8

The Lateral Geniculate Nucleus

S. Murray Sherman

The circuitry of the thalamus is among the most thoroughly studied and best understood exemplars of functional connectivity in the brain (for details, see Sherman and Guillery, 2006; Jones, 2007). Here, we shall focus on the A laminae of the cat's lateral geniculate nucleus (LGN), which represents the relay of retinal input to cortex, because this has proven to be an excellent model for thalamus. There are two major payoffs for understanding this circuit: the basic plan revealed by LGN circuitry seems to be applied throughout thalamus, with some modifications, and so this provides general insights into overall thalamic functioning; and circuit principles first appreciated in the LGN may apply to other brain circuits.

Basic Cell Types

As shown in Figure 8.1A and B, the basic circuit in LGN is comprised of three main cell types, with one of these having two distinct subtypes. The *relay cell* receives direct input from the retina and projects to visual cortex. It is a classical excitatory neuron that uses glutamate as its neurotransmitter. In the A laminae of the cat's LGN, there are two relay cell classes, X and Y, and these represent subtle differences in circuitry. These are recipient, respectively, of input from distinct retinal ganglion cell classes also known as X and Y, and thus the relay cells are incorporated into two parallel streams of information from retina to cortex (Sherman, 1982).

The interneuron is a local, GABAergic, inhibitory cell that resides in the A laminae among relay cells. With some exceptions, the relay cell to interneuron ratio throughout thalamus and in all mammalian species is roughly 3 to 1 (Sherman and Guillery, 2006; Jones, 2007). The interneuron is an unusual cell,

