Exp Brain Res (1988) 73: 384–392

Experimental Brain Research

Identification of X versus Y properties for interneurons in the A-laminae of the cat's lateral geniculate nucleus

S.M. Sherman[1] and M.J. Friedlander[2]

[1] Department of Neurobiology and Behavior, State University of New York, Stony Brook, NY 11794–5230, USA
[2] Neurobiology Research Center, University of Alabama at Birmingham, Birmingham, AL 35294, USA

Summary. Roughly 25% of the neurons in the A-laminae of the cat's lateral geniculate nucleus are local interneurons, while the remaining 75% are relay cells that project to the visual cortex. The interneurons form the focus of our study. The relay cells are either X or Y cells and are thereby integral links in the parallel and independent retino-geniculocortical X and Y pathways. Little is known about the response properties of interneurons, largely because it is difficult to identify them clearly during electrophysiological recording. However, they can be identified by morphological criteria. We thus studied their response properties by recording intracellularly from geniculate neurons to characterize them and then injecting them with horseradish peroxidase (HRP); the HRP labeling subsequently allowed us to distinguish relay cells from interneurons. In this manner, we studied 171 relay cells (83 X and 88 Y) and 15 interneurons. The response properties tested for each of the interneurons were indistinguishable from those of the relay X cells. We conclude that these interneurons are directly innervated by retinogeniculate X axons and are firmly embedded in the X pathway. We found no evidence for interneurons in the Y pathway.

Key words: Interneurons – X cells – Y cells – Lateral geniculate nucleus – Visual pathways – Thalamus

Introduction

The main retinal projection to the cerebral cortex via the thalamus in the cat contains several parallel neuronal streams. Chief and best characterized among these are the X and Y pathways that pass through laminae A and A1 of the dorsal division of the lateral geniculate nucleus. These pathways originate with retinal ganglion X and Y cells, the axons of which proceed to laminae A and A1, where they innervate the appropriate relay cells. Typically, each retinogeniculate X axon innervates geniculate relay X cells but not Y cells, and the converse is true for each Y axon. Very little mixing occurs between these X and Y pathways in retinogeniculate circuitry (Stone et al. 1979; Sherman and Spear 1982; Sherman 1985). The geniculocortical components of these parallel neuronal streams thus convey signals that are fairly independent of one another. Different functions in terms of the neuronal processing that subserves visual perception have been suggested for each of these pathways (Ikeda and Wright 1972; Stone et al. 1979; Lennie 1980; Sherman 1985).

Although these X and Y streams pass through geniculate laminae A and A1 with little or no mixing, the lateral geniculate nucleus is not a simple relay of retinal signals. Indeed, considerable nonretinal influence of the relay cells exists (for reviews, see Singer 1977; Sherman and Koch 1986). Among the important sources of nonretinal input to the relay cells are the local interneurons that coexist amongst the relay cells in laminae A and A1. These interneurons represent 20–30% of all the neurons there, and they contain the neurotransmitter γ-aminobutyric acid, or GABA (LeVay and Ferster 1979; Fitzpatrick et al. 1984; Montero and Zempel 1985). They are believed to be the source of the feed-forward inhibition seen in the relay cells following electrical stimulation of the optic chiasm (Lindström 1982).

There is, however, considerable uncertainty concerning the nature of these interneurons. On the one hand, Lindström (1982) has described feed-forward inhibition (i.e., disynaptic inhibition) after electrical activation of the optic chiasm for both relay X and Y cells, from which he suggests that interneurons

Offprint requests to: S.M. Sherman (address see above)

should be present in both the X and Y pathways. This judgment supports an earlier one offered by Singer and Bedworth (1973), who concluded that their observed inhibition of geniculate relay X cells from activation of retinogeniculate Y axons most likely passes through interneurons driven by these Y axons. On the other hand, electron microscopic evidence from electrophysiologically identified geniculate neurons suggests that only relay X cells receive prominent input from interneurons (Wilson et al. 1984; Hamos et al. 1985, 1987). This latter observation suggests the possibility that interneurons are primarily or exclusively associated with the X pathway.

More direct attempts at investigating functional properties of interneurons suffer from the difficulty in unambiguously identifying them. Several studies have attempted an electrophysiological identification based on the failure to activate the axons of geniculate neurons antidromically from electrical stimulation of visual cortex. Such studies claim that both X and Y interneurons exist (Dubin and Cleland 1977; Lindström 1982). This strategy is flawed, because it is based on a negative finding. Indeed, many X and Y cells identified in this manner and subsequently labeled by intracellular iontophoresis of horseradish peroxidase (HRP) reveal somata too large to be interneurons and/or axons that enter the optic radiations (Friedlander et al. 1981; also our unpublished data).

