells have the same circular center-surround organization described previously for cats and monkeys, with on- and off- center channels, mostly linear spatial summation, and the existence of burst and tonic modes. Spatial resolution in mice is extremely poor compared to cats and monkeys, but temporal tuning is comparable (Grubb and Thompson, 2003; Grubb and Thompson, 2005; Tang *et al.*, 2016) (with rats similar to mice (Sriram *et al.*, 2016)). Removal of cortical feedback using knockout genetic techniques leads to complex excitatory and inhibitory effects in LGN that are difficult to interpret (Denman and Contreras, 2015). While such binocular driving is found only in koniocellular cells in primates, its occurrence is widespread in the LGN of rats and mice (Grieve, 2005; Howarth *et al.*, 2014).

Both orientation selectivity (Kondo and Ohki, 2016; Li *et al.*, 2013; Niell, 2013; Piscopo *et al.*, 2013; Scholl *et al.*, 2013; Sun *et al.*, 2016; Tang *et al.*, 2016; Zhao *et al.*, 2013) and motion direction selectivity (Cruz-Martín *et al.*, 2014; Marshel *et al.*, 2012; Piscopo *et al.*, 2013; Sun *et al.*, 2016) are more frequent in the mouse LGN compared to

cat and monkey. Cells with orientation and direction selectivity tend to be concentrated in the dorsal shell region. These dorsal shell cells may be analogous to koniocellular neurons in primates, based on their higher incidence of orientation and direction selectivity, in conjunction with their efferent projection to the superficial layers of striate cortex as well as the reception of afferent projections from superior colliculus.

3 LGN circuitry

3.1 Retinal inputs and synaptic triads

Although only about 10% of the synaptic inputs to geniculate neurons come from the retina (Guillery, 1971; Van Horn *et al.*, 2000; Wilson, 1993; Wilson *et al.*, 1984), that small percentage drives the thalamocortical cells and largely determines their basic receptive field properties (Weyand, 2015). Each LGN thalamocortical cell often receives a dominant driving input from just one retinal ganglion cell, with lesser inputs from two or three other ganglion cells (Cleland *et al.*, 1971a; Cleland and Lee, 1985; Mastronarde, 1987; Sincich *et al.*, 2007; Usrey *et al.*, 1999; Weyand, 2007). There is little excitatory crosstalk between thalamocortical cells (Bickford *et al.*, 2008), in contrast to extensive excitatory interactions between cortical cells. These observations would give the impression that properties of thalamocortical cells are essentially the same as those of retinal ganglion cells, other than some tweaks from inhibitory neurons and cortical feedback, and that signals from the M, P, and K streams transfer from retina to LGN in parallel with minimal crosstalk.

However, the neuroanatomy of ganglion cell projections to LGN presents a more complex picture than that. A recent connectomic analysis of retinal inputs to mouse LGN, based on a massive electron microscopy database covering the entire LGN, found that

retinogeniculate connectivity did not correlate with cell morphology (Morgan *et al.*, 2016). Individual ganglion cells projected to multiple LGN cell types, and individual thalamocortical cells received input from multiple ganglion cell types. The result was that LGN cells belonged to multiple fuzzy set classes and thus it was impossible to classify them uniquely, a different picture than that of parallel, non-interacting streams. Rompani *et al.* (2017) also find a more complex pattern of anatomical integration between retinal ganglion cells and LGN cells than previously envisioned. Going back to the older literature, we see similar anatomical mixing in the retinogeniculate projection of cats (Hamos *et al.*, 1987) (based on a much smaller data sampling constrained by the technology of the time), so it's not a peculiarity of the mouse visual system.

The anatomical mixing going from retina to LGN is not inconsistent with the findings that a single ganglion cell tends to dominate the activity of individual thalamocortical cells, as anatomy doesn't reveal the functional strengths amongst the multitude of retinogeniculate synapses. So the question is what functional consequences does this anatomical diversity have? There are multiple reports of thalamocortical cells showing mixed influences from multiple ganglion cells, in some cases mixing from different visual streams (Mastronarde, 1987; Mastronarde, 1992; Usrey *et al.*, 1999; Yeh *et al.*, 2009; Yeh *et al.*, 2003). Although dominated by input from one primary ganglion cell, convergence from weaker secondary inputs appears sufficient to diversify the receptive field properties of thalamocortical cells beyond what is received from retina. Given, in addition, that retinal inputs are already more diverse than frequently portrayed (Gollisch and Meister, 2010; Masland, 2012), all this presents a picture of more complex representations and processing in LGN than generally thought, even just looking at

feedforward retinal inputs without considering feedback loops and non-retinal inputs. As long as research is in the mindset to shoehorn all LGN cells into a small number of rigid categories, then a lot of the diversity of LGN responses, likely to be of major importance, will go unnoted. In this situation, there would seem to be advantages to developing high-dimensional multivariate classification schemes based on a broader range of response parameters than are currently considered.

The transfer function across the retinogeniculate synapse can be monitored by comparing thalamocortical output with input from retina. The input is observable in thalamocortical cells as S potentials, which are post-synaptic potentials of retinal origin (Cleland *et al.*, 1971a, b; Kaplan and Shapley, 1984). Because each thalamocortical cell receives its dominant input from just one or a few retinal ganglion cells, it is feasible to determine what input a thalamocortical cell receives. Thus, the signal transform going across the synapse from retina to LGN is far easier to characterize than the transform going across the synapse from LGN to cortex, where the cortical cell is driven by many inputs.

LGN thalamocortical cells typically transmit only about half the spikes they receive from retina (Cheng *et al.*, 1995; Cleland *et al.*, 1971a; Cleland and Lee, 1985; Hamamoto *et al.*, 1994; Kaplan *et al.*, 1987; Lee *et al.*, 1983b; Sincich *et al.*, 2007; Usrey *et al.*, 1998; Weyand, 2007). The ratio between retinal spikes and LGN spikes is called the "transfer ratio". The temporal pattern of retinal spikes arriving at thalamocortical cells affects the transfer ratio across retinogeniculate synapses. Short interspike intervals (less than 30 ms) enhance transmission across the retinogeniculate synapse through linear summation of EPSPs at the postsynaptic membrane (Carandini *et al.*, 2007; Rathbun *et*

al., 2010; Rowe and Fischer, 2001; Sincich *et al.*, 2007; Usrey *et al.*, 1998; Weyand, 2007). This is often described as a paired-spike enhancement in synaptic transmission efficacy. Spike timing effects are modeled by Carandini *et al.* (2007) and reviewed by Usrey (2002).

Losing half the spikes when crossing the retinogeniculate synapse might lead one to suspect that a high fraction of the information arriving from retina is leaking away, but that is not the case. It turns out that it is the less informative retinal spikes that are being edited out. The consequent LGN spike train carries more bits of information per spike than the retinal spike train, and most of the information arriving from retina is retained (Rathbun *et al.*, 2010; Sincich *et al.*, 2009; Uglesich *et al.*, 2009). An important mechanism for the editing process appears to be the enhanced transmission efficacy of retinal spikes that arrive within a short interval (Rathbun *et al.*, 2010; Sincich *et al.*, 2009). Thus, this temporal filtering property of the retinogeniculate synapse serves to increase sparseness and coding efficiency of the signal coming from retina.

Retinal inputs, in addition to stimulating LGN thalamocortical cells, stimulate LGN interneurons. The interneurons make up about 25% of the LGN cell population (Fitzpatrick *et al.*, 1984; LeVay and Ferster, 1979; Madarász *et al.*, 1985) and are all inhibitory. Upon receiving retinal input, these intrinsic interneurons in turn inhibit the thalamocortical cells and other interneurons, thus providing a feedforward inhibitory pathway (Wang *et al.*, 2011a; Wang *et al.*, 2007). In some cases thalamocortical cells receive excitatory and inhibitory inputs that are tightly time locked, indicating that the thalamocortical cell and inhibitory interneuron receive input from the same retinal ganglion cell (Blitz and Regehr, 2005). In other cases thalamocortical cells receive

excitatory and inhibitory inputs that are unlocked with respect to time, when the two inputs arrive from different retinal ganglion cells. Locked inhibition is associated with synaptic triads within the glomeruli, described below in this section, and may be involved in increasing the time precision of thalamocortical responses and in producing lagged thalamocortical cells (Section 2.4.2). Unlocked inhibition may be involved in increasing the strength of the spatial inhibitory surround in receptive fields compared to what occurs in retinal ganglion cells.

As was mentioned earlier, there appears to be very little excitatory crosstalk between LGN thalamocortical cells (Bickford *et al.*, 2008). Thalamocortical cell interactions are primarily inhibitory, indirectly mediated either through interneurons within LGN (feedforward inhibition) or inhibitory neurons in the TRN (feedback inhibition). These include binocular inhibitory interactions (Section 2.4.5). An early study reports inhibition between on- and off- center thalamocortical cells inhibit (Singer and Creutzfeldt, 1970). This inhibition may be mediated by feedback inhibition from TRN, which pools on- and off-center signals, as direct inhibition through intrinsic inhibitory neurons was not observed (Lindström and Wróbel, 2011). Inhibition has also been reported between X and Y thalamocortical cells (Bloomfield and Sherman, 1988; Singer and Bedworth, 1973). However, such cross inhibition between X and Y cells was not found by (Lindström and Wróbel, 1990c, 2011), neither for feedforward nor feedback inhibition.

LGN thalamocortical cells inherit the basic center-surround spatial organization of the ganglion cells. How does the thalamic inhibitory circuitry interact with this spatial organization? Two basic modes of interaction exist between inhibitory interneurons and

the thalamocortical, push-pull inhibition and same-sign inhibition (reviewed by (Hirsch *et al.*, 2015; Wang *et al.*, 2011a)). In push-pull inhibition, thalamocortical cells are inhibited by interneurons having opposite contrast polarity. For example, a thalamocortical cell on-center is inhibited by the off-center of an inhibitory interneuron, and likewise for the surrounds of the two cells. In same-sign inhibition, the thalamocortical cell receives inhibition from an interneuron with the same contrast polarity. Both types of inhibition were reported very early in LGN (Singer *et al.*, 1972).

An effect of push-pull inhibition is to extend the dynamic range of thalamocortical cells and restore linearity to rectified responses (Hirsch *et al.*, 2015). Same-sign inhibition may produce the stronger surround inhibition seen in thalamocortical cells compared to their retinal inputs. These two modes of inhibitory interactions apply to the feedforward interneurons, which have spatially distinct on and off zones. TRN cells, providing feedback inhibition, have complex properties with on and off responses throughout their entire receptive fields (Lindström and Wróbel, 2011; Wang *et al.*, 2007) and thus cannot fit into these schemas for inhibitory interactions. Push-pull inhibition has been often reported in thalamocortical cells (Hirsch *et al.*, 2015; Martinez *et al.*, 2014; Suresh *et al.*, 2016; Wang *et al.*, 2011a). Despite that, Lindström and Wróbel (2011) concluded from their intracellular recordings that feedforward inhibition to thalamocortical cells is always same-sign, suggesting that the observed 'pull' in push-pull may reflect disfacilitation inherited from retina rather than intrageniculate inhibition.

Synaptic triads, encapsulated within ball-like glomeruli formed by glial cells, are a distinctive feature of the synaptic organization of the thalamus generally, and the LGN

in particular (Famiglietti and Peters, 1972; Guillery, 1969; Hamos *et al.*, 1985; Sherman, 2004) (Figure 2b). Within a glomerulus, an incoming axon terminal from the retina makes synaptic contacts both with a dendrite from an LGN thalamocortical cell and a dendrite from an interneuron. The interneuron dendrite in turn makes synaptic contact with the thalamocortical cell dendrite, forming the third synapse of the triad. Therefore, the interneuron dendrite is presynaptic to the thalamocortical cell dendrite, and under some circumstances a signal can be transmitted from interneuron dendrite to thalamocortical dendrite without passing through the axon of the interneuron. Under this triadic arrangement, the excitation and inhibition that a thalamocortical cell receives originates from the same retinal ganglion cell, so inhibition with a glomerulus is same-sign inhibition. In addition to retinal inputs, afferents from the parabrachial complex often form similar synaptic triads within a glomerulus (Erişir *et al.*, 1997a). However, feedback from cortex does not usually participate in the glomerular complex.

In cats, where research on this topic is most extensive, triads are associated almost exclusively with X cells (analogous to magnocellular cells in primates). The inhibitory circuitry associated with the Y cells will generally occur outside glomeruli, although a small number of Y cell triads have been reported (Dankowski and Bickford, 2003; Datskovskaia *et al.*, 2001). Monkeys also have synaptic triads within glomeruli (Hámori *et al.*, 1991), with their prevalence being greater for magnocellular neurons (Wilson, 1989).

Different regions within the dendritic tree of a single cell may be conducting independent computations. Different branches of distal dendrites in the inhibitory interneurons appear to be electrically isolated from each other, as well as isolated from

the cell soma and dendrites near the soma (Bloomfield and Sherman, 1989; Cox *et al.*, 1998; Crandall and Cox, 2012). A consequence of this isolation is that different local-circuit computations involving presynaptic interneuron dendrites and postsynaptic thalamocortical dendrites, and using analog graded potentials rather than spikes, could be carried out in parallel in different regions of the dendritic tree, independent of the spiking output at the axon of the interneuron (Ralston, 1971). Adding another layer of complexity to dendritic processing is the observation that active potentials can occur in the dendrites of thalamocortical cells, either locally generated in distal dendrites (Augustinaite *et al.*, 2014) or backpropagated to proximal dendrites from the axonal spike initiation zone (Acuna-Goycolea *et al.*, 2008; Casale and McCormick, 2011; Williams and Stuart, 2000). Thus, interneurons in LGN can inhibit thalamocortical cells in three ways, through their axonal output, through presynaptic dendrites activated locally within triads, and through presynaptic dendrites activated globally by backpropagation from the soma and proximal dendrites.

The functional significance of dendritic local-circuit processing is not known. One suggestion is that it is involved in contrast gain control (Sherman, 2004) and another is that it is a gating mechanism for retinal input (Koch, 1985). Dendritic local-circuit processing, dependent on specific dendritic and synaptic geometries, is an aspect of LGN organization that has not been well integrated into current general models of LGN function (see London and Häusser (2005) for a review of dendritic computation). Aside from all the dendrodendritic interactions of interneurons with thalamocortical cells, the axonal output of interneurons reflects yet another independent parallel computation.

As we said earlier, retina is essentially the only driver to LGN activity and everything else is modulatory. Two biophysical characteristics distinguish connections that drive LGN (retina→LGN) from modulatory connections to LGN, which include both feedback connections (TRN→LGN; cortex→LGN) as well as brainstem→LGN connections. First is the location of synapses in the dendritic tree, and second is the type of neurotransmitter receptor in the postsynaptic membrane. Feedforward connections from retina form synapses at the base of dendrites, near the cell body, where they can most strongly influence the firing rate of the cell. Feedback and other modulatory synapses tend to be located at the periphery of the dendritic tree, where their influences are weaker (Erişir *et al.*, 1997b; Wilson *et al.*, 1984). Second, the retinal input has synapses with fast ionotropic receptors, while the modulatory synapses have a mixture of ionotropic and slower metabotropic receptors. Ionotropic receptors, directly activated when the neurotransmitter binds to them, produce postsynaptic potentials (PSPs) with a short latency (<1 ms) and short response duration (tens of milliseconds). Metabotropic receptors, involving an intermediary chain of biochemical interactions, produce PSPs with longer latencies (>10 ms) and longer response durations (hundreds of milliseconds or more) (Conn and Pin, 1997; Pin and Duvoisin, 1995). Using ionotropic receptors is an effective strategy for transmitting visual information from the retina about fast-paced events in the world, as fast PSPs transmit information with high temporal resolution and avoid information loss (Guillery and Sherman, 2002a; Sherman, 2007; Sherman and Guillery, 2002). In contrast, sustained PSPs via metabotropic receptors are more suitable to control longer-term changes in the response mode of LGN neurons.

3.2 Cortical projections

Magnocellular and parvocellular thalamocortical neurons project mainly to layer 4, with collaterals going to layer 6 (Blasdel and Lund, 1983; Callaway, 2005; LeVay and Gilbert, 1976). The same thalamic cell contacts both with principal cells and inhibitory interneurons in cortical layer 4 (Ferster and Lindström, 1983), with input to interneurons being stronger and faster than to principal cells (Hull *et al.*, 2009). The LGN input to cortical interneurons thus forms a disynaptic feedforward inhibitory circuit onto the principal cells, in some ways analogous to the organization of retinal inputs to thalamus but without the dendrodendritic glomerular organization. LGN input contributes less than 15% of synapses to the spiny stellate cells in layer 4, which are the principal cells receiving LGN input (Ahmed *et al.*, 1994; Benshalom and White, 1986; da Costa and Martin, 2009a; Latawiec *et al.*, 2000; Peters and Payne, 1993; Peters *et al.*, 1994).

LGN input can monosynaptically drive striate cells, as shown by cross-correlation analysis (Tanaka, 1983), with on-center LGN cells driving on-regions of striate cell receptive fields and off-center LGN cells driving striate Off regions (Tanaka, 1985). The number of converging LGN cells required to fire a cortical cell has estimates ranging from 10 (Tanaka, 1983) to 30 (Alonso *et al.*, 2001; Bruno and Sakmann, 2006), in contrast to LGN cells which are driven by much fewer retinal inputs.

Despite the small proportion of synapses contributed by LGN, they are sufficient to drive cortical activity without any cortical amplification mediated by recurrent circuits (Bruno and Sakmann, 2006). Nevertheless, cortical amplification does occur as thalamic excitation contributes less than half of the total response of layer 4 stellate cells (Chung and Ferster, 1998; Ferster *et al.*, 1996; Lien and Scanziani, 2013; Stratford *et al.*, 1996).

Overall, both cortical amplification (da Costa and Martin, 2009a; Douglas *et al.*, 1989) and convergent input from a synchronously firing LGN population may enhance the efficacy of the thalamocortical synapses (synchronous firing will be discussed in Section 4).

Projections of the M and P streams from LGN to striate layer 4 remain segregated in the recipient sublayers of layer 4, with M cells going to striate layer 4Cα and P cells going to layer 4Cβ (Blasdel and Fitzpatrick, 1984; Blasdel and Lund, 1983; Hendrickson *et al.*, 1978). From there, the two streams may be mixed downstream as they reach other striate layers (Callaway and Wiser, 1996; Lachica *et al.*, 1992; Sawatari and Callaway, 1996; Sincich and Horton, 2005; Yabuta and Callaway, 1998; Yoshioka *et al.*, 1994).

Layer 6 of striate cortex sends feedback to LGN. The visual signal can reach layer 6 from layer 4 through polysynaptic interlaminar microcircuits (Callaway, 1998; Hirsch and Martinez, 2006). Layer 6 cells can also receive direct LGN inputs, either through collaterals from LGN axons on their way to layer 4 or through dendrites of layer 6 cells that extend into layer 4 (Bannister *et al.*, 2002; Blasdel and Lund, 1983; Briggs, 2010; Hendrickson *et al.*, 1978; Wang *et al.*, 2013). However, it is the polysynaptic circuits rather than the direct LGN inputs that appear to be most important in defining the properties of the layer 6 (da Costa and Martin, 2009b). Layer 6 sends feedback not only to LGN, but to layer 4 as well (Bortone *et al.*, 2014; Briggs, 2010; Ferster and Lindström, 1983; Olsen *et al.*, 2012; Thomson, 2010). Thus, layer 6 is in position to modulate both LGN and the cortical target of LGN. As layer 6 feedback is the largest single source of input to LGN in terms of the number of synapses, being far greater than the retinal input to LGN, it will be a topic of more detailed consideration in Section 5.

For koniocellular neurons in LGN, cortical projections are quite different from M and P cells as they do not send efferents to layer 4. K cell projections to striate cortex fall into two categories (Casagrande *et al.*, 2007; Ding and Casagrande, 1997; Lachica and Casagrande, 1992). K cells associated with the two M layers in LGN (K1 and K2 layers) send projections to layer 1 and upper part of layer 3. K cells associated with the four P layers (K3-6 layers) project to the cytochrome oxidase blob areas in layers 2 and 3 (Figure 2a).

LGN also sends axons directly to extrastriate visual areas, entirely bypassing V1 (Fries, 1981). These LGN efferents come disproportionately from K cells (Rodman *et al.*, 2001). K cells (identified as "interlaminar zones" in the older literature) project to V2 (Bullier and Kennedy, 1983; Markov *et al.*, 2011; Yukie and Iwai, 1981), V3 (Benevento and Yoshida, 1981) and V4 (Benevento and Yoshida, 1981; Gattass *et al.*, 2014; Lyon and Rabideau, 2012). LGN also sends direct projections to posterior inferotemporal cortex (Webster *et al.*, 1994), though it was not identified whether or not those projections were from K cells.

Of particular interest have been the direct projections from LGN K cells to motion area MT (Jayakumar *et al.*, 2013; Lyon and Rabideau, 2012; Sincich *et al.*, 2004; Warner *et al.*, 2010), which is substantially stronger than direct projections to V4 (Lyon and Rabideau, 2012). A direct interaction between LGN and MT that bypasses V1 has also been shown using fMRI (Gaglianese *et al.*, 2012; Gaglianese *et al.*, 2015). The direct connection between LGN and area MT may be functionally significant for passing short-latency visual signals related to motion into the dorsal visual stream.

The extrastriate projections of LGN K cells appear to underlie the phenomenon of blindsight (Cowey, 2010b; Jayakumar *et al.*, 2013; Leopold, 2012). Blindsight occurs after striate cortex has been damaged. Despite a lesioned V1, subjects are still able to discriminate or detect visual stimuli located within the lesioned visual field at above chance levels when asked to guess (Cowey, 2010a; Cowey and Stoerig, 1995; Weiskrantz, 1996). This ability occurs even though subjects having no conscious awareness of the stimuli. Blindsight disappears when, in addition to the original striate lesion, LGN is also lesioned (Schmid *et al.*, 2010). When blindsight disappears upon lesioning LGN, visual activation of extrastriate cortex also disappears (Schmid *et al.*, 2010), an activation that survives when striate cortex alone is lesioned (Schmid *et al.*, 2009). Human MRI shows blindsight positive individuals have a tract connecting LGN to MT, while blindsight negative individuals lack this tract (Ajina *et al.*, 2015; Tamietto and Morrone, 2016). All these are consistent with blindsight being dependent on extrastriate projections of LGN, possibly involving K cells (Cowey, 2010b).

3.3 The role of the thalamic reticular nucleus

A key element in LGN circuitry is the thalamic reticular nucleus (TRN). As Crick (1984) described it: "If the thalamus is the gateway to the cortex, the reticular complex might be described as the guardian of the gateway." The TRN is a thin shell of inhibitory neurons between the thalamus and cortex, but closer to the thalamus and partially surrounding it. Reticular means net-like, and refers to the appearance of the TRN being pierced by many axons passing between thalamus and cortex. Different segments of the TRN shell are associated with different thalamic nuclei as well as their corresponding

cortical areas, such that the TRN segments form a crude map mirroring the various cortical areas.

The visual segment lies in the caudal portion of TRN, adjacent to LGN. In carnivores, the portion of visual TRN associated with LGN is displaced from the main body of TRN and called the perigeniculate nucleus, but for simplicity we shall use the term TRN for all species. In most taxa that have been examined (cat, rat, rabbit, and two primates, galago and marmoset), the visual segment of TRN is arranged into two parallel laminae or tiers (Baldauf, 2010; Coleman and Mitrofanis, 1996; Conley and Diamond, 1990; Crabtree and Killackey, 1989; Fitzgibbon *et al.*, 2007; Harting *et al.*, 1991b; Lozsádi *et al.*, 1996). The lateral tier, filling the lateral 2/3 of the nucleus, is associated with LGN and striate cortex, while the medial tier is associated with pulvinar and extrastriate cortex. An exception to this tiered arrangement may be macaque monkey, where it has not been reported (Bragg *et al.*, 2017). TRN has been reviewed by (Guillery *et al.*, 1998; Guillery and Harting, 2003; Pinault, 2004).

Primary inputs to the lateral tier of visual TRN come from LGN and striate cortex. Thalamocortical axons passing from LGN to striate cortex traverse TRN. Feedback corticothalamic axons from striate cortex layer 6 to LGN also traverse TRN. As they pass through, both thalamocortical axons (Ahlsén and Lindström, 1982; Ahlsén *et al.*, 1983; Dubin and Cleland, 1977) and corticothalamic axons (Boyapati and Henry, 1984; Ohara and Lieberman, 1981; Robson, 1984) give off collaterals that form excitatory synapses on TRN cells (Figure 2b). Striate and LGN inputs to TRN are both topographically organized (Coleman and Mitrofanis, 1996; Conley and Diamond, 1990;

Crabtree and Killackey, 1989; Fitzgibbon *et al.*, 1999; Harting *et al.*, 1991b; Lozsádi *et al.*, 1996; Montero *et al.*, 1977).

The output from TRN projects back to LGN (Cucchiaro *et al.*, 1991b; Uhlrich *et al.*, 2003; Wang *et al.*, 2001) and is inhibitory (Burke and Sefton, 1966; Lo and Sherman, 1994; Sanchez-Vives and McCormick, 1997; Sumitomo *et al.*, 1976). Thus, TRN neurons projecting to LGN are a source of feedback inhibition to LGN, in contrast to the intrinsic inhibitory neurons within LGN, which provide feedforward inhibition. There is physiological evidence that TRN neurons also project to the intrinsic inhibitory neurons in LGN (Ahlsén *et al.*, 1985), thus providing a disinhibitory influence on the LGN, although a substantial TRN projection to interneurons has not observed neuroanatomically (Cucchiaro *et al.*, 1991b; Wang *et al.*, 2001). The TRN inhibitory field in LGN is bell-shaped, overlapping and aligned with the excitatory field of LGN thalamocortical cells but with a broader diameter. Inhibitory feedback from TRN to LGN is topographically organized (Coleman and Mitrofanis, 1996; Conley and Diamond, 1990; Crabtree and Killackey, 1989; Harting *et al.*, 1991b; Lozsádi *et al.*, 1996; Pinault and Deschênes, 1998). Feedback is not just closed loop, in which a TRN neuron projects directly to the same thalamic cells that provides its input, but also nonreciprocal or open loop, in which the TRN projects to other thalamic cells (Lo and Sherman, 1994; Pinault and Deschênes, 1998). Speculations on the possible significance of open loop vs. closed loop inhibitory feedback by TRN are discussed by Willis *et al.* (2015) as well as Halassa and Acsády (2016).

The spatial receptive fields of TRN cells do not resemble the receptive fields of their inputs from either LGN or cortex. Rather, they have a diverse and complex

organization in which no stereotyped pattern emerges (Vaingankar *et al.*, 2012). TRN cells do not have a center/surround organization (Dubin and Cleland, 1977; Sanderson, 1971a; Wróbel, 1982). Their complicated stimulus responses indicate convergent inputs from on- and off-center LGN thalamocortical cells (Ahlsén *et al.*, 1983; Lindström and Wróbel, 2011; Wróbel and Tarnecki, 1984). Combined with the topographic nature of the feedback to LGN, cells with such receptive fields have the potential to perform localized modulation of LGN transmission based on high-order features in visual stimuli, though this has never been demonstrated. In addition to convergence of on- and off- center inputs, there is convergence of signals from the two eyes, so that many TRN cells are binocular (Sanderson, 1971a; Uhlrich *et al.*, 1991; Vaingankar *et al.*, 2012; Xue *et al.*, 1988).

Perhaps another factor contributing to the complexity of TRN spatial responses are lateral interactions between nearby TRN cells. For chemical synapses, these interactions are inhibitory (Ahlsén and Lindström, 1982; Lam *et al.*, 2006; Sanchez-Vives *et al.*, 1997)(although Hou *et al.* (2016) failed to observe these intrinsic inhibitory connections). There are also electrical synapses. Neighboring pairs of TRN cells may be electrically coupled (Deleuze and Huguenard, 2006; Landisman *et al.*, 2002), correlating their activities. Correlations achieved through electrical synapses may be important in synchronizing population activities within the geniculocortical loop (Fuentealba and Steriade, 2005), a topic that will be further discussed in Section 4.

A question to consider is the role of the different functional streams in the processing of visual signals by TRN. In cat TRN (perigeniculate), there is evidence that the X and Y streams remain segregated (Ahlsén *et al.*, 1983; Wróbel and Bekisz, 1994).

We don't know if the M, P, and K streams in primates are segregated. However, it is known in primates that individual TRN cells project through multiple LGN layers, making synaptic contacts with many LGN cell types along the way regardless of ocularity or functional stream (Bragg *et al.*, 2017; Uhlrich *et al.*, 2003). Therefore, whether or not TRN cells themselves retain a stream identity in primates, the indiscriminate nature of their connections in LGN means that the TRN feedback to LGN is not stream specific, unlike cortical feedback to LGN, which is stream specific. One interpretation of this indiscriminate TRN inhibitory feedback is that it is engaged in a nonspecific gating of activity through LGN, but it could also be argued that allowing crosstalk between different visual functional streams in indicative of more complex processing.

The thinner medial tier of TRN (medial 1/3) is connected to pulvinar rather than LGN (Coleman and Mitrofanis, 1996; Conley and Diamond, 1990; Crabtree and Killackey, 1989; Harting *et al.*, 1991b; Lozsádi *et al.*, 1996). It receives cortical inputs from a variety of extrastriate visual areas rather than V1 (Coleman and Mitrofanis, 1996; Conley and Diamond, 1990; Crabtree and Killackey, 1989; Graham *et al.*, 1979; Lozsádi *et al.*, 1996). The microcircuitry by which the lateral and medial tiers of TRN tiers interact is not yet known.

In addition to inputs from thalamus and visual cortex, the visual segment of TRN receives direct projections from some areas of prefrontal cortex, primarily orbitofrontal cortex and dorsolateral prefrontal cortex (Zikopoulos and Barbas, 2006), as well as projections from the amygdala (Zikopoulos and Barbas, 2012). The significance of the prefrontal projection for attentional modulation will be further discussed in Section 7.1.

The amygdalar projection raises the possibility of emotional modulation of LGN, which has not yet been explored. TRN also receives diffuse modulatory inputs from brain stem structures (Ahlsén *et al.*, 1984; Hallanger *et al.*, 1987; Morrison and Foote, 1986) and basal forebrain (Asanuma, 1994).

In contrast to the standard view that there is no communication between thalamic nuclei except through cortical feedback, TRN may serve as a nexus that allows different thalamic nuclei to interact without going through cortex (Crabtree, 1999; Crabtree and Isaac, 1998, 2002). A possible example of intrathalamic communication through TRN involves the modulation of LGN activity by saccadic eye movements (Section 2.4.6). In rabbits, stimulation of the superior colliculus, involved in generating eye movements, can lead to inhibition of LGN through a pathway in which the central lateral nucleus of the thalamus, a motor structure, and the LGN communicate with each other across TRN (Lo, 1988; Zhu and Lo, 1996). Projections of the pulvinar to the TRN sector for the LGN may also be a source of eye movement modulations to LGN, though this has not been investigated. Another possible example of intrathalamic communication involves cross-modal interactions between responses to visual and auditory stimuli in TRN (Kimura, 2014). Cross-modal interactions mediated by TRN appear to primarily involve individual TRN cells receiving afferents from multiple thalamic nuclei, or projecting to multiple thalamic nuclei, rather than direct projections between different TRN sectors (Crabtree and Isaac, 2002; Kimura, 2014; Kimura *et al.*, 2007; Lam and Sherman, 2011; Pinault and Deschênes, 1998). Overall, if LGN can communicate with other thalamic nuclei through TRN interactions without cortical involvement, then that would increase the

complexity of processing being carried out in LGN relative to the conventional, non-interacting stovepipe picture of LGN transmission of visual signals.

3.4 Brainstem inputs

Besides retinal and feedback inputs to LGN, another major non-thalamic input comes from brainstem structures, which accounts for another 30% of the total synapses. Of particular significance are the largely cholinergic inputs from parabrachial complex (De Lima and Singer, 1987; Fitzpatrick *et al.*, 1989). Also important are inputs involved in eye movements, coming from the superior colliculus (previously discussed in connection with LGN K cells in Section 2.4.3) and from the pretectum (Section 2.4.6) (Cucchiaro *et al.*, 1991a; Hughes and Mullikin, 1984). There are additional smaller noradrenergic input from the locus coeruleus and serotonergic input from the dorsal raphe nucleus (Ahlsén and Lo, 1982; Bickford *et al.*, 2000; De Lima and Singer, 1987), as well as histaminergic input from the tuberomamillary nucleus of the hypothalamus (Uhlrich *et al.*, 1993).

Brainstem inputs to LGN are associated with arousal from sleep (McCormick, 1989; Steriade, 2004) and more generally with switching LGN neuron dynamics from burst to tonic mode (Lu *et al.*, 1993; Sherman, 1996) (bursting will be covered in Section 6). Cholinergic inputs, either through stimulating the parabrachial nucleus or through applying acetylcholine to mimic parabrachial activity, activate LGN responses. The activation can occur indirectly through reducing activity of inhibitory cells (disinhibition) (Ahlsén, 1984; Ahlsén *et al.*, 1984; Singer, 1977) or directly by depolarizing thalamocortical cells (Hu *et al.*, 1989; McCormick and Prince, 1987; Sillito *et al.*, 1983) (reviewed by (McCormick, 1989)). Parabrachial stimulation increases contrast gain in

LGN (Fjeld *et al.*, 2002; Hartveit and Heggelund, 1995) and enhances LGN responses to drifting sinusoidal grating stimuli (Uhlrich *et al.*, 1995), consistent with a general role connected with arousal.

Although the retina is not generally thought of as receiving inputs from the brain, it does receive histaminergic and serotonergic projections from the posterior hypothalamus and dorsal raphe nucleus respectively, which may be involved in arousal (Gastinger *et al.*, 2006). These inputs to retina would serve as another, indirect, route for subcortical modulations of LGN.

4 Precision, coherence, and oscillations

Traditionally, theories of neural processing have emphasized average firing rate in response to stimuli as the central carriers of visual information. In recent years, however, there has been increasing appreciation that temporal aspects of neural responses on a millisecond time scale may be critically important as well (Abeles *et al.*, 1994; Bruno, 2011; Buzsáki and Draguhn, 2004; Fries, 2005; Singer, 1999; Softky, 1995; Tiesinga *et al.*, 2008; Uhlhaas *et al.*, 2009) (but see Shadlen and Movshon (1999) as well as Ray and Maunsell (2015) for critiques of this viewpoint). Of particular interest has been coherent (synchronous) firing in a population of cells, which may take the form of phase locked oscillations, both within a given brain area and across areas. With this in mind, a number of studies have examined coherence and oscillations in LGN responses, and how such temporal factors affect information transmission from LGN to cortex.

One indication that LGN may be using a temporal code rather than average firing rate derives from observations of high temporal precision in LGN spike timings. With repeat presentations of the same stimulus, a particular LGN cell will fire at reproducible

times with a precision of around 1 ms (Butts *et al.*, 2007; Reich *et al.*, 1997; Reinagel and Reid, 2000), greater than the precision in cortical cells (Veredas *et al.*, 2005), and greater than the precision required by the relatively slowly varying stimuli. The data of Blitz and Regehr (2005) indicate that inhibitory interneurons play a role in producing these precise thalamocortical responses. Increased response precision occurs when a thalamocortical cell receives time locked excitatory and inhibitory inputs arising from the same retinal ganglion cell (as would occur in glomerular synaptic triads). The time locked inhibition, having a reliable 1 ms latency relative to excitation, allows the initial spike in the thalamocortical response to occur but blocks subsequent ones. If neural processing were based on average firing rates, there would probably not be a need to create this level of spike time precision.

Beyond precision in responses of single LGN cells, there is also high precision in spike timings across nearby LGN cells in a manner that generates synchronous activity dependent on stimulus onset. Because LGN cells of the same type respond similarly to stimuli by producing reliable and precise spike timings, visual signals frequently arrive from LGN to cortex in the form of synchronized volleys. In general, spike-timing precision is about 10 ms for nearby LGN cells (Desbordes *et al.*, 2010; Desbordes *et al.*, 2008; Kumbhani *et al.*, 2007). Tight firing synchrony within 1 ms occurs for some LGN cells (Alonso *et al.*, 1996; Alonso *et al.*, 2008; Reinagel and Reid, 2002), with such cells possibly forming a functionally important subset. Tight synchronization across cells arises when multiple LGN cells receive divergent inputs from the same retinal ganglion cell. At the population level, it has been repeatedly emphasized, in both the experimental literature (Butts *et al.*, 2007; Desbordes *et al.*, 2008; Kara and Reid, 2003; Stanley *et al.*,

2012) and theoretical literature (Brette, 2012; Masquelier, 2012, 2013), that precision in relative spike timings across cells, and not just absolute spike timings, may be an important aspect of stimulus encoding.

Possible functional significance of synchronous LGN firing may lie in observations that such synchronous firing is more effective in driving cortical cells than non-synchronous firing (Alonso *et al.*, 1996; Bruno and Sakmann, 2006; Schoonover *et al.*, 2014; Usrey *et al.*, 2000). EPSPs that are aligned in time, arising from multiple LGN cells impinging on the same cortical cell, will drive the cortical cell more strongly through postsynaptic summation of EPSPs than the same number of EPSPs arriving in a more dispersed manner. These experimental observations are reinforced by modeling (Wang *et al.*, 2010a). Synchronous LGN firing appears to increase the sensitivity of cortical responses to weak stimuli, and increase the efficacy of information transfer from LGN to cortex.

However, the efficacy of thalamocortical communication depends not just on the state of the LGN cells transmitting a visual signal, but also on the state of the cortical cells receiving it. Cortical cells are more likely to respond to LGN input if their activity levels are already high and if they are oscillating at gamma frequencies, with the phase of the cortical gamma oscillation at the time the LGN input arrives possibly being of importance (Briggs and Usrey, 2007a).

While nearby LGN cells with synchronized firing have similar receptive field properties, they are not identical. A consequence of having non-identical receptive fields is that the degree of coherence amongst theses LGN cells will be dependent on the nature of the visual stimulus (Alonso *et al.*, 2008; Kelly *et al.*, 2014; Stanley *et al.*, 2012). In

other words, properties of the visual stimulus can be indicated by changes in coherence in a local population of LGN cells, without changes in their firing rates. Using coherence to encode stimulus properties has also been studied through modeling (Brette, 2012). Whether or not coherence-based coding of stimulus parameters is actually used, in addition to firing-rate based coding, is an open question.

A class of cells that may be particularly important for creating thalamocortical synchrony can be identified by neurochemical criteria (Jones, 2001, 2002). Jones divided thalamic neurons into two classes, core cells and matrix cells, arguing that matrix cells were involved in synchrony. Core cells are parvalbumin-immunoreactive, and matrix cells are calbindin-immunoreactive, where parvalbumin and calbindin are two classes of calcium binding proteins. This neurochemical difference extends to differences in neuroanatomical projections. Core neurons project in a topographically ordered fashion to middle layers of the cortex in an area-specific manner, while matrix project to superficial layers of cortex diffusely over wide areas, unconstrained by boundaries between areas. The diffuse projections of matrix cells allow localized oscillations to become synchronized across multiple cortical areas, according to the hypothesis of (Jones, 2001, 2002).

In LGN, core cells appear to correspond to M and P cells. Matrix cells may match with K cells, given the presence of matrix cell neurochemical markers on K cells (Hendry and Yoshioka, 1994; Xu *et al.*, 2001). Therefore, under this neuroanatomically based argument, K cells in LGN should have a larger role than M and P cells in establishing widespread corticothalamic synchrony, an idea that has yet to be tested neurophysiologically.

Synchronized firing limits the amount of information that can be extracted from averaging activities in a population (Mazurek and Shadlen, 2002; Shadlen and Newsome, 1998). However, modeling by (Kenyon *et al.*, 2004; Stephens *et al.*, 2006) has shown that the nature of the correlations themselves can contain information about the stimulus and thus enhance stimulus discriminability. While their work was done in retina, the same principle should apply in LGN. Although correlated firing across a population of LGN cells might seem to reduce the amount of information being transmitted, the precision of spike timings allows more information to be carried by the temporal pattern of responses, beyond that carried by mean firing rate alone. The amount of information being transmitted may actually be greater for correlated activity when spike timings are considered (Butts *et al.*, 2007; Dan *et al.*, 1998).