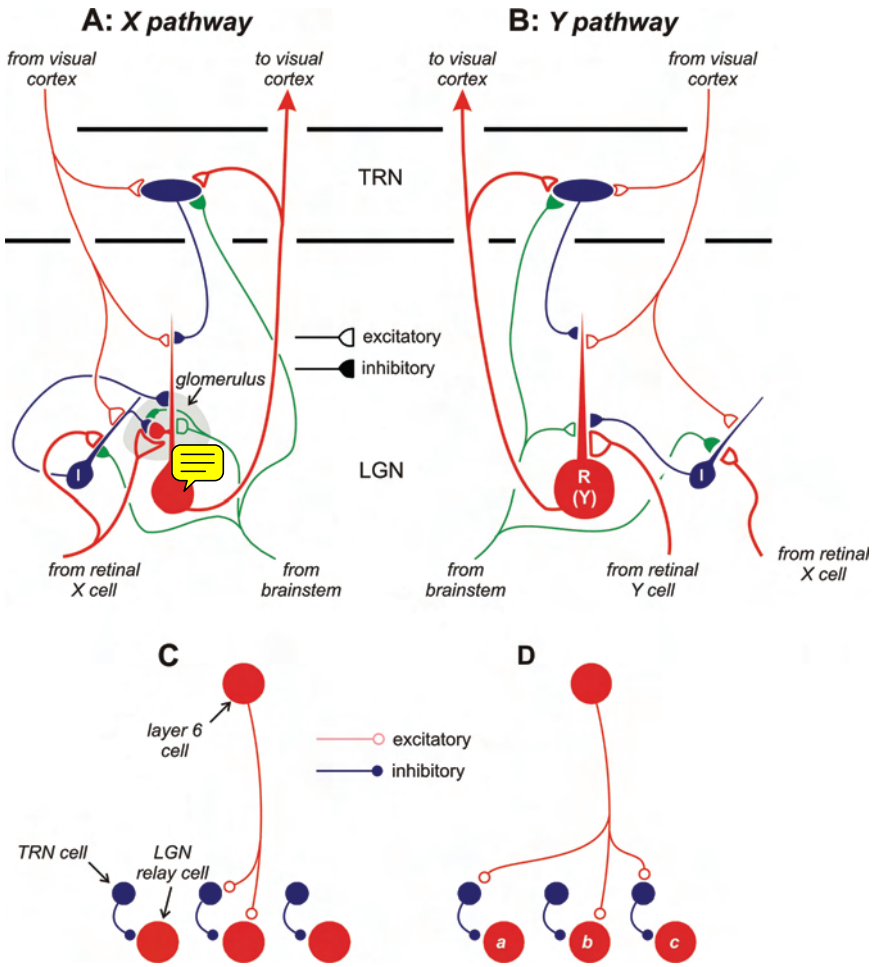

FIGURE 8–1. Overview of circuitry of LGN. (*A* and *B*) Detailed circuitry for X and Y relay cells of the LGN of the cat. (Redrawn from Sherman and Guillery, 2004). (*C* and *D*) Two possible patterns among others for corticogeniculate projection. (*C*) shows excitation and feedforward inhibition. (*D*) shows a more complicated pattern whereby a cortical axon can excite some relay cells directly (e.g., cell *b*) and inhibit others indirectly (e.g., cells *a* and *c*). I, interneuron; LGN, lateral geniculate nucleus; R, LGN relay cell; TRN, thalamic reticular nucleus. (Redrawn from Sherman and Guillery, 2004)

because while it has a conventional axon producing synaptic outputs, most of its synaptic efferents derive from its distal dendrites (Sherman, 2004). Furthermore, these dendritic terminals are both presynaptic to relay cells and postsynaptic to retinal or brainstem inputs (see also the section "Triads and Glomeruli") and are thus the only synaptic terminal type in thalamus with a postsynaptic status. One suggestion for the interneuron's function is that the axonal output is controlled conventionally by proximal inputs that determine

the cell's firing, but that the inputs onto the dendritic terminals are so far electronically from the soma that they have little effect on the axonal output (Sherman, 2004). In this sense, the interneuron can multiplex by having separate input/output circuits operating through the axonal and dendritic terminals. As shown in Figure 8.1A, the retinal input to interneurons that determines its receptive field properties and axonal output is from axons of the X type (Sherman and Friedlander, 1988).

Finally, the cell located in the thalamic reticular nucleus (TRN),[1] a shell of neurons adjacent to the thalamus and through which all thalamocortical and corticothalamic axons pass, is another local, GABAergic, inhibitory cell.

Circuitry

General Circuit Features

Figure 8.1A and B also shows the major inputs to the relay cells. In addition to the retinal input, which represents the information relayed to cortex, there are a number of other inputs. These include inhibitory inputs from interneurons and TRN cells, a feedback, glutamatergic input from visual cortex, and assorted inputs from scattered cells in the brainstem. This last group represents mostly cholinergic inputs, but there are also inputs from serotonergic, noradrenergic, and histaminergic cells in the brainstem (for further details, see Sherman and Guillery, 2006; Jones, 2007).

Figure 8.2A shows a more detailed view of how these inputs innervate relay cells. Note that the different input types innervate different parts of the dendritic arbor (reviewed in Sherman and Guillery, 2006; Jones, 2007). Thus, retinal, brainstem, and interneuronal inputs innervate proximal dendrites, while cortical and TRN inputs innervate distal dendrites. Generally, it is thought the more distal the input, the less effective it is due to properties of electrotonic transmission, but this assumes passive cable properties of the relay cell dendrites, and this is one issue for which sufficient relevant information is unavailable. Thus, the significance of the differential distribution of synaptic inputs onto relay cell dendritic arbors remains to be fully determined. One difference between X and Y cells is the relationship of triadic inputs in glomeruli seen in X but not Y cells (Sherman, 2004; Sherman and Guillery, 2006); triads and glomeruli are considered more fully in the section "Triads and Glomeruli." Also note that interneuron axons, whose output is dominated by retinal X input, inhibit both X and Y relay cells, so at the level of LGN, there is some inhibitory mixture of these pathways.