While there are problems with the electrophysiological criteria generally used to identify interneurons in the lateral geniculate nucleus, they can be more readily identified with morphological criteria as the class 3 cells described by Guillery (1966) based on Golgi impregnations (see also Discussion). These cells have thin, sinuous dendrites with prominent clusters of bouton-like appendages. The dendrites have an axoniform appearance, and indeed their bouton-like appendages are the terminals that give rise to a major synaptic output of the cell (Famiglietti and Peters 1972; Hamos et al. 1985). These interneurons thus have many presynpatic dendritic terminals. Finally, class 3 cells have been identified as the neurons containing GABA that fail to transport HRP retrogradely from visual cortex (Fitzpatrick et al. 1984; Montero and Zempel 1985; see also Discussion). This fairly unambiguous morphological identification of interneurons as class 3 cells permits a retrospective means of studying their response properties. That is, cells can be studied electrophysiologically with micropipettes filled with HRP, which is subsequently iontophoresed into the cell (e.g., Friedlander et al. 1981). Once the morphological features of the cell are determined, it is possible to amass information retrospectively about the interneuron's response properties.

This is, however, an inefficient means of studying interneurons, since one cannot know during the recording whether or not an interneuron is under study. Also, the small size of these neurons added to their minority status probably biases against the likelihood that a given neuron being recorded is an interneuron. We have thus not designed experiments specifically to study interneurons with this approach. However, during the course of experiments aimed at other questions, our laboratories have separately accumulated an acceptable sample of interneurons labeled intracellularly with HRP following electrophysiological analysis. Since many of these properties are consistent among the cells, we have decided to describe these results briefly.

Material and methods

We have used precisely the same approach and techniques that we have described in detail in the past (Friedlander et al. 1981; Stanford et al. 1983) and shall thus relate these very briefly. Normal fully adult cats were anesthetized and prepared for electrophysiological recording. We dilated their pupils with topical application of atropine sulfate and fitted their corneas with contact lenses chosen by retinoscopy to focus their eyes on the visual stimuli. For the 24–48 h recording session, we maintained the anesthesia, paralyzed the cats, artificially ventilated them, and monitored their vital signs, including end-tidal CO_2 and core body temperature; in later experiments, we also monitored blood pressure and EEG. Wound margins and pressure points were periodically infused with long-lasting local anesthetics.

We recorded activity of single neurons in laminae A and A1 of the lateral geniculate nucleus with fine-tipped micropipettes filled with a buffered solution of HRP and KCl. Both extracellular and intracellular recordings were routinely achieved in this manner. Bipolar stimulating electrodes were placed across the optic chiasm and, in some experiments, into visual cortex (areas 17, 18, and 19) as well. Visual stimuli consisted both of targets positioned manually on a frontal tangent screen and of sinusoidal gratings generated on a cathode ray tube. For these gratings, we could continuously change the contrast (up to 0.6), spatial frequency, spatial phase, and temporal frequency. We imposed different temporal frequencies either by drifting the grating or counterphase modulating it.

We classified cells as X or Y on a standard battery of tests (reviewed by Stone et al. 1979; Sherman and Spear 1982; Sherman 1985; Shapley and Lennie 1985), including response latency to electrical stimulation of the optic chiasm, linearity of spatial and temporal summation in response to visual stimuli (particularly the grating stimuli), receptive field center diameter, center/surround organization, the tonic or phasic nature of responses to sustained stimulation of the receptive field center, and responsiveness to visual targets moving at high speed. For the experiments in which stimulating electrodes were placed into the visual cortex, geniculate neurons were also tested for responses to electrical stimulation of cortex. In particular, we determined if an action potential evoked in a geniculate neuron from cortical stimulation could be canceled by a prior one traveling from that neuron in the orthograde direction. Such cancellation (or "collision") demons-