Oscillations have become increasingly central to theoretical ideas about information processing in the brain (Buzsáki and Draguhn, 2004; Fries, 2005; Wang, 2010). The frequency bands of greatest interest in awake, behaving animals are alpha (8-12 Hz), beta (15-30 Hz), and gamma (60-120 Hz). These are measured as either EEG signals or local field potentials (LFP), both of which are largely dependent on dendritic currents. These oscillations are frequently synchronized over broad regions of a cortical or thalamic area, and synchronized between different areas in a manner that depends on the state of the animal (McCormick *et al.*, 2015). The phase of synchronous oscillatory activity is often not locked to stimulus onset. Rather, there is phase locking of oscillations in neurons to each other across a population, randomly set relative to stimulus onset. While a common stimulus input is a feedforward source of synchrony for LGN neurons, synchrony can also occur in a manner independent from the stimulus through

thalamocortical feedback interactions, perhaps reflecting more abstracts aspects of the visual signal processing.

High frequency gamma oscillations are associated with feedforward processing, while the lower frequency beta oscillations (often including the alpha band) are associated with feedback (Bastos *et al.*, 2014; Bastos *et al.*, 2015; Buffalo *et al.*, 2011; Buschman and Miller, 2007; Jensen *et al.*, 2015; Michalareas *et al.*, 2016; Zheng and Colgin, 2015). Wróbel (2000) presents the hypothesis that alpha oscillations are preferentially involved in arousal, beta oscillations in attention, and gamma oscillations in feature binding (see Roskies (1999) and von der Malsburg (1999) for discussions of binding). However, all these three functions may have both bottom-up and top-down aspects to them, so this idea may have to be further elaborated in light of the recent evidence associating particular frequency bands with feedback or feedforward processing.

Evidence for the involvement of gamma oscillations in LGN for feature binding comes from Neuenschwander and Singer (1996) and Castelo-Branco *et al.* (1998). They found that if the stimulus formed a single, large object, then LGN spikes were phase locked to long-range coherent gamma oscillations spread over a broad area of LGN, possibly covering over 20^{o} of visual angle. If the stimulus was broken into disjoint parts, the coherence was lost. These LGN oscillations appear to be inherited from the retina, through the intrinsic dynamics of networks of retinal neurons. The role of synchronized oscillations in LGN for attention will be considered in Section 7.1.

Coherence within a system is an indication of redundancy. There is therefore a tension between the observations outlined in this section indicating benefits of redundancy in visual representations, and approaches to visual function emphasizing

redundancy reduction and efficient coding. Within information theory, it is recognized that, in addition to the advantages of efficient coding, there are also advantages to redundancy with respect to the reliable transmission of information in a noisy system. Therefore, within LGN there is a need to strike a balance between the reliability of redundancy (when the signal to noise ratio is low) and the efficiency of redundancy reduction (when the signal to noise ratio is high). Designing the system to maximize the amount of information transmitted at all times is one way to reconcile these two goals (Van Hateren, 1993). Furthermore, as Hillenbrand and van Hemmen (2002) point out, representational coherence and redundancy reduction need not be mutually exclusive goals. Representational coherence could be occurring at the time scale of individual perceptual events (hundreds of milliseconds) while redundancy reduction could track longer-term regularities in the environment.

5 How is the LGN affected by cortical feedback connections?

The function of the massive feedback projections from striate cortex to LGN, far outnumbering feedforward retinal projections, has been an enduring mystery. Although a broad range of effects have been noted when feedback has been experimentally manipulated, it is difficult to assess which if any of them reflect core LGN functionality under natural conditions. Reviews of feedback to LGN from striate cortex include (Briggs, 2010; Briggs and Usrey, 2008, 2011; Sillito *et al.*, 2006). LGN also receives direct feedback projections from V2 and MT in some species of monkeys (Briggs *et al.*, 2016; Lin and Kaas, 1977), but their physiology has been less studied.

Striate cortex sends topographically organized projections from layer 6 back to LGN thalamocortical cells, as well as to LGN inhibitory interneurons and TRN inhibitory

neurons (Gilbert and Kelly, 1975; Ichida and Casagrande, 2002; Montero, 1991). The excitatory nature of these feedback projections is shown by the presence of EPSPs in the thalamocortical cells when corticothalamic cells are stimulated (e.g., Deschênes and Bin, 2006; Lindström and Wróbel, 1990a; McCormick and von Krosigk, 1992). In addition, there are disynaptic inhibitory effects on thalamocortical cells mediated by striate projections to both inhibitory interneurons in LGN and inhibitory neurons in TRN. Of these two, the TRN appears to be a more potent source of inhibitory feedback (Jurgens *et al.*, 2012). Synapses from corticogeniculate axons occur primarily on distal dendrites (Wilson, 1989; Wilson *et al.*, 1984), in contrast to synapses from retinal inputs to LGN which occur predominantly on proximal dendrites. Although the distal location of cortical feedback is frequently interpreted as indicating a modulatory role for the feedback, there is evidence that voltage dependent amplification mechanisms in the dendrites can allow distal inputs to be as effective as proximal inputs in their effects at the soma, in some cases allowing the distal inputs to drive the cell (Connelly *et al.*, 2016). Cortical feedback stimulates both fast ionotropic (Deschênes and Hu, 1990; Scharfman *et al.*, 1990) and slow metabotropic (Godwin *et al.*, 1996; McCormick and von Krosigk, 1992) glutamate receptors. Involvement of both classes of receptors means that feedback can dynamically influence the visual signal passing through LGN as it occurs, as well as provide long-lasting state changes to the visual processing.

Cortical feedback to LGN involves both simple and complex cells. There is disagreement, however, whether these corticothalamic cells are predominantly simple or complex. Some studies have reported that feedback cells in Layer 6 are mostly simple (Ferster and Lindström, 1983; Grieve and Sillito, 1995; Harvey, 1980) while others have

reported mostly complex receptive fields (Gilbert, 1977; Tsumoto *et al.*, 1978). Perhaps resolving this disagreement, Briggs and Usrey (2009) found an association between the simple/complex classification and the M/P/K streams, in which P feedback was simple while both M and K feedback were complex. As simple cells have spatially separate on/off regions and complex cells don't, the spatial organization of interactions of corticothalamic cells with LGN thalamocortical cells (which do have spatially separate on/off regions) may be quite different for P cells compared to M and K cells.

The striate feedback is oriented, directionally selective for motion, and in the case of complex cells also binocular (Gilbert, 1977). Latency of feedback can be as short as 5 ms (Briggs and Usrey, 2007b). Feedback from a particular cortical cell extends over those LGN cells whose retinotopic positions overlap those of the cortical cell (Angelucci and Sainsbury, 2006; Ichida and Casagrande, 2002; Murphy and Sillito, 1996). Thus, arborizations of feedback axons are anisotropic, in most cases synapsing upon row of LGN cells corresponding to the preferred orientation of the cortical neuron (Murphy *et al*., 1999; Sillito *et al*., 1994; Wang *et al*., 2016; Wang *et al*., 2006). Thus, the feedback inverts the classic Hubel and Wiesel (1962) model for cortical orientation selectivity.

The parallel channels found in LGN thalamocortical cells (magnocellular, parvocellular, and koniocellular) maintain their separate identities within the cortical feedback projections both functionally and anatomically (Briggs *et al.*, 2016; Briggs and Usrey, 2009; Conley and Raczkowski, 1990; Ichida *et al.*, 2014; Ichida and Casagrande, 2002; Lund *et al.*, 1975; Tsumoto and Suda, 1980). The separation of streams can be seen in the anatomy of the feedback from layer 6, in which the upper part of layer 6 projects to the parvocellular regions of LGN while the lower part projects to the magnocellular and

koniocellular regions (Briggs *et al.*, 2016; Fitzpatrick *et al.*, 1994; Usrey and Fitzpatrick, 1996).

Cortical feedback produces complex mixtures of excitatory and inhibitory effects on LGN thalamocortical cells, although it appears that excitatory effects predominate for spatially restricted stimuli (de Labra *et al.*, 2007; Deschênes and Bin, 2006; Geisert *et al.*, 1981; Gulyás *et al.*, 1990; Hull, 1968; Kalil and Chase, 1970; Vastola, 1967; Waleszczyk *et al.*, 2005; Webb *et al.*, 2002). Excitatory feedback would reflect direct interaction of cortical cells with LGN thalamocortical cells, while inhibitory feedback would reflect indirect interactions, mediated primarily by TRN but also intrinsic LGN neurons.

How one might explain this diversity of excitatory and inhibitory interactions in terms of the spatial organization of cortical feedback depends on whether one is dealing with simple or complex feedback cells. Tsumoto *et al.* (1978), based on a sample of feedback cells that were mostly complex and therefore did not have discrete on/off regions, reported that feedback was excitatory when the cortical and LGN receptive fields centers where close to each other, and inhibitory when they were further apart. Potent suppression of LGN by cortical feedback neurons was interpreted as consistent with this center-surround model of cortical feedback effects, in which suppression dominates when using full-field stimuli (Olsen *et al.*, 2012).

On the other hand Wang *et al.* (2006), examining feedback cells that were simple, reported quite a different functional organization for cortical feedback based on the presence of discrete on- and off- regions in those simple cells. They interpreted their data as showing that on- and off-regions of cortical feedback cells are phase-reversed relative to the on-center and off-center cells in their LGN target population, when having

overlapping receptive fields. For example, an on-center LGN cell will receive an inhibitory influence from an on-center cortical feedback cell, and an excitatory influence from an off-center feedback cell, forming a push-pull feedback arrangement. This orderly arrangement breaks down when LGN and cortex do not have overlapping receptive fields. Note that this phase-reversed organization for simple cell feedback is significant for predictive coding models of LGN function (Jehee and Ballard, 2009; Zabbah *et al.*, 2014).

Whether cortical feedback is excitatory or inhibitory depends not only on the spatial organization of the feedback, but also on feedback strength. The balance between direct feedback excitation and indirect feedback inhibition mediated by TRN is controlled by the firing rate of feedback. High feedback firing rates lead to excitation of thalamocortical cells while low firing rates leads to inhibition (Crandall *et al.*, 2015).

The visual responsiveness of LGN cells differ in anesthetized and alert animals (Alitto *et al.*, 2011; Wróbel *et al.*, 1984). Although feedback has been studied in awake cats (Waleszczyk *et al.*, 2005), at present most studies related to the dynamics of LGN cell responses rely on data obtained from anesthetized animals. While anesthetized studies are certainly informative, our understanding of corticothalamic dynamics and the role of corticogeniculate feedback could be improved by performing similar studies with alert animals.

Functional effects of cortical feedback on LGN responses can be placed into three categories: 1. changes in contrast gain; 2. changes in spatial properties; 3. changes in temporal properties.

5.1 Response gain

The idea that cortical feedback modulations act as a non-selective contrast gain control for LGN transmissions has been suggested since early times (Burke and Cole, 1978; Singer, 1977), and in more detail by Ahlsén *et al.* (1985). By non-selective we mean that the gain can be adjusted without altering the spatial or temporal properties of the visual signal, in a manner suitable for retinotopically localized enhancement of particular regions of the visual field. Such a mechanism may be engaged during spatial attention, for example. The localized nature of the cortical feedback modulation differentiates it from brainstem modulations involved in general arousal.

Evidence that cortical feedback can control LGN gain in such a manner comes from Przybyszewski *et al.* (2000), who report that the feedback signal multiplicatively modulates contrast gain of LGN thalamocortical cells (see also Olsen *et al.* (2012)). Others that have interpreted their data in a manner consistent with feedback producing non-selective gain control include (Cudeiro *et al.*, 2000; Granseth, 2004; Granseth *et al.*, 2002; Granseth and Lindström, 2003; Lindström and Wróbel, 1990a). Ichida *et al.* (2014) suggest, based on their neuroanatomical data, that feedback from P and M channels are involved in gain control but feedback from the K channel has a temporal function and is organized to synchronize activity in cortex and LGN.

5.2 Spatial properties

Cortical feedback can change the spatial characteristics of LGN cells by altering the balance of activity between the center and surround of receptive fields, particularly the extraclassical surround (Andolina *et al.*, 2013; Geisert *et al.*, 1981; Jones *et al.*, 2012; Kalil and Chase, 1970; Marrocco *et al.*, 1982; McClurkin and Marrocco, 1984; Webb *et*

al., 2002). Corticofugal feedback effects on center/surround antagonism have been modeled by Hayot and Tranchina (2001).

One manifestation of the dependence of receptive field spatial organization on feedback is the observation that length tuning to moving bars is much tighter (i.e., tuning is to shorter bars) with feedback than without it (Jones and Sillito, 1991; Murphy and Sillito, 1987; Rivadulla *et al.*, 2002). There are similar effects on size tuning for the diameter of patches of drifting gratings (Cudeiro and Sillito, 1996). It is significant that these effects occur only for moving stimuli, as that may reflect feedback influences of area MT on striate layer 6, which in turn feeds back to LGN (Jones *et al.*, 2013). Possibly other extrastriate areas besides MT, such as V2 (Briggs *et al.*, 2016), may exert direct or indirect feedback effects on LGN responses. One would expect that cortical feedback from V1 would impose some of the spatial properties of striate cells upon LGN responses, and we see that in the observation of orientation-specific suppression in the LGN (Cudeiro and Sillito, 1996; Sillito *et al.*, 1993). Further indication that cortical feedback can modulate the spatial organization of LGN receptive fields comes observations that such feedback can expand or shrink receptive field diameter as well as shift the receptive field center (Wang *et al.*, 2016).

These spatial modulations raise the possibility that LGN may act as an adaptive spatial filter in which cortical feedback serves to enhance transmission of features which higher-level analyses in cortex finds significant. This idea is discussed by (Wróbel, 2000, 2014) and finds support in the psychophysical data of Han and VanRullen (2016). Modeling from this perspective is presented in Section 8.5. In contrast to non-specific modification of contrast gain, which can be thought of as related to location-based

attention, feedback modification of LGN spatial properties may be related to object-based attention (see (Carrasco, 2011; Chen, 2012) for reviews of object-based attention).

Another aspect of LGN spatial properties possibly affected by cortical feedback is binocular processing. Schmielau and Singer (1977) reported results suggesting that feedback enhances transmission of fused contours on the fixation plane (zero or small disparities). Varela and Singer (1987) observed binocular interactions in LGN that were abolished upon ablation of the cortex. These early findings have not been subsequently extended, as the binocular aspects of LGN function is currently a rather neglected topic.

5.3 Temporal properties

Cortical feedback also affects the temporal characteristics of LGN responses. One notable example of this is the ability of feedback to cause a shift in the balance between burst and tonic firing in LGN corticothalamic cells (Andolina *et al.*, 2013; Godwin *et al.*, 1996; McCormick and von Krosigk, 1992; Ortuño *et al.*, 2014; Sillito and Jones, 2002; Wang *et al.*, 2006; Wolfart *et al.*, 2005) (burst and tonic modes are discussed in Section 6). Cortical feedback also increases the precision of LGN responses (Andolina *et al.*, 2007; Funke *et al.*, 1996; Wörgötter *et al.*, 1998). As outlined in Section 4, possible consequences of increased precision are higher rates of information transmission, as well as more effective activation of cortical cells due to increased synchrony of LGN activity. Increased synchrony induced by cortical feedback has been observed in LGN cells that are aligned along the contour of a bar stimulus (Kirkland *et al.*, 2000; Sillito *et al.*, 1994); modeled by Kirkland and Gerstein (1998). Such synchrony indicates a role of top-down cortical modulations in producing feature binding through synchronization, in addition to bottom-up retina-derived synchronization for feature binding observed by

Neuenschwander and Singer (1996). The importance of cortical feedback in producing synchronized oscillations between cortex and LGN is a central idea that we shall discuss in various contexts, already introduced in Section 4 on oscillations, and to which we shall return to in Section 7 on cognition and Section 8 on modeling.

From a theoretical perspective, an interesting possible role for cortical feedback is in modulating the biphasic temporal impulse response of LGN cells. Although the LGN impulse response is in part inherited from retina, it shows a larger rebound magnitude (larger second phase), as presented in Section 2.4.2. Several models have explored how cortical feedback may sculpt the LGN impulse response (Norheim *et al.*, 2012; Yousif and Denham, 2007). At the moment, however, the hypothesis that removing cortical feedback will affect temporal impulse responses and temporal tuning has not been directly tested experimentally. However, indirect support may come from observation that feedback affects the balance of activity between intrinsic inhibitory cells and thalamocortical cells in LGN (Augustinaite *et al.*, 2011), which would be expected to modify temporal properties of the network (as well as spatial properties).

6 The role of LGN tonic and burst firing modes in visual processing

Action potentials of LGN cells occur in two dynamic modes, burst and tonic (Jahnsen and Llinás, 1984a; Steriade and Deschenes, 1984; Steriade and Llinás, 1988). During burst mode a cluster of 2-6 spikes occurs in rapid succession. In tonic mode, individual spikes occur in isolation, with some random interval between them. Although burst and tonic firing are often thought of as two discrete modes, some have suggested that there is a continuum in the prevalence of burst and tonic firing (Guido *et al.*, 1992; Guido *et al.*,

1995; Guido and Weyand, 1995; Mukherjee and Kaplan, 1995; Wolfart *et al.*, 2005). Bursting in LGN is reviewed by Alitto and Usrey (2005).

The biophysics of bursting has been fairly well worked out (Bessaïh *et al.*, 2008; Coulter *et al.*, 1989; Crunelli *et al.*, 1989; Deschênes *et al.*, 1984; Hernández-Cruz and Pape, 1989; Huguenard, 1996; Jahnsen and Llinás, 1984b; Zhou *et al.*, 1997). Bursts are dependent on T-type Ca^{2+} channels (T=transient), located in the soma and proximal dendrites of thalamocortical cells (Destexhe *et al.*, 1998; Williams and Stuart, 2000; Zhan *et al.*, 2000; Zhou *et al.*, 1997) (in contrast, Connelly *et al.* (2015) report channels in the distal dendrites are most important). When the membrane potential is in a hyperpolarized state, the Ca^{2+} channels are activated. In the activated state, membrane depolarization may be sufficient to cause a slow, low-threshold calcium dependent spike. It is called low threshold because less depolarization is needed to trigger this calcium spike than a conventional sodium spike. The low-threshold spike has been reported to occur synchronously throughout the entire dendritic tree and soma to form a "global spike" (Connelly *et al.*, 2015).

The membrane depolarization caused by the slow calcium spike will generally be sufficient to reach the threshold for triggering conventional Na^{+} dependent spikes, and a burst of such Na^{+} dependent spikes will occur riding on the back of the Ca^{2+} depolarization. When a cell is in a depolarized state, the transient Ca^{2+} channels rapidly switch from an activated to an inactivated state after about 100 ms, so that no further burst firing occurs in response to maintained synaptic input. To return the Ca^{2+} channels to an active state (deinactivate them), the membrane must become re-hyperpolarized for

an extended period of at least 100 ms. When that occurs, burst firing becomes possible again.

Given the biophysics of T-type Ca^{2+} channels, the dynamic mode of LGN can be switched from burst to tonic by any modulatory input that depolarizes the membranes of thalamocortical cells, thereby inactivating the Ca^{2+} channels. Among modulatory inputs that can switch LGN cells from burst to tonic mode are cortical feedback from striate cortex (Section 5.3), as well as parabrachial brainstem input (Section 3.4).

Calcium-dependent bursts can have their appearance superficially mimicked by transient tonic responses to strong stimuli (tonic responses that adapt quickly). However, transient tonic responses have different ionic underpinnings, and would not be classified as bursts in this context. Bursts can only be identified with certainty using intracellular recording, which can monitor in detail the time course of membrane potential changes for various ion channels. In practice, however, bursts are usually identified using spike patterns monitored during extracellular recordings (e.g., Bakkum *et al.*, 2014), typically based on the occurrence of a cluster of spikes with very short interspike interval (<4 ms; Figure 3) together with a preceding quiescent period (~100 ms) (Lo *et al.*, 1991).

Bursting occurs not just in LGN but also throughout the thalamus, with the prevalence of bursting being lowest in the LGN (Ramcharan *et al.*, 2000; Ramcharan *et al.*, 2005; Wei *et al.*, 2011). Bursting also occurs in visual TRN (Bal *et al.*, 1995; Mulle *et al.*, 1986). It is a cortical phenomenon as well, present in striate cortex (Legéndy and Salcman, 1985) as well as other sensory cortices (e.g., Bair *et al.*, 1994; Nicolelis, 2005). Sincich *et al.* (2007) report bursting in adult retina, though this bursting may not be based

on T-type Ca^{2+} channels (Toychiev *et al.*, 2013) and therefore could represent a different phenomenon than the LGN bursting.

In the past, bursting in LGN was associated with slow-wave sleep while tonic firing was exclusively associated with transmission during the awake state (Livingstone and Hubel, 1981; Steriade and Llinás, 1988). However, rather than being exclusively in one mode or the other, Wang *et al.* (2007) showed a mixed response in anesthetized cats exposed to naturalistic videos, with occasional bursting together with predominantly tonic firing. There have been demonstrations that burst firing occurs in the awake state, with early reports of that happening by (Guido and Weyand, 1995; Ramcharan *et al.*, 2000; Weyand *et al.*, 2001). Although bursts clearly do occur in the awake state they are rare. Estimates of the prevalence of spikes associated with bursts typically range from 1-6% (Guido and Weyand, 1995; Ramcharan *et al.*, 2000; Ruiz *et al.*, 2006; Sincich *et al.*, 2007; Weyand *et al.*, 2001; Wolfart *et al.*, 2005), though with an outlier estimate of 25% by Lesica and Stanley (2004). Use of natural stimuli appears to increase the frequency of bursting (Lesica and Stanley, 2004; Reinagel *et al.*, 1999). On the other hand, Ruiz *et al.* (2006) found no difference in LGN bursting in awake monkeys between viewing natural images and viewing a blank screen, which Ortuño *et al.* (2014) attributes to the low attentional load of the Ruiz *et al.* (2006) task.

The functional significance of awake bursting remains debated, with (Hong *et al.*, 2014; Ruiz *et al.*, 2006; Steriade, 2001) among those downplaying the significance of the bursts. For those advocating an important role for bursting in the awake state, a widespread theory is that LGN bursting is a mechanism for attentional orienting by allowing weak or novel stimuli to more effectively activate cortex than through tonic

spikes. Supporting this viewpoint is the finding that LGN burst responses allow enhanced detection of weak stimuli compared to tonic responses (Godwin *et al.*, 1996; Grubb and Thompson, 2005; Guido *et al.*, 1995; Lesica and Stanley, 2004; Lesica *et al.*, 2006; Mukherjee and Kaplan, 1995; Ortuño *et al.*, 2014). Underlying the enhanced detection is a greater signal to noise ratio in burst responses (Guido *et al.*, 1995; Lesica *et al.*, 2006; Reinagel *et al.*, 1999), as well as the ability of the low-threshold calcium spike during bursts to produce a nonlinear, step-function-like amplification of the stimulus (leading to a highly distorted stimulus representation) (Guido *et al.*, 1995; Lesica and Stanley, 2004; Lesica *et al.*, 2006; Smith *et al.*, 2000; Williams and Stuart, 2000). Such nonlinear signal amplification during bursting contrasts with a more linear transfer function during tonic firing.

In turn, the low threshold, amplified burst responses from thalamus are more effective in activating cortex than tonic spikes (Swadlow and Gusev, 2001). The increased effectiveness of bursts for transmitting signals across thalamocortical synapses may be due to the ability of burst firing to enhance the reliability of synaptic transmission (Lisman, 1997), involving either increased postsynaptic summation of EPSPs or increased presynaptic release of transmitter when multiple incoming spikes arrive within a short time interval, as occurs during bursting. (We already saw postsynaptic summation increasing the efficacy of cortical activation for synchronized LGN input in Section 4.) Providing a different perspective, Izhikevich *et al.* (2003) interpret the higher effectiveness of LGN bursts for driving cortical in terms of a resonance mechanism.

Connecting LGN bursts with orienting to new features in the environment are observations that bursts disproportionately occur near the beginning of the stimulus

presentation (Guido *et al.*, 1992; Ortuño *et al.*, 2014), likely a consequence of the biophysical requirement of T-type Ca^{2+} channels for a 100 ms quiescent period in order to become deinactivated. Also, frequency of bursting decreases with stimulus repetition (Ortuño *et al.*, 2014), as stimulus novelty declines.

The general idea here is that bursting provides high sensitivity, short latency detection of stimuli, but with a heavily distorted representation. After this initial response reaches cortex, cortical feedback switches the dynamic mode of LGN from burst to tonic, allowing a low distortion representation of the stimulus to be transmitted. The initial bursting is frequently described as the LGN bursts giving a "wake up call" to cortex, which can be viewed as a form of bottom up or exogenous attention. Attentional aspects of the LGN will be further discussed in Section 7.1.

A second theory focuses on observations that repeated stimulus presentations lead to burst timings that are more precise than those of tonic cells (i.e., lower variance in the response latency) (Alitto *et al.*, 2005; Grubb and Thompson, 2005; Guido and Sherman, 1998). The increased precision can lead to increased information capacity within the visual channel (Borst and Theunissen, 1999; Kara *et al.*, 2000; Kepecs and Lisman, 2003; Reinagel *et al.*, 1999; Zador, 1998).

A third theory holds that LGN bursts signal specific stimulus attributes, different from those signaled by tonic spikes. It has been shown that bursts in the awake state can carry information about the visual stimulus (Denning and Reinagel, 2005; Reinagel *et al.*, 1999). For repeated displays of movies containing naturalistic images, bursts are reliably generated at specific, reproducible times within the image sequence (Figure 3), as shown experimentally by Lesica and Stanley (2004) and through simulations by Lesica *et al.*

(2006). An LGN cell is stimulated when a particular spatial pattern enters its receptive field, but in order to be in a state that allows bursting it also needs a particular temporal organization in the sequence of spatial patterns, enforced by the requirement of T-type Ca^{2+} channels the 100 ms quiescent period before they can produce bursts. Moreover, the spatial organization of the receptive field is itself somewhat different under burst mode compared to tonic mode (Alitto *et al.*, 2005; Rivadulla *et al.*, 2003). Thus, the implication here is that bursting cells respond to a different portion of the stimulus spatiotemporal domain than cells in tonic mode. We shall return to the idea that bursts can encode stimulus attributes in Section 8.4.

Taking a radically different viewpoint, there are reports suggesting that the major function of T-type Ca^{2+} channels is not to create bursts but to modulate response properties during tonic firing. Even though most T-type Ca^{2+} are inactivated at the membrane depolarization levels occurring during tonic mode, the absolute number of such channels is large enough such that even having only a small percentage of them active is still sufficient to produce physiologically significant effects (Deleuze *et al.*, 2012). These data suggest that T-type Ca^{2+} channels may stabilize the input-output transfer function of thalamocortical cells across variations in membrane potential while remaining within tonic mode. The observation that T-type Ca^{2+} channels can modulate tonic firing properties was confirmed by Hong *et al.* (2014), who interpreted those channels as implementing a homeostatic mechanism for efficient coding, maintaining membrane excitability at the optimum level so that spike generation is maximally sensitive to small gradations in synaptic drive (Fiorillo *et al.*, 2014; Hong *et al.*, 2014). Therefore, from this perspective it is possible that the occasional appearance of bursts in

awake animals is an epiphenomenon, due to side effects from T-type Ca^{2+} channels that are engaged in something entirely unrelated.

7 Cognition and higher-level visual processing

It is increasingly clear that the thalamus is involved in cognitive functions. Such involvement is reflected in a recent collection of papers about cognition in higher-order thalamic nuclei introduced by Saalmann and Kastner (2015). It also appears to be true for sensory thalamic nuclei such as the LGN, as presented below.

7.1 Effects of attention in LGN

Attention plays a significant role in selecting preferred visual information in cluttered natural scenes, as reviewed by Carrasco (2011). Visual responses in all cortical areas that have been examined exhibit attention-related modulations in their activities, as shown by both single-cell neurophysiological studies and brain imaging studies (Bosman *et al.*, 2012; Chelazzi *et al.*, 2011; Corbetta and Shulman, 2002; Harris and Thiele, 2011; Kanwisher and Wojciulik, 2000; Miller and Buschman, 2012; Peelen and Kastner, 2014; Pessoa *et al.*, 2003; Squire *et al.*, 2013). Attentional modulations extend into subcortical structures, including the superior colliculus (Krauzlis *et al.*, 2013) as well as thalamic structures.

Much of the early cognitive work in the thalamus examined attentional influences on the thalamic reticular nucleus (TRN) rather than directly studying the thalamus (including LGN). The motivation for this was the neuroanatomical organization of TRN. As described earlier (Section 3.3), the visual sector of TRN receives excitatory inputs from both the LGN and V1, and projects inhibitory inputs back to the thalamocortical

neurons. Such inhibition of LGN that integrates thalamic and cortical information suggests the possibility that TRN could be a locus for attentional gating of LGN. Seminal in this respect was the work of (Yingling and Skinner, 1976, 1977), who showed that different TRN sectors could independently gate different thalamic nuclei in a modality specific manner. Observation of modality specific inhibition opened the possibility that thalamic gating was controlled by selective attention to particular aspects of the stimulus rather than by nonspecific shifts in the general level of arousal. Interest in TRN was strongly boosted by the influential "searchlight hypothesis" model of Crick (1984), which posited a key role for TRN in attentional gating of the thalamus.

Behavioral evidence for TRN involvement in attentional orienting first came from Weese *et al.* (1999), who showed lesioning of TRN disrupted behavioral performance in a variant of the Posner attentional cueing task (see Posner, 1980). Further evidence of attentional modulation of TRN was provided by McAlonan *et al.* (2000) using a classical conditioning task. They showed activity in visual TRN was selectively activated by the attended stimulus, as measured by elevated levels of expression of c-fos protein (see Kawashima *et al.* (2014) for a review of the c-fos method). More recent studies supporting a role of TRN in attention include (Halassa *et al.*, 2014; McAlonan *et al.*, 2006; Petrof and Brown, 2010), all showing changes in TRN activation correlating with changes in the attentional state of the animal.

Using genetically modified knockout mice provides a new approach to examining attentional effects in TRN. Behavioral studies on a knockout mouse strain, in which inhibition in LGN was reduced through a gene deletion attenuating TRN activity, led to visual attentional deficits in which irrelevant stimuli that were normally suppressed

became highly distracting (Wells *et al.*, 2016). The increased susceptibility to attentional focus being disrupted by distractors when TRN function is attenuated supports the "leaky thalamus" model of attentional deficits. A different knockout mouse strain in which TRN activity was enhanced rather than attenuated led to an opposite behavioral deficit. Rather than being impaired by increased distractibility to irrelevant stimuli, these mice showed behavioral perseveration with an inability to switch attention from an irrelevant cue to a relevant one as the goal of the task changed (Ahrens *et al.*, 2015). Both these studies strengthen the possibility that abnormal TRN responses contribute to human cognitive disorders with an attentional component, such as attention deficit hyperactivity disorder and autism spectrum disorder, as well as schizophrenia (Ferrarelli and Tononi, 2011; Pinault, 2011).

Particularly significant are observations that prefrontal cortex can modulate TRN activity during an attention task, as prefrontal cortex is high-level executive region which may be the ultimate source of top-down attentional effects (Miller, 2000). Wimmer *et al.* (2015) found that during a visual attention task in mice, TRN responses were modulated by perturbations in prefrontal cortex activity but not perturbations in sensory cortex. The attentional behavior of the mice correlated with the TRN modulations. These data suggest direct top-down causal links between prefrontal cortex and thalamus for cognitive control, bypassing striate and extrastriate visual cortex. Such direct prefrontal control of thalamic gating is reinforced by the finding that prefrontal cortex directly projects to visual TRN in monkeys (Zikopoulos and Barbas, 2006, 2007). In addition to high-level control of LGN transmission by attention, it is possible that LGN transmission is affected

by emotional factors, as TRN also receives direct projections from orbitofrontal cortex and the amygdala (Zikopoulos and Barbas, 2012).

Overall, the evidence is in accord with the broad hypothesis of Crick (1984) that TRN is a critical structure involved in attentional gating of visual signals at the LGN, although some of the particular neural mechanisms he proposed are not supported. The recent report that TRN can be imaged using fMRI (Viviano and Schneider, 2015) broadens the scope of future studies on its role in attention.

Concerning the LGN itself, early work examined attentional effects on field potential oscillations, as attention is often associated with coherent oscillations (Wang, 2010). Attention directed at visual stimuli produced elevated power at beta frequencies in local field potentials in cat LGN (Bekisz and Wróbel, 1993, 1999; Wróbel *et al.*, 1994), leading to the hypothesis that beta frequency cortical feedback activity causes subthreshold depolarization in LGN within the focus of attention, leading to lower stimulus thresholds (Wróbel, 2000, 2014). Recent observations confirm that beta frequency oscillations are associated with cortical feedback in various brain areas (Bastos *et al.*, 2015; Michalareas *et al.*, 2016). Beta frequency cortical feedback to LGN circuitry appears to be a promising area for future research on cognitive modulation in LGN.

Moving to brain imaging studies, attentional modulation in LGN was shown in a radiolabeled 2-deoxyglucose study of macaque monkeys conducted by Vanduffel *et al.* (2000), who found attention-dependent suppression of activity in the magnocellular layers of LGN. Attention-related observations in LGN were extended by human fMRI observations showing enhanced responses to attended stimuli and suppressed responses to non-attended stimuli (O'Connor *et al.*, 2002). Notably contradicting this report, Gouws

et al. (2014) did not observe enhanced fMRI responses from LGN at attended locations, but did agree there was suppression at unattended locations, interpreting this suppression as supporting the normalization model of attention from Reynolds and Heeger (2009). However, as we shall see below, single-cell recordings do support the existence of attentional enhancement of LGN responses.

Among other fMRI findings, attentional modulations are stronger in LGN than in V1 cortex (O'Connor *et al.*, 2002) but weaker than in superior colliculus (Schneider, 2011; Schneider and Kastner, 2009), possibly an indication that LGN is receiving attentional signals not only via cortical feedback but also from subcortical structures. LGN attentional modulations appear to be stronger in magnocellular layers than parvocellular layers, though they occur in both (Schneider, 2011; Schneider and Kastner, 2009). While there is psychophysical evidence for associating attention more strongly with magnocellular pathways, that remains controversial (Laycock *et al.*, 2008; Skottun and Skoyles, 2008). Finally, fMRI studies show that LGN is modulated not only by spatial attention (O'Connor *et al.*, 2002; Schneider and Kastner, 2009), but by feature attention as well (Ling *et al.*, 2015; Schneider, 2011).

Further evidence for cognitive modulations in LGN comes from an fMRI study showing LGN activation by visual imagery (Chen *et al.*, 1998), presumably through cortical feedback; (see Kosslyn *et al.* (2001) for general background on imagery). A graph-theoretic network analysis of resting-state human fMRI data provides support for the idea that LGN participates in cognitive networks that spread widely across the brain (Hwang *et al.*, 2016). The analysis showed the LGN had characteristics of a “connector

hub", capable of integrating multimodal information across diverse cortical functional networks.

Given the strong anatomical and physiological links between TRN and LGN, it would seem straightforward that the well-established attentional modulations in TRN would lead to observations of similar modulations in LGN in single-cell recordings. Nevertheless, the first monkey single-cell neurophysiological studies failed to such attentional effects in LGN (Bender and Youakim, 2001; Mehta *et al.*, 2000a, b).

Attentional modulation of LGN activity at the single-cell level was finally demonstrated by McAlonan *et al.* (2008). They recorded simultaneously from TRN and LGN in macaque monkey performing a spatial attention task. Attending to a saccade target location had the effect of decreasing TRN firing rate and increasing LGN firing rates (both magnocellular and parvocellular) during the early period of the response. A second enhancement in LGN firing occurred during the late period, with no modulation of TRN firing during that period. Possibly the early attentional modulation reflects LGN input from subcortical structures (Wilson *et al.*, 1995), such as superior colliculus, while the late modulation may be due to cortical feedback (Briggs and Usrey, 2007b; Sillito et al., 2006). A second single-cell study showing attentional enhancement in LGN responses is that of Ortuño *et al.* (2014), focusing on burst responses and recording only in the parvocellular layers. Both McAlonan *et al.* (2008) and Ortuño *et al.* (2014) used endogenous (top down) attentional tasks rather than exogenous (bottom up) attentional tasks (See Posner (1980) for a discussion of endogenous vs. exogenous attention).

Strong LGN modulation has also been shown neurophysiologically in an exogenous attentional task involving figure-ground discrimination, using a stimulus

figure defined by motion contrast (Jones *et al.*, 2015; Self and Roelfsema, 2015) (Figure 4). It is not clear if this modulation reflected the perceptual figure-ground segregation aspect of the task (determination of the location of the figure relative to motion-direction borders), which would involve integrating contextual motion information from far beyond the LGN receptive field, likely based on cortical feedback, or the spatial attention aspect of the task (saccade to the location of the detected figure), also based on cortical feedback. In either case, it indicates more complex LGN processing than simple linear filtering. Another task compared LGN responses during a task requiring detection of low-contrast stimuli (an exogenous attention task) with responses during a passive fixation task (Jiang *et al.*, 2015). This study failed to show enhanced LGN responses at the attended location (in fact, showed decreased responses), possibly due to a confound involving different eye movements during the two tasks (saccade to detected target in the attentional task, versus no eye movement during the passive fixation task).

Attention is closely associated with mechanisms for the control of eye movements, as overt attention generally involves a saccade to the target of attention (Moore *et al.*, 2003). There is a high degree of overlap between neural structures for saccade control and attention (Nobre *et al.*, 2000). Eye movements modulate LGN activity, as was outlined earlier in Section 2.4.6. Inhibition of LGN activity begins as early as 300 ms before the start of a saccade (Lee and Malpeli, 1998). Such early inhibition may be related to motor planning (Casagrande *et al.*, 2005; Royal *et al.*, 2006), or, from a different perspective, may be viewed as attention related. The early saccade-related modulation in LGN could therefore be another indication of LGN participation in cognitive processing.

7.2 Neural correlates of binocular rivalry in the LGN

Binocular rivalry occurs when incompatible images are presented to each eye, for example, a horizontal grating to the left eye and a vertical grating to the right eye (Blake and Logothetis, 2001). Such stimulus conditions cause the visual system to go into oscillation, such that the image to only one eye is visible at any given time. The entire visual field need not oscillate in unison, but rather may form an undulating patchwork of image fragments from the two eyes. The period of the oscillations is irregular with a typical mean of around two seconds. The laminar organization of LGN, each layer driven by one eye, has long led to speculations that inhibition between LGN layers might play a role in producing rivalry.

Interest in rivalry with respect to cognition often arises in connection with visual awareness (the visual aspect of consciousness) (e.g., Logothetis, 1998), because although the stimuli to both eyes are stimulating the retinae, only one becomes manifest in consciousness. However, Blake *et al.* (2014) provides a skeptical discussion of the utility of binocular rivalry as a tool for discovering neural correlates of consciousness, with which we agree.

Correlates of binocular rivalry have been observed in a number of visual cortical areas. In monkey single-cell recordings, the spiking responses of most inferotemporal cells correlate with the rivalrous percept, while in V1, V2, V4, and MT rivalrous spiking activity occurs in a smaller fraction of cells (Leopold and Logothetis, 1996; Logothetis and Schall, 1989; Sheinberg and Logothetis, 1997). Correlates of rivalry in cortex have also been shown using fMRI (Lee and Blake, 2002; Lee *et al.*, 2005; Lumer *et al.*, 1998; Polonsky *et al.*, 2000; Tong and Engel, 2001; Tong *et al.*, 1998), MEG (Srinivasan and

Petrovic, 2006; Srinivasan *et al.*, 1999; Tononi *et al.*, 1998), and EEG (Srinivasan, 2004; Sutoyo and Srinivasan, 2009).

Moving to LGN, two monkey neurophysiological studies have failed to find evidence for rivalry in that structure (Lehky and Maunsell, 1996; Wilke *et al.*, 2009). However, correlates of rivalry have been found in human fMRI studies (Haynes *et al.*, 2005; Wunderlich *et al.*, 2005).