Figure 8.2A also represents each input type in roughly proportional numbers. Each relay cell receives approximately 5000 synaptic inputs (Sherman and Guillery, 2006). Of these, about 5% are retinal in origin, and most of the rest are roughly equally divided among cortical, brainstem, and local

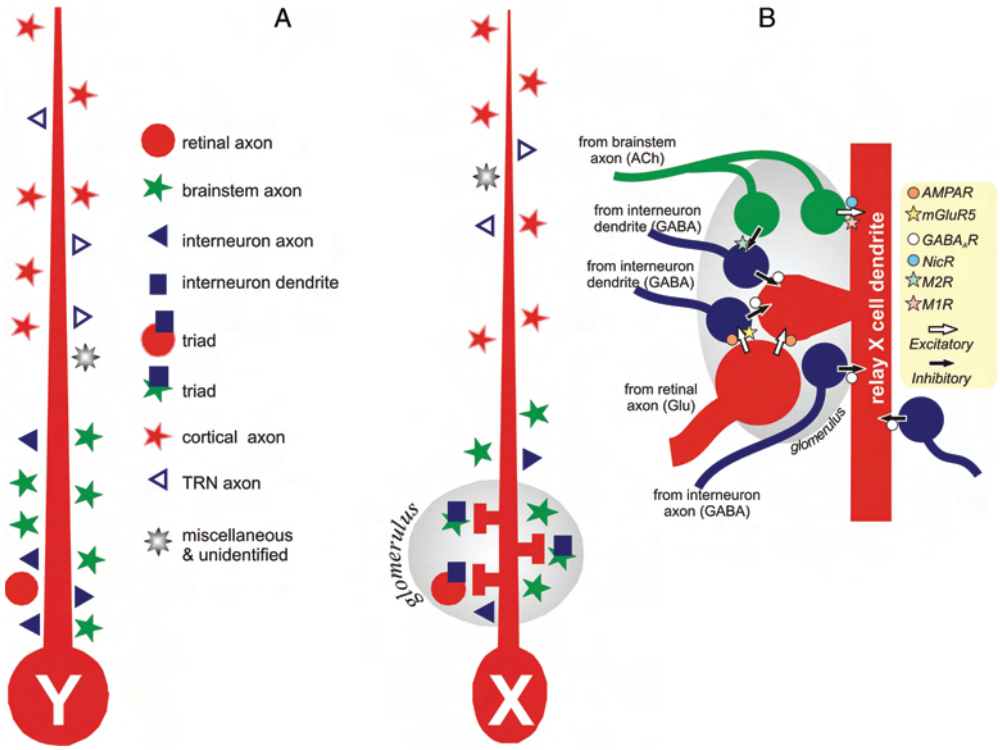

FIGURE 8–2. Schematic views of synaptic inputs onto relay cells and in triads within glomeruli. (*A*) Inputs onto schematic, reduced dendrite of an X and Y cell. Synaptic types are shown in relative numbers and locations. The main difference between X and Y cells is that the former has most retinal input filtered through triads in glomeruli, while the latter has a simpler pattern of retinal input. The triadic inputs and glomeruli typically occur on dendritic appendages of X cells. (Redrawn from Sherman and Guillery, 2004). (*B*) Triads and glomerulus. Shown are the various synaptic contacts (arrows), whether they are inhibitory or excitatory, and the related postsynaptic receptors. The "classical" triad includes the lower interneuron dendritic terminal and involves the retinal terminal. Another type of triad includes the upper interneuron dendritic terminal and also involves the brainstem terminals. For simplicity, the NMDA receptor on the relay cell postsynaptic to the retinal input has been left off. ACh, acetylcholine; AMPAR, (RS)-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor; GABA, γ-aminobutyric acid; $GABA_AR$, type A receptor for GABA; Glu, glutamate; M1R and M2R, two types of muscarinic receptor; mGluR5, type 5 metabotropic glutamate receptor; NicR, nicotinic receptor; TRN, thalamic reticular nucleus. (Redrawn from Sherman, 2004)

GABAergic sources (Sherman and Guillery, 2006). Finally, roughly 5% cannot be identified as one of these major types.