The question naturally arises as to why there is a discrepancy between neurophysiology and fMRI results in the LGN rivalry data. A promising explanation is that the fMRI BOLD signal is primarily sensitive to local field potentials (LFPs) arising within the dendritic inputs to neurons, and not spiking activity. The preferential sensitivity of the BOLD signal to LFPs has been observed both in visual cortex (Logothetis *et al.*, 2001; Magri *et al.*, 2012; Martuzzi *et al.*, 2009) and LGN (Yen *et al.*, 2011). As cortical feedback provides most of the synapses in LGN, then, as was suggested by Chang *et al.* (2016), a technique such as fMRI that is most sensitive to synaptic activity in dendrites would be biased towards reporting response characteristics of the cortical feedback rather than those of LGN thalamocortical cells. Perhaps feedback of rivalrous activity from striate cortex is sufficient to modulate LFPs in LGN but is not potent enough to affect spiking activity.

Correlations between the BOLD signal and spiking activity in LGN have been examined by (Li and Freeman, 2007, 2012), but these reports neglect to consider LFPs which are likely to be the critical factor. Boynton (2011) provides a general discussion of the relationship between fMRI BOLD signals and neurophysiological responses, relevant to not only binocular rivalry but attentional effects as well.

7.3 Consciousness

A number of influential theories of consciousness revolve around the idea that while cortex is the key substrate for consciousness, critical thalamocortical interactions are required to support cortex in that role. There are also theories reversing that, in which consciousness occurs primarily in the thalamus or some other subcortical structure and cortex plays a supportive role. Discussions about the role of the thalamus in consciousness often don't explicitly consider LGN separately from the rest of the thalamus, but in many cases, the LGN can be considered as a special case of whatever theory is being proposed. Under any of these theories concerning the function of the thalamus in consciousness, the LGN would play a far more complex role than simply transmitting a lightly processed copy of retinal inputs to striate cortex.

A currently popular idea is that consciousness involves cortical assemblies of neurons having synchronized firing, with the synchronization often taking the form of oscillatory behavior (Singer, 1998, 2001). In thalamocortical theories of consciousness, notably those of Llinás *et al.* (1998) as well as (Edelman, 2003; Edelman *et al.*, 2011), the thalamus acts as a nexus which coordinates activity across broad areas of cortex, possibly mediated by synchronized oscillatory behavior, and in which the attentional aspects of thalamus and TRN help select regions of cortex destined for binding. This general perspective on thalamocortical interactions found early expression by Koch and Ullman (1985) and has been further discussed by Min (2010) and modeled by Newman *et al.* (1997).

Anesthetics obviously cause loss of consciousness. When considering a large range of anesthetics, their strongest common effect is depression of thalamic activity. The

effect of anesthetics on the thalamus has led to a "thalamic switch" theory for deactivating consciousness, in which consciousness in cortex is disrupted by scrambled activity in thalamocortical loops, as presented by (Alkire *et al.*, 2008; Alkire and Miller, 2005; Mashour and Alkire, 2013).

There is also an ongoing minority view that focuses on subcortical structures rather than cortex as dominant in higher cognitive functions. An early expression of this viewpoint was the centrencephalic theory of Penfield, developed in the 1950s (Thompson, 1993). The centrencephalic theory emphasized the role of mid to low level brainstem structures in cognition, based on, among other things, Penfield's observation that excision of large areas of cortex during human surgery had little effect on consciousness. A more recent expression of this perspective comes from Merker (2007), who places consciousness in the superior colliculus. More directly related to the concerns here is the thalamic dynamic core theory of consciousness of Ward (2011). His theory posits that consciousness occurs solely in the thalamus, which acts as an active blackboard to display the results of cortical computations. Presumably under this theory, LGN would then be the prime locus of visual awareness. Alkire and colleagues (Alkire *et al.*, 2008; Mashour and Alkire, 2013) accept this general class of thalamic readout theory of consciousness (thalamocentric viewpoint) as a viable alternative to their thalamic switch theory (corticocentric viewpoint).

In the future, as fMRI technology develops, it is likely to be of central importance in characterizing cognitive functions of LGN. In human cognitive studies, fMRI would of course be indispensable. In animal studies, use of fMRI would serve to situate LGN responses within the context of wide ranging cognitive networks across the brain, and

helps select promising locations for electrode placement when examining LGN interactions with other (non-striate) brain structures during cognitive tasks. Resolution in fMRI has developed to the point where it can resolve activity in individual LGN layers (Denison *et al.*, 2014; Zhang *et al.*, 2015), as well as activity in the thin shell of cells that forms the thalamic reticular nucleus (Viviano and Schneider, 2015). However, reliance of fMRI on the sluggish BOLD signal will limit its usefulness in studies involving dynamic aspects of cognitive processing. Furthermore, the selective sensitivity of the fMRI signal to local field potentials within dendritic trees rather than thalamocortical cell action potentials (Logothetis *et al.*, 2001; Yen *et al.*, 2011) also places limits on its usefulness for many questions.

8 Modeling

The LGN has been extensively modeled at a variety of levels, from the biophysical properties of its neurons to feedback network models dealing with issues in high-level vision.

8.1 Biophysical models of individual neurons

At the most basic level, biophysical models of the intrinsic properties of individual LGN neurons have examined contributions of a variety of ion channel types to neural activity using Hodgkin-Huxley type approaches. An influential general model in this genre is McCormick and Huguenard (1992), sometimes used as the starting point for more specialized models. The role of low-threshold calcium currents in burst generation has been of particular interest, not only in thalamocortical cells but also interneurons and TRN neurons (Destexhe *et al.*, 1996; Destexhe *et al.*, 1998; Halnes *et al.*, 2011;

McMullen and Ly, 1988; Rhodes and Llinás, 2005; Wang *et al.*, 1991; Zomorrodi *et al.*, 2008). Intrinsic non-burst oscillations within single LGN neurons have been modeled for alpha frequencies (Vijayan and Kopell, 2012) and gamma frequencies (Rhodes and Llinás, 2005). Detailed biophysical modeling of the dynamic behavior of individual thalamic neurons, including calcium related oscillations, from the perspective of nonlinear dynamical systems (state space diagrams and stability analysis) has been carried out by (Rose and Hindmarsh, 1989a, b, c). This modeling builds upon earlier more qualitative dynamical models centered on tonic single-spike activity (Hindmarsh and Rose, 1984; Rose and Hindmarsh, 1985); see also Reinker *et al.* (2003) for elaborations on this approach. A central interest in these models is in defining the ionic conditions that lead to burst or tonic modes in thalamic neurons.

Integration of dendritic inputs in individual cells has also been the subject of biophysical modeling. Briska *et al.* (2003) modeled conduction of electrical signals between dendrites and soma in LGN thalamocortical cells with passive membrane properties. They found burst EPSPs were conducted especially well, indicating that the temporal characteristics of EPSP inputs can have a strong effect on the functional geometry of LGN dendritic trees. Rhodes and Llinás (2005) focused on the consequences of active, regenerative behavior in dendrites involving calcium currents. Transmission across the retinothalamic synapse was modeled by Carandini *et al.* (2007), predicting the spike train on a thalamocortical cell given the spike train of the retinal ganglion cell providing its major input. Temporally clustered EPSPs from the retina were found to be more effective in driving LGN responses than isolated EPSPs. Moving to the other end of the system, modeling of transmission across thalamocortical synapses showed that

temporally clustered EPSPs from the LGN (in the form of synchronized firing from multiple LGN cells) were more effective in driving a cell in V1 cortex than isolated EPSPs (Wang *et al.*, 2010a). The general point these low-level models demonstrate is that the temporal organization of spiking activity beyond mean firing rate, for example in the form of intrinsic bursting behavior or population synchrony, can affect the transmission of information through the LGN, an aspect of system behavior often omitted from models of high-level vision (Figure 5).

A notable low-level aspect of LGN organization is the formation of dendro-dendritic synapses between the LGN thalamocortical cells and interneurons, which, together with the retinal axonal input, form synaptic triads ensheathed in a glial glomerulus. Although LGN synaptic triads have been modeled by Koch (1985) and Bloomfield and Sherman (1989), the functional significance of this arrangement for high-level vision remains largely unknown. Beyond the specific synaptic triad arrangement in LGN, the possibility that thalamocortical cell dendrites and interneuron dendrites in general may be engaged in complex, nonlinear computations of specific visual algorithms is also largely unexplored territory. One example of the use of dendritic interactions in high-level LGN modeling is the attentional gating model of Taylor and Alavi (1993). Dendritic computation has been reviewed by (Kath, 2005; Mel, 1994; Segev and London, 2000).

Although these biophysical models have only considered a few basic properties of real neurons, they nevertheless could serve as building blocks within high-level network models and enrich those models by introducing a variety of computationally significant nonlinearities.

8.2 Spatial receptive field properties and feedforward response models

The most common description of the classical spatial receptive field in LGN is the Difference-of-Gaussians (DoG) model, based on the difference of two circular 2D Gaussians with a common center. The DoG model was originally proposed by Rodieck (1965) and Enroth-Cugell and Robson (1966) as a description of receptive fields in cat retinal ganglion cells. As LGN spatial receptive fields closely resemble those of retinal ganglion cells (Ruksenas *et al.*, 2000), the DoG model was transferred to LGN (Irvin *et al.*, 1993; White *et al.*, 2001; Xu *et al.*, 2002a) and has found widespread use in descriptions of LGN responses. Direct physiological support for the DoG model of LGN receptive fields comes from intracellular recording in cat by Lindström and Wróbel (2011), who found bell-shaped excitatory and inhibitory (both feedforward and feedback) fields that overlapped and were aligned with each other. There are biologically inspired alternatives to the DoG model for describing LGN receptive fields, involving close consideration of particular experimental data. An example of this is the Difference Of Offset Gaussians (DOOG) model (Young, 1987). Another DoG alternative comes from Einevoll and Heggelund (2000), who developed a receptive field model based on LGN microcircuitry modifying the retinal input.

Besides descriptive models of LGN receptive fields outlined above, there are also normative or prescriptive models. Normative models are based on application of various engineering optimality principles to derive the shape a receptive field ideally ought to have in order to perform a particular signal processing or computational function. Approaching the issue from a signal processing engineering perspective, Cho and Choi (2014) suggest describing early visual receptive fields in terms of an orthogonal basis set

of wavelets has advantages over DoGs in terms of efficiency of coding. An alternative to the DoG that comes with a long history in computer vision oriented modeling is the Laplacian of a Gaussian (for example, Marr and Hildreth, 1980). The Laplacian is a mathematical operator that takes the second derivative of a circular 2D Gaussian to produce a center/surround receptive field shape closely resembling a DoG. In recent work, a theory of receptive fields based on Gaussian derivatives has been developed by (Lindeberg, 2013, 2016). Lindeberg's theory starts by postulating mathematical requirements for an ideal front end of a visual system, developed from the perspective of scale space theory (Koenderink and van Doorn, 1990). Receptive fields fulfilling those requirements are then derived, ending up with Laplacian receptive fields resembling those observed experimentally in LGN. Mahmoodi (2016) presents a conceptually similar, though more biologically motivated model, mathematically implemented as an electronic circuit representing a grid of neurons.

Other models have focused on more specialized aspects of LGN spatial organization. Ferreiroa and Sánchez (2013) were interested in the effect different center-surround organizations had on processing borders, as well as effects of spike adaptation and bursting behavior. Wielaard and Sajda (2007) used a feedforward model to generate orientation bias in LGN cells as well as other spatial effects, while Kosmidis and Vibert (2002) found that the proper configuration of feedforward inhibition could increase spatial resolution of the representation. Although modeling of LGN color processing is a largely neglected topic, Valberg and Seim (2013) have modeled the organization of color opponent receptive fields and how they respond to different spatial patterns. Notably absent are feedforward models considering the binocular inhibitory interactions within

LGN that were described in Section 2.4.5.

A number of models have found that LGN responses can be accurately predicted using feedforward processing only. LGN spike trains could be predicted from simultaneously recorded retinal inputs, using either the estimated LGN temporal impulse response to retinal spikes (Keat *et al.*, 2001) or applying a leaky integrate-and-fire model to the retinal input potentials (Casti *et al.*, 2008; Lesica and Stanley, 2004). These models were successful without including consideration of feedback from cortex or TRN, or of a diverse array of ion channels. Modeling similar data, Babadi *et al.* (2010), found that for spot stimuli the non-retinal inputs (cortical, TRN, brainstem, etc.) accounted for a negligible amount of the variance in the data. Wang *et al.* (2007) constructed a feedforward model that predicted LGN responses to naturalistic videos by estimating spatiotemporal receptive fields based on retinal input potentials. Mante *et al.* (2008), building upon earlier modeling by Carandini *et al.* (2007), also predicted LGN responses to naturalistic images, using estimates of LGN spatiotemporal receptive fields plus nonlinear fast adaptation to luminance and contrast, again with no feedback mechanisms of any sort. For our interests in identifying the function of the LGN, a limitation of this last model was that it lumped retinal and LGN processing together into a single peripheral processing stage, as the model used the stimulus light distribution as input rather than retinal responses as input.

Going in the opposite direction, instead of predicting LGN responses from inputs it is possible to regenerate the stimulus from the recorded responses of a population of cells. An early example of this is Wróbel *et al.* (1983), using moving parallel stripes as stimuli. Stanley *et al.* (1999) applied an inverse transfer function to LGN population

responses to reconstruct videos of natural scenes, with impressive results for such complex stimuli.

If simple feedforward models can give good account of LGN response data under a variety of stimulus conditions, including naturalistic videos (Lesica and Stanley, 2004; Mante *et al.*, 2008; Wang *et al.*, 2007), this again raises questions about the significance of cortical feedback. However, even though the models that fitted data (Babadi *et al.*, 2010; Casti *et al.*, 2008; Keat *et al.*, 2001; Lesica and Stanley, 2004; Mante *et al.*, 2008; Stanley *et al.*, 1999) did not explicitly include cortical feedback, that doesn't mean that there weren't feedback effects embedded in the data that were being fitted. Those models could thus be viewed as feedforward approximations to a feedback system. Some LGN models have deliberately modified feedforward transfer characteristics to implicitly include feedback effects without having an explicit feedback loop in the model (Einevoll and Plesser, 2002, 2012; Nirody, 2014). Perhaps other models that describe themselves as purely feedforward are also in fact including implicit feedback modulations when they fit LGN data.

Another topic connected to spatial modeling of the LGN is contrast gain control (Section 2.4.4). Contrast gain control is generally modeled by divisive normalization due to the extraclassical suppressive field (Ayaz and Chance, 2009; Bonin *et al.*, 2005; Carandini and Heeger, 2012; Cope *et al.*, 2013, 2014; Mante *et al.*, 2008). The suppressive field in these models is described in functional terms without consideration of the biophysical mechanisms producing it.

Two general approaches to neural modeling are to either use spiking models or firing rate models. Spiking models, which generate action potentials for each neuron and

integrate those potentials across the network through biophysical synaptic mechanisms, are more realistic but also require greater computational resources than firing rate models. Firing rate models specify the activity of each neuron as an analogue value, a simpler procedure that allows firing rates to be used directly as variables in coupled differential equations. Heiberg *et al.* (2013), comparing the two modeling approaches, reported that firing rate models of LGN thalamocortical cells can produce acceptable approximations to more complex spiking models. However, it is likely that firing rate models would not do so well under conditions where spike timing at synapses becomes a significant consideration, as examined in the modeling of (Carandini *et al.*, 2007; Casti *et al.*, 2008) and data of (Blitz and Regehr, 2003; Rathbun *et al.*, 2010); see also spike timing review of Usrey (2002). Firing rate models would have problems dealing with spike timing and synchrony effects at synapses.

A general limitation of feedforward models fitting LGN data is that they were based on data from anesthetized animals, so that attentional or other cognitive modulations were never a factor. They may have therefore underestimated the contribution of cortical feedback. Preferably one would build comprehensive models of the LGN transfer function using data from awake animals performing some demanding visual task that went beyond simple fixation (e.g., an objection recognition task if investigating the role of LGN in higher vision).

Going to the next step beyond modeling response properties of LGN thalamocortical cells, feedforward spatial models have also been used to examine the role of LGN in generating or modulating receptive field structure in striate cortex. While general coverage of this topic is too broad to be included here, one area of intense interest

that can serve as an example of computational and modeling issues is the origin of orientation selectivity in striate cortex. That topic has been reviewed by (Ben-Yishai *et al.*, 1995; Ferster and Miller, 2000; Priebe and Ferster, 2012; Vidyasagar and Eysel, 2015; Vidyasagar *et al.*, 1996). The original feedforward model of Hubel and Wiesel (1962) still stands today as the standard for comparison. Under the Hubel and Wiesel (1962) model, cortical orientation selectivity is built up from circular LGN receptive fields by summing LGN inputs aligned in a row. The general question under debate is whether such a feedforward model by itself can account for the data or if recurrent interactions within cortex are necessary. The larger issue here is whether visual computation follows a predominantly hierarchical feedforward organization or if recurrent processing plays a critical role at each stage.

The standard Hubel-Wiesel model is sufficient to generate a cortical orientation tuning curve (e.g., Hesam Shariati and Freeman, 2012). However, besides the basic orientation tuning of striate simple cells, models of orientation selectivity need to account for other nonlinear facets of the cortical data, such as cross-orientation suppression or the contrast independence of tuning curve width (Ferster and Miller, 2000). Elaborating on the Hubel and Wiesel feedforward approach, (Priebe and Ferster, 2006, 2012) outlined a model in which cortical orientation selectivity is due purely to feedforward inputs from the LGN without any recurrent cortical processing. Critical to making this model work is the incorporation of numerous nonlinear biophysical features within LGN responses, such as spike threshold, synaptic depression (e.g., Cimenser and Miller, 2014), response saturation, and driving force nonlinearities on synaptic currents. So even for something as basic as generating orientation selectivity in cortex, it would not be possible to treat LGN

cells as linear filters as is conventionally done.

Orientation models that include intracortical interactions come in a variety of flavors. Some take the full Hubel-Wiesel geometrical alignment of LGN inputs to produce cortical orientation selectivity, with the intracortical processing limited to producing contrast nonlinearities (Kayser *et al.*, 2001; Krukowski and Miller, 2001; Troyer *et al.*, 1998). Other models postulate a weak feedforward orientation bias in the LGN input to cortex as a symmetry-breaking mechanism, with intracortical interactions providing a sharpening in tuning as well as the various nonlinear effects observed in the data. Such symmetry breaking can occur through the standard Hubel-Wiesel alignment of LGN receptive fields, except with a smaller number of receptive fields lined up (Ben-Yishai *et al.*, 1995; Somers *et al.*, 1995; Teich and Qian, 2006, 2010). Alternatively, the orientation symmetry breaking could occur through slight orientation biases that occur in the responses of individual LGN cells (Kuhlmann and Vidyasagar, 2011; Vidyasagar *et al.*, 2015; Viswanathan *et al.*, 2011, 2015). These orientation biases in LGN cells are generally ignored as not being functionally significant, though the modeling of Kuhlmann and Vidyasagar (2011) raises the possibility that is not necessarily true. Going to the extreme opposite of a feedforward model, it is also possible to generate orientation tuning using purely intracortical mechanisms, without any LGN contribution to the orientation-specific aspect of cortical responses (Adorján *et al.*, 1998).

We have seen in these orientation models that details of LGN responses that are frequently ignored during modeling, such as various nonlinearities or the orientation bias, can become important in defining responses at the next stage. Another example of this comes from Lin *et al.* (2012), who found that LGN spike train statistics affected

orientation tuning bandwidth in their model. Using a more realistic leaky integrate-and-fire model for spike statistics produced greater orientation selectivity than using inhomogeneous Poisson spike statistics.

8.3 Models with feedback from TRN

These models include feedback from TRN but not cortex, so that there is no higher-level information being passed back into the system. The model of Musial *et al.* (1996) showed that feedback inhibition from TRN could account for spatial nonlinearities in LGN thalamocortical cells when stimulated by two small stimuli in different parts of the same thalamocortical cell receptive field. A detailed biophysical integrate-and-fire model of the LGN/TRN system was developed by (Huertas *et al.*, 2005; Huertas and Smith, 2006). They found fluctuating proportions of tonic and burst firing depending on stimulus conditions, which they attributed to a varying inhibitory influence of TRN on the LGN. Chelian and Srinivasa (2013) incorporated intrinsic inhibitory and TRN inputs to thalamocortical cells as well as separate M and P channels to model LGN responses to naturalistic videos. While this model qualitatively reproduced some characteristics of those responses, they did not have access to neurophysiological recordings of LGN responses to the same stimuli in order to make a quantitative comparison, unlike the feedforward models described in the previous section. Not made clear in any of these models is what additional capability does this TRN feedback inhibition provide to visual processing that cannot be provided by the feedforward inhibition of the intrageniculate inhibitory neurons.

8.4 Temporal models

Differences between the temporal properties of LGN neurons and their retinal inputs are stronger than differences in their spatial properties, as was outlined in Section 2.4. One theoretical interpretation of the enhanced biphasic temporal response found in LGN is that it may be performing temporal decorrelation of the visual signal for purposes of efficient coding (Dong and Atick, 1995). The general idea that Dong and Atick (1995) were proposing was that efficient coding in the periphery was a two-step process, in which the retina performed a spatial decorrelation based upon on- and off-center cells with an antagonistic center-surround organization, while the LGN performed a temporal decorrelation. They found in their modeling that when the rectifying property of neurons was taken into account (i.e., spike rates must be non-negative), two classes of cells with phase-shifted temporal responses were required to effect the temporal decorrelation. These resembled the lagged and non-lagged LGN cells observed experimentally (Saul, 2008a; Saul and Humphrey, 1990).

Understanding the mechanisms that differentiate lagged and non-lagged cells has been a concern for modelers. Whether an LGN cell has lagged or non-lagged properties may depend on the balance of feedforward excitation and inhibition received by the cell, with increased inhibition leads to lagged responses (Hillenbrand and van Hemmen, 2001). In this model, T-type calcium channels are required to produce lagged responses, an example where these channels might play a role in tonic responses of LGN neurons and not just bursting behavior. Dealing with this issue at a more abstract level, the spatial receptive field models of (Lindeberg, 2013, 2016; Mahmoodi, 2016) described in Section 8.2 are in fact full spatiotemporal models. They therefore provide predictions of temporal

receptive fields as well as spatial ones, including a consideration of lagged and non-lagged cells. In particular, the model of Lindeberg (2016) predicts triphasic impulse responses for lagged cells. Although lagged cells generally been observed to have biphasic temporal impulse responses (Saul, 2008a), there is a report in cats of triphasic impulse responses in lagged cells under chromatic stimulus conditions (as opposed to the usual achromatic conditions) (Saul, 2008b). Why chromaticity would affect the shape of lagged impulse responses remains to be investigated, though the answer might involve the relative levels of activity in X and Y cells and interactions between them.

Both Yousif and Denham (2007) and Zabbah *et al.* (2014) used cortical feedback to model temporal decorrelation, requiring that the feedback be organized in the phase reversed push-pull manner observed in the data of Wang *et al.* (2006). The temporal decorrelation took the form of an enhanced second phase of the temporal impulse response in LGN, or in the frequency domain a whitening of the temporal power spectrum as has been observed experimentally by Dan *et al.* (1996) in LGN. However, as shown by modeling of Norheim *et al.* (2012), temporal decorrelation in LGN doesn't necessarily require cortical feedback inhibition, as it could also be done by feedforward inhibition via LGN interneurons. Resolving the relative contributions of feedback and feedforward inhibition in sculpting the LGN response remains an issue for future experimental work.

The temporal frequency response of thalamocortical cells was modeled by Mukherjee and Kaplan (1995). The temporal frequency response is the Fourier transform of the temporal impulse response, if the system is linear. Mukherjee and Kaplan (1995) found that the temporal frequency response depended on the resting membrane potential

of the model neurons, changing from low-pass to band-pass as the neuron became increasingly hyperpolarized. That is equivalent to hyperpolarization causing the temporal impulse response to have a larger secondary rebound phase. This modeling provides a mechanism by which the temporal filtering properties of LGN can be placed under dynamic control, either using cortical feedback or intrinsic circuitry within the LGN/TRN complex. Temporal properties may thus vary across the LGN in response to local stimulus or attentional conditions.

Another aspect of LGN temporal responses that has attracted the attention of modelers is the role of spike bursts in coding stimulus features. As outlined in Section 6, there is experimental evidence that aspects of spike bursts reliably correlate with stimulus features in awake animals. Modeling has focused on the number of spikes in a burst (Elijah *et al.*, 2015; Kepecs and Lisman, 2003; Samengo and Montemurro, 2010) or the interspike interval (Oswald *et al.*, 2004) as key parameters encoding stimulus features. For a single cell, such parameters would likely allow only a coarse characterization of a stimulus, but a population could refine stimulus categorization. Critical to the generation of bursts is that the stimulus has a particular temporal organization as well as spatial organization, dependent on the dynamics of low-threshold calcium channels. Reflecting that aspect of burst generation, Elijah *et al.* (2015) noted that in their model bursting neurons responded to complex spatiotemporal patterns that were difficult to describe in simple terms.

While it is commonplace to treat the temporal and spatial aspects of a neuron's responses separately when considering its function, Eyherabide and Samengo (2010) argue that there are computational advantages to considering the response within a

common spatiotemporal framework, as space and time may not be independent channels of information defining a stimulus. Burst coding may allow the representation of more complex interactions between the spatial and temporal aspects of stimuli than can be coded by simple spike trains. The potential ability of LGN neurons to use nonlinear bursting mechanisms to encode complex spatiotemporal features further expands the complexity of processing the LGN may be engaged in.

Approaching temporal properties of LGN from a more computational perspective, Jehee and Ballard (2009) and Ballard and Jehee (2012) used a learning algorithm to train a two-layer network (LGN and V1) to implement a predictive coding model of early visual processing, using natural image stimuli. Cortical predictions of LGN responses were fed back to LGN, while LGN sent the prediction errors forward to cortex, with network weights adapting to minimize prediction error as the signal reverberating around the thalamocortical loop. The network learned a strong biphasic temporal impulse response. These models therefore also interpret cortical feedback to LGN as a mechanism for producing efficient temporal coding.

Under a predictive coding model, the LGN can be considered as a comparator that is capable of sending to cortex the difference between sensory input and an anticipatory prediction of sensory input, reducing redundancy in the feedforward signal. Such an approach is very similar to what is used in the comparator part of a Kalman filter (Maybeck, 1979; Rao and Ballard, 1997). Thus, from a predictive coding perspective the LGN can be modeled as a Kalman filter that predicts next sequences of input stimuli. A neural mechanism can be proposed for such a prediction procedure using simple time-order sensitive Hebbian synapses (Montague *et al.*, 1996), or by using neural networks

that are either trained to predict a dynamic input (Fielding and Ruck, 1995; Schmidhuber, 1992; Softky, 1996) or reconstruct a static one (Hinton *et al.*, 1995; Jehee and Ballard, 2009; Mumford, 1994).

An entirely different and interesting view of cortical feedback and temporal processing in LGN comes from (Hillenbrand and van Hemmen, 2000, 2001, 2002), who emphasize *coherence representation* rather than *redundancy reduction*. From their perspective the ultimate task of sensory processing is extraction of features relevant for achieving behavioral goals, not efficient coding that maximizes transmission of information in a channel constrained to use a minimal amount of resources (Hillenbrand and van Hemmen, 2002). Behaviorally relevant parts of a stimulus, such as individual objects, are usually marked by a temporally coherent structure. On this view, visual responses –at some level in the hierarchy and some time scale— should be modulated to enhance object-related correlations (so that they are segmented against a non-coherent background), rather than suppress correlations for purposes of efficient coding. Coming from this theoretical viewpoint, (Hillenbrand and van Hemmen, 2000, 2001) developed a model in which cortical feedback adjusts the temporal properties of lagged and non-lagged LGN cells (with lagged cells acting as delay lines for Reichardt motion detectors (Reichardt 1961)), such as to modify the velocity tuning of cortical cells. Tuning preferences are adjusted to cluster around the velocity of a coherently moving object. Thus, the representation of the object is enhanced relative to background.

The idea of using enhanced correlation rather than decorrelation to represent stimulus attributes is also the basis of some models of retinal processing (Kenyon *et al.*, 2004; Schnitzer and Meister, 2003; Stephens *et al.*, 2006), consistent with observation of

enhanced correlations in retinal responses beyond that imposed by correlations in the stimulus images (Meister *et al.*, 1995). Retinal correlations are inherited by the LGN (Neuenschwander and Singer, 1996), so the retinal coherence representation models are relevant to LGN processing.

8.5 Cortical feedback models

Models of striate feedback to LGN follow many paths, as there is no consensus about what the feedback might be doing.

Retinal synapses onto LGN thalamocortical cells are on proximal dendrites, while cortical feedback, synapses are on distal dendrites. Despite that, biophysical modeling indicates that cortical feedback from layer 6 can have potent effects on thalamocortical cell firing. Modeling shows that, under some input signal conditions, the dendritic location of synaptic inputs has little effect on the size of somatic responses in thalamocortical cells (Briska *et al.*, 2003; Destexhe, 2000; Lajeunesse *et al.*, 2013; Perreault and Raastad, 2006), so that cortical feedback can mimic at the biophysical level the effects of retinal input. The results from this modeling is supported by the data of Connelly *et al.* (2016), who found that cortical feedback can occasionally drive thalamocortical cells and are not always modulatory. As pointed out by Destexhe (2000), the ability of feedback to mimic retinal input in some manner would appear to be a prerequisite for predictive coding models, based on the premise that feedback is communicating down to LGN specific predictions about the stimulus, and not just providing non-specific gain modulations.

The possibility that corticothalamic feedback on distal dendrites might have a driving rather than modulatory impact on somatic activity under some input conditions

does not obviate a modulatory role for them under other, perhaps most, conditions, in particular a role in local dendritic computations that only indirectly affect the soma (London and Häusser, 2005). The relationship between distal feedback and somatic response at the biophysical level is an open area for both experimental and theoretical work.

In addition to direct excitatory feedback onto thalamocortical cells, there are indirect, disynaptic inhibitory feedback connections mediated by both the intrinsic interneurons and TRN neurons. Modeling indicates that the inhibitory cortical feedback mediated via TRN is functionally more significant than that through the intrinsic interneurons (Rogala *et al.*, 2013), a result supported by the data of (Jurgens *et al.*, 2012). The dual pathways for striate influence upon thalamocortical cells leads to the question of which dominates: direct excitatory feedback or the indirect inhibitory feedback. Modeling by Destexhe (2000) found that the relative magnitudes of excitatory and inhibitory feedback influences depend on the depolarization state of TRN dendrites. Depolarized TRN dendrites lead to a predominantly excitatory feedback effect due to inactivation of low threshold Ca^{+2} currents in the TRN dendrites. The modeling results of Destexhe (2000) are consistent with the experimental observations of Crandall *et al.* (2015), which showed that high cortical feedback firing rates, which would produce strongly depolarized TRN dendrites, leads to the predominance of excitatory effects upon thalamocortical cells, while low firing rates leads to inhibitory effects (also see the commentary by Alitto and Usrey (2015a)).

As mentioned in Section 3.2, the neurons sending feedback to LGN also send collaterals within striate cortex that have a predominantly inhibitory effect (through

interneurons) on other cortical layers (Bortone *et al.*, 2014; Olsen *et al.*, 2012). This aspect of the thalamocortical loop has not been incorporated into models.

Modulation of LGN thalamocortical cell firing rates, through either excitation or inhibition, is one way cortical feedback could exert its effects. Another possibility is for cortical feedback to change statistical characteristics of LGN responses at the population level without necessarily changing mean firing rate. Destexhe *et al.* (1999) showed that cortical feedback could, in principle, trigger and synchronize oscillations in LGN. Synchronized LGN activity leads to greater efficacy in transmitting information across thalamocortical synapses and activating cortical cells (Section 4). Modeling by Agarwal and Sarma (2012) found that the frequency of oscillations in cortical feedback also affects the efficacy of information transmission, with stronger feedback activity improving thalamocortical communication. Using cortical feedback to inject synaptic noise into the LGN may produce beneficial effects for visual processing. For example, noise introduced from the cortex may smooth out the transition between burst and tonic response modes, improving the linearity of responses over a broader range of LGN membrane potentials (Wolfart *et al.*, 2005). Feedback noise may also improve the reliability of spike transmission from LGN to cortex by reducing noise correlation across an LGN population (but not the response correlation of the population) (Béhuret *et al.*, 2015; Béhuret *et al.*, 2013), through what is essentially a stochastic resonance mechanism (McDonnell and Abbott, 2009). All these are possible feedback mechanisms by which attention may act on LGN.

A significant experimental observation related to feedback and synchronization is the finding by Sillito *et al.* (1994) that synchronized firing across LGN cells in response

to drifting gratings disappeared upon removal of cortical feedback. Modeling by (Kirkland and Gerstein, 1998; Kirkland *et al.*, 2000) has reproduced these data, in which correlated LGN activity required the conjunction of cortical feedback and the presence of T-type calcium channels in the LGN cells. Here is another example in which T-type calcium channels could play a role in tonic responses of LGN neurons and not just bursting behavior.

There has also been some interest in modeling the role of the thalamocortical loop in producing synchronous oscillations in cortex, particularly alpha, beta, and gamma oscillations. Oscillations can arise through intrinsic properties of individual neurons (Llinás, 2014), and they can also arise through a variety of network interactions. Network interactions can include cortical interlaminar loops, local horizontal loops within a cortical layer, loops between different cortical areas, and loops between cortex and thalamus (Lumer *et al.*, 1997). Llinás and colleagues have long advocated the idea that resonance in the thalamocortical loop serves to amplify certain frequencies among the variety of oscillations produced by different mechanisms (Llinás and Ribary, 1993; Llinás and Paré, 1991). More recent biophysical modeling of the thalamocortical loop supports that idea, indicating resonant frequencies, determined by axonal conduction delays between striate cortex and LGN, can occur at alpha, beta, and gamma frequencies (Henke *et al.*, 2014; Robinson, 2006; Robinson *et al.*, 2001a; Robinson *et al.*, 2001b; Rowe *et al.*, 2004). If thalamocortical resonance is the source of prominent oscillations seen throughout the brain, then that raises the importance of the thalamus with respect to whatever functional significance one ascribes to those oscillations (attention, feature binding, consciousness, etc.)

Another possible role for cortical feedback is indicated by the "shifter circuits" model of Anderson and Van Essen (1987). Under this model, feedback from cortex may cause the LGN to shift the spatial alignment of the retinal sheets and the cortical sheet to achieve binocular registration at the cortical level during stereopsis. To some extent this can be seen as a formalization of qualitative theorizing by McIlwain (1995) as well as Schmielau and Singer (1977) that the LGN provides fine adjustments of cortical disparity tuning curves. Anderson and Van Essen (1987) also suggest a role of shifter circuits in motion deblurring and shifts in spatial attention. Convincing physiological evidence supporting the existence of shifter circuits in LGN has not developed over the years.

Cortical feedback can also affect integration of stimulus information beyond the classical LGN receptive field. Extended center/surround interactions in LGN neurons, as measured by sensitivity to orientation discontinuity, are feedback dependent (Cudeiro and Sillito, 1996; Sillito *et al.*, 1993). Their data was modeled by Hayot and Tranchina (2001). In a similar vein, Einevoll and Plesser (2012) modeled the effects of the stimulus diameter on LGN responses, which depends on whether feedback is present or not according to the data of (Cudeiro and Sillito, 1996; Sillito and Jones, 2002). The enhancement of the suppressive surround in LGN receptive fields by cortical feedback can also lead to dynamical shifts in the spatial frequency tuning of LGN cells. According to the modeling of Nirody (2014), late LGN responses are tuned to higher spatial frequencies than early responses, in accord with experimental observations by Allen and Freeman (2006) of a coarse-to-fine sequence in LGN spatial responses.

Gollo *et al.* (2010) went beyond modeling cortical effects on thalamus and examine how corticothalamic feedback affects subsequent activity in the cortex itself as

the signal reverberates around the thalamocortical loop. Their modeling indicates cortical feedback allows the thalamus to act as a central hub that can switch on or off synchrony between distant cortical areas, through a process of dynamic relaying (Fischer *et al.*, 2006). The LGN acting as a central hub could perhaps synchronize different cortical visual areas with each other, and would presumably involve the various extrastriate projections from LGN described earlier. Such a central hub role for thalamus had previously been suggested by Brody (1992) and Miller (1996), and is supported by the fMRI data of Hwang *et al.* (2016).

There is an important class of models that focus on the idea that cortical feedback is providing higher-level expectations about the stimulus to LGN. Seminal here is the model of Harth *et al.* (1987), with (Mumford, 1991, 1994) proceeding along similar lines. The general idea is that bottom-up stimulus inputs that match top-down expectations are selectively enhanced and passed upward. Mutual interactions between top-down and bottom-up signals can be an iterative process in which expectations are refined until the system relaxes into a state in which the top level has created a sufficiently accurate generative model of inputs from the world. We shall call this class of models "expectation coding" models.

This view of cortical feedback is opposite to ideas about cortical processing centering on efficient coding, such as predictive feedback models (Bastos *et al.*, 2012; Huang and Rao, 2011), in which aspects of the stimuli matching expectations are suppressed rather than enhanced. It is compatible, however, with the coherence representation models described in Section 8.4, as well as biased competition models of attention (Desimone, 1998). Under expectation coding models, LGN responses,

integrating bottom-up and top-down activations in a self-consistent manner, will reflect a richer, more complex representation of the world than under feedforward models or under feedback models centering on spatial attentional gating.

Notable within the broad genre of expectation coding is a family of related models based on Adaptive Resonance Theory (ART) (Grossberg, 2013). The basic module for an ART model includes two layers with feedforward and feedback connections between them. The feedforward population activity will activate each upper-level unit to a varying degree, and in return, the feedback from each upper-level signals the expectation of that unit to the lower level. When the bottom-up inputs match top-down expectations of an upper-level unit, there is "resonance" in the system. These models can be extended to include multiple layers and lateral interactions (Grossberg, 1999; Grossberg, 2007).

ART models in their various manifestations generally include an input layer corresponding to LGN that receives expectations from V1 as feedback. These models have been applied to a wide variety of visual phenomena, particularly psychophysical phenomena, including binocular effects (Cao and Grossberg, 2005, 2012; Grossberg and Howe, 2003; Grossberg *et al.*, 2015; Grunewald and Grossberg, 1998), Gestalt perceptual grouping (Ross *et al.*, 2000), illusionary contours (Gove *et al.*, 1995), surface perception and transparency (Grossberg and Yazdanbakhsh, 2005), texture processing (Bhatt *et al.*, 2007; Grossberg *et al.*, 2007), and object recognition (Cao *et al.*, 2011; Rajaei *et al.*, 2012).

8.6 Future directions

Many current models of high-level vision, including object recognition models, do not include a separate stage for LGN processing, perhaps viewing LGN as a simple linear filter that wouldn't add interesting capabilities. We have seen throughout this review that LGN is substantially more complex than that, including a number of nonlinearities. Perhaps the most interesting nonlinearity involves feedback from cortex.

Feedback synapses greatly exceed feedforward synapses. Purely feedforward models, in which the majority of synapses serve no purpose, clearly cannot be the entire story. We anticipate that an area with strong potential for advancing the field is further development of feedback models of high-level vision that also include consideration of LGN processing (Figure 5b). Interesting work in this direction include the predictive coding models (Bastos *et al.*, 2012; Huang and Rao, 2011) and the expectation coding models (e.g., Harth *et al.*, 1987), with the ART models (Grossberg, 2007) being well developed examples in the latter category.

A currently popular approach to modeling object recognition involves feedforward convolution neural networks using a multistage Hubel-Wiesel architecture (Figure 5a). Such models consist of alternating layers of units, one layer acting as convolutional feature detectors similar to simple cells in striate cortex, and the next layer implementing a pooling operation to create invariance, similar to complex cells. The first to implement this architecture was the Neocognitron model (Fukushima, 1980, 1988), followed by the ConvNet model (LeCun and Bengio, 1995; LeCun *et al.*, 2010), the HMAX model (Ghodrati *et al.*, 2012; Rajaei *et al.*, 2012; Riesenhuber and Poggio, 1999; Serre *et al.*, 2007a; Serre *et al.*, 2007b), and deep convNet models (Ciresan *et al.*, 2012; Khaligh-Razavi and Kriegeskorte, 2014; Kheradpisheh *et al.*, 2016a, b; Krizhevsky *et al.*,

2012; Yamins *et al.*, 2014). A recognized limitation of this class of model is the lack of feedback (Serre *et al.*, 2007b).

Recently the multistage Hubel-Wiesel architecture has been extended to include feedback effects using probabilistic graphical models. Graphical models consist of nodes representing the values of random variables, with the nodes connected by directed or undirected edges (lines) with weights to form a graph expressing the dependencies between variables (Bengio *et al.*, 2013; Bishop, 2012; Koller and Friedman, 2009). Adding feedback is clearly an arrangement that fits better with the organization of the central nervous system (Figure 5b). One manifestation of graphical models can be deep learning networks (deep in the sense of many layers) with feedback, which has attracted considerable interest (LeCun *et al.*, 2015). Models of high-level vision that use graphical models include (Dura-Bernal *et al.*, 2012; George and Hawkins, 2009; Lee *et al.*, 2011). Adding semantic content to visual signals is an area of expanding interest involving feedback modeling, as emphasized by Lehky and Tanaka (2016) (for example, see Karpathy and Fei-Fei (2015)).

However, all those models start at striate cortex as the input layer and do not include consideration of LGN (Figure 5a). We suggest that those feedback models could expand and improve their object recognition capabilities by including an LGN layer. In particular, including feedback brings a temporal factor into the network models, and the LGN strongly distinguishes itself from retina in its temporal properties (enhanced temporal impulse response, lagged and non-lagged cells, strong disynaptic interactions with motion cortex MT via V1). Possibly LGN may contribute to temporal aspects of object processing, such as identification of moving objects. We see an example of

inclusion of LGN in a higher level model in Zabbah *et al.* (2014), in which adding an LGN layer with cortical feedback improved the correspondence between object classification performance of the model and human classification performance. An unexplored area is the contribution LGN might make to depth perception related to object processing, an interesting possibility given the precise alignment of left and right monocular layers in primate and carnivore LGN.

A general question is what level of biophysical detail is necessary to capture the essential features of LGN network behavior relevant for high-level vision. Dendrite geometry, including the glomerular dendritic structure in LGN, as well as ion channel properties such as those of T type calcium channels, are a rich source on nonlinear behavior that are likely to be computationally significant. It may be that networks that are far more powerful can be constructed if, instead of treating individual neurons as fundamental atoms within a network, we move down a level and consider neurons as complex entities with interacting parts.

9 Conclusions

What difference would it make if retinal outputs were to go directly to primary visual cortex without going through the LGN? What are the functions of this subcortical visual area? The reason why researchers have ignored the role of the LGN in high-level visual information processing may lie with the conventional view that these high-level processes are dominated by cortex and the role of thalamus is negligible. However, recent advances during the past decade in fMRI, anatomical, electrophysiological, and computational studies have provided precise information about response properties and functional characteristics of the LGN. These advances have dramatically changed our

view about LGN from a simple linear filter –for transferring retinal visual information – to a vital area for early visual processing. Accumulating evidence during recent years suggests that the LGN has a fundamental role in regulating information transmission to the visual cortex in a cognitively and computationally important manner. It is likely such evidence will grow as the early visual system is probed not only with naturalistic stimuli but also with cognitively demanding behavioral tasks. We believe that models of high-level vision will not be complete without a fuller consideration of computations occurring in LGN.

Acknowledgements

We thank Ian Andolina, Ann Butler, and Andrzej Wróbel for comments on the manuscript.

References

Abeles, M., Prut, Y., Bergman, H., Vaadia, E., 1994. Synchronization in neuronal transmission and its importance for information processing. *Prog. Brain Res.* 102**,** 395-404.

Acuna-Goycolea, C., Brenowitz, S.D., Regehr, W.G., 2008. Active dendritic conductances dynamically regulate GABA release from thalamic interneurons. *Neuron* 57**,** 420-431.

Adelson, E.H., Bergen, J.R., 1985. Spatiotemporal energy models for the perception of motion. *J. Opt. Soc. Am. A.* 2**,** 284-299.

Adey, W.R., Noda, H., 1973. Influence of eye movements on geniculo-striate excitability in the cat. *J. Physiol. (Lond).* 235**,** 805-821.

Adorján, P., Levitt, J.B., Lund, J.S., Obermayer, K., 1998. A model for the intracortical origin of orientation preference and tuning in macaque striate cortex. *Vis. Neurosci.* 16**,** 303-318.

Adrian, E.D., 1926. The impulses produced by sensory nerve endings: Part 1. *J. Physiol. (Lond).* 61**,** 49-72.

Agarwal, R., Sarma, S.V., 2012. Performance limitations of relay neurons. *PLoS Comput Biol* 8**,** e1002626.

Ahlsén, G., 1984. Brain stem neurones with differential projection to functional subregions of the dorsal lateral geniculate complex in the cat. *Neuroscience* 12**,** 817-838.

Ahlsén, G., Lindström, S., 1982. Mutual inhibition between perigeniculate neurones. *Brain Res.* 236**,** 482-486.

Ahlsén, G., Lindström, S., Lo, F.-S., 1985. Interaction between inhibitory pathways to principal cells in the lateral geniculate nucleus of the cat. *Exp. Brain Res.* 58**,** 134-143.

Ahlsén, G., Lindström, S., Lo, F.S., 1983. Excitation of perigeniculate neurones from X and Y principal cells in the lateral geniculate nucleus of the cat. *Acta Physiol. Scand.* 118**,** 445-448.

Ahlsén, G., Lindström, S., Lo, F.S., 1984. Inhibition from the brain stem of inhibitory interneurones of the cat's dorsal lateral geniculate nucleus. *J. Physiol. (Lond).* 347**,** 593-609.

Ahlsén, G., Lo, F.S., 1982. Projection of brain stem neurons to the perigeniculate nucleus and the lateral geniculate nucleus in the cat. *Brain Res.* 238**,** 433-438.

Ahmed, B., Anderson, J.C., Douglas, R.J., Martin, K.A., Nelson, J.C., 1994. Polyneuronal innervation of spiny stellate neurons in cat visual cortex. *J. Comp. Neurol.* 341**,** 39-49.

Ahmed, B., Hammond, P., 1991. Orientation bias of cat dorsal lateral geniculate cells: directional analysis of the major axis of the receptive field centre. *Exp. Brain Res.* 84**,** 676-679.

Ahrens, S., Jaramillo, S., Yu, K., Ghosh, S., Hwang, G.-R., Paik, R., Lai, C., He, M., Huang, Z.J., Li, B., 2015. ErbB4 regulation of a thalamic reticular nucleus circuit for sensory selection. *Nat. Neurosci.* 18**,** 104-111.

Ajina, S., Pestilli, F., Rokem, A., Kennard, C., Bridge, H., 2015. Human blindsight is mediated by an intact geniculo-extrastriate pathway. *eLife***,** e08935.

Albright, T.D., Stoner, G.R., 2002. Contextual influences on visual processing. *Annu. Rev. Neurosci.* 25**,** 339-379.

Alitto, H.J., Moore, B.D.t., Rathbun, D.L., Usrey, W.M., 2011. A comparison of visual responses in the lateral geniculate nucleus of alert and anaesthetized macaque monkeys. *J. Physiol. (Lond).* 589**,** 87-99.

Alitto, H.J., Usrey, W.M., 2004. Influence of contrast on orientation and temporal frequency tuning in ferret primary visual cortex. *J. Neurophysiol.* 91**,** 2797-2808.

Alitto, H.J., Usrey, W.M., 2005. Dynamic properties of thalamic neurons for vision. *Prog. Brain Res.* 149**,** 83-90.

Alitto, H.J., Usrey, W.M., 2008. Origin and dynamics of extraclassical suppression in the lateral geniculate nucleus of the macaque monkey. *Neuron* 57**,** 135-146.

Alitto, H.J., Usrey, W.M., 2015a. Dissecting the dynamics of corticothalamic feedback. *Neuron* 86**,** 605-607.

Alitto, H.J., Usrey, W.M., 2015b. Surround suppression and temporal processing of visual signals. *J. Neurophysiol.* 113**,** 2605-2617.

Alitto, H.J., Weyand, T.G., Usrey, W.M., 2005. Distinct properties of stimulus-evoked bursts in the lateral geniculate nucleus. *J. Neurosci.* 25**,** 514-523.

Alkire, M.T., Hudetz, A.G., Tononi, G., 2008. Consciousness and anesthesia. *Science* 322**,** 876-880.

Alkire, M.T., Miller, J., 2005. General anesthesia and the neural correlates of consciousness. *Prog. Brain Res.* 50**,** 229-244.

Allen, E.A., Freeman, R.D., 2006. Dynamic spatial processing originates in early visual pathways. *J. Neurosci.* 6**,** 11763-11774.

Alonso, J.-M., Usrey, W.M., Reid, R.C., 1996. Precisely correlated firing in cells of the lateral geniculate nucleus. *Nature* 383**,** 815-819.

Alonso, J.-M., Yeh, C.-I., Stoelzel, C.R., 2008. Visual stimuli modulate precise synchronous firing within the thalamus. *Thalamus Relat. Syst.* 4**,** 21-34.

Alonso, J.M., Usrey, W.M., Reid, R.C., 2001. Rules of connectivity between geniculate cells and simple cells in cat primary visual cortex. *J. Neurosci.* 21**,** 4002-4015.

Anderson, C.H., Van Essen, D.C., 1987. Shifter circuits: a computational strategy for dynamic aspects of visual processing. *Proc. Natl. Acad. Sci. U. S. A.* 84**,** 6297-6301.

Andolina, I.M., Jones, H.E., Sillito, A.M., 2013. Effects of cortical feedback on the spatial properties of relay cells in the lateral geniculate nucleus. *J. Neurophysiol.* 109**,** 889-899.

Andolina, I.M., Jones, H.E., Wang, W., Sillito, A.M., 2007. Corticothalamic feedback enhances stimulus response precision in the visual system. *Proc. Natl. Acad. Sci. U. S. A.* 104**,** 1685-1690.

Angelucci, A., Sainsbury, K., 2006. Contribution of feedforward thalamic afferents and corticogeniculate feedback to the spatial summation area of macaque V1 and LGN. *J. Comp. Neurol.* 498**,** 330-351.

Asanuma, C., 1994. GABAergic and pallidal terminals in the thalamic reticular nucleus of squirrel monkeys. *Exp. Brain Res.* 101**,** 439-451.

Augustinaite, S., Kuhn, B., Helm, P.J., Heggelund, P., 2014. NMDA spike/plateau potentials in dendrites of thalamocortical neurons. *J. Neurosci.* 34**,** 10892-10905.

Augustinaite, S., Yanagawa, Y., Heggelund, P., 2011. Cortical feedback regulation of input to visual cortex: role of intrageniculate interneurons. *J. Physiol. (Lond).* 589**,** 2963-2977.

Ayaz, A., Chance, F.S., 2009. Gain modulation of neuronal responses by subtractive and divisive mechanisms of inhibition. *J. Neurophysiol.* 101**,** 958-968.

Babadi, B., Casti, A., Xiao, Y., Kaplan, E., Paninski, L., 2010. A generalized linear model of the impact of direct and indirect inputs to the lateral geniculate nucleus. *J. Vision* 10**,** 22.

Baccus, S.A., Meister, M., 2002. Fast and slow contrast adaptation in retinal circuitry. *Neuron* 36**,** 909-919.

Bair, W., Koch, C., Newsome, W.T., Britten, K., 1994. Power spectrum analysis of bursting cells in area MT in the behaving monkey. *J. Neurosci.* 14**,** 2870-2892.

Bakkum, D.J., Radivojevic, M., Frey, U., Franke, F., Hierlemann, A., Takahashi, H., 2014. Parameters for burst detection. *Front. Comput. Neurosci.* 7**,** 193.

Bal, T., von Krosigk, M., McCormick, D.A., 1995. Role of the ferret perigeniculate nucleus in the generation of synchronized oscillations in vitro. *J. Physiol. (Lond).* 15**,** 665-685.

Baldauf, Z.B., 2010. Dual chemoarchitectonic lamination of the visual sector of the thalamic reticular nucleus. *Neuroscience* 165**,** 801-818.

Ballard, D.H., Jehee, J.F.M., 2012. Dynamic coding of signed quantities in cortical feedback circuits. *Front. Psychol.* 3**,** 254.

Bannister, N.J., Nelson, J.C., Jack, J.J.B., 2002. Excitatory inputs to spiny cells in layers 4 and 6 of cat striate cortex. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 357**,** 1793-1808.

Bartlett, J.R., Doty, R.W., Lee, B.B.S., Sakakura, H., 1976. Influence of saccadic eye movements on geniculostriate excitability in normal monkeys. *Exp. Brain Res.* 25**,** 487-509.

Bastos, A.M., Briggs, F., Alitto, H.J., Mangun, G.R., Usrey, W.M., 2014. Simultaneous recordings from the primary visual cortex and lateral geniculate nucleus reveal rhythmic interactions and a cortical source for gamma-band oscillations. *J. Neurosci.* 34**,** 7639-7644.

Bastos, A.M., Usrey, W.M., Adams, R.A., Mangun, G.R., Fries, P., Friston, K.J., 2012. Canonical microcircuits for predictive coding. *Neuron* 76**,** 695-711.

Bastos, A.M., Vezoli, J., Bosman, C.A., Schoffelen, J.M., Oostenveld, R., Dowdall, J.R., De Weerd, P., Kennedy, H., Fries, P., 2015. Visual areas exert feedforward and feedback influences through distinct frequency channels. *Neuron* 85**,** 390-401.

Bayram, A., Karahan, E., Bilgiç, B., Ademoglu, A., Demiralp, T., 2016. Achromatic temporal-frequency responses of human lateral geniculate nucleus and primary visual cortex. *Vision Res.* 127**,** 177-185.

Béhuret, S., Deleuze, C., Bal, T., 2015. Corticothalamic Synaptic Noise as a Mechanism for Selective Attention in Thalamic Neurons. *Front. Neural Circuits* 9**,** 80.

Béhuret, S., Deleuze, C., Gomez, L., Frégnac, Y., Bal, T., 2013. Cortically-controlled population stochastic facilitation as a plausible substrate for guiding sensory transfer across the thalamic gateway. *PLoS Comput Biol* 9**,** e1003401.

Bekisz, M., Wróbel, A., 1993. 20 Hz rhythm of activity in visual system of perceiving cat. *Acta Neurobiologiae Experimentalis* 53**,** 175-182.

Bekisz, M., Wróbel, A., 1999. Coupling of beta and gamma activity in corticothalamic system of cats attending to visual stimuli. *Neuroreport* 10**,** 3589-3594.

Ben-Yishai, R., Bar-Or, R.L., Sompolinsky, H., 1995. Theory of orientation tuning in visual cortex. *Proc. Natl. Acad. Sci. U. S. A.* 92**,** 3844-3848.

Bender, D.B., Youakim, M., 2001. Effect of attentive fixation in macaque thalamus and cortex. *J. Neurophysiol.* 85**,** 219-234.

Benevento, L.A., Yoshida, K., 1981. The afferent and efferent organization of the lateral geniculo-prestriate pathways in the macaque monkey. *J. Comp. Neurol.* 203**,** 455-474.

Bengio, Y., Courville, A., Vincent, P., 2013. Representation learning: A review and new perspectives. *IEEE Trans. Pattern Anal. Mach. Intell.* 35**,** 1798-1828.

Benshalom, G., White, E.L., 1986. Quantification of thalamocortical synapses with spiny stellate neurons in layer IV of mouse somatosensory cortex. *J. Comp. Neurol.* 253**,** 303-314.

Bessaïh, T., Leresche, N., Lambert, R.C., 2008. T current potentiation increases the occurrence and temporal fidelity of synaptically evoked burst firing in sensory thalamic neurons. *Proc. Natl. Acad. Sci. U. S. A.* 105**,** 11376-11381.

Bhatt, R., Carpenter, G.A., Grossberg, S., 2007. Texture segregation by visual cortex: perceptual grouping, attention, and learning. *Vision Res.* 47**,** 3173-3211.

Bickford, M.E., Ramcharan, E., Godwin, D.W., Erisir, A., Gnadt, J., Sherman, S.M., 2000. Neurotransmitters contained in the subcortical extraretinal inputs to the monkey lateral geniculate nucleus. *J. Comp. Neurol.* 424**,** 701-717.

Bickford, M.E., Wei, H., Eisenback, M.A., Chomsung, R.D., Slusarczyk, A.S., Dankowski, A.B., 2008. Synaptic organization of thalamocortical axon collaterals in the perigeniculate nucleus and dorsal lateral geniculate nucleus. 508**,** 264-285.

Bishop, C.M., 2012. Model-based machine learning. *Philos. Trans. R. Soc. Lond. A.* 371**,** 20120222.

Bishop, G.H., 1959. The relation between nerve fiber size and sensory modality: phylogenetic implications of the afferent innervation of cortex. *J. Nerv. Ment. Dis.* 128**,** 89-114.

Blake, R., Brascamp, J., Heeger, D.J., 2014. Can binocular rivalry reveal neural correlates of consciousness? *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 369**,** 20130211.

Blake, R., Logothetis, N., 2001. Visual competition. *Nature Rev. Neurosci.* 3**,** 13-21.

Blakemore, C., Vital-Durand, F., 1986. Organization and post-natal development of the monkey's lateral geniculate nucleus. *J. Physiol. (Lond).* 380**,** 453-491.

Blasdel, G.G., Fitzpatrick, D., 1984. Physiological organization of layer 4 in macaque striate cortex. *J. Neurosci.* 4**,** 880-895.

Blasdel, G.G., Lund, J.S., 1983. Termination of afferent axons in macaque striate cortex. *J. Neurosci.* 3**,** 1389-1413.

Blitz, D.M., Regehr, W.G., 2003. Retinogeniculate synaptic properties controlling spike number and timing in relay neurons. *J. Neurophysiol.* 90**,** 2438-2450.

Blitz, D.M., Regehr, W.G., 2005. Timing and specificity of feed-forward inhibition within the LGN. *Neuron* 45**,** 917-928.

Bloomfield, S.A., Sherman, S.M., 1988. Postsynaptic potentials recorded in neurons of the cat's lateral geniculate nucleus following electrical stimulation of the optic chiasm. *J. Neurophysiol.* 60**,** 1924-1945.

Bloomfield, S.A., Sherman, S.M., 1989. Dendritic current flow in relay cells and interneurons of the cat's lateral geniculate nucleus. *Proc. Natl. Acad. Sci. U. S. A.* 86**,** 3911-3914.

Bonin, V., Mante, V., Carandini, M., 2005. The suppressive field of neurons in lateral geniculate nucleus. *J. Neurosci.* 25**,** 10844-10856.

Bonin, V., Mante, V., Carandini, M., 2006. The statistical computation underlying contrast gain control. *J. Neurosci.* 26**,** 6346-6353.

Borst, A., Theunissen, F.E., 1999. Information theory and neural coding. *Nat. Neurosci.* 2**,** 947-957.

Bortone, D.S., Olsen, S.R., Scanziani, M., 2014. Translaminar inhibitory cells recruited by layer 6 corticothalamic neurons suppress visual cortex. *Neuron* 82**,** 474-485.

Bosman, C.A., Schoffelen, J.-M., Brunet, N., Oostenveld, R., Bastos, A.M., Womelsdorf, T., Rubehn, B., Stieglitz, T., De Weerd, P., Fries, P., 2012. Attentional stimulus selection through selective synchronization between monkey visual areas. *Neuron* 75**,** 875-888.

Boyapati, J., Henry, G., 1984. Corticofugal axons in the lateral geniculate nucleus of the cat. *Exp. Brain Res.* 53**,** 335-340.

Boynton, G.M., 2011. Spikes, BOLD, attention, and awareness: a comparison of electrophysiological and fMRI signals in V1. *J. Vision* 11**,** 12.

Bragg, E.M., Fairless, E.A., Liu, S., Briggs, F., 2017. Morphology of visual sector thalamic reticular neurons in the macaque monkey suggests retinotopically

specialized, parallel stream-mixed input to the lateral geniculate nucleus. *J. Comp. Neurol.* 525**,** 1273-1290.

Brette, R., 2012. Computing with neural synchrony. *PLoS Comput Biol* 8**,** e1002561.

Briggs, F., 2010. Organizing principles of cortical layer 6. *Front. Neural Circuits* 4**,** 3.

Briggs, F., Kiley, C.W., Callaway, E.M., Usrey, W.M., 2016. Morphological Substrates for Parallel Streams of Corticogeniculate Feedback Originating in Both V1 and V2 of the Macaque Monkey. *Neuron* 90**,** 388-399.

Briggs, F., Mangun, G.R., Usrey, W.M., 2013. Attention enhances synaptic efficacy and the signal-to-noise ratio in neural circuits. *Nature* 499**,** 476-480.

Briggs, F., Usrey, W.M., 2007a. Cortical activity influences geniculocortical spike efficacy in the macaque monkey. *Front. Integr. Neurosci.* 1**,** 3.

Briggs, F., Usrey, W.M., 2007b. A fast, reciprocal pathway between the lateral geniculate nucleus and visual cortex in the macaque monkey. *J. Neurosci.* 27**,** 5431-5436.

Briggs, F., Usrey, W.M., 2008. Emerging views of corticothalamic function. *Curr. Opin. Neurobiol.* 8**,** 403-407.

Briggs, F., Usrey, W.M., 2009. Parallel processing in the corticogeniculate pathway of the macaque monkey. *Neuron* 62**,** 135-146.

Briggs, F., Usrey, W.M., 2011. Corticogeniculate feedback and visual processing in the primate. *J. Physiol. (Lond).* 589**,** 33-40.

Briska, A.M., Uhlrich, D.J., Lytton, W.W., 2003. Computer model of passive signal integration based on whole-cell in vitro studies of rat lateral geniculate nucleus. *Eur. J. Neurosci.* 17**,** 1531-1541.

Brody, C. 1992. A model of feedback to the lateral geniculate nucleus. In: *Advances in Neural Information Processing Systems 5*. pp. 409-416. Morgan Kaufmann: San Francisco.

Bruno, R.M., 2011. Synchrony in sensation. *Curr. Opin. Neurobiol.* 21**,** 701-708.

Bruno, R.M., Sakmann, B., 2006. Cortex is driven by weak but synchronously active thalamocortical synapses. *Science* 312**,** 1622-1627.

Buffalo, E.A., Fries, P., Landman, R., Buschman, T.J., Desimone, R., 2011. Laminar differences in gamma and alpha coherence in the ventral stream. *Proc. Natl. Acad. Sci. U. S. A.* 108**,** 11262-11267.

Bullier, J., Kennedy, H., 1983. Projection of the lateral geniculate nucleus onto cortical area V2 in the macaque monkey. *Exp. Brain Res.* 53**,** 168-172.

Bullier, J., Norton, T.T., 1979. Comparison of receptive-field properties of X and Y ganglion cells with X and Y lateral geniculate cells in the cat. *J. Neurophysiol.* 42**,** 274-291.

Burke, W., Cole, A.M., 1978. Extraretinal influences on the lateral geniculate nucleus. *Rev. Physiol. Biochem. Pharmacol.* 80**,** 105-166.

Burke, W., Sefton, A.J., 1966. Inhibitory mechanisms in lateral geniculate nucleus of rat. *J. Physiol. (Lond).* 187**,** 231-246.

Buschman, T.J., Miller, E.K., 2007. Top-down versus bottom-up control of attention in the prefrontal and posterior parietal cortices. *Science* 315**,** 1860-1862.

Butler, A.B., 1994a. The evolution of the dorsal pallium in the telencephalon of amniotes: cladistic analysis and a new hypothesis. *Brain. Res. Rev.* 19**,** 66-101.

Butler, A.B., 1994b. The evolution of the dorsal thalamus of jawed vertebrates, including mammals: cladistic analysis and a new hypothesis. *Brain. Res. Rev.* 19**,** 29-65.

Butler, A.B., 2008. Evolution of the thalamus: a morphological and functional review. *Thalamus Relat. Syst.* 4**,** 35-38.

Butts, D.A., Weng, C., Jin, J., Yeh, C.-I., Lesica, N.A., Alonso, J.-M., Stanley, G.B., 2007. Temporal precision in the neural code and the timescales of natural vision. *Nature* 449**,** 92-95.

Buzás, P., Kóbor, P., Petykó, Z., Telkes, I., Martin, P.R., Lénárd, L., 2013. Receptive field properties of color opponent neurons in the cat lateral geniculate nucleus. *J. Neurosci.* 33**,** 1451-1461.

Buzsáki, G.r., Draguhn, A., 2004. Neuronal oscillations in cortical networks. *Science* 304**,** 1926-1929.

Cai, D., DeAngelis, G.C., Freeman, R.D., 1997. Spatiotemporal receptive field organization in the lateral geniculate nucleus of cats and kittens. *J. Neurophysiol.* 78**,** 1045-1061.

Callaway, E.M., 1998. Local circuits in primary visual cortex of the macaque monkey. *Annu. Rev. Neurosci.* 21**,** 47-74.

Callaway, E.M., 2005. Structure and function of parallel pathways in the primate early visual system. *J. Physiol. (Lond).* 566**,** 13-19.

Callaway, E.M., Wiser, A.K., 1996. Contributions of individual layer 2-5 spiny neurons to local circuits in macaque primary visual cortex. *Vis. Neurosci.* 13**,** 907-922.

Camp, A.J., Tailby, C., Solomon, S.G., 2009. Adaptable mechanisms that regulate the contrast response of neurons in the primate lateral geniculate nucleus. *J. Neurosci.* 29**,** 5009-5021.

Campbell, C.B.G., 1972. Evolutionary patterns in mammalian diencephalic visual nuclei and their fiber connections. *Brain. Behav. Evol.* 6**,** 218-236.

Cao, Y., Grossberg, S., 2005. A laminar cortical model of stereopsis and 3D surface perception: closure and da Vinci stereopsis. *Spat. Vis.* 18**,** 515-578.

Cao, Y., Grossberg, S., 2012. Stereopsis and 3D surface perception by spiking neurons in laminar cortical circuits: a method for converting neural rate models into spiking models. *Neural Networks* 26**,** 75-98.

Cao, Y., Grossberg, S., Markowitz, J., 2011. How does the brain rapidly learn and reorganize view-invariant and position-invariant object representations in the inferotemporal cortex? *Neural Networks* 24**,** 1050-1061.

Carandini, M., Heeger, D.J., 2012. Normalization as a canonical neural computation. *Nature Rev. Neurosci.* 13**,** 51-62.

Carandini, M., Horton, J.C., Sincich, L.C., 2007. Thalamic filtering of retinal spike trains by postsynaptic summation. *J. Vision* 7**,** 20.

Carrasco, M., 2011. Visual attention: the past 25 years. *Vision Res.* 51**,** 1484-1525.

Casagrande, V.A., 1994. A third parallel visual pathway to primate area V1. *Trends Neurosci.* 17**,** 305-310.

Casagrande, V.A., Boyd, J.D., 1996. The neural architecture of binocular vision. *Eye* 10**,** 153-160.

Casagrande, V.A., Sáry, G., Royal, D., Ruiz, O., 2005. On the impact of attention and motor planning on the lateral geniculate nucleus. *Prog. Brain Res.* 149**,** 11-29.

Casagrande, V.A., Yazar, F., Jones, K.D., Ding, Y., 2007. The morphology of the koniocellular axon pathway in the macaque monkey. *Cereb. Cortex* 17**,** 2334-2345.

Casale, A.E., McCormick, D.A., 2011. Active action potential propagation but not initiation in thalamic interneuron dendrites. *J. Neurosci.* 31**,** 18289-18302.

Castelo-Branco, M., Neuenschwander, S., Singer, W., 1998. Synchronization of visual responses between the cortex, lateral geniculate nucleus, and retina in the anesthetized cat. *J. Neurosci.* 18**,** 6395-6410.

Casti, A., Hayot, F., Xiao, Y., Kaplan, E., 2008. A simple model of retina-LGN transmission. *J. Comput. Neurosci.* 24**,** 235-252.

Cavanaugh, J., Berman, R.A., Joiner, W.M., Wurtz, R.H., 2016. Saccadic corollary discharge underlies stable visual perception. *J. Neurosci.* 36**,** 31-42.

Chang, D.H., Hess, R.F., Mullen, K.T., 2016. Color responses and their adaptation in human superior colliculus and lateral geniculate nucleus. *Neuroimage* 138**,** 211-220.

Chatterjee, S., Callaway, E.M., 2003. Parallel colour-opponent pathways to primary visual cortex. *Nature* 426**,** 668-671.

Chelazzi, L., Della Libera, C., Sani, I., Santandrea, E., 2011. Neural basis of visual selective attention. *Wiley Interdisciplinary Reviews: Cognitive Science* 2**,** 392–407.

Chelian, S.E., Srinivasa, N. (2013) A spiking thalamus model for form and motion processing of images. In: *International Joint Conference on Neural Networks (IJCNN)*. IEEE: Dallas, TX.

Chen, C., Blitz, D.M., Regehr, W.G., 2002. Contributions of receptor desensitization and saturation to plasticity at the retinogeniculate synapse. *Neuron* 779-788**,** 33.

Chen, W., Kato, T., Zhu, X.-H., Ogawa, S., Tank, D.W., Ugurbil, K., 1998. Human primary visual cortex and lateral geniculate nucleus activation during visual imagery. *Neuroreport* 9**,** 3669-3674.

Chen, Z., 2012. Object-based attention: a tutorial review. *Atten. Percept. Psychophys.* 74**,** 784-802.

Cheng, H., Chino, Y.M., Smith, E.L.r., Hamamoto, J., Yoshida, K., 1995. Transfer characteristics of lateral geniculate nucleus X neurons in the cat: effects of spatial frequency and contrast. *J. Neurophysiol.* 74**,** 2548-2557.

Cheong, S.K., Pietersen, A.N., 2014. Antidromic latency of magnocellular, parvocellular, and koniocellular (Blue-ON) geniculocortical relay cells in marmosets. *Vis. Neurosci.* 31**,** 263-273.

Cheong, S.K., Tailby, C., Solomon, S.G., Martin, P.R., 2013. Cortical-like receptive fields in the lateral geniculate nucleus of marmoset monkeys. *J. Neurosci.* 33**,** 6864-6876.

Cho, M.W., Choi, M.Y., 2014. A model for the receptive field of retinal ganglion cells. *Neural Networks* 49**,** 51-58.

Chung, S., Ferster, D., 1998. Strength and orientation tuning of the thalamic input to simple cells revealed by electrically evoked cortical suppression. *Neuron* 20**,** 1177-1189.

Cimenser, A., Miller, K.D., 2014. The effects of short-term synaptic depression at thalamocortical synapses on orientation tuning in cat V1. *PLoS ONE* 9**,** e106046.

Ciresan, D., Meier, U., Schmidhuber, J. (2012) Multi-column deep neural networks for image classification. In: *IEEE Conf. on Computer Vision and Pattern Recognition CVPR 2012*. pp. 3642–3649.

Cleland, B.G., Dubin, M.W., Levick, W.R., 1971a. Simultaneous recording of input and output of lateral geniculate neurones. *Nature New Biology* 231**,** 191-192.

Cleland, B.G., Dubin, M.W., Levick, W.R., 1971b. Sustained and transient neurones in the cat's retina and lateral geniculate nucleus. *J. Physiol. (Lond).* 217**,** 473-496.

Cleland, B.G., Lee, B.B., 1985. A comparison of visual responses of cat lateral geniculate nucleus neurones with those of ganglion cells afferent to them. *J. Physiol. (Lond).* 369**,** 249-268.

Coleman, K.A., Mitrofanis, J., 1996. Organization of the visual reticular thalamic nucleus of the rat. *Eur. J. Neurosci.* 8**,** 388-404.

Conley, M., 1988. Laminar organization of geniculostriate projections. A common organizational plan based on layers rather than individual functional classes. *Brain. Behav. Evol.* 32**,** 187-192.

Conley, M., Diamond, I.T., 1990. Organization of the visual sector of the thalamicreticular nucleus in Galago. *Eur. J. Neurosci.* 2**,** 211-226.

Conley, M., Raczkowski, D., 1990. Sublaminar organization within layer VI of the striate cortex in Galago. *J. Comp. Neurol.* 302**,** 425-436.

Conn, P.J., Pin, J.-P., 1997. Pharmacology and functions of metabotropic glutamate receptors. *Annu. Rev. Pharmacol. Toxicol.* 37**,** 205-237.

Connelly, W.M., Crunelli, V., Errington, A.C., 2015. The Global Spike: Conserved Dendritic Properties Enable Unique Ca2+ Spike Generation in Low-Threshold Spiking Neurons. *J. Neurosci.* 35**,** 15505-15522.

Connelly, W.M., Crunelli, V., Errington, A.C., 2016. Passive Synaptic Normalization and Input Synchrony-Dependent Amplification of Cortical Feedback in Thalamocortical Neuron Dendrites. *J. Neurosci.* 36**,** 3735-3754.

Connolly, M., Van Essen, D.C., 1984. The representation of the visual field in parvicellular and magnocellular layers of the lateral geniculate nucleus in the macaque monkey. *J. Comp. Neurol.* 226**,** 544-564.

Cope, D., Blakeslee, B., McCourt, M.E., 2013. Modeling lateral geniculate nucleus response with contrast gain control. Part 1: formulation. *J. Opt. Soc. Am. A.* 30**,** 2401-2408.

Cope, D., Blakeslee, B., McCourt, M.E., 2014. Modeling lateral geniculate nucleus response with contrast gain control. Part 2: analysis. *J. Opt. Soc. Am. A.* 31**,** 348-362.

Corbetta, M., Shulman, G.L., 2002. Control of goal-directed and stimulus-driven attention in the brain. *Nature Rev. Neurosci.* 3**,** 201-215.

Coulter, D.A., Huguenard, J.R., Prince, D.A., 1989. Calcium currents in rat thalamocortical relay neurones: kinetic properties of the transient, low-threshold current. *J. Physiol. (Lond)*. 414**,** 587-604.

Cowey, A., 2010a. The blindsight saga. *Exp. Brain Res.* 200**,** 3-24.

Cowey, A., 2010b. Visual system: how does blindsight arise? *Curr. Biol.* 20**,** R702-704.

Cowey, A., Stoerig, P., 1995. Blindsight in monkeys. *Nature* 373**,** 247-249.

Cox, C.L., Zhou, Q., Sherman, S.M., 1998. Glutamate locally activates dendritic outputs of thalamic interneurons. *Nature* 394**,** 478-482.

Crabtree, J.W., 1999. Intrathalamic sensory connections mediated by the thalamic reticular nucleus. *Cell. Mol. Life Sci.* 56**,** 683-700.

Crabtree, J.W., Isaac, J.T.R., 1998. A new intrathalamic pathway linking modality-related nuclei in the dorsal thalamus. *Nat. Neurosci.* 1**,** 389-394.

Crabtree, J.W., Isaac, J.T.R., 2002. New intrathalamic pathways allowing modality-related and cross-modality switching in the dorsal thalamus. *J. Neurosci.* 22**,** 8754-8761.

Crabtree, J.W., Killackey, H.P., 1989. The topographic organization and axis of projection within the visual sector of the rabbit's thalamic reticular nucleus. *Eur. J. Neurosci.* 1**,** 94-109.

Crandall, S.R., Cox, C.L., 2012. Local dendrodendritic inhibition regulates fast synaptic transmission in visual thalamus. *J. Neurosci.* 32**,** 2513-2522.

Crandall, S.R., Cruikshank, S.J., Connors, B.W., 2015. A corticothalamic switch: controlling the thalamus with dynamic synapses. *Neuron*.

Crick, F., 1984. Function of the thalamic reticular complex: the searchlight hypothesis. *Proc. Natl. Acad. Sci. U. S. A.* 81**,** 4586-4590.

Crunelli, V., Lightowler, S., Pollard, C.E., 1989. A T-type Ca2+ current underlies low-threshold Ca2+ potentials in cells of the cat and rat lateral geniculate nucleus. *J. Physiol. (Lond)*. 413**,** 543-561.

Cruz-Martín, A., El-Danaf, R.N., Osakada, F., Sriram, B., Dhande, O.S., Nguyen, P.L., Callaway, E.M., Ghosh, A., Huberman, A.D., 2014. A dedicated circuit links direction-selective retinal ganglion cells to the primary visual cortex. *Nature* 507**,** 358-361.

Cucchiaro, J.B., Bickford, M.E., Sherman, S.M., 1991a. A GABAergic projection from the pretectum to the dorsal lateral geniculate nucleus in the cat. *Neuroscience* 41**,** 213-226.

Cucchiaro, J.B., Uhlrich, D.J., Sherman, S.M., 1991b. Electron-microscopic analysis of synaptic input from the perigeniculate nucleus to the A-laminae of the lateral geniculate nucleus in cats. *J. Comp. Neurol.* 310**,** 316-336.

Cudeiro, J., Rivadulla, C., Grieve, K.L., 2000. Visual response augmentation in cat (and macaque) LGN: potentiation by corticofugally mediated gain control in the temporal domain. *Eur. J. Neurosci.* 12**,** 1135-1144.

Cudeiro, J., Sillito, A.M., 1996. Spatial frequency tuning of orientation-discontinuity-sensitive corticofugal feedback to the cat lateral geniculate nucleus. *J. Physiol. (Lond).* 490**,** 481-492.

Cudeiro, J., Sillito, A.M., 2006. Looking back: corticothalamic feedback and early visual processing. *Trends Neurosci.* 29**,** 298-306.

da Costa, N.M., Martin, K.A.C., 2009a. The proportion of synapses formed by the axons of the lateral geniculate nucleus in layer 4 of area 17 of the cat. *J. Comp. Neurol.* 516**,** 264-276.

da Costa, N.M., Martin, K.A.C., 2009b. Selective targeting of the dendrites of corticothalamic cells by thalamic afferents in Area 17 of the cat. *J. Neurosci.* 29**,** 13919-13928.

Dacey, D.M., Lee, B.B., 1994. The 'blue-on' opponent pathway in primate retina originates from a distinct bistratified ganglion cell type. *Nature* 367**,** 731-735.

Dan, Y., Alonso, J.-M., Usrey, W.M., Reid, R.C., 1998. Coding of visual information by precisely correlated spikes in the lateral geniculate nucleus. *Nat. Neurosci.* 1**,** 501-507.

Dan, Y., Atick, J.J., Reid, R.C., 1996. Efficient coding of natural scenes in the lateral geniculate nucleus: experimental test of a computational theory. *J. Neurosci.* 16**,** 3351-3362.

Dankowski, A., Bickford, M.E., 2003. Inhibitory circuitry involving Y cells and Y retinal terminals in the C laminae of the cat dorsal lateral geniculate nucleus. *J. Comp. Neurol.* 460**,** 368-379.

Datskovskaia, A., Carden, W.B., Bickford, M.E., 2001. Y retinal terminals contact interneurons in the cat dorsal lateral geniculate nucleus. *J. Comp. Neurol.* 430**,** 85-100.

Dawis, S., Shapley, R., Kaplan, E., Trachina, D., 1984. The receptive field organization of X-cells in the cat: spatiotemporal coupling and asymmetry. *Vision Res.* 24**,** 549-564.

de Labra, C., Rivadulla, C., Grieve, K., Mariño, J., Espinosa, N., Cudeiro, J., 2007. Changes in visual responses in the feline dLGN: selective thalamic suppression induced by transcranial magnetic stimulation of V1. *Cereb. Cortex* 17**,** 1376-1385.

De Lima, A.D., Singer, W., 1987. The brainstem projection to the lateral geniculate nucleus in the cat: identification of cholinergic and monoaminergic elements. *J. Comp. Neurol.* 259**,** 92-121.

de Sousa, A.A., Sherwood, C.C., Hof, P.R., Zilles, K., 2013. Lamination of the lateral geniculate nucleus of catarrhine primates. *Brain. Behav. Evol.* 81**,** 93-108.

De Valois, R.L., Abramov, I., Jacobs, G.H., 1966. Analysis of response patterns of LGN cells. *J. Opt. Soc. Am.* 56**,** 966-977.

De Valois, R.L., Cottaris, N.P., Elfar, S.D., Mahon, L.E., Wilson, J.A., 2000. Some transformations of color information from lateral geniculate nucleus to striate cortex. *Proc. Natl. Acad. Sci. U. S. A.* 97**,** 4997-5002.

DeAngelis, G.C., Ohzawa, I., Freeman, R.D., 1995. Receptive-field dynamics in the central visual pathways. *Trends Neurosci.* 18**,** 451-458.