Drivers and Modulators

At first glance the above ratios of different inputs to relay cells seem quite surprising, because the major information to be relayed is retinal, and yet this

comprises only 5% of the synaptic input. Although small in number anatomically, retinal input is nonetheless quite powerful in driving relay cells, and so we refer to this as the *driver* input (Sherman and Guillery, 1998, 2006). If the retinal driver input represents the main information to be relayed, what of the other nonretinal inputs? These have been lumped together as *modulators*, because their main role seems to be one of modulating retinogeniculate transmission.

Driver (retinal) and modulator (nonretinal) inputs can be distinguished on a number of criteria (for a complete list and other details, see Sherman and Guillery, 1998, 2006), but the main ones are as follows:

- Driver inputs have large, powerful synapses, while modulator inputs are small and weak.
- Driver synapses have a high probability of release and produce large excitatory postsynaptic potentials (EPSPs) with paired-pulse depression, while modulator synapses generally have a low probability of release and produce small EPSPs (or inhibitory postsynaptic potentials [IPSPs]) with paired pulse facilitation.
- Driver synapses activate only ionotropic receptors (iGluRs; mostly AMPA but also NMDA), while modulator synapses in addition activate metabotropic receptors (i.e., metabotropic glutamate receptors, mGluRs, for cortical input, $GABA_B$ receptors for interneuron and TRN input, muscarinic receptors for brainstem input, etc.; for more information on metabotropic receptors, see Kandel et al., 2000).

Modulation can take many forms, including affecting the gain of retinogeniculate transmission, altering relay cell excitability, and controlling a number of voltage- and time-gated ionic conductances, such as I_T, I_A, and I_h (Jahnsen and Llinás, 1984; McCormick, 2004; Sherman and Guillery, 2006). I_T, a Ca^{2+} current, is particularly interesting, because it determines in which of two firing modes, burst or tonic, relay cells respond to retinal input, and this has important consequences for the relay of information (Sherman, 2001). If a relay cell is depolarized sufficiently (in amplitude and time), I_T is inactivated, and the cell responds in tonic mode; if instead the cell is sufficiently hyperpolarized, inactivation of I_T is removed, and the next effective excitatory input will activate I_T, leading to a burst of action potentials in the relay cell. The activation of metabotropic receptors is particularly important here, because they produce prolonged EPSPs or IPSPs, lasting hundreds of milliseconds to several seconds, and thus these produce membrane potential changes sufficient in amplitude and time to control the inactivation state of I_T and other such conductances. Ionotropic receptor activation typically produces postsynaptic potentials that are too brief to have a major effect on the inactivation state of these conductances.

This division of inputs to relay cells into drivers and modulators seems to be a general principle of thalamus, and identifying the driver input to a

thalamic nucleus identifies the information to be relayed. The key point is that inputs to relay cells do not act equally as some sort of anatomical democracy. A study of most circuits laid out in textbooks will reveal that they are based on anatomical numbers almost exclusively. If one were just to consider numbers as the important variable, one might conclude that the LGN relays information mainly from brainstem cholinergic inputs, since these produce 30% of synapses onto relay cells, while the small number of retinal inputs represents an obscure, unimportant input. An open question is the extent to which this driver versus modulator division of inputs to neurons extends to other areas of the brain, such as cortex (Lee and Sherman, 2008).

Effects of Extrinsic Modulatory Input

The two major extrinsic sources of modulatory input arrive from the brainstem and visual cortex.

Brainstem Input

The brainstem input, as noted earlier, is mostly cholinergic. A glance at Figure 8.1A shows an unusual feature of this input: different branches of the same brainstem axon excite relay cells and inhibit the inhibitory GABAergic cells (Sherman and Guillery, 2006). This remarkable trick is managed due to the different postsynaptic receptors involved. Relay cells respond to the cholinergic input with a depolarizing nicotinic receptor as well as one type of muscarinic receptor (M1), activation of which closes a leak K^+ channel, resulting in further depolarization. In contrast, interneurons and TRN cells respond mainly with another type of muscarinic receptor (M2) that leads to the opening of K^+ channels, resulting in a hyperpolarization. The net result is that increased activity in these brainstem axons leads to a direct depolarization of relay cells and indirect depolarization due to inhibition of GABAergic inputs to these cells. Thus, brainstem activation makes relay cells more responsive and less bursty (because the depolarization inactivates I_T). Indeed, as animals pass from sleep through drowsiness to vigilance, these cholinergic brainstem cells become more active, and LGN cells, in turn, become more active and less bursty (Datta and Siwek, 2002).

Less is known about the other modulatory neurotransmitter systems, such as serotonergic, noradrenergic, and histaminergic inputs, but their overall effects seem similar to those of the cholinergic inputs (McCormick, 2004; Sherman and Guillery, 2006).

Cortical Input

The cortical input, which emanates from layer 6 cells, is glutamatergic. Its overall effect on relay cells is difficult to predict and depends on the details

of circuitry, details that remain mostly obscure. That is, different branches of the same axon innervate relay cells and the local GABAergic cells, exciting all. Thus, from Figure 8.1A, it appears that the effect of this input is to directly excite and indirectly inhibit relay cells, but this may be an oversimplification.

As noted, the actual effects depend on circuit details, and two variants among others are illustrated in Figure 8.1C and D. Figure 8.1C shows the conventional view, which is a feedback inhibitory circuit. Since activation of the corticothalamic axons in this arrangement will provide a somewhat balanced direct depolarizing and indirect hyperpolarizing response in the relay cell, at first glance this might seem to be a fairly useless circuit. However, as Chance et al. (2002) have shown, increasing a fairly balanced inhibitory and excitatory input to a cell reduces its excitability, or in this case, activation of the corticothalamic axon reduces the gain of retinogeniculate transmission, a very effective modulatory function. This is achieved without a major change in the relay cell's membrane potential, partly by increasing synaptic conductance, which reduces neuronal input resistance, and partly by the increase in synaptic noise. Figure 8.1D shows something else altogether. In this circuit, activation of the corticothalamic axon directly excites some relay cells (e.g., cell *b*), thereby promoting tonic firing, while it indirectly inhibits others (e.g., cells *a* and *c*), promoting burst firing. There is some indirect evidence for such a circuit (Tsumoto et al., 1978).

Obviously, we must have a much better understanding of the details of corticothalamic circuitry before we can really understand its function. One key to this understanding is an appreciation that there may be no one function, but rather, many, and that multiple variations in the circuit such as those shown in Figure 8.1C and D, and other possible variants not considered here, may participate in the corticothalamic feedback.

Triads and Glomeruli

General Structure

Triads and glomeruli are ubiquitous features of thalamus, related to interneurons and found in most nuclei and species.[2] This is shown schematically in Figure 8.2B. A triad is a synaptic configuration comprised of three elements. The most common involves a single retinal terminal that contacts both a dendritic terminal of an interneuron and a relay X cell, with the dendritic terminal contacting the same X cell (Sherman, 2004). The three synapses involved are retinal to dendritic terminal, retinal to relay cell, and dendritic terminal to relay cell. A variant of this involves a cholinergic brainstem axon that functionally replaces the retinal terminal: the brainstem axon contacts the interneuronal dendritic terminal and a relay X cell axon, via different brainstem terminals, with the dendritic terminal contacting the same relay cell.