Deleuze, C., David, F., Béhuret, S., Sadoc, G., Shin, H.-S., Uebele, V.N., Renger, J.J., Lambert, R.C., Leresche, N., Bal, T., 2012. T-type calcium channels consolidate tonic action potential output of thalamic neurons to neocortex. *J. Neurosci.* 32**,** 12228-12236.

Deleuze, C., Huguenard, J.R., 2006. Distinct electrical and chemical connectivity maps in the thalamic reticular nucleus: potential roles in synchronization and sensation. *J. Neurosci.* 26**,** 8633-8645.

Demb, J.B., 2008. Functional circuitry of visual adaptation in the retina. *J. Physiol. (Lond).* 586**,** 4377-4384.

Denison, R.N., Vu, A.T., Yacoub, E., Feinberg, D.A., Silver, M.A., 2014. Functional mapping of the magnocellular and parvocellular subdivisions of human LGN. *Neuroimage* 102**,** 358-369.

Denman, D.J., Contreras, D., 2015. Complex effects on in vivo visual responses by specific projections from mouse cortical layer 6 to dorsal lateral geniculate nucleus. *J. Neurosci.* 35**,** 9265-9280.

Denman, D.J., Contreras, D., 2016. On Parallel Streams through the Mouse Dorsal Lateral Geniculate Nucleus. *Front. Neural Circuits* 10**,** 20.

Denning, K.S., Reinagel, P., 2005. Visual control of burst priming in the anesthetized lateral geniculate nucleus. *J. Neurosci.* 25**,** 3531-3538.

Derrington, A.M., Felisberti, F., 1998. Peripheral shift reduces visual sensitivity in cat geniculate neurones. *Vis. Neurosci.* 15**,** 875-880.

Derrington, A.M., Fuchs, A.F., 1979. Spatial and temporal properties of X and Y cells in the cat lateral geniculate nucleus. *J. Physiol. (Lond).* 293**,** 347-364.

Derrington, A.M., Krauskopf, J., Lennie, P., 1984. Chromatic mechanisms in lateral geniculate nucleus of macaque. *J. Physiol. (Lond).* 357**,** 241-265.

Derrington, A.M., Lennie, P., 1984. Spatial and temporal contrast sensitivities of neurones in lateral geniculate nucleus of macaque. *J. Physiol. (Lond).* 357**,** 219-240.

Desbordes, G., Jin, J., Alonso, J.-M., Stanley, G.B., 2010. Modulation of temporal precision in thalamic population responses to natural visual stimuli. *Nat. Neurosci.* 4**,** 151.

Desbordes, G., Jin, J., Weng, C., Lesica, N.A., Stanley, G.B., Alonso, J.-M., 2008. Timing precision in population coding of natural scenes in the early visual system. *PLOS Biology* 6**,** e324.

Deschênes, M., Bin, H., 2006. Electrophysiology and pharmacology of the corticothalamic input to lateral thalamic nuclei: an intracellular study in the cat. *Eur. J. Neurosci.* 2**,** 140-152.

Deschênes, M., Hu, B., 1990. Membrane resistance increase induced in thalamic neurons by stimulation of brainstem cholinergic afferents. *Brain Res.* 513**,** 339-342.

Deschênes, M., Paradis, M., Roy, J.P., Steriade, M., 1984. Electrophysiology of neurons of lateral thalamic nuclei in cat: resting properties and burst discharges. *J. Neurophysiol.* 51**,** 1196-1219.

Desimone, R., 1998. Visual attention mediated by biased competition in extrastriate visual cortex. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 353**,** 1245-1255.

Destexhe, A., 2000. Modelling corticothalamic feedback and the gating of the thalamus by the cerebral cortex. *J. Physiol. Paris* 94**,** 391-410.

Destexhe, A., Contreras, D., Steriade, M., 1999. Cortically-induced coherence of a thalamic-generated oscillation. *Neuroscience* 92**,** 427-443.

Destexhe, A., Contreras, D., Steriade, M., Sejnowski, T.J., Huguenard, J.R., 1996. In vivo, in vitro, and computational analysis of dendritic calcium currents in thalamic reticular neurons. *J. Neurosci.* 16**,** 169-185.

Destexhe, A., Neubig, M., Ulrich, D., Huguenard, J., 1998. Dendritic low-threshold calcium currents in thalamic relay cells. *J. Neurosci.* 18**,** 3574-3588.

DeYoe, E.A., Van Essen, D.C., 1988. Concurrent processing streams in monkey visual cortex. *Trends Neurosci.* 11**,** 219-226.

Ding, Y., Casagrande, V.A., 1997. The distribution and morphology of LGN K pathway axons within the layers and CO blobs of owl monkey V1. *Vis. Neurosci.* 14**,** 691-704.

Donaldson, I.M., Dixon, R.A., 1980. Excitation of units in the lateral geniculate and contiguous nuclei of the cat by stretch of extrinsic ocular muscles. *Exp. Eye Res.* 38**,** 245-255.

Dong, D.W., Atick, J.J., 1995. Temporal decorrelation: a theory of lagged and nonlagged responses in the lateral geniculate nucleus. *Network: Comput. Neural Syst.* 6**,** 159–178.

Douglas, R.J., Martin, K.A.C., Whitteridge, D., 1989. A canonical microcircuit for neocortex. *Neural Comput.* 1**,** 480-488.

Dreher, B., Fukada, Y., Rodieck, R.W., 1976. Identification, classification and anatomical segregation of cells with X-like and Y-like properties in the lateral geniculate nucleus of old-world primates. *J. Physiol. (Lond).* 258**,** 433-452.

Dubin, M.W., Cleland, B.G., 1977. Organization of visual inputs to interneurons of lateral geniculate nucleus of the cat. *J. Neurophysiol.* 40**,** 410-427.

Dura-Bernal, S., Wennekers, T., Denham, S.L., 2012. Top-down feedback in an HMAX-like cortical model of object perception based on hierarchical Bayesian networks and belief propagation. *PLoS ONE* 7**,** e48216.

Eckhorn, R., Krause, F., Nelson, J.I., 1993. The RF-cinematogram. A cross-correlation technique for mapping several visual receptive fields at once. *Biol. Cybern.* 69**,** 37-55.

Edelman, G.M., 2003. Naturalizing consciousness: a theoretical framework. *Proc. Natl. Acad. Sci. U. S. A.* 100**,** 5520-5524.

Edelman, G.M., Gally, J.A., Baars, B.J., 2011. Biology of consciousness. *Front. Psychol.* 2**,** 4.

Einevoll, G.T., Heggelund, P., 2000. Mathematical models for the spatial receptive-field organization of nonlagged X-cells in dorsal lateral geniculate nucleus of cat. *Vis. Neurosci.* 17**,** 871-885.

Einevoll, G.T., Plesser, H.E., 2002. Linear mechanistic models for the dorsal lateral geniculate nucleus of cat probed using drifting-grating stimuli. *Network: Comput. Neural Syst.* 13**,** 503-530.

Einevoll, G.T., Plesser, H.E., 2012. Extended difference-of-Gaussians model incorporating cortical feedback for relay cells in the lateral geniculate nucleus of cat. *Cogn. Neurodyn.* 6**,** 307–324.

Elijah, D.H., Samengo, I., Montemurro, M.A., 2015. Thalamic neuron models encode stimulus information by burst-size modulation. *Front. Comput. Neurosci.* 9**,** 113.

Enroth-Cugell, C., Robson, J.G., 1966. The contrast sensitivity of retinal ganglion cells of the cat. *J. Physiol. (Lond).* 187**,** 517-552.

Erişir, A., Van Horn, S.C., Bickford, M.E., Sherman, S.M., 1997a. Immunocytochemistry and distribution of parabrachial terminals in the lateral geniculate nucleus of the cat: a comparison with corticogeniculate terminals. *J. Comp. Neurol.* 377**,** 535-549.

Erişir, A., Van Horn, S.C., Sherman, S.M., 1997b. Relative numbers of cortical and brainstem inputs to the lateral geniculate nucleus. *Proc. Natl. Acad. Sci. U. S. A.* 94**,** 1517-1520.

Eyherabide, H.G., Samengo, I., 2010. Time and category information in pattern-based codes. *Front. Comput. Neurosci.* 4**,** 145.

Fain, G.L., Matthews, H.R., Cornwall, M.C., Koutalos, Y., 2001. Adaptation in vertebrate photoreceptors. *Physiol. Rev.* 81**,** 117-151.

Famiglietti, E.V.J., Peters, A., 1972. The synaptic glomerulus and the intrinsic neuron in the dorsal lateral geniculate nucleus of the cat. *J. Comp. Neurol.* 144**,** 285-334.

Felisberti, F., Derrington, A.M., 1999. Long-range interactions modulate the contrast gain in the lateral geniculate nucleus of cats. *Vis. Neurosci.* 16**,** 943-956.

Felisberti, F., Derrington, A.M., 2001. Long-range interactions in the lateral geniculate nucleus of the New-World monkey, Callithrix jacchus. *Vis. Neurosci.* 18**,** 209-218.

Ferrarelli, F., Tononi, G., 2011. The thalamic reticular nucleus and schizophrenia. *Schizophr. Bull.* 37**,** 306-315.

Ferreiroa, R., Sánchez, E., 2013. Functional properties of a realistic model of dLGN. *Neurocomputing* 114**,** 36–44.

Ferrera, V.P., Nealey, T.A., Maunsell, J.H., 1992. Mixed parvocellular and magnocellular geniculate signals in visual area V4. *Nature* 358**,** 756-761.

Ferster, D., Chung, S., Wheat, H., 1996. Orientation selectivity of thalamic input to simple cells of cat visual cortex. *Nature* 380**,** 249-252.

Ferster, D., Lindström, S., 1983. An intracellular analysis of geniculo-cortical connectivity in area 17 of the cat. *J. Physiol. (Lond).* 342**,** 181-215.

Ferster, D., Miller, K.D., 2000. Neural mechanisms of orientation selectivity in the visual cortex. *Annu. Rev. Neurosci.* 23**,** 441-471.

Fielding, K.H., Ruck, D.W., 1995. Recognition of moving light displays using hidden markov models. *Pattern Recognition* 28**,** 1415-1421.

Fiorillo, C.D., Kim, J.K., Hong, S.Z., 2014. The meaning of spikes from the neuron's point of view: predictive homeostasis generates the appearance of randomness. *Front. Comput. Neurosci.* 8**,** 49.

Fischer, I., Vicente, R., Buldú, J.M., Peil, M., Mirasso, C.R., Torrent, M.C., García-Ojalvo, J., 2006. Zero-lag long-range synchronization via dynamical relaying. *Phys. Rev. Lett.* 97**,** 123902.

Fischer, W.H., Schmidt, M., Hoffmann, K.P., 1998. Saccade-induced activity of dorsal lateral geniculate nucleus X- and Y-cells during pharmacological inactivation of the cat pretectum. *Vis. Neurosci.* 15**,** 197-210.

Fisher, T.G., Alitto, H.J., Usrey, W.M., 2016. Retinal and Non-Retinal Contributions to Extraclassical Surround Suppression in the Lateral Geniculate Nucleus. *J. Neurosci.* [Epub ahead of print].

Fitzgibbon, T., Bittar, R.G., Dreher, B., 1999. Projections from striate and extrastriate visual cortices of the cat to the reticular thalamic nucleus. *J. Comp. Neurol.* 410**,** 467-488.

Fitzgibbon, T., Szmajida, B.A., Martin, P.R., 2007. First order connections of the visual sector of the thalamic reticular nucleus in marmoset monkeys (Callithrix jacchus). *Vis. Neurosci.* 24**,** 857-874.

Fitzpatrick, D., Diamond, I.T., Raczkowski, D., 1989. Cholinergic and monoaminergic innervation of the cat's thalamus: comparison of the lateral geniculate nucleus with other principal sensory nuclei. *J. Comp. Neurol.* 288**,** 647-675.

Fitzpatrick, D., Penny, G.R., Schmechel, D.E., 1984. Glutamic acid decarboxylase-immunoreactive neurons and terminals in the lateral geniculate nucleus of the cat. *J. Neurosci.* 4**,** 1809-1829.

Fitzpatrick, D., Usrey, W.M., Schofield, B.R., Einstein, G., 1994. The sublaminar organization of corticogeniculate neurons in layer 6 of macaque striate cortex. *Vis. Neurosci.* 11**,** 307-315.

Fjeld, I.T., Ruksenas, O., Heggelund, P., 2002. Brainstem modulation of visual response properties of single cells in the dorsal lateral geniculate nucleus of cat. *J. Physiol. (Lond).* 543**,** 541-554.

Fries, P., 2005. A mechanism for cognitive dynamics: neuronal communication through neuronal coherence. *Trends Cogn. Sci.* 9**,** 474-478.

Fries, W., 1981. The projection from the lateral geniculate nucleus to the prestriate cortex of the macaque monkey. *Proc. R. Soc. Lond. B. Biol. Sci.* 213**,** 73-86.

Fuentealba, P., Steriade, M., 2005. The reticular nucleus revisited: intrinsic and network properties of a thalamic pacemaker. *Prog. Neurobiol.* 75**,** 125-141.

Fukushima, K., 1980. Neocognitron : A self-organizing neural network model for a mechanism of pattern recognition unaffected by shift in position. *Biol. Cybern.* 36**,** 193-202.

Fukushima, K., 1988. Neocognitron: A hierarchical neural network capable of visual pattern recognition. *Neural Networks* 1**,** 119-130.

Funke, K., Nelle, E., Li, B., Wörgötter, F., 1996. Corticofugal feedback improves the timing of retino-geniculate signal transmission. *Neuroreport* 7**,** 2130-2134.

Funke, K., Wörgötter, F., 1997. On the significance of temporally structured activity in the dorsal lateral geniculate nucleus (LGN). *Prog. Neurobiol.* 53**,** 67-119.

Gaglianese, A., Costagli, M., Bernardi, G., Ricciardi, E., Pietrini, P., 2012. Evidence of a direct influence between the thalamus and hMT+ independent of V1 in the human brain as measured by fMRI. *Neuroimage* 60**,** 1440-1447.

Gaglianese, A., Costagli, M., Ueno, K., Ricciardi, E., Bernardi, G., Pietrini, P., Cheng, K., 2015. The direct, not V1-mediated, functional influence between the thalamus and middle temporal complex in the human brain is modulated by the speed of visual motion. *Neuroscience* 284**,** 833-844.

Gastinger, M.J., Tian, N., Horvath, T., Marshak, D.W., 2006. Retinopetal axons in mammals: emphasis on histamine and serotonin. *Curr. Eye Res.* 31**,** 655-667.

Gattass, R., Galkin, T.W., Desimone, R., Ungerleider, L.G., 2014. Subcortical connections of area V4 in the macaque. *J. Comp. Neurol.* 522**,** 1941-1965.

Gawne, T.J., McClurkin, J.W., Richmond, B.J., Optican, L.M., 1991. Lateral geniculate neurons in behaving primates. III. Response predictions of a channel model with multiple spatial-to-temporal filters. *J. Neurophysiol.* 66**,** 809-823.

Geisert, E.E., Langsetmo, A., Spear, P.D., 1981. Influence of the cortico-geniculate pathway on response properties of cat lateral geniculate neurons. *Brain Res.* 208**,** 409-415.

George, D., Hawkins, J., 2009. Towards a mathematical theory of cortical micro-circuits. *PLoS Comput Biol* 5**,** e1000532.

Ghazanfar, A.A., Nicolelis, M.A., 2001. Feature article: the structure and function of dynamic cortical and thalamic receptive fields. *Cereb. Cortex* 11**,** 183-193.

Ghodrati, M., Khaligh-Razavi, S.-M., Ebrahimpour, R., Rajaei, K., Pooyan, M., 2012. How can selection of biologically inspired features improve the performance of a robust object recognition model? *PLoS ONE* 7**,** e32357.

Gilbert, C.D., 1977. Laminar differences in receptive field properties of cells in cat primary visual cortex. *J. Physiol. (Lond).* 268**,** 391-421.

Gilbert, C.D., Kelly, J.P., 1975. The projections of cells in different layers of the cat's visual cortex. *J. Comp. Neurol.* 163**,** 81-105.

Girardin, C.C., Kiper, D.C., Martin, K.A.C., 2002. The effect of moving textures on the responses of cells in the cat's dorsal lateral geniculate nucleus. *Eur. J. Neurosci.* 16**,** 2149-2156.

Godwin, D.W., Vaughan, J.W., Sherman, S.M., 1996. Metabotropic glutamate receptors switch visual response mode of lateral geniculate nucleus cells from burst to tonic. *J. Neurophysiol.* 76**,** 1800-1816.

Gollisch, T., Meister, M., 2010. Eye smarter than scientists believed: neural computations in circuits of the retina. *Neuron* 65**,** 150-164.

Gollo, L.L., Mirasso, C., Villa, A.E., 2010. Dynamic control for synchronization of separated cortical areas through thalamic relay. *Neuroimage* 52**,** 947-955.

Golomb, D., Kleinfeld, D., Reid, R.C., Shapley, R.M., Shraiman, B.I., 1994. On temporal codes and the spatiotemporal response of neurons in the lateral geniculate nucleus. *J. Neurophysiol.* 72**,** 2990-3003.

Gouws, A.D., Alvarez, I., Watson, D.M., Uesaki, M., Rodgers, J., Morland, A.B., 2014. On the role of suppression in spatial attention: evidence from negative BOLD in human subcortical and cortical structures. *J. Neurosci.* 34**,** 10347-10360.

Gove, A., Grossberg, S., Mingolla, E., 1995. Brightness perception, illusory contours, and corticogeniculate feedback. *Vis. Neurosci.* 12**,** 1027-1052.

Graham, J., Lin, C.-S., Kaas, J.H., 1979. Subcortical projections of six visual cortical areas in the owl monkey, Aotus trivirgatus. *J. Comp. Neurol.* 187**,** 557-580.

Granseth, B., 2004. Dynamic properties of corticogeniculate excitatory transmission in the rat dorsal lateral geniculate nucleus in vitro. *J. Physiol. (Lond).* 556**,** 135-146.

Granseth, B., Ahistrand, E., Lindström, S., 2002. Paired pulse facilitation of corticogeniculate EPSCs in the dorsal lateral geniculate nucleus of the rat investigated in vitro. *J. Physiol. (Lond).* 544**,** 477-486.

Granseth, B., Lindström, S., 2003. Unitary EPSCs of corticogeniculate fibers in the rat dorsal lateral geniculate nucleus in vitro. *J. Neurophysiol.* 89**,** 2952-2960.

Grieve, K.L., 2005. Binocular visual responses in cells of the rat dLGN. *J. Physiol. (Lond).* 566**,** 119-124.

Grieve, K.L., Sillito, A.M., 1995. Differential properties of cells in the feline primary visual cortex providing the corticofugal feedback to the lateral geniculate nucleus and visual claustrum. *J. Neurosci.* 15**,** 4868-4874.

Grossberg, S., 1999. How does the cerebral cortex work? Learning, attention, and grouping by the laminar circuits of visual cortex. *Spat. Vis.* 12**,** 163-185.

Grossberg, S., 2007. Towards a unified theory of neocortex: laminar cortical circuits for vision and cognition. *Prog. Brain Res.* 165**,** 79-104.

Grossberg, S., 2013. Adaptive Resonance Theory: how a brain learns to consciously attend, learn, and recognize a changing world. *Neural Networks* 37**,** 1-47.

Grossberg, S., Howe, P.D., 2003. A laminar cortical model of stereopsis and three-dimensional surface perception. *Vision Res.* 43**,** 801-829.

Grossberg, S., Kuhlmann, L., Mingolla, E., 2007. A neural model of 3D shape-from-texture: multiple-scale filtering, boundary grouping, and surface filling-in. *Vision Res.* 47**,** 634-672.

Grossberg, S., Srinivasan, K., Yazdanbakhsh, A., 2015. Binocular fusion and invariant category learning due to predictive remapping during scanning of a depthful scene with eye movements. *Front. Psychol.* 5**,** 1457.

Grossberg, S., Yazdanbakhsh, A., 2005. Laminar cortical dynamics of 3D surface perception: stratification, transparency, and neon color spreading. *Vision Res.* 45**,** 1725-1743.

Grubb, M.S., Thompson, I.D., 2003. Quantitative characterization of visual response properties in the mouse dorsal lateral geniculate nucleus. *J. Neurophysiol.* 90**,** 3594-3607.

Grubb, M.S., Thompson, I.D., 2004. Biochemical and anatomical subdivision of the dorsal lateral geniculate nucleus in normal mice and in mice lacking the beta2 subunit of the nicotinic acetylcholine receptor. *Vision Res.* 44**,** 3365-3376.

Grubb, M.S., Thompson, I.D., 2005. Visual response properties of burst and tonic firing in the mouse dorsal lateral geniculate nucleus. *J. Neurophysiol.* 93**,** 3224-3247.

Grunewald, A., Grossberg, S., 1998. Self-organization of binocular disparity tuning by reciprocal corticogeniculate interactions. *J. Cogn. Neurosci.* 10**,** 199-215.

Guido, W., Lu, S.-m., Sherman, S.M., 1992. Relative contributions of burst and tonic responses to the receptive field properties of lateral geniculate neurons in the cat. *J. Neurophysiol.* 68**,** 2199-2211.

Guido, W., Lu, S.-M., Vaughan, J.W., Godwin, D.W., Sherman, S.M., 1995. Receiver operating characteristic (ROC) analysis of neurons in the cat's lateral geniculate nucleus during tonic and burst response mode. *Vis. Neurosci.* 12**,** 723-741.

Guido, W., Sherman, S.M., 1998. Response latencies of cells in the cat's lateral geniculate nucleus are less variable during burst than tonic firing. *Vis. Neurosci.* 15**,** 231-237.

Guido, W., Tumosa, N., Spear, P.D., 1989. Binocular interactions in the cat's dorsal lateral geniculate nucleus. I. Spatial-frequency analysis of responses of X, Y, and W cells to nondominant-eye stimulation. *J. Neurophysiol.* 62**,** 526-543.

Guido, W., Weyand, T., 1995. Burst responses in thalamic relay cells of the awake behaving cat. *J. Neurophysiol.* 74**,** 1782-1786.

Guillery, R.W., 1966. A study of Golgi preparations from the dorsal lateral geniculate nucleus of the adult cat. *J. Comp. Neurol.* 128**,** 21-50.

Guillery, R.W., 1969. The organization of synaptic interconnections in the laminae of the dorsal lateral geniculate nucleus of the cat. *Z. Zellforsch. Mikrosk. Anat.* 96**,** 1-38.

Guillery, R.W., 1971. Patterns of synaptic interconnections in the dorsal lateral geniculate nucleus of cat and monkey: a brief review. *Vision Res.* Suppl 3**,** 211-227.

Guillery, R.W., Feig, S.L., Lozsádi, D.A., 1998. Paying attention to the thalamic reticular nucleus. *Trends Neurosci.* 21**,** 28-32.

Guillery, R.W., Harting, J.K., 2003. Structure and connections of the thalamic reticular nucleus: Advancing views over half a century. *J. Comp. Neurol.* 463**,** 360-371.

Guillery, R.W., Sherman, S.M., 2002a. Thalamic relay functions and their role in corticocortical communication: generalizations from the visual system. *Neuron* 33**,** 163-175.

Guillery, R.W., Sherman, S.M., 2002b. The thalamus as a monitor of motor outputs. *Proc. R. Soc. Lond. B. Biol. Sci.* 357**,** 1809-1821.

Gulyás, B., Lagae, L., Eysel, U., Orban, G.A., 1990. Corticofugal feedback influences the responses of geniculate neurons to moving stimuli. *Exp. Brain Res.* 79**,** 441-446.

Gulyás, B., Orban, G.A., Duysens, J., Maes, H., 1987. The suppressive influence of moving textured backgrounds on responses of cat striate neurons to moving bars. *J. Neurophysiol.* 57**,** 1767-1791.

Halassa, M., Acsády, L., 2016. Thalamic Inhibition: Diverse Sources, Diverse Scales. *Trends Neurosci.* 39**,** 680-693.

Halassa, M.M., Chen, Z., Wimmer, R.D., Brunetti, P.M., Zhao, S., Zikopoulos, B., Wang, F., Brown, E.N., Wilson, M.A., 2014. State-dependent architecture of thalamic reticular subnetworks. *Cell* 158**,** 808-821.

Hallanger, A.E., Levey, A.I., Lee, H.J., Rye, D.B., Wainer, B.H., 1987. The origins of cholinergic and other subcortical afferents to the thalamus in the rat. *J. Comp. Neurol.* 262**,** 105-124.

Halnes, G., Augustinaite, S., Heggelund, P., Einevoll, G.T., Migliore, M., 2011. A multi-compartment model for interneurons in the dorsal lateral geniculate nucleus. *PLoS Comput Biol* 7**,** e1002160.

Hamamoto, J., Cheng, H., Yoshida, K., Smith, E.L.I., Chino, Y.M., 1994. Transfer characteristics of lateral geniculate nucleus X-neurons in the cat: effects of temporal frequency. *Exp. Brain Res.* 98**,** 191-199.

Hámori, J., Pasik, P., Pasik, T., 1991. Different types of synaptic triads in the monkey dorsal lateral geniculate nucleus. *J. Hirnforsch.* 32**,** 369-379.

Hamos, J.E., Van Horn, S.C., Raczkowski, D., Sherman, S.M., 1987. Synaptic circuits involving an individual retinogeniculate axon in the cat. *J. Comp. Neurol.* 259**,** 165-192.

Hamos, J.E., Van Horn, S.C., Raczkowski, D., Uhlrich, D.J., Sherman, S.M., 1985. Synaptic connectivity of a local circuit neurone in lateral geniculate nucleus of the cat. *Nature* 317**,** 618-621.

Han, B., VanRullen, R., 2016. Shape perception enhances perceived contrast: evidence for excitatory predictive feedback? *Scientific Reports* 6**,** 22944.

Harris, K.D., Thiele, A., 2011. Cortical state and attention. *Nature Rev. Neurosci.* 12**,** 509-523.

Harth, E., Unnikrishnan, K.P., Pandya, A.S., 1987. The inversion of sensory processing by feedback pathways: a model of visual cognitive functions. *Science* 237**,** 184-187.

Harting, J.K., Huerta, M.F., Hashikawa, T., van Lieshout, D.P., 1991a. Projection of the mammalian superior colliculus upon the dorsal lateral geniculate nucleus: organization of tectogeniculate pathways in nineteen species. *J. Comp. Neurol.* 304**,** 275-306.

Harting, J.K., Van Lieshout, D.P., Feig, S.L., 1991b. Connectional studies of the primate lateral geniculate nucleus: distribution of axons arising from the thalamic reticular nucleus of Galago crassicaudatus. *J. Comp. Neurol.* 310**,** 411-427.

Hartveit, E., Heggelund, P., 1995. Brainstem modulation of signal transmission through the cat dorsal lateral geniculate nucleus. *Exp. Brain Res.* 103**,** 372-384.

Harvey, A.R., 1980. A physiological analysis of subcortical and commissural projections of areas 17 and 18 of the cat. *J. Physiol. (Lond).* 302**,** 507-534.

Hawken, M.J., Shapley, R.M., Grosof, D.H., 1996. Temporal-frequency selectivity in monkey visual cortex. *Vision Res.* 13**,** 477-492.

Haynes, J.-D., Deichmann, R., Rees, G., 2005. Eye-specific effects of binocular rivalry in the human lateral geniculate nucleus. *Nature* 438**,** 496-499.

Hayot, F., Tranchina, D., 2001. Modeling corticofugal feedback and the sensitivity of lateral geniculate neurons to orientation discontinuity. *Vis. Neurosci.* 18**,** 865-877.

Heiberg, T., Kriener, B., Tetzlaff, T., Casti, A., Einevoll, G.T., Plesser, H.E., 2013. Firing-rate models capture essential response dynamics of LGN relay cells. *J. Comput. Neurosci.* 35**,** 359-375.

Hendrickson, A.E., Wilson, J.R., Ogren, M.P., 1978. The neuroanatomical organization of pathways between the dorsal lateral geniculate nucleus and visual cortex in Old World and New World primates. *J. Comp. Neurol.* 182**,** 123-136.

Hendry, S.H.C., Reid, R.C., 2000. The koniocellular pathway in primate vision. *Annu. Rev. Neurosci.* 23**,** 127-153.

Hendry, S.H.C., Yoshioka, T., 1994. A neurochemically distinct third channel in the macaque dorsal lateral geniculate nucleus. *Science* 264**,** 575-577.

Henke, H., Robinson, P.A., Drysdale, P.M., Loxley, P.N., 2014. Spatiotemporally varying visual hallucinations: I. Corticothalamic theory. *J. Theor. Biol.* 357**,** 200-209.

Hernández-Cruz, A., Pape, H.C., 1989. Identification of two calcium currents in acutely dissociated neurons from the rat lateral geniculate nucleus. *J. Neurophysiol.* 61**,** 1270-1283.

Hesam Shariati, N., Freeman, A.W., 2012. A multi-stage model for fundamental functional properties in primary visual cortex. *PLoS ONE* 7**,** e34466.

Hess, R.F., Thompson, B., Gole, G., Mullen, K.T., 2009. Deficient responses from the lateral geniculate nucleus in humans with amblyopia. *Eur. J. Neurosci.* 29**,** 1064-1070.

Hickey, T.L., Guillery, R.W., 1979. Variability of laminar patterns in the human lateral geniculate nucleus. *J. Comp. Neurol.* 183**,** 221-246.

Hillenbrand, U., van Hemmen, J.L., 2000. Spatiotemporal adaptation through corticothalamic loops: a hypothesis. *Vis. Neurosci.* 17**,** 107-118.

Hillenbrand, U., van Hemmen, J.L., 2001. Does corticothalamic feedback control cortical velocity tuning? *Neural Comput.* 13**,** 327-355.

Hillenbrand, U., van Hemmen, J.L., 2002. Adaptation in the corticothalamic loop: computational prospects of tuning the senses. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 357**,** 1859-1867.

Hindmarsh, J.L., Rose, R.M., 1984. A model of neuronal bursting using three coupled first order differential equations. *Proc. R. Soc. Lond. B. Biol. Sci.* 221[87-102.

Hinton, G.E., Dayan, P., Frey, B.J., Neal, R.M., 1995. The "wake-sleep" algorithm for unsupervised neural networks. *Science* 268**,** 1158-1161.

Hirsch, J.A., Martinez, L.M., 2006. Laminar processing in the visual cortical column. *Curr. Opin. Neurobiol.* 16**,** 377-384.

Hirsch, J.A., Wang, X., Sommer, F.T., Martinez, L.M., 2015. How inhibitory circuits in the thalamus serve vision. *Annu. Rev. Neurosci.* 38**,** 309-329.

Hochstein, S., Shapley, R.M., 1976. Linear and nonlinear spatial subunits in Y cat retinal ganglion cells. *J. Physiol. (Lond).* 262**,** 265-284.

Hofmann, M.H., Northcutt, R.G., 2012. Forebrain organization in elasmobranchs. *Brain. Behav. Evol.* 80**,** 142-151.

Hong, S.Z., Kim, H.R., Fiorillo, C.D., 2014. T-type calcium channels promote predictive homeostasis of input-output relations in thalamocortical neurons of lateral geniculate nucleus. *Front. Comput. Neurosci.* 8**,** 98.

Hou, G., Smith, A.G., Zhang, Z.W., 2016. Lack of Intrinsic GABAergic Connections in the Thalamic Reticular Nucleus of the Mouse. *J. Neurosci.* 36**,** 7246-7252.

Howarth, M., Walmsley, L., Brown, T., 2014. Binocular integration in the mouse lateral geniculate nuclei. *Curr. Biol.* 24**,** 1241-1247.

Hu, B., Li, X., Zhou, Y., Shou, T., 2000. Effects of bicuculline on direction-sensitive relay cells in the dorsal lateral geniculate nucleus (LGNd) of cats. *Brain Res.* 885**,** 87-93.

Hu, B., Steriade, M., Deschênes, M., 1989. The effects of brainstem peribrachial stimulation on neurons of the lateral geniculate nucleus. *Neuroscience* 31**,** 13-24.

Huang, Y., Rao, R.P.N., 2011. Predictive Coding. *Wiley Interdisciplinary Reviews: Cognitive Science* 2**,** 580-593.

Hubel, D., Livingstone, M.S., 1990. Color and contrast sensitivity in the lateral geniculate body and primary visual cortex of the macaque monkey. *J. Neurosci.* 10**,** 2223-2237.

Hubel, D.H., Wiesel, T.N., 1961. Integrative action in the cat's lateral geniculate body. *J. Physiol. (Lond).* 155**,** 385-398.

Hubel, D.H., Wiesel, T.N., 1962. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *J. Physiol. (Lond).* 160**,** 106-154.

Huberman, A.D., Niell, C.M., 2011. What can mice tell us about how vision works? *Trends Neurosci.* 34**,** 464-473.

Huertas, M.A., Groff, J.R., Smith, G.D., 2005. Feedback inhibition and throughput properties of an integrate-and-fire-or-burst network model of retinogeniculate transmission. *J. Comput. Neurosci.* 19**,** 147-180.

Huertas, M.A., Smith, G.D., 2006. A multivariate population density model of the dLGN/PGN relay. *J. Comput. Neurosci.* 21**,** 171-189.

Hughes, H.C., Mullikin, W.H., 1984. Brainstem afferents to the lateral geniculate nucleus of the cat. *Exp. Brain Res.* 54**,** 253-258.

Huguenard, J.R., 1996. Low-threshold calcium currents in central nervous system neurons. *Annu. Rev. Physiol.* 58**,** 329-348.

Hull, C., Isaacson, J.S., Scanziani, M., 2009. Postsynaptic mechanisms govern the differential excitation of cortical neurons by thalamic inputs. *J. Neurosci.* 29**,** 9127-9136.

Hull, E.M., 1968. Corticofugal influence in the macaque lateral geniculate nucleus. *Vision Res.* 8**,** 1285-1298.

Humphrey, A.L., Saul, A.B., 1992. Action of brain stem reticular afferents on lagged and nonlagged cells in the cat lateral geniculate nucleus. *J. Neurophysiol.* 68**,** 673-691.

Humphrey, A.L., Weller, R.E., 1988. Functionally distinct groups of X-cells in the lateral geniculate nucleus of the cat. *J. Comp. Neurol.* 268**,** 429-447.

Hwang, K., Bertolero, M.A., Liu, W., D'Esposito, M., 2016. The human thalamus is an integrative hub for functional brain networks. http://biorxiv.org/content/early/2016/2006/2002/056630.

Ichida, J., Mavity-Hudson, J., Casagrande, V., 2014. Distinct patterns of corticogeniculate feedback to different layers of the lateral geniculate nucleus. *Eye and Brain* 6 Suppl 1**,** 57-73.

Ichida, J.M., Casagrande, V.A., 2002. Organization of the feedback pathway from striate cortex (V1) to the lateral geniculate nucleus (LGN) in the owl monkey (Aotus trivirgatus). *J. Comp. Neurol.* 454**,** 272-283.

Irvin, G.E., Casagrande, V.A., Norton, T.T., 1993. Center/surround relationships of magnocellular, parvocellular, and koniocellular relay cells in primate lateral geniculate nucleus. *Vis. Neurosci.* 10**,** 363-373.

Izhikevich, E.M., Desai, N.S., Walcott, E.C., Hoppensteadt, F.C., 2003. Bursts as a unit of neural information: selective communication via resonance. *Trends Neurosci.* 26**,** 161-167.

Jacobs, G.H., 2009. Evolution of colour vision in mammals. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 364**,** 2957-2967.

Jahnsen, H., Llinás, R., 1984a. Electrophysiological properties of guinea-pig thalamic neurones: an in vitro study. *J. Physiol. (Lond).* 349**,** 205-226.

Jahnsen, H., Llinás, R., 1984b. Ionic basis for the electro-responsiveness and oscillatory properties of guinea-pig thalamic neurones in vitro. *J. Physiol. (Lond).* 349**,** 227-247.

Jayakumar, J., Roy, S., Dreher, B., Martin, P.R., Vidyasagar, T.R., 2013. Multiple pathways carry signals from short-wavelength-sensitive ('blue') cones to the middle temporal area of the macaque. *J. Physiol. (Lond).* 591**,** 339-352.

Jeffries, A.M., Killian, N.J., Pezaris, J.S., 2014. Mapping the primate lateral geniculate nucleus: a review of experiments and methods. *J. Physiol. Paris* 108**,** 3-10.

Jehee, J.F.M., Ballard, D.H., 2009. Predictive feedback can account for biphasic responses in the lateral geniculate nucleus. *PLoS Comput Biol* 5**,** e1000373.

Jensen, O., Bonnefond, M., Marshall, T.R., Tiesinga, P., 2015. Oscillatory mechanisms of feedforward and feedback visual processing. *Trends Neurosci.* 38**,** 192-194.

Jiang, Y., Yampolsky, D., Purushothaman, G., Casagrande, V.A., 2015. Perceptual decision related activity in the lateral geniculate nucleus (LGN). *J. Neurophysiol.* jn.00068.2015.

Jones, E.G., 2001. The thalamic matrix and thalamocortical synchrony. *Trends Neurosci.* 24**,** 595-601.

Jones, E.G., 2002. Thalamic circuitry and thalamocortical synchrony. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 357**,** 1659-1673.

Jones, H.E., Andolina, I.M., Ahmed, B., Shipp, S.D., Clements, J.T.C., Grieve, K.L., Cudeiro, J., Salt, T.E., Sillito, A.M., 2012. Differential feedback modulation of center and surround mechanisms in parvocellular cells in the visual thalamus. *J. Neurosci.* 32**,** 15946-15951.

Jones, H.E., Andolina, I.M., Grieve, K.L., Wang, W., Salt, T.E., Cudeiro, J., Sillito, A.M., 2013. Responses of primate LGN cells to moving stimuli involve a constant background modulation by feedback from area MT. *Neuroscience* 246**,** 254-264.

Jones, H.E., Andolina, I.M., Shipp, S.D., Adams, D.L., Cudeiro, J., Salt, T.E., Sillito, A.M., 2015. Figure-ground modulation in awake primate thalamus. *Proc. Natl. Acad. Sci. U. S. A.* 112**,** 7085-7090.

Jones, H.E., Sillito, A.M., 1991. The length-response properties of cells in the feline dorsal lateral geniculate nucleus. *J. Physiol. (Lond).* 444**,** 329-348.

Jurgens, C.W., Bell, K.A., McQuiston, A.R., Guido, W., 2012. Optogenetic stimulation of the corticothalamic pathway affects relay cells and GABAergic neurons differently in the mouse visual thalamus. *PLoS ONE* 7**,** e45717.

Kaas, J.H., Guillery, R.W., Allman, J.M., 1972. Some principles of organization in the dorsal lateral geniculate nucleus. *Brain. Behav. Evol.* 6**,** 253-299.

Kaas, J.H., Huerta, M.F., Weber, J.T., Harting, J.K., 1978. Patterns of retinal terminations and laminar organization of the lateral geniculate nucleus of primates. *J. Comp. Neurol.* 182**,** 517-553.

Kalil, R.E., Chase, R., 1970. Corticofugal influence on activity of lateral geniculate neurons in the cat. *J. Neurophysiol.* 33**,** 459-474.

Kanwisher, N., Wojciulik, E., 2000. Visual attention: insights from brain imaging. *Nature Rev. Neurosci.* 1**,** 91-100.

Kaplan, E., Purpura, K., Shapley, R.M., 1987. Contrast affects the transmission of visual information through the mammalian lateral geniculate nucleus. *J. Physiol. (Lond).* 391**,** 267-288.

Kaplan, E., Shapley, R., 1984. The origin of the S (slow) potential in the mammalian lateral geniculate nucleus. *Exp. Brain Res.* 55**,** 111-116.

Kaplan, E., Shapley, R.M., 1982. X and Y cells in the lateral geniculate nucleus of macaque monkeys. *J. Physiol. (Lond).* 330**,** 125-143.

Kara, P., Reid, R.C., 2003. Efficacy of retinal spikes in driving cortical responses. *J. Neurosci.* 23**,** 8547-8557.

Kara, P., Reinagel, P., Reid, R.C., 2000. Low response variability in simultaneously recorded retinal, thalamic, and cortical neurons. *Neuron* 27**,** 635-646.