All of these triadic contacts (plus some other simpler synapses involving axonal inputs onto relay X cells, mostly from interneurons) are contained within a glomerulus, which is thus a site of complex synaptic interaction involving inputs to X cells. Y cells are generally devoid of triadic inputs and glomeruli, so this appears to be a common variant in thalamic circuitry. What makes the glomerulus further distinct is the fact that the entire synaptic structure is contained within a single glial sheath (Szentágothai, 1963; Sherman and Guillery, 2006). Generally, each individual synapse in the brain is surrounded by a glial sheath, the function of which is obscure but is thought to play some role in synaptic regulation and neurotransmitter uptake (Bacci et al., 1999). Whatever that role for individual synapses may be, it appears to be missing in glomeruli because the individual synapses are naked. This has led to a number of hypotheses, one of which is that neurotransmitters released in the glomeruli are not limited to their immediately adjacent targets but may spill over to affect other processes as well. Whatever its functional significance, the glomerulus is a prominent component of LGN circuitry, and it seems likely it plays an important role in modulating retinogeniculate transmission.

Triadic Synaptic Properties: Retinal Inputs

One key to understanding the triad is appreciating the properties of the component synapses. We can start with a consideration of the "classical" triad involving retinal input and ask how it affects retinogeniculate transmission. At first glance, it seems organized in a feedforward inhibitory manner, with a direct monosynaptic EPSP in the relay cell followed by a disynaptic IPSP, perhaps organized to curtail prolonged excitatory input or provide gain control of retinogeniculate transmission much like the circuit of Figure 8.1C.

However, a look at the postsynaptic receptors involved suggests another, more interesting function. Note that the retinal-to-relay cell synapse activates only iGluRs, whereas the relay cell-to-dendritic terminal activates both iGluRs and mGluRs (Cox and Sherman, 2000; Sherman, 2004; Govindaiah and Cox, 2006). Activation of iGluRs typically occurs even at low rates of afferent activity, and so one would expect that at low retinal firing rates a simple feedforward inhibitory circuit would be activated. Activation of mGluRs usually requires higher rates of afferent activity, and so the prediction is that, as the retinal input fires at higher levels, extra inhibition is brought to bear via activation of the mGluRs. Furthermore, this extra inhibition evoked by higher retinal activity would be long-lasting due to the prolonged effects of activation of mGluRs; estimates indicate an effect that would outlast retinal activity by several seconds (Govindaiah and Cox, 2006).

This overall effect, including its time course, seems an ideal neuronal substrate for the function of contrast adaptation (Sclar et al., 1989; Demb, 2002; Solomon et al., 2004). This is an important property of vision, namely, the

ability to adjust overall contrast sensitivity to the dynamic range of the visual stimuli, decreasing contrast sensitivity during epochs of high contrast, and vice versa. Evidence exists that retinal, LGN, and cortical circuitry all contribute to this (Sclar et al., 1989; Demb, 2002; Solomon et al., 2004). In general, retinal firing rates increase monotonically with increasing contrast in the stimulus. Thus, as increased contrast raises the firing of retinal inputs past a level sufficient to activate mGluRs on the interneuron dendritic terminals, extra inhibition of the relay cell kicks in, making the cell less sensitive, and this would outlast the increased period of contrast and elevated retinal firing by several seconds, all of which is precisely what occurs with contrast adaption. Note, however, that this property should be limited to the X system, since LGN Y cells lack triadic inputs. This, however, remains a hypothesis for the X system that has yet to be tested.

Triadic Synaptic Properties: Brainstem Cholinergic Inputs

The other sort of triad involving brainstem cholinergic inputs (see Fig. 8.2A) seems easier to understand (Cox and Sherman, 2000; Sherman, 2004). The terminal contacting the relay X cell activates M1 (metabotropic) and nicotinic (ionotropic) receptors, both producing excitation. The terminal contacting the interneuron dendritic terminal, in contrast, activates M2 (metabotropic) receptors, thereby inhibiting the terminal. Thus, in this circuit, just like that described in Figure 8.1A, the cholinergic brainstem input directly excites and indirectly disinhibits the relay X cell.

Concluding Remarks

As noted, LGN circuitry reflects that seen throughout thalamus, with some variations between species and nuclei. Thus, an appreciation of this circuitry helps us to understand the function of thalamus more generally. If we consider the role of the LGN in the visual system from the perspective of information processing, it appears to have a rather unique function. We can understand information processing at one level by determining how each stage in visual processing enhances and elaborates receptive field properties as one ascends the synaptic hierarchies (Van Essen and Maunsell, 1983; Hubel and Wiesel, 1998). Thus, as one passes within retina from receptors through interneurons to ganglion cells, at each stage receptive fields become more elaborate. The same is true as one ascends the hierarchy from LGN to and through the various levels of cortical processing. One clear exception to this pattern is the retinogeniculate synapse, because there seems little receptive field elaboration here. That is, the basic center-surround receptive field of the ganglion cell is seen also in the LGN relay cell, with only minor changes.

This means either that the retinogeniculate synaptic level has little real function (and the LGN was often in the past seen as an uninteresting, machine-like relay), which on the face of it seems absurd, or that this synapse has a unique role in visual processing. That role is not to further elaborate receptive field properties but rather to control the flow of retinal information to cortex. This control is accomplished via modulatory inputs that affect retinogeniculate transmission. One can see this control in a number of different forms, from obvious to fairly subtle. For instance, a glance at Figure 8.1A reveals that, if the local GABAergic (interneuron and TRN) cells are sufficiently active, relay cells will be so inhibited as to fail to relay any retinal information, and in this case, the thalamic gate is shut; conversely, silencing of the local GABAergic cells would open the gate. More subtle examples have been discussed earlier and include more continuously variable gain control of retinogeniculate transmission and control of burst versus tonic response modes. Many other modulatory functions are likely.

Behaviorally, control of information transfer might be related to arousal and attentional mechanisms. Indeed, LGN as well as other thalamic nuclei have been implicated in such behavioral phenomena (LaBerge, 2002; Kastner et al., 2004; McAlonan et al., 2006, 2008). This may well be the main role of thalamus, including LGN. All information reaching cortex must pass through thalamus, and as far as we know, all cortical regions receive a thalamic input. Thus, thalamus appears to play a key role in the flow of information to cortex, and this flow is related to behavioral states such as wakefulness and selective attention. This overview of LGN circuit properties is meant to provide some insights into how this function is achieved. While much is known, clearly this remains a ripe research area so that we can improve our knowledge of these thalamic relay functions.

1. This structure in the cat is actually named the perigeniculate nucleus, but it appears that this is indeed part of the TRN.
2. Exceptions seem to be rats and mice, which have interneurons in their LGN, but few if any are found in other thalamic nuclei (Arcelli et al., 1997). Because triads and glomeruli seem related to interneuronal dendritic terminals, these structures are also rare in these animals outside of the LGN. Other mammals so far studied, including other rodents, generally have interneurons, triads, and glomeruli throughout thalamus.

REFERENCES

Arcelli P, Frassoni C, Regondi MC, De Biasi S, Spreafico R (1997) GABAergic neurons in mammalian thalamus: a marker of thalamic complexity? *Brain Res Bull* 42:27–37.

Bacci A, Verderio C, Pravettoni E, Matteoli M (1999) The role of glial cells in synaptic function. *Philos Trans R Soc Lond B Biol Sci* 354:403–409.