Karpathy, A., Fei-Fei, L., 2015. Deep visual-semantic alignments for generating image descriptions. http://arxiv.org/abs/1412.2306.

Kastner, S., O'Connor, D.H., Fukui, M.M., Fehd, H.M., Herwig, U., Pinsk, M.A., 2004. Functional imaging of the human lateral geniculate nucleus and pulvinar. *J. Neurophysiol.* 91**,** 438-448.

Kastner, S., Schneider, K.A., Wunderlich, K., 2006. Beyond a relay nucleus: neuroimaging views on the human LGN. *Prog. Brain Res.* 155**,** 125-143.

Kath, W.L., 2005. Computational modeling of dendrites. *J. Neurobiol.* 64**,** 91-99.

Kawashima, T., Okuno, H., Bito, H., 2014. A new era for functional labeling of neurons: activity-dependent promoters have come of age. *Front. Neural Circuits* 8**,** 37.

Kayser, A., Priebe, N.J., Miller, K.D., 2001. Contrast-dependent nonlinearities arise locally in a model of contrast-invariant orientation tuning. *J. Neurophysiol.* 85**,** 2130-2149.

Keat, J., Reinagel, P., Reid, R.C., Meister, M., 2001. Predicting every spike: a model for the responses of visual neurons. *Neuron* 30**,** 803-817.

Kelly, S.T., Kremkow, J., Jin, J., Wang, Y., Wang, Q., Alonso, J.-M., Stanley, G.B., 2014. The role of thalamic population synchrony in the emergence of cortical feature selectivity. *PLoS Comput Biol* 10**,** e1003418.

Kenyon, G.T., Theiler, J., George, J.S., Travis, B.J., Marshak, D.W., 2004. Correlated firing improves stimulus discrimination in a retinal model. *Neural Comput.* 16**,** 2261-2291.

Kepecs, A., Lisman, J., 2003. Information encoding and computation with spikes and bursts. *Network* 14**,** 103-118.

Khaligh-Razavi, S.-M., Kriegeskorte, N., 2014. Deep supervised, but not unsupervised, models may explain IT cortical representation. *PLoS Comput Biol* 10**,** e1003915.

Kheradpisheh, S.R., Ghodrati, M., Ganjtabesh, M., Masquelier, T., 2016a. Deep Networks Can Resemble Human Feed-forward Vision in Invariant Object Recognition. *Scientific Reports* 6**,** 32672.

Kheradpisheh, S.R., Ghodrati, M., Ganjtabesh, M., Masquelier, T., 2016b. Humans and Deep Networks Largely Agree on Which Kinds of Variation Make Object Recognition Harder. *Front. Comput. Neurosci.* 10**,** 92.

Kimura, A., 2014. Diverse subthreshold cross-modal sensory interactions in the thalamic reticular nucleus: implications for new pathways of cross-modal attentional gating function. *Eur. J. Neurosci.* 39**,** 1405-1418.

Kimura, A., Imbe, H., Donishi, T., Tamai, Y., 2007. Axonal projections of single auditory neurons in the thalamic reticular nucleus: implications for tonotopy-related gating function and cross-modal modulation. *Eur. J. Neurosci.* 26**,** 3524-3535.

Kimura, A., Shimegi, S., Hara, S.i., Okamoto, M., Sato, H., 2013. Role of GABAergic inhibition in shaping the spatial frequency tuning of neurons and its contrast dependency in the dorsal lateral geniculate nucleus of cat. *Eur. J. Neurosci.* 37**,** 1270-1783.

Kirkland, K.L., Gerstein, G.L., 1998. A model of cortically induced synchronization in the lateral geniculate nucleus of the cat: a role for low-threshold calcium channels. *Vision Res.* 38**,** 2007-2022.

Kirkland, K.L., Sillito, A.M., Jones, H.E., West, D.C., Gerstein, G.L., 2000. Oscillations and long-lasting correlations in a model of the lateral geniculate nucleus and visual cortex. *J. Neurophysiol.* 84**,** 1863-1868.

Koch, C., 1985. Understanding the intrinsic circuitry of the cat's lateral geniculate nucleus: electrical properties of the spine-triad arrangement. *Proc. R. Soc. Lond. B. Biol. Sci.* 225**,** 365-390.

Koch, C., Ullman, S., 1985. Shifts in selective visual attention: towards the underlying neural circuitry. *Hum. Neurobiol.* 4**,** 219-227.

Koenderink, J.J., van Doorn, A.J., 1990. Receptive field families. *Biol. Cybern.* 63**,** 291-297

Koller, D., Friedman, N., 2009. *Probabilistic Graphical Models: Principles and Techniques* MIT Press: Cambridge, MA.

Kondo, S., Ohki, K., 2016. Laminar differences in the orientation selectivity of geniculate afferents in mouse primary visual cortex. *Nat. Neurosci.* 19**,** 316-319.

Kosmidis, E.K., Vibert, J.-F., 2002. Feed-forward inhibition in the visual thalamus. *Neurocomputing* 44**,** 479 – 487.

Kosslyn, S.M., Ganis, G., Thompson, W.L., 2001. Neural foundations of imagery. *Nature Rev. Neurosci.* 2**,** 635-642.

Krahe, T.E., El-Danaf, R.N., Dilger, E.K., Henderson, S.C., Guido, W., 2011. Morphologically distinct classes of relay cells exhibit regional preferences in the dorsal lateral geniculate nucleus of the mouse. *J. Neurosci.* 31**,** 17437-17448.

Krauzlis, R.J., Lovejoy, L.P., Zénon, A., 2013. Superior colliculus and visual spatial attention. *Annu. Rev. Neurosci.* 36**,** 165-182.

Kremers, J., Weiss, S., 1997. Receptive field dimensions of lateral geniculate cells in the common marmoset (Callithrix jacchus). *Vision Res.* 37**,** 2171-2181.

Krizhevsky, A., Sutskever, I., Hinton, G.E. 2012. ImageNet Classification with Deep Convolutional Neural Networks. In: *Adv. Neural. Inf. Process. Syst.* pp. 1097-1105. Eds. F. Pereira, C.J.C. Burges, L. Bottou, K.Q. Weinberger.

Krüger, J., 1977. The shift-effect in the lateral geniculate body of the rhesus monkey. *Exp. Brain Res.* 29**,** 387-392.

Krukowski, A.E., Miller, K.D., 2001. Thalamocortical NMDA conductances and intracortical inhibition can explain cortical temporal tuning. *Nat. Neurosci.* 4**,** 424-430.

Kuhlmann, L., Vidyasagar, T.R., 2011. A computational study of how orientation bias in the lateral geniculate nucleus can give rise to orientation selectivity in primary visual cortex. *Front. Syst. Neurosci.* 5**,** 81.

Kumbhani, R.D., Nolt, M.J., Palmer, L.A., 2007. Precision, reliability, and information-theoretic analysis of visual thalamocortical neurons. *J. Neurophysiol.* 98**,** 2647-2663.

Lachica, E.A., Beck, P.D., Casagrande, V.A., 1992. Parallel pathways in macaque monkey striate cortex: anatomically defined columns in layer III. *Proc. Natl. Acad. Sci. U. S. A.* 89**,** 3566-3570.

Lachica, E.A., Casagrande, V.A., 1992. Direct W-like geniculate projections to the cytochrome oxidase (CO) blobs in primate visual cortex: axon morphology. *J. Comp. Neurol.* 319**,** 141-145.

Lajeunesse, F., Kröger, H., Timofeev, I., 2013. Regulation of AMPA and NMDA receptor-mediated EPSPs in dendritic trees of thalamocortical cells. *J. Neurophysiol.* 109**,** 13-30.

Lal, R., Friedlander, M.J., 1989. Gating of retinal transmission by afferent eye position and movement signals. *Science* 243**,** 93-96.

Lal, R., Friedlander, M.J., 1990. Effect of passive eye position changes on retinogeniculate transmission in the cat. *J. Neurophysiol.* 63**,** 502-522.

Lam, Y.-W., Nelson, C.S., Sherman, S.M., 2006. Mapping of the functional interconnections between thalamic reticular neurons using photostimulation. *J. Neurophysiol.* 96**,** 2593-2600.

Lam, Y.W., Sherman, S.M., 2011. Functional organization of the thalamic input to the thalamic reticular nucleus. *J. Neurosci.* 31**,** 6791-6799.

Landisman, C.E., Long, M., Beierlein, M., Deans, M.R., Paul, D.L., Connors, B.W., 2002. Electrical synapses in the thalamic reticular nucleus. *J. Neurosci.* 22**,** 1002-1009.

Latawiec, D., Martin, K.A., Meskenaite, V., 2000. Termination of the geniculocortical projection in the striate cortex of macaque monkey: a quantitative immunoelectron microscopic study. *J. Comp. Neurol.* 419**,** 306-319.

Laycock, R., Crewther, D.P., Crewther, S.G., 2008. The advantage in being magnocellular: a few more remarks on attention and the magnocellular system. *Neurosci. Biobehav. Rev.* 32**,** 1409-1415.

LeCun, Y., Bengio, Y. 1995. Convolutional networks for images, speech and time- series. In: *The Handbook of Brain Theory and Neural Networks*. pp. 255–258. Ed. M. Arbib. MIT Press: Cambridge, MA.

LeCun, Y., Bengio, Y., Hinton, G., 2015. Deep learning. *Nature* 521**,** 436-444.

LeCun, Y., Kavukcuoglu, K., Farabet, C. (2010) Convolutional networks and applications in vision. In: *IEEE International Symposium on Circuits and Systems (ISCAS)*. pp. 253–256: Paris.

Lee, B., Virsu, V., Elepfandt, A., 1983a. Cell responses in dorsal layers of macaque lateral geniculate nucleus as a function of intensity and wavelength. *J. Neurophysiol.* 50**,** 849-863.

Lee, B.B., Virsu, V., Creutzfeldt, O.D., 1983b. Linear signal transmission from prepotentials to cells in the macaque lateral geniculate nucleus. *Exp. Brain Res.* 52**,** 50-56.

Lee, D., Malpeli, J.G., 1998. Effects of saccades on the activity of neurons in the cat lateral geniculate nucleus. *J. Neurophysiol.* 79**,** 922-936.

Lee, H., Grosse, R., Ranganath, R., Ng, A.Y., 2011. Unsupervised learning of hierarchical representations with convolutional deep belief networks. *Communications of the ACM* 54**,** 95-103.

Lee, S.-H., Blake, R., 2002. V1 activity is reduced during binocular rivalry. *J. Vision* 2**,** 618-626.

Lee, S.-H., Blake, R., Heeger, D.J., 2005. Traveling waves of activity in primary visual cortex during binocular rivalry. *Nat. Neurosci.* 8**,** 22-23.

Legéndy, C.R., Salcman, M., 1985. Bursts and recurrences of bursts in the spike trains of spontaneously active striate cortex neurons. *J. Neurophysiol.* 53**,** 926-939.

Lehky, S.R., Maunsell, J.H.R., 1996. No binocular rivalry in LGN of alert macaque monkeys. *Vision Res.* 36**,** 1225-1234.

Lehky, S.R., Tanaka, K., 2016. Neural representation for object recognition in inferotemporal cortex. *Curr. Opin. Neurobiol.* 37**,** 23-35.

Leopold, D.A., 2012. Primary visual cortex: awareness and blindsight. *Annu. Rev. Neurosci.* 35**,** 91-109.

Leopold, D.A., Logothetis, N.K., 1996. Activity changes in early visual cortex reflect monkeys' percepts during binocular rivalry. *Nature* 379**,** 549-553.

Lesica, N.A., Jin, J., Weng, C., Yeh, C.-I., Butts, D.A., Stanley, G.B., Alonso, J.-M., 2007. Adaptation to stimulus contrast and correlations during natural visual stimulation. *Neuron* 55**,** 479-491.

Lesica, N.A., Stanley, G.B., 2004. Encoding of natural scene movies by tonic and burst spikes in the lateral geniculate nucleus. *J. Neurosci.* 24**,** 10731-10740.

Lesica, N.A., Weng, C., Jin, J., Yeh, C.-I., Alonso, J.-M., Stanley, G.B., 2006. Dynamic encoding of natural luminance sequences by LGN bursts. *PLOS Biology* 4**,** e209.

LeVay, S., Ferster, D., 1979. Proportion of interneurons in the cat's lateral geniculate nucleus. *Brain Res.* 164**,** 304-308.

LeVay, S., Gilbert, C.D., 1976. Laminar patterns of geniculocortical projection in the cat. *Brain Res.* 113**,** 1-19.

Leventhal, A.G., Rodieck, R.W., Dreher, B., 1981. Retinal ganglion cell classes in the Old World monkey: morphology and central projections. *Science* 213**,** 1139-1142.

Levick, W.R., Cleland, B.G., Dubin, M.W., 1972. Lateral geniculate neurons of cat: retinal inputs and physiology. *Invest. Ophthalmol.* 11**,** 302-311.

Levine, M.W., Troy, J.B., 1986. The variability of the maintained discharge of cat dorsal lateral geniculate cells. *J. Physiol. (Lond).* 375**,** 339-359.

Levitt, J.B., Schumer, R.A., Sherman, S.M., Spear, P.D., Movshon, J., 2001. Visual response properties of neurons in the LGN of normally reared and visually deprived macaque monkeys. *J. Neurophysiol.* 85**,** 2111-2129.

Li, B., Freeman, R.D., 2007. High-resolution neurometabolic coupling in the lateral geniculate nucleus. *J. Neurosci.* 27**,** 10223-10229.

Li, B., Freeman, R.D., 2012. Spatial summation of neurometabolic coupling in the central visual pathway. *Neuroscience* 213**,** 112-121.

Li, Y.-T., Ibrahim, L.A., Liu, B.-H., Zhang, L.I., Tao, H.W., 2013. Linear transformation of thalamocortical input by intracortical excitation. *Nat. Neurosci.* 16**,** 1324-1330.

Lien, A.D., Scanziani, M., 2013. Tuned thalamic excitation is amplified by visual cortical circuits. *Nat. Neurosci.* 6**,** 1315-1323.

Lin , C.S., Kaas, J.H., 1977. Projections from cortical visual areas 17, 18, and MT onto the dorsal lateral geniculate nucleus in owl monkeys. *J. Comp. Neurol.* 173**,** 457-474.

Lin, I.-C., Xing, D., Shapley, R., 2012. Integrate-and-fire vs Poisson models of LGN input to V1 cortex: noisier inputs reduce orientation selectivity. *J. Comput. Neurosci.* 33**,** 559-572.

Lindeberg, T., 2013. A computational theory of visual receptive fields. *Biol. Cybern.* 107**,** 589-635.

Lindeberg, T., 2016. Time-causal and time-recursive spatio-temporal receptive fields. *J. Math. Imaging. Vis.* 55**,** 50–88.

Lindsley, D.F., Chow, K.L., Gollender, M., 1967. Dichoptic interactions of lateral geniculate neurons of cats to contralateral and ipsilateral eye stimulation. *J. Neurophysiol.* 30**,** 628-644.

Lindström, S., Wróbel, A., 1990a. Frequency dependent corticofugal excitation of principal cells in the cat's dorsal lateral geniculate nucleus. *Exp. Brain Res.* 79**,** 313-318.

Lindström, S., Wróbel, A., 1990b. Intracellular recordings from binocularly activated cells in the cat's dorsal lateral geniculate nucleus. *Acta Neurobiol. Exp. (Wars).* 50**,** 61-70.

Lindström, S., Wróbel, A., 1990c. Private inhibitory systems for the X and Y pathways in the dorsal lateral geniculate nucleus of the cat. *J. Physiol. (Lond).* 429**,** 259-280.

Lindström, S., Wróbel, A., 2011. Feedforward and recurrent inhibitory receptive fields of principal cells in the cat's dorsal lateral geniculate nucleus. *Pflugers Arch. Gesamte Physiol. Menschen Tiere* 461**,** 277-294.

Ling, S., Pratte, M.S., Tong, F., 2015. Attention alters orientation processing in the human lateral geniculate nucleus. *Nat. Neurosci.* 18**,** 496-498.

Lisman, J.E., 1997. Bursts as a unit of neural information: making unreliable synapses reliable. *Trends Neurosci.* 20**,** 38-43.

Livingstone, M., Hubel, D., 1988. Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science* 240**,** 740-749.

Livingstone, M.S., Hubel, D.H., 1981. Effects of sleep and arousal on the processing of visual information in the cat. *Nature* 291**,** 554-561.

Llinás, R., Ribary, U., 1993. Coherent 40-Hz oscillation characterizes dream state in humans. *Proc. Natl. Acad. Sci. U. S. A.* 90**,** 2078-2081.

Llinás, R., Ribary, U., Contreras, D., Pedroarena, C., 1998. The neuronal basis for consciousness. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 353**,** 1841-1849.

Llinás, R.R., 2014. Intrinsic electrical properties of mammalian neurons and CNS function: a historical perspective. *Frontiers in Cellular Neuroscience* 8**,** 320.

Llinás, R.R., Paré, D., 1991. Of dreaming and wakefulness. *Neuroscience* 44**,** 521-535.

Lo, F.-S., 1988. A study of neuronal circuitry mediating the saccadic suppression in the rabbit. *Exp. Brain Res.* 71**,** 618-622.

Lo, F.-S., Lu, S.-M., Sherman, S.M., 1991. Intracellular and extracellular in vivo recording of different response modes for relay cells of the cat's lateral geniculate nucleus. *Exp. Brain Res.* 83**,** 317-328.

Lo, F.-S., Sherman, S.M., 1994. Feedback inhibition in the cat's lateral geniculate nucleus. *Exp. Brain Res.* 100**,** 365-368.

Logothetis, N.K., 1998. Single units and conscious vision. *Proc. R. Soc. Lond. B. Biol. Sci.* 353**,** 1801-1818.

Logothetis, N.K., Pauls, J., Augath, M., Trinath, T., Oeltermann, A., 2001. Neurophysiological investigation of the basis of the fMRI signal. *Nature* 412**,** 150-157.

Logothetis, N.K., Schall, J.D., 1989. Neuronal correlates of subjective visual perception. *Science* 245**,** 761-763.

London, M., Häusser, M., 2005. Dendritic computation. *Annu. Rev. Neurosci.* 28**,** 503-532.

Lozsádi, D.A., Gonzalez-Soriano, J., Guillery, R.W., 1996. The course and termination of corticothalamic fibres arising in the visual cortex of the rat. *Eur. J. Neurosci.* 8**,** 2416-2427.

Lu, S.-m., Guido, W., Sherman, S.M., 1993. The brain-stem parabrachial region controls mode of response to visual stimulation of neurons in the cat's lateral geniculate nucleus. *Vis. Neurosci.* 10**,** 631-642.

Lumer, E.D., Edelman, G.M., Tononi, G., 1997. Neural dynamics in a model of the thalamocortical system. I. Layers, loops and the emergence of fast synchronous rhythms. *Cereb. Cortex* 7**,** 207-227.

Lumer, E.D., Friston, K.J., Rees, G., 1998. Neural correlates of perceptual rivalry in the human brain. *Science* 280**,** 1930-1934.

Lund, J.S., Lund, R.D., Hendrickson, A.E., Bunt, A.H., Fuchs, A.F., 1975. The origin of efferent pathways from the primary visual cortex, area 17, of the macaque monkey as shown by retrograde transport of horseradish peroxidase. *J. Comp. Neurol.* 164**,** 287-303.

Lyon, D.C., Rabideau, C., 2012. Lack of robust LGN label following transneuronal rabies virus injections into macaque area V4. *J. Comp. Neurol.* 520**,** 2500-2511.

Madarász, M., Somogyi, G., Somogyi, J., Hámori, J., 1985. Numerical estimation of gamma-aminobutyric acid (GABA)-containing neurons in three thalamic nuclei of the cat: direct GABA immunocytochemistry. *Neurosci. Lett.* 61**,** 73-78.

Maffei, L., Fiorentini, A., 1973. The visual cortex as a spatial frequency analyser. *Vision Res.* 13**,** 1255-1267.

Magri, C., Schridde, U., Murayama, Y., Panzeri, S., Logothetis, N.K., 2012. The amplitude and timing of the BOLD signal reflects the relationship between local field potential power at different frequencies. *J. Neurosci.* 32**,** 1395-1407.

Mahmoodi, S., 2016. Linear Neural Circuitry Model for Visual Receptive Fields. *J. Math. Imaging. Vis.* 54**,** 138–161.

Malpeli, J.G., Baker, F.H., 1975. The representation of the visual field in the lateral geniculate nucleus of Macaca mulatta. *J. Comp. Neurol.* 161**,** 569-594.

Malpeli, J.G., Lee, D., Baker, F.H., 1996. Laminar and retinotopic organization of the macaque lateral geniculate nucleus: magnocellular and parvocellular magnification functions. *J. Comp. Neurol.* 375**,** 363-377.

Mante, V., Bonin, V., Carandini, M., 2008. Functional mechanisms shaping lateral geniculate responses to artificial and natural stimuli. *Neuron* 58**,** 625-638.

Mante, V., Frazor, R.A., Bonin, V., Geisler, W.S., Carandini, M., 2005. Independence of luminance and contrast in natural scenes and in the early visual system. *Nat. Neurosci.* 8**,** 1690-1697.

Marchiafava, P., 1966. Binocular reciprocal interaction upon optic fiber endings in the lateral geniculate nucleus of the cat. *Brain Res.* 2**,** 188-192.

Markov, N.T., Misery, P., Falchier, A., Lamy, C., Vezoli, J., Quilodran, R., Gariel, M.A., Giroud, P., Ercsey-Ravasz, M., Pilaz, L.J., Huissoud, C., Barone, P., Dehay, C., Toroczkai, Z., Van Essen, D.C., Kennedy, H., Knoblauch, K., 2011. Weight Consistency Specifies Regularities of Macaque Cortical Networks. *Cerebal Cortex* 21**,** 1254-1272.

Marr, D., 1982. *Vision: A Computational Investigation into the Human Representation and Processing of Visual Information*. MIT Press: Cambridge, MA.

Marr, D., Hildreth, E., 1980. Theory of edge detection. *Proc. R. Soc. Lond. B. Biol. Sci.* 207**,** 187-217.

Marrocco, R.T., McClurkin, J.W., 1979. Binocular interaction in the lateral geniculate nucleus of the monkey. *Brain Res.* 168**,** 633-637.

Marrocco, R.T., McClurkin, J.W., Young, R.A., 1982. Modulation of lateral geniculate nucleus cell responsiveness by visual activation of the corticogeniculate pathway. *J. Neurosci.* 2**,** 256-263.

Marshel, J.H., Kaye, A., Nauhaus, I., Callaway, E.M., 2012. Anterior-posterior direction opponency in the superficial mouse lateral geniculate nucleus. *Neuron* 76**,** 713-720.

Martin, P.R., 2004. Colour through the thalamus. *Clinical and Experimental Optometry* 87**,** 249-257.

Martin, P.R., Blessing, E.M., Buzás, P., Szmajda, B.A., Forte, J.D., 2011. Transmission of colour and acuity signals by parvocellular cells in marmoset monkeys. *J. Physiol. (Lond).* 589**,** 2795-2812.

Martin, P.R., Lee, B.B., 2014. Distribution and specificity of S-cone ("blue cone") signals in subcortical visual pathways. *Vis. Neurosci.* 20**,** 1-11.

Martin, P.R., White, A.J., Goodchild, A.K., Wilder, H.D., Sefton, A.E., 1997. Evidence that blue-on cells are part of the third geniculocortical pathway in primates. *Eur. J. Neurosci.* 9**,** 1536-1541.

Martinez, L.M., Molano-Mazón, M., Wang, X., Sommer, F.T., Hirsch, J.A., 2014. Statistical wiring of thalamic receptive fields optimizes spatial sampling of the retinal image. *Neuron* 81**,** 943-956.

Martuzzi, R., Murray, M.M., Meuli, R.A., Thiran, J.-P., Maeder, P.P., Michel, C.M., Grave de Peralta Menendez, R., Gonzalez Andino, S.L., 2009. Methods for determining frequency- and region-dependent relationships between estimated LFPs and BOLD responses in humans. *J. Neurophysiol.* 101**,** 491-502.

Mashour, G.A., Alkire, M.T., 2013. Consciousness, anesthesia, and the thalamocortical system. *Anesthesiology* 118**,** 13-15.

Masland, R.H., 2012. The neuronal organization of the retina. *Neuron* 76**,** 266-280.

Masquelier, T.e., 2012. Relative spike time coding and STDP-based orientation selectivity in the early visual system in natural continuous and saccadic vision: a computational model. *J. Comput. Neurosci.* 32**,** 425-441.

Masquelier, T.e., 2013. Neural variability, or lack thereof. *Front. Comput. Neurosci.* 7**,** 7.

Mastronarde, D.N., 1987. Two classes of single-input X-cells in cat lateral geniculate nucleus. I. Receptive-field properties and classification of cells. *J. Neurophysiol.* 57**,** 357-380.

Mastronarde, D.N., 1992. Nonlagged relay cells and interneurons in the cat lateral geniculate nucleus: receptive-field properties and retinal inputs. *Vis. Neurosci.* 8**,** 407-441.

Mastronarde, D.N., Humphrey, A.L., Saul, A.B., 1991. Lagged Y cells in the cat lateral geniculate nucleus. *Vis. Neurosci.* 7**,** 191-200.

Maunsell, J.H.R., Ghose, G.M., Assad, J.A., McAdams, C.J., Boudreau, C.E., Noerager, B.D., 1999. Visual response latencies of magnocellular and parvocellular LGN neurons in macaque monkeys. *Vis. Neurosci.* 16**,** 1-14.

May, P.J., 2006. The mammalian superior colliculus: laminar structure and connections. *Prog. Brain Res.* 151**,** 321-378.

Maybeck, P.S., 1979. *Stochastic models, estimation, and control*. Academic Press: New York.

Mazurek, M.E., Shadlen, M.N., 2002. Limits to the temporal fidelity of cortical spike rate signals. *Nat. Neurosci.* 5**,** 463-471.

McAlonan, K., Brown, V.J., Bowman, E.M., 2000. Thalamic reticular nucleus activation reflects attentional gating during classical conditioning. *J. Neurosci.* 20**,** 8897-8901.

McAlonan, K., Cavanaugh, J., Wurtz, R.H., 2006. Attentional modulation of thalamic reticular neurons. *J. Neurosci.* 26**,** 4444-4450.

McAlonan, K., Cavanaugh, J., Wurtz, R.H., 2008. Guarding the gateway to cortex with attention in visual thalamus. *Nature* 456**,** 391-394.

McClurkin, J.W., Gawne, T.J., Optican, L.M., Richmond, B.J., 1991a. Lateral geniculate neurons in behaving primates. II. Encoding of visual information in the temporal shape of the response. *J. Neurophysiol.* 66**,** 794-808.

McClurkin, J.W., Gawne, T.J., Richmond, B.J., Optican, L.M., Robinson, D.L., 1991b. Lateral geniculate neurons in behaving primates. I. Responses to two-dimensional stimuli. *J. Neurophysiol.* 66**,** 777-793.

McClurkin, J.W., Marrocco, R.T., 1984. Visual cortical input alters spatial tuning in monkey lateral geniculate nucleus cells. *J. Physiol. (Lond).* 348**,** 135-152.

McCormick, D.A., 1989. Cholinergic and noradrenergic modulation of thalamocortical processing. *Trends Neurosci.* 12**,** 215-221.

McCormick, D.A., Bal, T., 1997. Sleep and arousal: thalamocortical mechanisms. *Annu. Rev. Neurosci.* 20**,** 185-215.

McCormick, D.A., Huguenard, J.R., 1992. A model of the electrophysiological properties of thalamocortical relay neurons. *J. Neurophysiol.* 68**,** 1384-1400.

McCormick, D.A., McGinley, M.J., Salkoff, D.B., 2015. Brain state dependent activity in the cortex and thalamus. *Curr. Opin. Neurobiol.* 31**,** 133-140.

McCormick, D.A., Prince, D.A., 1987. Actions of acetylcholine in the guinea-pig and cat medial and lateral geniculate nuclei, in vitro. *J. Physiol. (Lond).* 392**,** 147-165.

McCormick, D.A., von Krosigk, M., 1992. Corticothalamic activation modulates thalamic firing through glutamate "metabotropic" receptors. *Proc. Natl. Acad. Sci. U. S. A.* 89**,** 2774-2778.

McDonnell, M.D., Abbott, D., 2009. What is stochastic resonance? Definitions, misconceptions, debates, and its relevance to biology. *PLoS Comput Biol* 5**,** e1000348.

McFarland, J.M., Bondy, A.G., Saunders, R.C., Cumming, B.G., Butts, D.A., 2015. Saccadic modulation of stimulus processing in primary visual cortex. *Nat. Commun.* 6**,** 8110.

McIlwain, J.T., 1995. Lateral geniculate lamination and the corticogeniculate projection: a potential role in binocular vision in the quadrants. *J. Theor. Biol.* 172**,** 329-333.

McMullen, T., Ly, N., 1988. Model of oscillatory activity in thalamic neurons: role of voltage- and calcium-dependent ionic conductances. *Biol. Cybern.* 58**,** 243-259.

Mehta, A.D., Ulbert, I., Schroeder, C.E., 2000a. Intermodal selective attention in monkeys. I: distribution and timing of effects across visual areas. *Cereb. Cortex* 10**,** 343-358.

Mehta, A.D., Ulbert, I., Schroeder, C.E., 2000b. Intermodal selective attention in monkeys. II: physiological mechanisms of modulation. *Cereb. Cortex* 10**,** 359-370.

Meister, M., Berry II, M.J., 1999. The neural code of the retina. *Neuron* 22**,** 435-450.

Meister, M., Lagnado, L., Baylor, D.A., 1995. Concerted signaling by retinal ganglion cells. *Science* 270**,** 1207-1210.

Mel, B.W., 1994. Information Processing in Dendritic Trees. *Neural Comput.* 6**,** 1031-1085.

Merker, B., 2007. Consciousness without a cerebral cortex: a challenge for neuroscience and medicine. *Behav. Brain Sci.* 30**,** 63-81.

Michalareas, G., Vezoli, J., van Pelt, S., Schoffelen, J.M., Kennedy, H., Fries, P., 2016. Alpha-Beta and Gamma Rhythms Subserve Feedback and Feedforward Influences among Human Visual Cortical Areas. *Neuron* 89**,** 384-397.

Miller, E.K., 2000. The prefrontal cortex and cognitive control. *Nature Rev. Neurosci.* 1**,** 59-65.

Miller, E.K., Buschman, T.J., 2012. Cortical circuits for the control of attention. *Curr. Opin. Neurobiol.* 23**,** 216-222.

Miller, R., 1996. Cortico-thalamic interplay and the security of operation of neural assemblies and temporal chains in the cerebral cortex. *Biol. Cybern.* 75**,** 263-275.

Min, B.-K., 2010. A thalamic reticular networking model of consciousness. *Theor. Biol. Med. Model.* 7**,** 10.

Mollon, J.D., 1989. "Tho' she kneel'd in that place where they grew..." The uses and origins of primate colour vision. *J. Exp. Biol.* 146**,** 21-38.

Molotchnikoff, S., Casanova, C., 1985. Reactions of the geniculate cells to extraocular proprioceptive activation in rabbits. *J. Neurosci. Res.* 14**,** 105-115.

Montague, P.R., Dayan, P., Sejnowski, T.J., 1996. A framework for mesencephalic dopamine systems based on predictive Hebbian learning. *J. Neurosci.* 16**,** 1936-1947.

Montero, V.M., 1991. A quantitative study of synaptic contacts on interneurons and relay cells of the cat lateral geniculate nucleus. *Exp. Brain Res.* 86**,** 257-270.

Montero, V.M., Guillery, R.W., Woolsey, C.N., 1977. Retinotopic organization within the thalamic reticular nucleus demonstrated by a double label autoradiographic technique. *Brain Res.* 138**,** 407-421.

Moore, B.D.t., Rathbun, D.L., Usrey, W.M., Freeman, R.D., 2014. Spatiotemporal flow of information in the early visual pathway. *Eur. J. Neurosci.* 39**,** 593-601.

Moore, T., Armstrong, K.M., Fallah, M., 2003. Visuomotor origins of covert spatial attention. *Neuron* 40**,** 671-683.

Morgan, J.L., Berger, D.R., Wetzel, A.W., Lichtman, J.W., 2016. The Fuzzy Logic of Network Connectivity in Mouse Visual Thalamus. *Cell* 165**,** 192-206.

Morrison, J.H., Foote, S.L., 1986. Noradrenergic and serotoninergic innervation of cortical, thalamic, and tectal visual structures in Old and New World monkeys. *J. Comp. Neurol.* 243**,** 117-138.

Mukherjee, P., Kaplan, E., 1995. Dynamics of neurons in the cat lateral geniculate nucleus: in vivo electrophysiology and computational modeling. *J. Neurophysiol.* 74**,** 1222-1243.

Mulle, C., Madariaga, A., Deschênes, M., 1986. Morphology and electrophysiological properties of reticularis thalami neurons in cat: in vivo study of a thalamic pacemaker. *J. Neurosci.* 6**,** 2134-2145.

Mumford, D., 1991. On the computational architecture of the neocortex. I. The role of the thalamo-cortical loop. *Biol. Cybern.* 65**,** 135-145.

Mumford, D. 1994. Neuronal architectures for pattern-theoretic problems. In: *Large scale theories of the cortex*. pp. 125-152. Eds. C. Koch, J.L. Davis. MIT Press: Cambridge, MA.

Murphy, P.C., Duckett, S.G., Sillito, A.M., 1999. Feedback connections to the lateral geniculate nucleus and cortical response properties. *Science* 286**,** 1552-1554.

Murphy, P.C., Sillito, A.M., 1987. Corticofugal feedback influences the generation of length tuning in the visual pathway. *Nature* 329**,** 727-729.

Murphy, P.C., Sillito, A.M., 1989. The binocular input to cells in the feline dorsal lateral geniculate nucleus (dLGN). *J. Physiol. (Lond).* 415**,** 393-408.

Murphy, P.C., Sillito, A.M., 1996. Functional morphology of the feedback pathway from area 17 of the cat visual cortex to the lateral geniculate nucleus. *J. Neurosci.* 16**,** 1180-1192.

Musial, P., Panecki, S., Gerstein, G.L., Wróbel, A., 1996. Nonlinearities within the cat LGN cell receptive fields in simulated network with recurrent inhibition. *Acta Neurobiol. Exp. (Wars).* 56**,** 927-942.

Naito, T., Okamoto, M., Sadakane, O., Shimegi, S., Osaki, H., Hara, S., Kimura, A., Ishikawa, A., Suematsu, N., Sato, H., 2013. Effects of stimulus spatial frequency, size, and luminance contrast on orientation tuning of neurons in the dorsal lateral geniculate nucleus of cat. *Neurosci. Res.* 77**,** 143-154.

Nassi, J.J., Lyon, D.C., Callaway, E.M., 2006. The parvocellular LGN provides a robust disynaptic input to the visual motion area MT. *Neuron* 50**,** 319-327.

Neuenschwander, S., Singer, W., 1996. Long-range synchronization of oscillatory light responses in the cat retina and lateral geniculate nucleus. *Nature* 379**,** 728-732.

Newman, J., Baars, B.J., Cho, S.-B., 1997. A Neural Global Workspace Model for Conscious Attention. *Neural Networks* 10**,** 1195-1206.

Nicolelis, M.A., 2005. Computing with thalamocortical ensembles during different behavioural states. *J. Physiol. (Lond).* 566**,** 37-47.

Niell, C.M., 2013. Vision: more than expected in the early visual system. *Curr. Biol.* 23**,** R681-R684.

Nirody, J.A., 2014. Development of spatial coarse-to-fine processing in the visual pathway. *J. Comput. Neurosci.* 36**,** 401-414.

Nobre, A.C., Gitelman, D.R., Dias, E.C., Mesulam, M.M., 2000. Covert visual spatial orienting and saccades: overlapping neural systems. *Neuroimage* 11**,** 210-216.

Noda, H., Tamaki, Y., Iwama, K., 1972. Binocular units in the lateral geniculate nucleus of chronic cats. *Brain Res.* 41**,** 81-99.

Nolt, M.J., Kumbhani, R.D., Palmer, L.A., 2004. Contrast-dependent spatial summation in the lateral geniculate nucleus and retina of the cat. *J. Neurophysiol.* 92**,** 1708-1717.

Nolt, M.J., Kumbhani, R.D., Palmer, L.A., 2007. Suppression at high spatial frequencies in the lateral geniculate nucleus of the cat. *J. Neurophysiol.* 98**,** 1167-1180.

Norheim, E.S., Wyller, J., Nordlie, E., Einevoll, G.T., 2012. A minimal mechanistic model for temporal signal processing in the lateral geniculate nucleus. *Cogn. Neurodyn.* 6**,** 259-281.

Northcutt, R.G., 2011. Do teleost fishes possess a homolog of mammalian isocortex? *Brain. Behav. Evol.* 78**,** 136-138.

O'Connor, D.H., Fukui, M.M., Pinsk, M.A., Kastner, S., 2002. Attention modulates responses in the human lateral geniculate nucleus. *Nat. Neurosci.* 5**,** 1203-1209.

O'Keefe, L.P., Levitt, J.B., Kiper, D.C., Shapley, R.M., Movshon, J.A., 1998. Functional organization of owl monkey lateral geniculate nucleus and visual cortex. *J. Neurophysiol.* 80**,** 594-609.

Ohara, P.T., Lieberman, A.R., 1981. Thalamic reticular nucleus: anatomical evidence that cortico-reticular axons establish monosynaptic contact with reticulo-geniculate projection cells. *Brain Res.* 207**,** 153-156.

Olsen, S.R., Bortone, D.S., Adesnik, H., Scanziani, M., 2012. Gain control by layer six in cortical circuits of vision. *Nature* 483**,** 47-52.

Olson, M., 2012. The developmental renaissance in adaptationism. *Trends Ecol. Evol. (Amst.)* 27**,** 278-287.

Ortuño, T., Grieve, K.L., Cao, R., Cudeiro, J., Rivadulla, C., 2014. Bursting thalamic responses in awake monkey contribute to visual detection and are modulated by corticofugal feedback. *Front. Behav. Neurosci.* 8**,** 198.

Oswald, A.M., Chacron, M.J., Doiron, B., Bastian, J., Maler, L., 2004. Parallel processing of sensory input by bursts and isolated spikes. *J. Neurosci.* 24**,** 4351-4362.

Ozeki, H., Sadakane, O., Akasaki, T., Naito, T., Shimegi, S., Sato, H., 2004. Relationship between excitation and inhibition underlying size tuning and contextual response modulation in the cat primary visual cortex. *J. Neurosci.* 24**,** 1428-1438.

Pape, H., Eysel, U., 1986. Binocular interactions in the lateral geniculate nucleus of the cat: GABAergic inhibition reduced by dominant afferent activity. *Exp. Brain Res.* 1**,** 265-271.

Peelen, M.V., Kastner, S., 2014. Attention in the real world: toward understanding its neural basis. *Trends Cogn. Sci.* 18**,** 242-250.

Perreault, M.C., Raastad, M., 2006. Contribution of morphology and membrane resistance to integration of fast synaptic signals in two thalamic cell types. *J. Physiol. (Lond).* 577**,** 205-220.

Perry, V.H., Oehler, R., Cowey, A., 1984. Retinal ganglion cells that project to the dorsal lateral geniculate nucleus in the macaque monkey. *Neuroscience* 12**,** 1101-1123.

Pessoa, L., Kastner, S., Ungerleider, L.G., 2003. Neuroimaging studies of attention: from modulation of sensory processing to top-down control. *J. Neurosci.* 23**,** 3990-3998.

Peters, A., Payne, B.R., 1993. Numerical relationships between geniculocortical afferents and pyramidal cell modules in cat primary visual cortex. *Cereb. Cortex* 3**,** 69-78.

Peters, A., Payne, B.R., Budd, J., 1994. A numerical analysis of the geniculocortical input to striate cortex in the monkey. *Cereb. Cortex* 4**,** 215-229.

Peterson, M.R., Li, B., Freeman, R.D., 2004. The derivation of direction selectivity in the striate cortex. *J. Neurosci.* 24**,** 3583-3591.

Petrof, I., Brown, V.J., 2010. Attention to visual, but not tactile, properties of a stimulus results in activation of FOS protein in the visual thalamic reticular nucleus of rats. *Behav. Brain Res.* 211**,** 248-252.

Pietersen, A.N., Cheong, S.K., Solomon, S.G., Tailby, C., Martin, P.R., 2014. Temporal response properties of koniocellular (blue-on and blue-off) cells in marmoset lateral geniculate nucleus. *J. Neurophysiol.* 112**,** 1421–1438.

Pin, J.-P., Duvoisin, R., 1995. The metabotropic glutamate receptors: structure and functions. *Neuropharmacology* 34**,** 1-26.

Pinault, D., 2004. The thalamic reticular nucleus: structure, function and concept. *Brain. Res. Rev.* 46**,** 1-31.

Pinault, D., 2011. Dysfunctional thalamus-related networks in schizophrenia. *Schizophr. Bull.* 37**,** 238-243.

Pinault, D., Deschênes, M., 1998. Anatomical evidence for a mechanism of lateral inhibition in the rat thalamus. *Eur. J. Neurosci.* 10**,** 3462-3469.

Piscopo, D.M., El-Danaf, R.N., Huberman, A.D., Niell, C.M., 2013. Diverse visual features encoded in mouse lateral geniculate nucleus. *J. Neurosci.* 33**,** 4642-4656.

Podvigin, N.F., Jokeit, H., Pöppel, E., Chizh, A.N., Kiselyeva, N.B., 1992. Stimulus-dependent oscillatory activity in the lateral geniculate body of the cat. *Naturwissenschaften* 79**,** 428-431.

Podvigin, N.F., Poeppel, E., Kiseleva, N.B., Kozlov, I.V., Vershinina, E.A., Granstrem, M.P., 2001. The sensitivity of neurons in the lateral geniculate body of the cat to the orientation vectors of brightness gradients. *Neurosci. Behav. Physiol.* 31**,** 657-668.

Polonsky, A., Blake, R., Braun, J., Heeger, D.J., 2000. Neuronal activity in human primary visual cortex correlates with perception during binocular rivalry. *Nat. Neurosci.* 3**,** 1153-1159.

Posner, M.I., 1980. Orienting of attention. *Q. J. Exp. Psychol.* 32**,** 3-25.

Price, D.J., Morgan, J.E., 1987. Spatial properties of neurones in the lateral geniculate nucleus of the pigmented ferret. *Exp. Brain Res.* 68**,** 28-36.

Priebe, N.J., Ferster, D., 2006. Mechanisms underlying cross-orientation suppression in cat visual cortex. *Nat. Neurosci.* 9**,** 552-561.

Priebe, N.J., Ferster, D., 2012. Mechanisms of neuronal computation in mammalian visual cortex. *Neuron* 75**,** 194-208.

Przybyszewski, A.W., Gaska, J.P., Foote, W., Pollen, D.A., 2000. Striate cortex increases contrast gain of macaque LGN neurons. *Vis. Neurosci.* 17**,** 485-494.

Purushothaman, G., Chen, X., Yampolsky, D., Casagrande, V.A., 2014. Neural mechanisms of coarse-to-fine discrimination in the visual cortex. *J. Neurophysiol.* 112**,** 2822-2833.

Rajaei, K., Khaligh-Razavi, S.-M., Ghodrati, M., Ebrahimpour, R., Shiri Ahmad Abadi, M.E., 2012. A stable biologically motivated learning mechanism for visual feature extraction to handle facial categorization. *PLoS ONE* 7**,** e38478.

Ralston, H.J.r., 1971. Evidence for presynaptic dendrites and a proposal for their mechanism of action. *Nature* 230**,** 585-587.

Ramcharan, E.J., Gnadt, J.W., Sherman, S.M., 2000. Burst and tonic firing in thalamic cells of unanesthetized, behaving monkeys. *Vis. Neurosci.* 17**,** 55-62.

Ramcharan, E.J., Gnadt, J.W., Sherman, S.M., 2005. Higher-order thalamic relays burst more than first-order relays. *Proc. Natl. Acad. Sci. U. S. A.* 102**,** 12236-12241.

Rao, R.P.N., Ballard, D.H., 1997. Dynamic model of visual recognition predicts neural response properties in the visual cortex. *Neural Comput.* 9**,** 721-763.

Rathbun, D.L., Alitto, H.J., Warland, D.K., Usrey, W.M., 2016. Stimulus Contrast and Retinogeniculate Signal Processing. *Front. Neural Circuits* 10**,** 8.

Rathbun, D.L., Warland, D.K., Usrey, W.M., 2010. Spike timing and information transmission at retinogeniculate synapses. *J. Neurosci.* 30**,** 13558-13566.

Ray, S., Maunsell, J.H., 2015. Do gamma oscillations play a role in cerebral cortex? *Trends Cogn. Sci.* 19**,** 78-85.

Reese, B.E., 1984. The projection from the superior colliculus to the dorsal lateral geniculate nucleus in the rat. *Brain Res.* 305**,** 162-168.

Reese, B.E., 1988. 'Hidden lamination' in the dorsal lateral geniculate nucleus: the functional organization of this thalamic region in the rat. *Brain Res.* 472**,** 119-137.

Reich, D.S., Victor, J.D., Knight, B.W., Ozaki, T., Kaplan, E., 1997. Response variability and timing precision of neuronal spike trains in vivo. *J. Neurophysiol.* 77**,** 2836-2841.

Reichardt , W. 1961. Autocorrelation, a Principle for the Evaluation of Sensory Information by the Central Nervous System. In: *Sensory Communication*. pp. 303-317. Ed. W.A. Rosenblith. MIT Press: Cambridge, MA.

Reid, R., Shapley, R., 2002. Space and time maps of cone photoreceptor signals in macaque lateral geniculate nucleus. *J. Neurosci.* 22**,** 6158-6175.

Reid, R.C., Victor, J.D., Shapley, R.M., 1997. The use of m-sequences in the analysis of visual neurons: linear receptive field properties. *Vis. Neurosci.* 14**,** 1015-1027.

Reinagel, P., Godwin, D., Sherman, S.M., Koch, C., 1999. Encoding of visual information by LGN bursts. *J. Neurophysiol.* 81**,** 2558-2569.

Reinagel, P., Reid, R.C., 2000. Temporal coding of visual information in the thalamus. *J. Neurosci.* 20**,** 5392-5400.

Reinagel, P., Reid, R.C., 2002. Precise firing events are conserved across neurons. *J. Neurosci.* 22**,** 6837-6841.

Reinker, S., Puil, E., Miura, R.M., 2003. Resonances and noise in a stochastic Hindmarsh-Rose model of thalamic neurons. *Bull. Math. Biol.* 65**,** 641-663.

Reppas, J.B., Usrey, W.M., Reid, R.C., 2002. Saccadic eye movements modulate visual responses in the lateral geniculate nucleus. *Neuron* 35**,** 961-974.

Reynolds, J.H., Heeger, D.J., 2009. The normalization model of attention. *Neuron* 61**,** 168-185.

Rhodes, P.A., Llinás, R., 2005. A model of thalamocortical relay cells. *J. Physiol. (Lond).* 565(Pt 3)**,** 765-781.

Riesenhuber, M., Poggio, T., 1999. Hierarchical models of object recognition in cortex. *Nat. Neurosci.* 2**,** 1019-1025.

Rivadulla, C., Martinez, L., Grieve, K.L., Cudeiro, J., 2003. Receptive field structure of burst and tonic firing in feline lateral geniculate nucleus. *J. Physiol. (Lond).* 553(Pt 2)**,** 601-610.

Rivadulla, C., Martínez, L.M., Varela, C., Cudeiro, J., 2002. Completing the corticofugal loop: a visual role for the corticogeniculate type 1 metabotropic glutamate receptor. *J. Neurosci.* 22**,** 2956-2962.

Robinson, P.A., 2006. Patchy propagators, brain dynamics, and the generation of spatially structured gamma oscillations. *Phys. Rev. E Stat. Nonlin. Soft Matter Phys.* 73**,** 041904.

Robinson, P.A., Loxley, P.N., O'Connor, S.C., Rennie, C.J., 2001a. Modal analysis of corticothalamic dynamics, electroencephalographic spectra, and evoked potentials. *Phys. Rev. E Stat. Nonlin. Soft Matter Phys.* 63**,** 041909.

Robinson, P.A., Rennie, C.J., Wright, J.J., Bahramali, H., Gordon, E., Rowe, D.L., 2001b. Prediction of electroencephalographic spectra from neurophysiology. *Phys. Rev. E Stat. Nonlin. Soft Matter Phys.* 63**,** 021903.

Robson, J.A., 1984. Reconstructions of corticogeniculate axons in the cat. *J. Comp. Neurol.* 225**,** 193-200.

Rodieck, R., Dreher, B., 1979. Visual suppression from nondominant eye in the lateral geniculate nucleus: a comparison of cat and monkey. *Exp. Brain Res.* 35**,** 465-477.

Rodieck, R.W., 1965. Quantitative analysis of cat retinal ganglion cell response to visual stimuli. *Vision Res.* 5**,** 583-601.

Rodman, H.R., Sorenson, K.M., Shim, A.J., Hexter, D.P., 2001. Calbindin immunoreactivity in the geniculo-extrastriate system of the macaque: implications

for heterogeneity in the koniocellular pathway and recovery from cortical damage. *J. Comp. Neurol.* 431**,** 168-181.

Rogala, J., Waleszczyk, W.J., Lęski, S., Wróbel, A., Wójcik, D.K., 2013. Reciprocal inhibition and slow calcium decay in perigeniculate interneurons explain changes of spontaneous firing of thalamic cells caused by cortical inactivation. *J. Comput. Neurosci.* 34**,** 461-476.

Rompani, S.B., Müllner, F.E., Wanner, A., Zhang, C., Roth, C.N., Yonehara, K., Roska, B., 2017. Different Modes of Visual Integration in the Lateral Geniculate Nucleus Revealed by Single-Cell-Initiated Transsynaptic Tracing. *Neuron* 93**,** 767-776.

Rose, R.M., Hindmarsh, J.L., 1985. A model of a thalamic neuron. *Proc. R. Soc. Lond. B. Biol. Sci.* 225**,** 161-193.

Rose, R.M., Hindmarsh, J.L., 1989a. The assembly of ionic currents in a thalamic neuron. I. The three-dimensional model. *Proc. R. Soc. Lond. B. Biol. Sci.* 237**,** 267-288.

Rose, R.M., Hindmarsh, J.L., 1989b. The assembly of ionic currents in a thalamic neuron. II. The stability and state diagrams. *Proc. R. Soc. Lond. B. Biol. Sci.* 237**,** 289-312.

Rose, R.M., Hindmarsh, J.L., 1989c. The assembly of ionic currents in a thalamic neuron. III. The seven-dimensional model. *Proc. R. Soc. Lond. B. Biol. Sci.* 237**,** 313-334.

Roskies, A.L., 1999. The binding problem. *Neuron* 24**,** 7-9, 111-125.

Ross, J., Morrone, M.C., Goldberg, M.E., Burr, D.C., 2001. Changes in visual perception at the time of saccades. *Trends Neurosci.* 24**,** 13-21.

Ross, W.D., Grossberg, S., Mingolla, E., 2000. Visual cortical mechanisms of perceptual grouping: interacting layers, networks, columns, and maps. *Neural Networks* 13**,** 571-588.

Rowe, D.L., Robinson, P.A., Rennie, C.J., 2004. Estimation of neurophysiological parameters from the waking EEG using a biophysical model of brain dynamics. *J. Theor. Biol.* 231**,** 413-433.

Rowe, M.H., Fischer, Q., 2001. Dynamic properties of retino-geniculate synapses in the cat. *Vis. Neurosci.* 18**,** 219-231.

Roy, S., Jayakumar, J., Martin, P.R., Dreher, B., Saalmann, Y.B., Hu, D., Vidyasagar, T.R., 2009. Segregation of short-wavelength-sensitive (S) cone signals in the macaque dorsal lateral geniculate nucleus. *Eur. J. Neurosci.* 30**,** 1517-1526.

Royal, D.W., Sáry, G., Schall, J.D., Casagrande, V.A., 2006. Correlates of motor planning and postsaccadic fixation in the macaque monkey lateral geniculate nucleus. *Exp. Brain Res.* 168**,** 62-75.

Ruiz, O., Royal, D., Sáry, G., Chen, X., Schall, J.D., Casagrande, V.A., 2006. Low-threshold Ca2+-associated bursts are rare events in the LGN of the awake behaving monkey. *J. Neurophysiol.* 95**,** 3401-3413.

Ruksenas, O., Fjeld, I.T., Heggelund, P., 2000. Spatial summation and center-surround antagonism in the receptive field of single units in the dorsal lateral geniculate nucleus of cat: comparison with retinal input. *Vis. Neurosci.* 17**,** 855-870.

Saalmann, Y.B., Kastner, S., 2009. Attention modulates responses in the human lateral geniculate nucleus. *Curr. Opin. Neurobiol.* 19**,** 408-414.

Saalmann, Y.B., Kastner, S., 2011. Cognitive and perceptual functions of the visual thalamus. *Neuron* 71**,** 209-223.

Saalmann, Y.B., Kastner, S., 2015. The cognitive thalamus. *Front. Syst. Neurosci.*

Saini, K.D., Garey, L.J., 1981. Morphology of neurons in the lateral geniculate nucleus of the monkey. A Golgi study. *Exp. Brain Res.* 42**,** 235-248.

Samengo, I., Montemurro, M.A., 2010. Conversion of phase information into a spike-count code by bursting neurons. *PLoS ONE* 5**,** e9669.

Sanchez-Vives, M.V., Bal, T., McCormick, D.A., 1997. Inhibitory interactions between perigeniculate GABAergic neurons. *J. Neurosci.* 17**,** 8894-8908.

Sanchez-Vives, M.V., McCormick, D.A., 1997. Functional properties of perigeniculate inhibition of dorsal lateral geniculate nucleus thalamocortical neurons in vitro. *J. Neurosci.* 17**,** 8880-8893.

Sanderson, K., Bishop, P., Darian-Smith, I., 1971. The properties of the binocular receptive fields of lateral geniculate neurons. *Exp. Brain Res.* 13**,** 178-207.

Sanderson, K., Darian-Smith, I., Bishop, P., 1969. Binocular corresponding receptive fields of single units in the cat dorsal lateral geniculate nucleus. *Vision Res.* 9**,** 1297-1303.

Sanderson, K.J., 1971a. The projection of the visual field to the lateral geniculate and medial interlaminar nuclei in the cat. *J. Comp. Neurol.* 143**,** 101-108.

Sanderson, K.J., 1971b. Visual field projection columns and magnification factors in the lateral geniculate nucleus of the cat. *Exp. Brain Res.* 13**,** 159-177.

Saul, A.B., 2008a. Lagged cells. *Neurosignals* 16**,** 209–225.

Saul, A.B., 2008b. Lagged cells in alert monkey lateral geniculate nucleus. *Vis. Neurosci.* 25**,** 647-659.

Saul, A.B., Humphrey, A.L., 1990. Spatial and temporal response properties of lagged and nonlagged cells in cat lateral geniculate nucleus. *J. Neurophysiol.* 64**,** 206-224.

Sawatari, A., Callaway, E.M., 1996. Convergence of magno- and parvocellular pathways in layer 4B of macaque primary visual cortex. *Nature* 380**,** 442-446.

Sceniak, M.P., Chatterjee, S., Callaway, E.M., 2006. Visual spatial summation in macaque geniculocortical afferents. *J. Neurophysiol.* 96**,** 3474-3484.

Scharfman, H.E., Lu, S.M., Guido, W., Adams, P.R., Sherman, S.M., 1990. N-methyl-D-aspartate receptors contribute to excitatory postsynaptic potentials of cat lateral geniculate neurons recorded in thalamic slices. *Proc. Natl. Acad. Sci. U. S. A.* 87**,** 4548-4552.

Schein, S.J., de Monasterio, F.M., 1987. Mapping of retinal and geniculate neurons onto striate cortex of macaque. *J. Neurosci.* 7**,** 996-1009.

Schiller, P.H., 2010. Parallel information processing channels created in the retina. *Proc. Natl. Acad. Sci. U. S. A.* 107**,** 17087-17094.

Schiller, P.H., Malpeli, J.G., 1978. Functional specificity of lateral geniculate nucleus laminae of the rhesus monkey. *J. Neurophysiol.* 41**,** 788-797.

Schmid, M.C., Mrowka, S.W., Turchi, J., Saunders, R.C., Wilke, M., Peters, A.J., Ye, F.Q., Leopold, D.A., 2010. Blindsight depends on the lateral geniculate nucleus. *Nature* 466**,** 373-377.

Schmid, M.C., Panagiotaropoulos, T., Augath, M.A., Logothetis, N.K., Smirnakis, S.M., 2009. Visually driven activation in macaque areas V2 and V3 without input from the primary visual cortex. *PLoS ONE* 4**,** e5527.

Schmidhuber, J., 1992. Learning complex, extended sequences using the principle of history compression. *Neural Comput.* 4**,** 234–242.

Schmidt, M., 1996. Neurons in the cat pretectum that project to the dorsal lateral geniculate nucleus are activated during saccades. *J. Neurophysiol.* 76**,** 2907-2918.

Schmielau, F., Singer, W., 1977. The role of visual cortex for binocular interactions in the cat lateral geniculate nucleus. *Brain Res.* 120**,** 354-361.

Schmolesky, M.T., Wang, Y., Hanes, D., Thompson, K.G., Leutgeb, S., Schall, J.D., Leventhal, A.G., 1998. Signal timing across the macaque visual system. *J. Neurophysiol.* 79**,** 3272-3278.

Schneider, K.A., 2011. Subcortical mechanisms of feature-based attention. *J. Neurosci.* 31**,** 8643-8653.

Schneider, K.A., Kastner, S., 2009. Effects of sustained spatial attention in the human lateral geniculate nucleus and superior colliculus. *J. Neurosci.* 29**,** 1784-1795.

Schneider, K.A., Richter, M.C., Kastner, S., 2004. Retinotopic organization and functional subdivisions of the human lateral geniculate nucleus: a high-resolution functional magnetic resonance imaging study. *J. Neurosci.* 24**,** 8975-8985.

Schnitzer, M.J., Meister, M., 2003. Multineuronal firing patterns in the signal from eye to brain. *Neuron* 37**,** 499-511.

Scholl, B., Latimer, K.W., Priebe, N.J., 2012. A retinal source of spatial contrast gain control. *J. Neurosci.* 32**,** 9824-9830.

Scholl, B., Tan, A.Y., Corey, J., Priebe, N.J., 2013. Emergence of orientation selectivity in the mammalian visual pathway. *J. Neurosci.* 33**,** 10616-10624.

Schoonover, C.E., Tapia, J.-C., Schilling, V.C., Wimmer, V., Blazeski, R., Zhang, W., Mason, C.A., Bruno, R.M., 2014. Comparative strength and dendritic organization of thalamocortical and corticocortical synapses onto excitatory layer 4 neurons. *J. Neurosci.* 34**,** 6746-6758.

Schroeder, C.E., Tenke, C.E., Arezzo, J.C., Vaughan, H.G.J., 1990. Binocularity in the lateral geniculate nucleus of the alert macaque. *Brain Res.* 521**,** 303-310.

Schwartz, O., Simoncelli, E.P., 2001. Natural signal statistics and sensory gain control. *Nat. Neurosci.* 4**,** 819-825.

Segev, I., London, M., 2000. Untangling dendrites with quantitative models. *Science* 290**,** 744-750.

Seim, T., Valberg, A., 2013. Spatial sensitivity, responsivity, and surround suppression of LGN cell responses in the macaque. *Vis. Neurosci.* 30**,** 153-167.

Self, M.W., Roelfsema, P.R., 2015. Scene perception in early vision: Figure-ground organization in the lateral geniculate nucleus. *Proc. Natl. Acad. Sci. U. S. A.* 112**,** 6784-6785.

Sengpiel, F., Sen, A., Blakemore, C., 1997. Characteristics of surround inhibition in cat area 17. *Exp. Brain Res.* 116**,** 216-228.

Serre, T., Krieman, G., Kouh, M., Cadieu, C., Knoblich, U., Poggio, T., 2007a. A quantitative theory of immediate visual recognition. *Prog. Brain Res.* 165**,** 33-56.

Serre, T., Wolf, L., Bileschi, S., Riesenhuber, M., Poggio, T., 2007b. Robust object recognition with cortex-like mechanisms. *IEEE Trans. Pattern Anal. Mach. Intell.* 29**,** 411-426.

Sestokas, A.K., Lehmkuhle, S., 1986. Visual response latency of X- and Y-cells in the dorsal lateral geniculate nucleus of the cat. *Vision Res.* 26**,** 1041-1054.

Shadlen, M.N., Movshon, J.A., 1999. Synchrony unbound: a critical evaluation of the temporal binding hypothesis. *Neuron* 24**,** 67-77.

Shadlen, M.N., Newsome, W.T., 1998. The variable discharge of cortical neurons: implications for connectivity, computation, and information coding. *J. Neurosci.* 18**,** 3870-3896.

Shapley, R., Enroth-Cugell, C. 1984. Visual Adaptation and Retinal Gain Controls. In: *Progress in Retinal Research*. pp. 263-346. Eds. N. Osborne, G. Chade. Pergamon: New York.

Shapley, R., Hochstein, S., 1975. Visual spatial summation in two classes of geniculate cells. *Nature* 256**,** 411-413.

Shapley, R., Kaplan, E., Soodak, R., 1981. Spatial summation and contrast sensitivity of X and Y cells in the lateral geniculate nucleus of the macaque. *Nature* 292**,** 543-545.

Shapley, R., Perry, V.H., 1986. Cat and monkey retinal ganglion cells and their visual functional roles. *Trends Neurosci.* 9**,** 225-235.

Shapley, R.M., Victor, J.D., 1978. The effect of contrast on the transfer properties of cat retinal ganglion cells. *J. Physiol. (Lond).* 285**,** 275-298.

Sheinberg, D.L., Logothetis, N.K., 1997. The role of temporal cortical areas in perceptual organization. *Proc. Natl. Acad. Sci. U. S. A.* 94**,** 3408-3413.

Sherman, S.M., 1996. Dual response modes in lateral geniculate neurons: mechanisms and functions. *Vis. Neurosci.* 13**,** 205-213.

Sherman, S.M., 2004. Interneurons and triadic circuitry of the thalamus. *Trends Neurosci.* 27**,** 670-675.

Sherman, S.M., 2007. The thalamus is more than just a relay. *Curr. Opin. Neurobiol.* 17**,** 417-422.

Sherman, S.M., Guillery, R.W., 2002. The role of the thalamus in the flow of information to the cortex. *Proc. R. Soc. Lond. B. Biol. Sci.* 357**,** 1695-1708.

Shou, T., Ruan, D., Zhou, Y., 1986. The orientation bias of LGN neurons shows topographic relation to area centralis in the cat retina. *Exp. Brain Res.* 64**,** 233-236.

Shou, T.D., Leventhal, A.G., 1989. Organized arrangement of orientation-sensitive relay cells in the cat's dorsal lateral geniculate nucleus. *J. Neurosci.* 9**,** 4287-4302.

Shyue, S.-K., Hewett-Emmett, D., Sperling, H.G., Hunt, D.M., Bowmaker, J.K., Mollon, J.D., Li, W.-H., 1995. Adaptive evolution of color vision genes in higher primates. *Science* 269**,** 1265-1267.

Sillito, A.M., Cudeiro, J., Jones, H.E., 2006. Always returning: feedback and sensory processing in visual cortex and thalamus. *Trends Neurosci.* 29**,** 307-316.

Sillito, A.M., Cudeiro, J., Murphy, P.C., 1993. Orientation sensitive elements in the corticofugal influence on centre-surround interactions in the dorsal lateral geniculate nucleus. *Exp. Brain Res.* 93**,** 6-16.

Sillito, A.M., Jones, H.E., 2002. Corticothalamic interactions in the transfer of visual information. *Proc. R. Soc. Lond. B. Biol. Sci.* 357**,** 1739-1752.

Sillito, A.M., Jones, H.E., Gerstein, G.L., West, D.C., 1994. Feature-linked synchronization of thalamic relay cell firing induced by feedback from the visual cortex. *Nature* 369**,** 479-482.

Sillito, A.M., Kemp, J.A., 1983. The influence of GABAergic inhibitory processes on the receptive field structure of X and Y cells in cat dorsal lateral geniculate nucleus (dLGN). *Brain Res.* 277**,** 63-77.

Sillito, A.M., Kemp, J.A., Berardi, N., 1983. The cholinergic influence on the function of the cat dorsal lateral geniculate nucleus (dLGN). *Brain Res.* 280**,** 299-307.

Sincich, L.C., Adams, D.L., Economides, J.R., Horton, J.C., 2007. Transmission of spike trains at the retinogeniculate synapse. *J. Neurosci.* 27**,** 2683-2692.

Sincich, L.C., Horton, J.C., 2005. The circuitry of V1 and V2: integration of color, form, and motion. *Annu. Rev. Neurosci.* 28**,** 303-326.

Sincich, L.C., Horton, J.C., Sharpee, T.O., 2009. Preserving information in neural transmission. *J. Neurosci.* 29**,** 6207-6216.

Sincich, L.C., Park, K.F., Wohlgemuth, M.J., Horton, J.C., 2004. Bypassing V1: a direct geniculate input to area MT. *Nat. Neurosci.* 7**,** 1123-1128.

Singer, W., 1970. Inhibitory binocular interaction in the lateral geniculate body of the cat. *Brain Res.* 18**,** 165-170.

Singer, W., 1977. Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system. *Physiol. Rev.* 57**,** 386-420.

Singer, W., 1998. Consciousness and the structure of neuronal representations. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 353**,** 1829-1840.

Singer, W., 1999. Neuronal synchrony: a versatile code for the definition of relations? *Neuron* 24**,** 49-65.

Singer, W., 2001. Consciousness and the binding problem. *Ann. N. Y. Acad. Sci.* 929**,** 123-146.

Singer, W., Bedworth, N., 1973. Inhibitory interaction between X and Y units in the cat lateral geniculate nucleus. *Brain Res.* 49**,** 291-307.

Singer, W., Creutzfeldt, O.D., 1970. Reciprocal lateral inhibition of on- and off-center neurones in the lateral geniculate body of the cat. *Exp. Brain Res.* 10**,** 311-330.

Singer, W., Pöppel, E., Creutzfeldt, O., 1972. Inhibitory interaction in the cat's lateral geniculate nucleus. *Exp. Brain Res.* 14**,** 210-226.

Skottun, B.C., Skoyles, J.R., 2008. Visual search: magno- and parvocellular systems or color and luminance processes? *Int. J. Neurosci.* 118**,** 1259-1267.

Smith, G.D., Cox, C.L., Sherman, S.M., Rinzel, J., 2000. Fourier analysis of sinusoidally driven thalamocortical relay neurons and a minimal integrate-and-fire-or-burst model. *J. Neurophysiol.* 83**,** 588-610.

So, Y.S., Shapley, R., 1981. Spatial tuning of cells in and around lateral geniculate nucleus of the cat: X and Y relay cells and perigeniculate interneurons. *J. Physiol. (Lond).* 45**,** 107-120.

So, Y.T., Shapley, R., 1979. Spatial properties of X and Y cells in the lateral geniculate nucleus of the cat and conduction veolcities of their inputs. *Exp. Brain Res.* 36**,** 533-550.

Softky, W.R., 1995. Simple codes versus efficient codes. *Curr. Opin. Neurobiol.* 5**,** 239-247.

Softky, W.R., 1996. Unsupervised pixel-prediction. *Adv. Neural. Inf. Process. Syst.* 8**,** 809-815.

Solomon, S.G., Lee, B.B., Sun, H., 2006. Suppressive surrounds and contrast gain in magnocellular-pathway retinal ganglion cells of macaque. *J. Neurosci.* 26**,** 8715-8726.

Solomon, S.G., Peirce, J.W., Dhruv, N.T., Lennie, P., 2004. Profound contrast adaptation early in the visual pathway. *Neuron* 42**,** 155-162.

Solomon, S.G., Tailby, C., Cheong, S.K., Camp, A.J., 2010. Linear and nonlinear contributions to the visual sensitivity of neurons in primate lateral geniculate nucleus. *J. Neurophysiol.* 104**,** 1884-1898.

Solomon, S.G., White, A.J., Martin, P.R., 1999. Temporal contrast sensitivity in the lateral geniculate nucleus of a New World monkey, the marmoset Callithrix jacchus. *J. Physiol. (Lond).* 517**,** 907-917.

Solomon, S.G., White, A.J., Martin, P.R., 2002. Extraclassical receptive field properties of parvocellular, magnocellular, and koniocellular cells in the primate lateral geniculate nucleus. *J. Neurosci.* 22**,** 338-349.

Somers, D.C., Nelson, S.B., Sur, M., 1995. An emergent model of orientation selectivity in cat visual cortical simple cells. *J. Neurosci.* 15**,** 5448-5465.

Soodak, R.E., Shapley, R.M., Kaplan, E., 1987. Linear mechanism of orientation tuning in the retina and lateral geniculate nucleus of the cat. *J. Neurophysiol.* 58**,** 267-275.

Squire, R.F., Noudoost, B., Schafer, R.J., Moore, T., 2013. Prefrontal contributions to visual selective attention. *Annu. Rev. Neurosci.* 36**,** 451-466.

Srinivasan, R., 2004. Internal and external neural synchronization during conscious perception. *Int. J. Bifurc. Chaos.* 14**,** 825-842.

Srinivasan, R., Petrovic, S., 2006. MEG phase follows conscious perception during binocular rivalry induced by visual stream segregation. *Cerebal Cortex* 16**,** 597-608.

Srinivasan, R., Russell, D.P., Edelman, G.M., Tononi, G., 1999. Increased synchronization of neuromagnetic responses during conscious perception. *J. Neurosci.* 19**,** 5435-5448.

Sriram, B., Meier, P.M., Reinagel, P., 2016. Temporal and spatial tuning of dorsal lateral geniculate nucleus neurons in unanesthetized rats. *J. Neurophysiol.* 115**,** 2658-2671.

Stanley, G.B., Jin, J., Wang, Y., Desbordes, G., Wang, Q., Black, M.J., Alonso, J.-M., 2012. Visual orientation and directional selectivity through thalamic synchrony. *J. Neurosci.* 32**,** 9073-9088.

Stanley, G.B., Li, F.F., Dan, Y., 1999. Reconstruction of natural scenes from ensemble responses in the lateral geniculate nucleus. *J. Neurosci.* 19**,** 8036-8042.

Stephens, G.J., Neuenschwander, S., George, J.S., Singer, W., Kenyon, G.T., 2006. See globally, spike locally: oscillations in a retinal model encode large visual features. *Biol. Cybern.* 95**,** 327-348.

Steriade, M., 2001. To burst, or rather, not to burst. *Nat. Neurosci.* 4**,** 671.

Steriade, M., 2004. Acetylcholine systems and rhythmic activities during the waking--sleep cycle. *Prog. Brain Res.* 145**,** 179-196.

Steriade, M., Deschenes, M., 1984. The thalamus as a neuronal oscillator. *Brain Res.* 320**,** 1-63.

Steriade, M., Llinás, R.R., 1988. The functional states of the thalamus and the associated neuronal interplay. *Physiol. Rev.* 68**,** 649-742.

Steriade, M., McCormick, D.A., Sejnowski, T.J., 1993. Thalamocortical oscillations in the sleeping and aroused brain. *Science* 262**,** 679-685.

Stevens, J.K., Gerstein, G.L., 1976. Spatiotemporal organization of cat lateral geniculate receptive fields. *J. Neurophysiol.* 39**,** 213-238.

Stratford, K.J., Tarczy-Hornoch, K., Martin, K.A., Bannister, N.J., Jack, J.J., 1996. Excitatory synaptic inputs to spiny stellate cells in cat visual cortex. *Nature* 382**,** 258-261.

Striedter, G.F., 2005. *Principles of Brain Evolution*. Sinauer Associates: Sunderland, MA, USA.

Suematsu, N., Naito, T., Sato, H., 2012. Relationship between orientation sensitivity and spatiotemporal receptive field structures of neurons in the cat lateral geniculate nucleus. *Neural Networks* 35**,** 10-20.

Sumitomo, I., Nakamura, M., Iwama, K., 1976. Location and function of the so-called interneurons of rat lateral geniculate body. *Exp. Neurol.* 51**,** 110-123.

Sun, W., Tan, Z., Mensh, B.D., Ji, N., 2016. Thalamus provides layer 4 of primary visual cortex with orientation- and direction-tuned inputs. *Nat. Neurosci.* 19**,** 308-315.

Sur, M., Sherman, S.M., 1982. Linear and nonlinear W-cells in C-laminae of the cat's lateral geniculate nucleus. *J. Neurophysiol.* 47**,** 869-884.

Suresh, V., Çiftçioğlu, U.M., Wang, X., Lala, B.M., Ding, K.R., Smith, W.A., Sommer, F.T., Hirsch, J.A., 2016. Synaptic Contributions to Receptive Field Structure and Response Properties in the Rodent Lateral Geniculate Nucleus of the Thalamus. *J. Neurosci.* 36**,** 10949-10963.

Sutoyo, D., Srinivasan, R., 2009. Nonlinear SSVEP responses are sensitive to the perceptual binding of visual hemifields during conventional 'eye' rivalry and interocular 'percept' rivalry. *Brain Res.* 1251**,** 245-255.

Suzuki, H., Kato, E., 1966. Binocular interaction at cat's lateral geniculate body. *J. Neurophysiol.* 29**,** 909-920.

Suzuki, H., Takahashi, M., 1970. Organization of lateral geniculate neurons in binocular inhibition. *Brain Res.* 23**,** 261-264.

Swadlow, H.A., Gusev, A.G., 2001. The impact of 'bursting' thalamic impulses at a neocortical synapse. *Nat. Neurosci.* 4**,** 402-408.

Sylvester, R., Haynes, J.-D., Rees, G., 2005. Saccades differentially modulate human LGN and V1 responses in the presence and absence of visual stimulation. *Curr. Biol.* 15**,** 37-41.

Sylvester, R., Rees, G., 2006. Extraretinal saccadic signals in human LGN and early retinotopic cortex. *Neuroimage* 30**,** 214-219.

Szmajda, B.A., Buzás, P., Fitzgibbon, T., Martin, P.R., 2006. Geniculocortical relay of blue-off signals in the primate visual system. *Proc. Natl. Acad. Sci. U. S. A.* 103**,** 19512-19517.

Szmajda, B.A., Grünert, U., Martin, P.R., 2008. Retinal ganglion cell inputs to the koniocellular pathway. *J. Comp. Neurol.* 510**,** 251-268.

Tadmor, Y., Tolhurst, D.J., 2000. Calculating the contrasts that retinal ganglion cells and LGN neurones encounter in natural scenes. *Vision Res.* 40**,** 3145-3157.

Tailby, C., Szmajda, B.A., Buzás, P., Lee, B.B., Martin, P.R., 2008. Transmission of blue (S) cone signals through the primate lateral geniculate nucleus. *J. Physiol. (Lond).* 586**,** 5947-5967.

Tamietto, M., Morrone, M.C., 2016. Visual Plasticity: Blindsight Bridges Anatomy and Function in the Visual System. *Curr. Biol.* 26**,** R70-73.

Tanaka, K., 1983. Cross-correlation analysis of geniculostriate neuronal relationships in cats. *J. Neurophysiol.* 49**,** 1303-1318.

Tanaka, K., 1985. Organization of geniculate inputs to visual cortical cells in the cat. *Vision Res.* 25**,** 357-364.

Tang, J., Ardila Jimenez, S.C., Chakraborty, S., Schultz, S.R., 2016. Visual Receptive Field Properties of Neurons in the Mouse Lateral Geniculate Nucleus. *PLoS One* 11**,** e0146017.

Taylor, J.G., Alavi, F.N., 1993. A global competitive network for attention. *Neural Network World* 5**,** 477-502.

Teich, A.F., Qian, N., 2006. Comparison among some models of orientation selectivity. *J. Neurophysiol.* 96**,** 404-419.

Teich, A.F., Qian, N., 2010. V1 orientation plasticity is explained by broadly tuned feedforward inputs and intracortical sharpening. *Vis. Neurosci.* 27**,** 57-73.

Thilo, K.V., Santoro, L., Walsh, V., Blakemore, C., 2004. The site of saccadic suppression. *Nat. Neurosci.* 7**,** 13-14.

Thompson, K.G., Leventhal, A.G., Zhou, Y., Liu, D., 1994a. Stimulus dependence of orientation and direction sensitivity of cat LGNd relay cells without cortical inputs: a comparison with area 17 cells. *Vis. Neurosci.* 11**,** 939-951.

Thompson, K.G., Zhou, Y., Leventhal, A.G., 1994b. Direction-sensitive X and Y cells within the A laminae of the cat's LGNd. *Vis. Neurosci.* 11**,** 927-938.

Thompson, R., 1993. Centrencephalic theory, the general learning system, and subcortical dementia. *Ann. N. Y. Acad. Sci.* 702**,** 197-223.

Thomson, A.M., 2010. Neocortical layer 6, a review. *Front. Neuroanat.* 4**,** 13.

Tiesinga, P., Fellous, J.-M., Sejnowski, T.J., 2008. Regulation of spike timing in visual cortical circuits. *Nature Rev. Neurosci.* 9**,** 97-107.

Tong, F., Engel, S.A., 2001. Interocular rivalry revealed in the human cortical blind-spot representation. *Nature* 411**,** 195-199.

Tong, F., Nakayama, K., Vaughan, J.T., Kanwisher, N., 1998. Binocular rivalry and visual awareness in human extrastriate cortex. *Neuron* 21**,** 753-759.

Tononi, G., Srinivasan, R., Russell, D.P., Edelman, G.M., 1998. Investigating neural correlates of conscious perception by frequency-tagged neuromagnetic responses. *Proc. Natl. Acad. Sci. U. S. A.* 95**,** 3198-3203.

Toychiev, A.H., Yee, C.W., Sagdullaev, B.T., 2013. Correlated spontaneous activity persists in adult retina and is suppressed by inhibitory inputs. *PLoS ONE* 8**,** e77658.

Troyer, T.W., Krukowski, A.E., Priebe, N.J., Miller, K.D., 1998. Contrast-invariant orientation tuning in cat visual cortex: thalamocortical input tuning and correlation-based intracortical connectivity. *J. Neurosci.* 18**,** 5908-5927.

Tsumoto, T., Creutzfeldt, O.D., Legéndy, C.R., 1978. Functional organization of the corticofugal system from visual cortex to lateral geniculate nucleus in the cat (with an appendix on geniculo-cortical mono-synaptic connections). *Exp. Brain Res.* 32**,** 345-364.

Tsumoto, T., Suda, K., 1980. Three groups of cortico-geniculate neurons and their distribution in binocular and monocular segments of cat striate cortex. *J. Comp. Neurol.* 193**,** 223-236.

Uglesich, R., Casti, A., Hayot, F., Kaplan, E., 2009. Stimulus size dependence of information transfer from retina to thalamus. *Front. Syst. Neurosci.* 3**,** 10.

Uhlhaas, P.J., Pipa, G., Lima, B., Melloni, L., Neuenschwander, S., Nikolić, D., Singer, W., 2009. Neural synchrony in cortical networks: history, concept and current status. *Front. Integr. Neurosci.* 3**,** 17.

Uhlrich, D.J., Cucchiaro, J.B., Humphrey, A.L., Sherman, S.M., 1991. Morphology and axonal projection patterns of individual neurons in the cat perigeniculate nucleus. *J. Neurophysiol.* 65**,** 1528-1541.

Uhlrich, D.J., Manning, K.A., Feig, S.L., 2003. Laminar and cellular targets of individual thalamic reticular nucleus axons in the lateral geniculate nucleus in the prosimian primate Galago. *J. Comp. Neurol.* 458**,** 128-143.

Uhlrich, D.J., Manning, K.A., Pienkowski, T., 1993. The histaminergic innervation of the lateral geniculate complex in the cat. *Vis. Neurosci.* 10**,** 225-235.

Uhlrich, D.J., Tamamaki, N., Murphy, P.C., Sherman, S.M., 1995. Effects of brain stem parabrachial activation on receptive field properties of cells in the cat's lateral geniculate nucleus. *J. Neurophysiol.* 73**,** 2428-2447.