Chance FS, Abbott LF, Reyes A (2002) Gain modulation from background synaptic input. *Neuron* 35:773–782.
Cox CL, Sherman SM (2000) Control of dendritic outputs of inhibitory interneurons in the lateral geniculate nucleus. *Neuron* 27:597–610.
Datta S, Siwek DF (2002) Single cell activity patterns of pedunculopontine tegmentum neurons across the sleep-wake cycle in the freely moving rats. *J Neurosci Res* 70: 611–621.
Demb JB (2002) Multiple mechanisms for contrast adaptation in the retina. *Neuron* 36: 781–783.
Govindaiah G, Cox CL (2006) Metabotropic glutamate receptors differentially regulate GABAergic inhibition in thalamus. *J Neurosci* 26:13443–13453.
Hubel DH, Wiesel TN (1998) Early exploration of the visual cortex. *Neuron* 20:401–412.
Jahnsen H, Llinás R (1984) Electrophysiological properties of guinea-pig thalamic neurones: an *in vitro* study. *J Physiol (Lond)* 349:205–226.
Jones EG (2007) *The Thalamus*, 2nd ed. Cambridge, England: Cambridge University Press.
Kandel ER, Schwartz JH, Jessell TM (2000) *Principles of Neural Science*. New York: McGraw Hill.
Kastner S, O'Connor DH, Fukui MM, Fehd HM, Herwig U, Pinsk MA (2004) Functional imaging of the human lateral geniculate nucleus and pulvinar. *J Neurophysiol* 91: 438–448.
LaBerge D (2002) Attentional control: brief and prolonged. *Psychol Res* 66:220–233.
Lee CC, Sherman SM (2008) Synaptic properties of thalamic and intracortical inputs to layer 4 of the first- and higher-order cortical areas in the auditory and somatosensory systems. *J Neurophysiol* 100:317–326.
McAlonan K, Cavanaugh J, Wurtz RH (2006) Attentional modulation of thalamic reticular neurons. *J Neurosci* 26:4444–4450.
McAlonan K, Cavanaugh J, Wurtz RH (2008) Guarding the gateway to cortex with attention in visual thalamus. *Nature* 456:391–394.
McCormick DA (2004) Membrane properties and neurotransmitter actions. In: Shepherd GM, ed. *The Synaptic Organization of the Brain*, 3rd ed., pp. 39–77. New York: Oxford University Press.
Sclar G, Lennie P, DePriest DD (1989) Contrast adaptation in striate cortex of macaque. *Vision Res* 29:747–755.
Sherman SM (1982) Parallel pathways in the cat's geniculocortical system: W-, X-, and Y-cells. In. Morrison AR and Strick PL (eds.), *Changing Concepts of the Nervous System*, pp. 337–359. Academic Press, Inc.
Sherman SM (2001) Tonic and burst firing: dual modes of thalamocortical relay. *Trends in Neurosci* 24:122–126.
Sherman SM (2004) Interneurons and triadic circuitry of the thalamus. *Trends in Neurosci* 27:670–675.
Sherman SM, Friedlander MJ (1988) Identification of X versus Y properties for interneurons in the A-laminae of the cat's lateral geniculate nucleus. *Exp Brain Res* 73: 384–392.
Sherman SM, Guillery RW (1998) On the actions that one nerve cell can have on another: distinguishing "drivers" from "modulators". *Proc Natl Acad Sci USA* 95:7121–7126.
Sherman SM, Guillery RW (2004) Thalamus. In: Shepherd GM, ed. *The Synaptic Organization of the Brain*, 3rd ed., pp 311–359. New York: Oxford University Press.
Sherman SM, Guillery RW (2006) *Exploring the Thalamus and Its Role in Cortical Function*. Cambridge, MA: MIT Press.

1 Solomon SG, Peirce JW, Dhruv NT, Lennie P (2004) Profound contrast adaptation early in
2 the visual pathway. *Neuron* 42:155–162.
3 Szentágothai J (1963) The structure of the synapse in the lateral geniculate nucleus. *Acta*
4 *Anat* 55:166–185.
5 Tsumoto T, Creutzfeldt OD, Legendy CR (1978) Functional organization of the cortifu-
6 gal system from visual cortex to lateral geniculate nucleus in the cat. *Exp Brain Res*
7 32:345–364.
8 Van Essen DC, Maunsell JHR (1983) Hierarchical organization and functional streams in
9 the visual cortex. *Trends in Neurosci* 6:370–375.