Uhlrich, D.J., Tamamaki, N., Sherman, S.M., 1990. Brainstem control of response modes in neurons of the cat's lateral geniculate nucleus. *Proc. Natl. Acad. Sci. U. S. A.* 87**,** 2560-2563.

Usrey, W.M., 2002. The role of spike timing for thalamocortical processing. *Curr. Opin. Neurobiol.* 12**,** 411-417.

Usrey, W.M., Alonso, J.-M., Reid, R.C., 2000. Synaptic interactions between thalamic inputs to simple cells in cat visual cortex. *J. Neurosci.* 20**,** 5461-5467.

Usrey, W.M., Fitzpatrick, D., 1996. Specificity in the axonal connections of layer VI neurons in tree shrew striate cortex: evidence for distinct granular and supragranular systems. *J. Neurosci.* 16**,** 1203-1218.

Usrey, W.M., Reid, R.C., 2000. Visual physiology of the lateral geniculate nucleus in two species of new world monkey: Saimiri sciureus and Aotus trivirgatis. *J. Physiol. (Lond).* 523**,** 755-769.

Usrey, W.M., Reppas, J.B., Reid, R.C., 1998. Paired-spike interactions and synaptic efficacy of retinal inputs to the thalamus. *Nature* 395**,** 384-387.

Usrey, W.M., Reppas, J.B., Reid, R.C., 1999. Specificity and strength of retinogeniculate connections. *J. Neurophysiol.* 82**,** 3527-3540.

Vaingankar, V., Soto-Sanchez, C., Wang, X., Sommer, F.T., Hirsch, J.A., 2012. Neurons in the thalamic reticular nucleus are selective for diverse and complex visual features. *Front. Integr. Neurosci.* 6**,** 118.

Valberg, A., Lee, B.B., Tigwell, D.A., Creutzfeldt, O.D., 1985. A simultaneous contrast effect of steady remote surrounds on responses of cells in macaque lateral geniculate nucleus. *Exp. Brain Res.* 58**,** 604-608.

Valberg, A., Seim, T., 2013. Neurophysiological correlates of color vision: a model. *Psychol. Neurosci.* 6**,** 213 - 218.

Van Hateren, J.H., 1993. Spatiotemporal contrast sensitivity of early vision. *Vision Res.* 33**,** 257-267.

Van Horn, S.C., Erisir, A., Sherman, S.M., 2000. Relative distribution of synapses in the A-laminae of the lateral geniculate nucleus of the cat. *J. Comp. Neurol.* 416**,** 509-520.

Vanduffel, W., Tootell, R.B., Orban, G.A., 2000. Attention-dependent suppression of metabolic activity in the early stages of the macaque visual system. *Cerebal Cortex* 10**,** 109-126.

Varela, F.J., Singer, W., 1987. Neuronal dynamics in the visual corticothalamic pathway revealed through binocular rivalry. *Exp. Brain Res.* 66**,** 10-20.

Vastola, E., 1960. Binocular inhibition in the lateral geniculate body. *Exp. Neurol.* 2**,** 221-231.

Vastola, E.F., 1967. Steady-state effects of visual cortex on geniculate cells. *Vision Res.* 7**,** 599-609.

Veredas, F.J., Vico, F.J., Alonso, J.M., 2005. Factors determining the precision of the correlated firing generated by a monosynaptic connection in the cat visual pathway. *J. Physiol. (Lond).* 567**,** 1057-1078.

Victor, J.D., 1999. Temporal aspects of neural coding in the retina and lateral geniculate. *Network* 10**,** R1-66.

Victor, J.D., Shapley, R.M., 1979. Receptive field mechanisms of cat X and Y retinal ganglion cells. *J. Gen. Physiol.* 74**,** 275-298.

Vidyasagar, T.R., 1984. Contribution of inhibitory mechanisms to the orientation sensitivity of cat dLGN Neurones. *Exp. Brain Res.* 55**,** 192-195.

Vidyasagar, T.R., Eysel, U.T., 2015. Origins of feature selectivities and maps in the mammalian primary visual cortex. *Trends Neurosci.* 38**,** 475-485.

Vidyasagar, T.R., Jayakumar, J., Lloyd, E., Levichkina, E.V., 2015. Subcortical orientation biases explain orientation selectivity of visual cortical cells. *Physiological Reports* 3**,** e12374.

Vidyasagar, T.R., Pei, X., Volgushev, M., 1996. Multiple mechanisms underlying the orientation selectivity of visual cortical neurones. *Trends Neurosci.* 19**,** 272-277.

Vidyasagar, T.R., Urbas, J.V., 1982. Orientation sensitivity of cat LGN neurones with and without inputs from visual cortical areas 17 and 18. *Exp. Brain Res.* 46**,** 157-169.

Vigeland, L.E., Contreras, D., Palmer, L.A., 2013. Synaptic mechanisms of temporal diversity in the lateral geniculate nucleus of the thalamus. *J. Neurosci.* 33**,** 1887-1896.

Vijayan, S., Kopell, N.J., 2012. Thalamic model of awake alpha oscillations and implications for stimulus processing. *Proc. Natl. Acad. Sci. U. S. A.* 109**,** 18553-18558.

Viswanathan, S., Jayakumar, J., Vidyasagar, T.R., 2011. Role of feedforward geniculate inputs in the generation of orientation selectivity in the cat's primary visual cortex. *J. Physiol. (Lond).* 589**,** 2349-2361.

Viswanathan, S., Jayakumar, J., Vidyasagar, T.R., 2015. Contrast invariance of orientation tuning in the lateral geniculate nucleus of the feline visual system. *Eur. J. Neurosci.* 42**,** 2250-2257.

Viviano, J.D., Schneider, K.A., 2015. Interhemispheric interactions of the human thalamic reticular nucleus. *J. Neurosci.* 35**,** 2026-2032.

von der Malsburg, C., 1999. The what and why of binding: the modeler's perspective. *Neuron* 24**,** 95-104, 111-125.

Waleszczyk, W.J., Bekisz, M., Wróbel, A., 2005. Cortical modulation of neuronal activity in the cat's lateral geniculate and perigeniculate nuclei. *Exp. Neurol.* 196**,** 54-72.

Walker, G.A., Ohzawa, I., Freeman, R.D., 2000. Suppression outside the classical cortical receptive field. *Vis. Neurosci.* 17**,** 369-379.

Wallace, D.J., Fitzpatrick, D., Kerr, J.N., 2016. Primate Thalamus: More Than Meets an Eye. *Curr. Biol.* 26**,** R60-61.

Wallis, G., Rolls, E.T., 1997. Invariant face and object recognition in the visual system. *Prog. Neurobiol.* 51**,** 167-194.

Walls, G.L., 1953. *The lateral geniculate nucleus and visual histophysiology*. University of California Press: Berkeley.

Wang, H.-P., Spencer, D., Fellous, J.-M., Sejnowski, T.J., 2010a. Synchrony of thalamocortical inputs maximizes cortical reliability. *Science* 328**,** 106-109.

Wang, L., Kloc, M., Gu, Y., Ge, S., Maffei, A., 2013. Layer-specific experience-dependent rewiring of thalamocortical circuits. *J. Neurosci.* 33**,** 4181-4191.

Wang, S., Bickford, M.E., Van Horn, S.C., Godwin, D.W., Sherman, S.M., 2001. Synaptic targets of thalamic reticular nucleus terminals in the visual thalamus of the cat. *J. Comp. Neurol.* 440**,** 321-341.

Wang, W., Andolina, I.M., Lu, Y., Jones, H.E., Sillito, A.M., 2016. Focal Gain Control of Thalamic Visual Receptive Fields by Layer 6 Corticothalamic Feedback. *Cereb. Cortex* [Epub ahead of print].

Wang, W., Jones, H.E., Andolina, I.M., Salt, T.E., Sillito, A.M., 2006. Functional alignment of feedback effects from visual cortex to thalamus. *Nat. Neurosci.* 9**,** 1330-1336.

Wang, X., Hirsch, J.A., Sommer, F.T., 2010b. Recoding of sensory information across the retinothalamic synapse. *J. Neurosci.* 30**,** 13567-13577.

Wang, X., Sommer, F.T., Hirsch, J.A., 2011a. Inhibitory circuits for visual processing in thalamus. *Curr. Opin. Neurobiol.* 21**,** 726-733.

Wang, X., Vaingankar, V., Soto Sanchez, C., Sommer, F.T., Hirsch, J.A., 2011b. Thalamic interneurons and relay cells use complementary synaptic mechanisms for visual processing. *Nat. Neurosci.* 14**,** 224-231.

Wang, X., Wei, Y., Vaingankar, V., Wang, Q., Koepsell, K., Sommer, F.T., Hirsch, J.A., 2007. Feedforward excitation and inhibition evoke dual modes of firing in the cat's visual thalamus during naturalistic viewing. *Neuron* 55**,** 465-478.

Wang, X.-J., 2010. Neurophysiological and computational principles of cortical rhythms in cognition. *Physiol. Rev.* 90**,** 1195-1268.

Wang, X.-J., Rinzel, J., Rogawski, M.A., 1991. A model of the T-type calcium current and the low-threshold spike in thalamic neurons. *J. Neurophysiol.* 66**,** 839-850.

Ward, L.M., 2011. The thalamic dynamic core theory of conscious experience. *Conscious. Cogn.* 20**,** 464-486.

Warner, C.E., Goldshmit, Y., Bourne, J.A., 2010. Retinal afferents synapse with relay cells targeting the middle temporal area in the pulvinar and lateral geniculate nuclei. *Front. Neuroanat.* 4**,** 8.

Webb, B.S., Tinsley, C.J., Barraclough, N.E., Easton, A., Parker, A., Derrington, A.M., 2002. Feedback from V1 and inhibition from beyond the classical receptive field modulates the responses of neurons in the primate lateral geniculate nucleus. *Vis. Neurosci.* 19**,** 583-592.

Webb, B.S., Tinsley, C.J., Vincent, C.J., Derrington, A.M., 2005. Spatial distribution of suppressive signals outside the classical receptive field in lateral geniculate nucleus. *J. Neurophysiol.* 94**,** 1789-1797.

Webster, M.J., Bachevalier, J., Ungerleider, L.G., 1994. Connections of inferior temporal areas TEO and TE with parietal and frontal cortex in macaque monkeys. *Cereb. Cortex* 4**,** 470-483.

Weese, G.D., Phillips, J.M., Brown, V.J., 1999. Attentional orienting is impaired by unilateral lesions of the thalamic reticular nucleus in the rat. *J. Neurosci.* 19**,** 10135-10139.

Wei, H., Bonjean, M., Petry, H.M., Sejnowski, T.J., Bickford, M.E., 2011. Thalamic burst firing propensity: a comparison of the dorsal lateral geniculate and pulvinar nuclei in the tree shrew. *J. Neurosci.* 31**,** 17287-17299.

Weiskrantz, L., 1996. Blindsight revisited. *Curr. Opin. Neurobiol.* 6**,** 215-220.

Wells, M.F., Wimmer, R.D., Schmitt, L.I., Feng, G., Halassa, M.M., 2016. Thalamic reticular impairment underlies attention deficit in Ptchd1(Y/-) mice. *Nature* 532**,** 58-63.

Weng, C., Yeh, C.I., Stoelzel, C.R., Alonso, J.M., 2005. Receptive field size and response latency are correlated within the cat visual thalamus. *J. Neurophysiol.* 93**,** 3537-3547.

Weyand, T.G., 2007. Retinogeniculate transmission in wakefulness. *J. Neurophysiol.* 98**,** 769-785.

Weyand, T.G., 2015. The multifunctional lateral geniculate nucleus. *Rev. Neurosci.* 27**,** 135-157.

Weyand, T.G., Boudreaux, M., Guido, W., 2001. Burst and tonic response modes in thalamic neurons during sleep and wakefulness. *J. Neurophysiol.* 85**,** 1107-1118.

White, A.J.R., Solomon, S.G., Martin, P.R., 2001. Spatial properties of koniocellular cells in the lateral geniculate nucleus of the marmoset Callithrix jacchus. *J. Physiol. (Lond).* 533(Pt 2)**,** 519-535.

Wielaard, J., Sajda, P., 2007. Dependence of response properties on sparse connectivity in a spiking neuron model of the lateral geniculate nucleus. *J. Neurophysiol.* 98**,** 3292-3308.

Wiesel, T.N., Hubel, D., 1966. Spatial and chromatic interactions in the lateral geniculate body of the rhesus monkey. *J. Neurophysiol.* 29**,** 1115-1156.

Wilke, M., Mueller, K.-M., Leopold, D.A., 2009. Neural activity in the visual thalamus reflects perceptual suppression. *Proc. Natl. Acad. Sci. U. S. A.* 106**,** 9465-9470.

Williams, S.R., Stuart, G.J., 2000. Action potential backpropagation and somato-dendritic distribution of ion channels in thalamocortical neurons. *J. Neurosci.* 20**,** 1307-1317.

Willis, A.M., Slater, B.J., Gribkova, E.D., Llano, D.A., 2015. Open-loop organization of thalamic reticular nucleus and dorsal thalamus: a computational model. *J. Neurophysiol.* 114**,** 2353-2367.

Wilson, J.R., 1989. Synaptic organization of individual neurons in the macaque lateral geniculate nucleus. *J. Neurosci.* 9**,** 2931-2953.

Wilson, J.R., 1993. Circuitry of the dorsal lateral geniculate nucleus in the cat and monkey. *Acta Anat. (Basel).* 147**,** 1-13.

Wilson, J.R., Friedlander, M.J., Sherman, S.M., 1984. Fine structural morphology of identified X- and Y-cells in the cat's lateral geniculate nucleus. *Proc. R. Soc. Lond. B. Biol. Sci.* 221**,** 411-436.

Wilson, J.R., Hendrickson, A.E., Sherk, H., Tigges, J., 1995. Sources of subcortical afferents to the macaque's dorsal lateral geniculate nucleus. *Anat. Rec.* 242**,** 566-574.

Wimmer, R.D., Schmitt, L.I., Davidson, T.J., Nakajima, M., Deisseroth, K., Halassa, M.M., 2015. Thalamic control of sensory selection in divided attention. *Nature* 526**,** 705-709.

Wolfart, J., Debay, D., Le Masson, G., Destexhe, A., Bal, T., 2005. Synaptic background activity controls spike transfer from thalamus to cortex. *Nat. Neurosci.* 8**,** 1760-1767.

Wolfe, J., Palmer, L.A., 1998. Temporal diversity in the lateral geniculate nucleus of cat. *Vis. Neurosci.* 15**,** 653-675.

Wong-Riley, M.T., 1972. Neuronal and synaptic organization of the normal dorsal lateral geniculate nucleus of the squirrel monkey, Saimiri sciureus. *J. Comp. Neurol.* 144**,** 25-59.

Wörgötter, F., Nelle, E., Li, B., Funke, K., 1998. The influence of corticofugal feedback on the temporal structure of visual responses of cat thalamic relay cells. *J. Physiol. (Lond).* 509**,** 797-815.

Wróbel, A., 1982. Inhibitory mechanisms within the receptive fields of the lateral geniculate body of the cat. *Acta Neurobiologiae Experimentalis* 42**,** 93-107.

Wróbel, A., 2000. Beta activity: a carrier for visual attention. *Acta Neurobiologiae Experimentalis* 60**,** 247-260.

Wróbel, A. 2014. Attentional Activation in Corticothalamic Loops of the Visual System. In: *The new visual neurosciences*. pp. 339–349. Eds. J.S. Werner, L.M. Chalupa. MIT Press: Cambridge, MA.

Wróbel, A., Bekisz, M., 1994. Visual classification of X and Y perigeniculate neurons of the cat. *Exp. Brain Res.* 101**,** 307-313.

Wróbel, A., Bekisz, M., Kublik, E., Waleszczyk, W., 1994. 20 Hz bursting beta activity in the cortico-thalamic system of visually attending cats. *Acta Neurobiologiae Experimentalis* 54**,** 95-107.

Wróbel, A., Podvigin, N.F., Tarnecki, R., 1983. Multiunit activity of the cat lateral geniculate neurons evoked by moving light pattern of variable intensity. *Acta Neurobiol. Exp. (Wars).* 43**,** 135-140.

Wróbel, A., Sarna, M.F., Dec, K., Tarnecki, R., 1984. Barbiturate influence upon organization of lateral geniculate receptive fields in cats. *Acta Neurobiologiae Experimentalis* 44**,** 73-81.

Wróbel, A., Tarnecki, R., 1984. Receptive fields of cat's non-relay lateral geniculate and perigeniculate neurons. *Acta Neurobiologiae Experimentalis* 44**,** 289-299.

Wunderlich, K., Schneider, K.A., Kastner, S., 2005. Neural correlates of binocular rivalry in the human lateral geniculate nucleus. *Nat. Neurosci.* 8**,** 1595-1602.

Xu, X., Bonds, A.B., Casagrande, V.A., 2002a. Modeling receptive-field structure of koniocellular, magnocellular, and parvocellular LGN cells in the owl monkey (Aotus trivigatus). *Vis. Neurosci.* 19**,** 703-711.

Xu, X., Ichida, J., Shostak, Y., Bonds, A.B., Casagrande, V.A., 2002b. Are primate lateral geniculate nucleus (LGN) cells really sensitive to orientation or direction? *Vis. Neurosci.* 19**,** 97-108.

Xu, X., Ichida, J.M., Allison, J.D., Boyd, J.D., Bonds, A.B., Casagrande, V.A., 2001. A comparison of koniocellular, magnocellular and parvocellular receptive field properties in the lateral geniculate nucleus of the owl monkey (Aotus trivirgatus). *J. Physiol. (Lond).* 531(Pt 1)**,** 203-218.

Xue, J., Carney, T., Ramoa, A., Freeman, R., 1988. Binocular interaction in the perigeniculate nucleus of the cat. *Exp. Brain Res.* 69**,** 497-508.

Xue, J., Ramoa, A., Carney, T., Freeman, R., 1987. Binocular interaction in the dorsal lateral geniculate nucleus of the cat. *Exp. Brain Res.* 68**,** 305-310.

Yabuta, N.H., Callaway, E.M., 1998. Functional streams and local connections of layer 4C neurons in primary visual cortex of the macaque monkey. *J. Neurosci.* 18**,** 9489-9499.

Yakimova, E.G., Chizhov, A.V., 2015. Experimental and Modeling Studies of Orientational Sensitivity of Neurons in the Lateral Geniculate Nucleus. *Neurosci. Behav. Physiol.* 45**,** 465-475.

Yamins, D.L., Hong, H., Cadieu, C.F., Solomon, E.A., Seiber, D., DiCarlo, J.J., 2014. Performance-optimized hierarchical models predict neural responses in higher visual cortex. *Proc. Natl. Acad. Sci. U. S. A.* 111**,** 8619-8624.

Yeh, C.-I., Stoelzel, C.R., Weng, C., Alonso, J.-M., 2009. Functional consequences of neuronal divergence within the retinogeniculate pathway. *J. Neurophysiol.* 101**,** 2166-2185.

Yeh, C.I., Stoelzel, C.R., Alonso, J.M., 2003. Two different types of Y cells in the cat lateral geniculate nucleus. *J. Neurophysiol.* 90**,** 1852-1864.

Yen, C.-C., Fukuda, M., Kim, S.-G., 2011. BOLD responses to different temporal frequency stimuli in the lateral geniculate nucleus and visual cortex: insights into the neural basis of fMRI. *Neuroimage* 58**,** 82-90.

Yingling, C.D., Skinner, J.E., 1976. Selective regulation of thalamic sensory relay nuclei by nucleus reticularis thalami. *Electroencephalogr. Clin. Neurophysiol.* 41**,** 476-482.

Yingling, C.D., Skinner, J.E. 1977. Gating of the thalamic input to cerebral cortex by nucleus reticularis thalami. In: *Progress in Clinical Neurophysiology, Vol. 1*. pp. 70-96. Ed. J.E. Desmedt. Karger: New York.

Yokoyama, S., Yokoyama, R., 1989. Molecular evolution of human visual pigment genes. *Mol. Biol. Evol.* 6**,** 186-197.

Yoshioka, T., Levitt, J.B., Lund, J.S., 1994. Independence and merger of thalamocortical channels within macaque monkey primary visual cortex: anatomy of interlaminar projections. *Vis. Neurosci.* 11**,** 467-489.

Young, R.A., 1987. The Gaussian derivative model for spatial vision: I. Retinal mechanisms. *Spat. Vis.* 2**,** 273-293.

Yousif, N., Denham, M., 2007. The role of cortical feedback in the generation of the temporal receptive field responses of lateral geniculate nucleus neurons: a computational modelling study. *Biol. Cybern.* 97**,** 269-277.

Yukie, M., Iwai, E., 1981. Direct projection from the dorsal lateral geniculate nucleus to the prestriate cortex in macaque monkeys. *J. Comp. Neurol.* 201**,** 81-97.

Zabbah, S., Rajaei, K., Mirzaei, A., Ebrahimpour, R., Khaligh-Razavi, S.-M., 2014. The impact of the lateral geniculate nucleus and corticogeniculate interactions on efficient coding and higher-order visual object processing. *Vision Res.* 101**,** 82-93.

Zador, A., 1998. Impact of synaptic unreliability on the information transmitted by spiking neurons. *J. Neurophysiol.* 79**,** 1219-1229.

Zeater, N., Cheong, S.K., Solomon, S.G., Dreher, B., Martin, P.R., 2015. Binocular Visual Responses in the Primate Lateral Geniculate Nucleus. *Curr. Biol.* 25**,** 3190–3195.

Zhan, X.J., Cox, C.L., Sherman, S.M., 2000. Dendritic depolarization efficiently attenuates low-threshold calcium spikes in thalamic relay cells. *J. Neurosci.* 20**,** 3909-3914.

Zhang, P., Zhou, H., Wen, W., He, S., 2015. Layer-specific response properties of the human lateral geniculate nucleus and superior colliculus. *Neuroimage* 111**,** 159-166.

Zhao, X., Chen, H., Liu, X., Cang, J., 2013. Orientation-selective responses in the mouse lateral geniculate nucleus. *J. Neurosci.* 33**,** 12751-12763.

Zheng, C., Colgin, L.L., 2015. Beta and gamma rhythms go with the flow. *Neuron* 85**,** 236-237.

Zhou, Q., Godwin, D.W., O'Malley, D.M., Adams, P.R., 1997. Visualization of calcium influx through channels that shape the burst and tonic firing modes of thalamic relay cells. *J. Neurophysiol.* 77**,** 2816-2825.

Zhou, Y., Yu, H., Yang, Y., Shou, T., 2003. Non-dominant eye responses in the dorsal lateral geniculate nucleus of the cat: an intracellular study. *Brain Res.* 987**,** 76-85.

Zhu, J.J., Lo, F.-S., 1996. Time course of inhibition induced by a putative saccadic suppression circuit in the dorsal lateral geniculate nucleus of the rabbit. *Brain Res. Bull.* 41**,** 281-291.

Zikopoulos, B., Barbas, H., 2006. Prefrontal projections to the thalamic reticular nucleus form a unique circuit for attentional mechanisms. *J. Neurosci.* 26**,** 7348-7361.

Zikopoulos, B., Barbas, H., 2007. Circuits for multisensory integration and attentional modulation through the prefrontal cortex and the thalamic reticular nucleus in primates. *Rev. Neurosci.* 18**,** 417-438.

Zikopoulos, B., Barbas, H., 2012. Pathways for emotions and attention converge on the thalamic reticular nucleus in primates. *J. Neurosci.* 32**,** 5338-5350.

Zomorrodi, R., Kröger, H., Timofeev, I., 2008. Modeling thalamocortical cell: impact of ca channel distribution and cell geometry on firing pattern. *Front. Comput. Neurosci.* 2**,** 5.

Figures

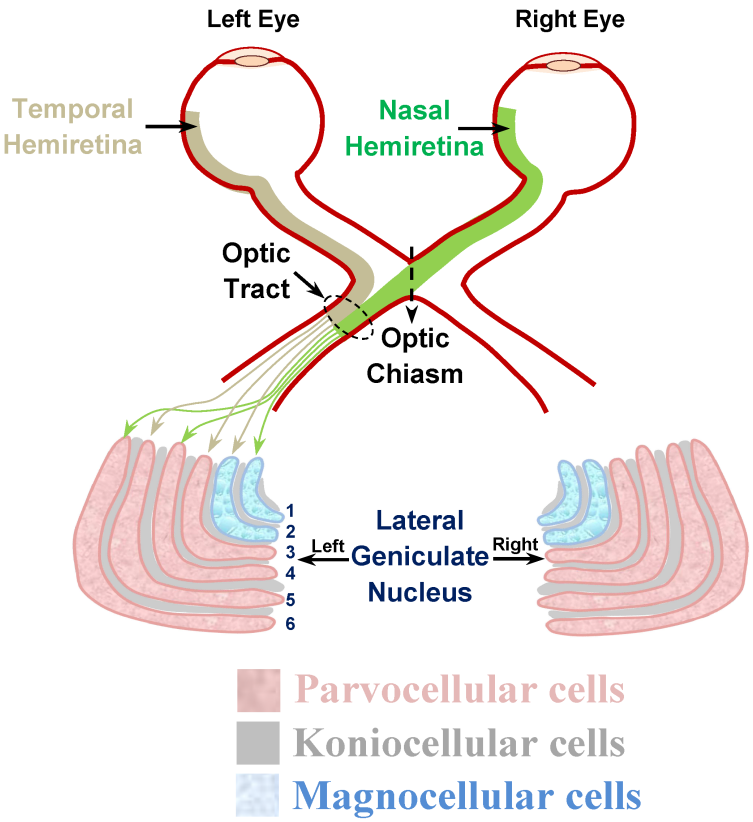

Figure 1. Retinal Inputs to Different Layers of LGN in Old World Monkeys, Apes, and Humans. Axons from retinal ganglion cells (RGC) reach the optic chiasm. Axons from the nasal halves of the two retinas cross the optic chiasm and go into the contralateral optic tract and those from the temporal halves go into the ipsilateral optic tract. For example, in the left LGN, retinal afferents from nasal hemiretina project to layers 1, 4, and 6 of the LGN, and those coming from temporal hemiretina go to layers 2, 3, and 5. Under this arrangement, the right visual field is processed by the left LGN, and the left visual field is processed by the right LGN.

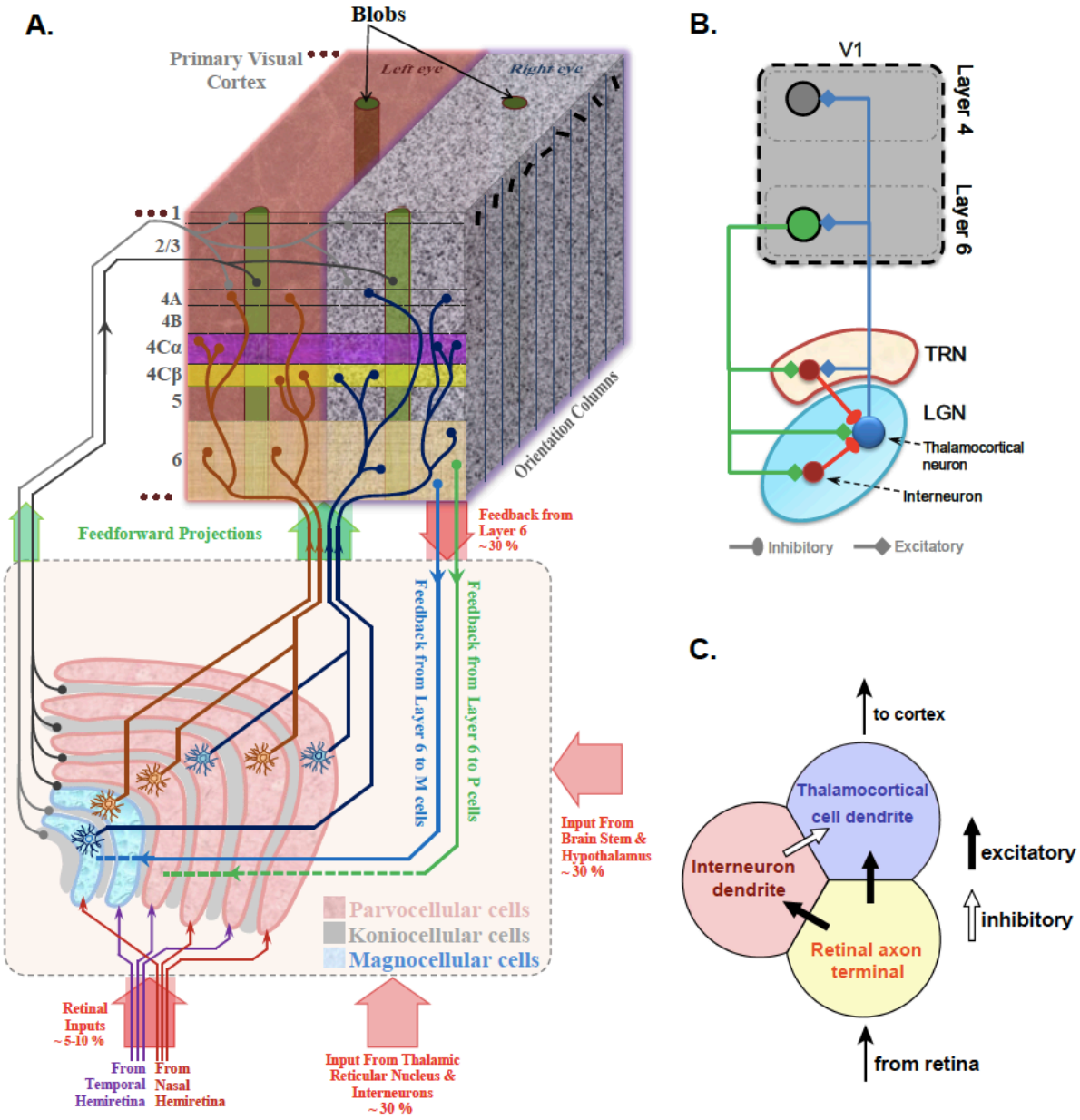

Figure 2. LGN Feedback and Feedforward Connections. A) Thalamocortical cells in LGN project to layer 4 of primary visual cortex (V1), sending collaterals to layer 6 along the way. Cells are color coded by eye of origin of visual input. Signals are projected from parvocellular layers of the LGN onto layer 4Cβ of V1 and magnocellular layers send their projections to layer 4Cα. Koniocellular layers of the LGN project to layers 1-3. Cortical feedback to LGN is provided by neurons in layer 6 of V1. The layer 6 feedback targets LGN thalamocortical cells and also contact inhibitory interneurons (not shown in the figure). In addition to receiving input from retina and cortical feedback from V1, LGN receives feedback from the thalamic reticular nucleus (TRN) as well as multiple neuromodulatory inputs from the brainstem. There are other afferents to the LGN not shown in the figure, such as neurons from the nucleus of the optic tract in the midbrain and from the tuberomamillary nucleus in the hypothalamus. **B)** The LGN-TRN-V1

circuitry**.** LGN thalamocortical neurons en route to V1 send excitatory collaterals to TRN. Feedback from layer 6 of V1 en route to LGN also sends excitatory collaterals to TRN. The output of TRN is an inhibitory projection back to LGN thalamocortical cells. **C**) Synaptic triad organization in the LGN. Within a glomerulus, an incoming axon terminal from the retina makes excitatory synaptic contacts both with a dendrite from an LGN thalamocortical cell (predominantly X cells) and a dendrite from an LGN interneuron. The interneuron dendrite makes an inhibitory synaptic contact with the thalamocortical cell dendrite, forming the third synapse of the triad. The interneuron dendrite is thus presynaptic to the thalamocortical cell dendrite, and under some circumstances a signal may be transmitted from dendrite to dendrite without passing through the soma of the interneuron. Alternatively, rather than being excited locally by the retinothalamic synapse within the triad, the presynaptic interneuron dendrite may also be activated by backpropagation of spike activity from the axonal output of the interneuron.

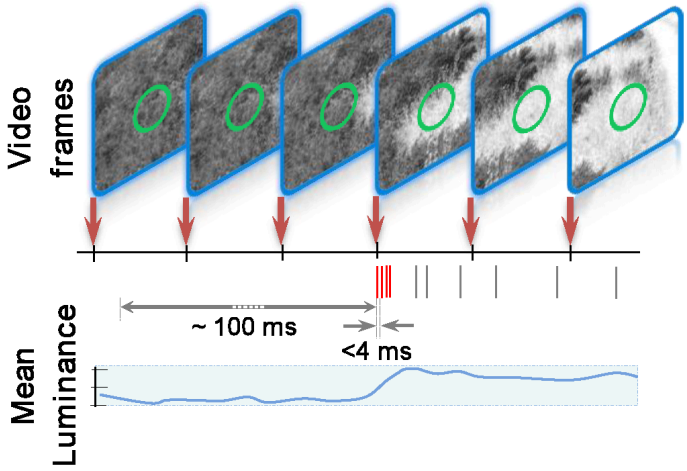

Figure 3. Schematic Summary of Burst Spikes during Natural Movie Stimulation. Burst spikes occur during specific, reproducible times during repeated showing of the same natural image movie. The specificity of stimuli inducing bursts raises the possibility that bursts are part of a nonlinear coding mechanism indicating specific aspects of the stimulus. Top: several sample frames of a natural scene sequence and the onset of frame presentation (specified with red arrows). The green circle shows neuron receptive field. Bottom: a schematic raster plot that shows burst spikes (red) and tonic spikes (gray). The blue curve indicates the mean luminance covering the receptive field of the neuron, showing that a burst occurs when a bright spot follows an extended dark period. The diagram is based on results of Lesica and Stanley (2004) and Lesica et al. (2006).

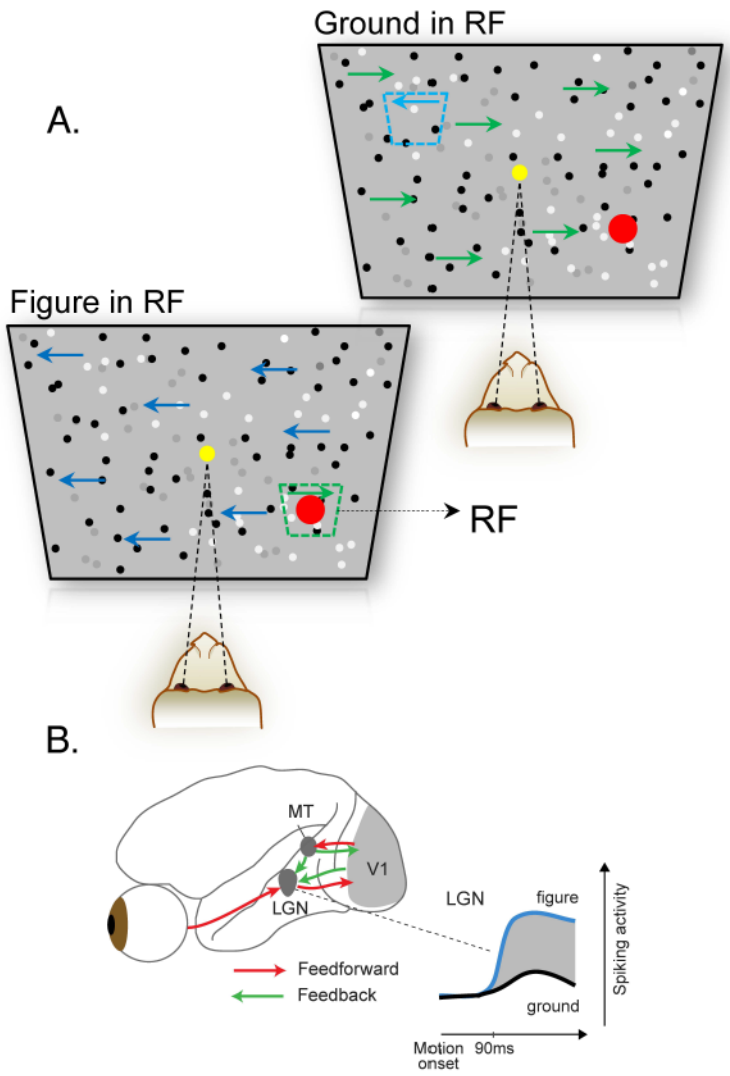

Figure 4. Figure-ground Modulation in the LGN of Awake Primate. This demonstrates how LGN responses might be affected by higher-order cortical information A) Experimental conditions. The monkey viewed moving random dot stimuli, with a figure-ground pattern defined by direction of motion. Sometimes the figure was placed on the LGN receptive field (red dot) and sometimes ground was placed on the receptive field. In either case, direction of motion over the receptive field remained the same. B) LGN response was stronger when the figure pattern covered the receptive field, even though the local stimulus was identical under both figure and ground conditions. A possible explanation is that the LGN is receiving globally integrated motion information through indirect feedback from MT via V1 (Jones *et al.*, 2013). Data from Jones *et al.* (2015), figure adapted from Self and Roelfsema (2015).

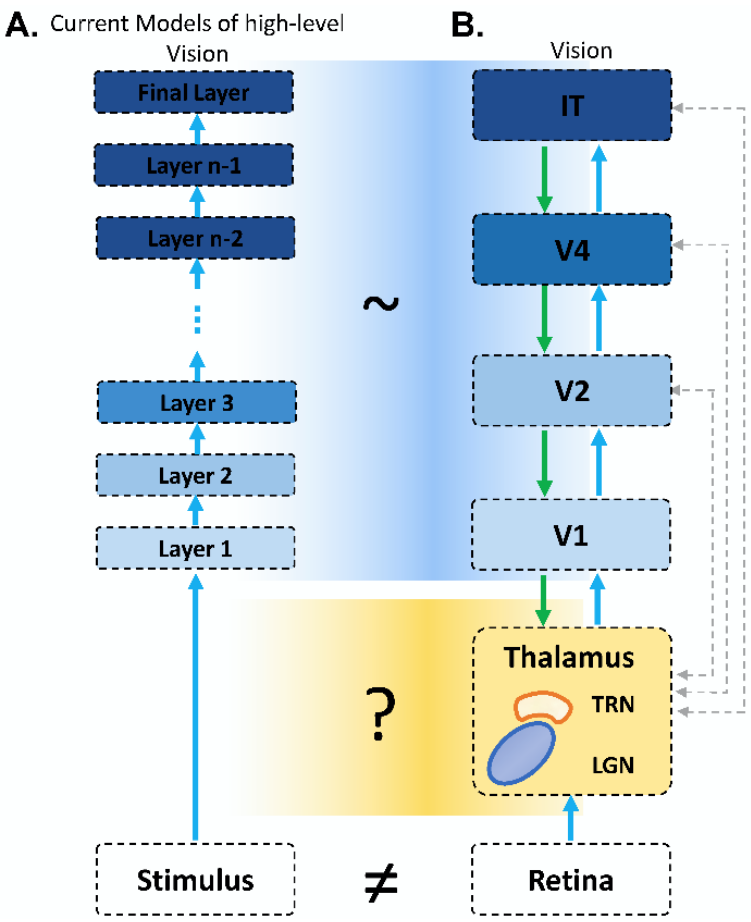

Figure 5. LGN Has Largely Been Ignored in Models of High-level Vision. Models of high-level vision mostly consist of several hierarchically organized computational layers/stages (A). In the early stages of models, low-level features such as oriented edges are usually extracted, implementing a functional approximation of neurons in V1. Motivated by the Simple-to-Complex model of Hubel and Wiesel (Fukushima, 1988; Hubel and Wiesel, 1962; Riesenhuber and Poggio, 1999), the mid- and high-level layers extract more complex features by pooling over the output of lower layers. The number of layers and the processing may slightly vary depending on the structure of network but the overall goal (e.g., object recognition) is the same. These models can explain some of functional properties of several cortical areas such as V1, V4, and IT. The processing in such models is performed in a fully feedforward manner (blue arrows) without any feedback connections between or within layers while biological vision (B) includes massive feedback connections between different cortical areas as well as corticothalamic feedback connections (green arrows). There are also small feedback projections from extrastriate cortices to LGN (grey dashed lines) whose functional significance is not known. More importantly, current models of high-level vision do not include a separate stage for LGN processing. However, we have seen throughout this review that LGN is substantially more complex than what has been thought. Purely feedforward models without LGN processing clearly cannot be the entire story. Finally, current feedforward models almost entirely ignore retinal processing (which we did not review) and directly send the stimulus to a V1-like layer for edge detection, entirely bypassing important processing happening in early vision.