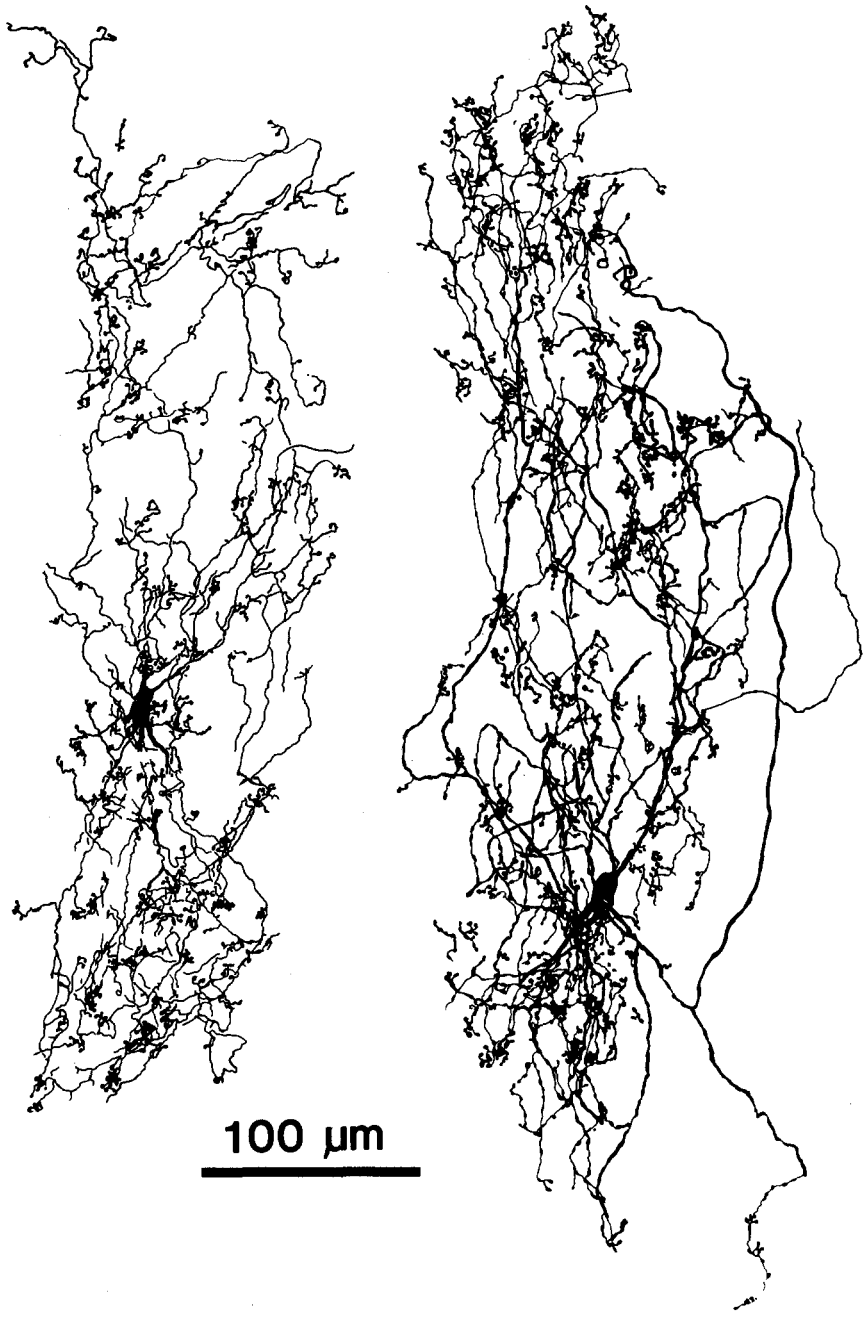

Fig. 1. Drawing of two typical examples of interneurons in the geniculate A-laminae; coronal view. These cells were studied physiologically, identified as X cells, and labeled intracellularly with HRP. The laminar borders (not shown) run horizontally, so the long axis of each dendritic arbor runs perpendicular to these borders. The cell on the left is further illustrated with photomicrographs in Fig. 2C, D

trates that the cortically evoked response was conducted antidromically down the geniculocortical axon, thus identifying the geniculate neuron under study as a relay cell.

Following physiological analysis, which was typically but not always performed extracellularly, the neuron's membrane was penetrated and HRP was iontophoresed into it. From 1–24 h after the HRP iontophoresis, the cat was more deeply anesthetized and perfused transcardially with a buffered rinse followed by fixatives. The brain was removed and histologically prepared to visualize the HRP. We cut sections coronally and reacted them with diaminobenzidine, usually with cobalt intensification. We reconstructed labeled neurons from serial sections with the aid of a drawing tube attached to a microscope.

Results

In the course of our various experiments involving the intracellular labeling of physiologically defined geniculate neurons, we have successfully recovered for morphological analysis many such cells from laminae A and A1. These include 83 relay X cells, 88 relay Y cells, and 15 cells identified as interneurons based on their morphological characteristics. Two examples of these cells are shown in Fig. 1. These 15 neurons form the main data base of this report.

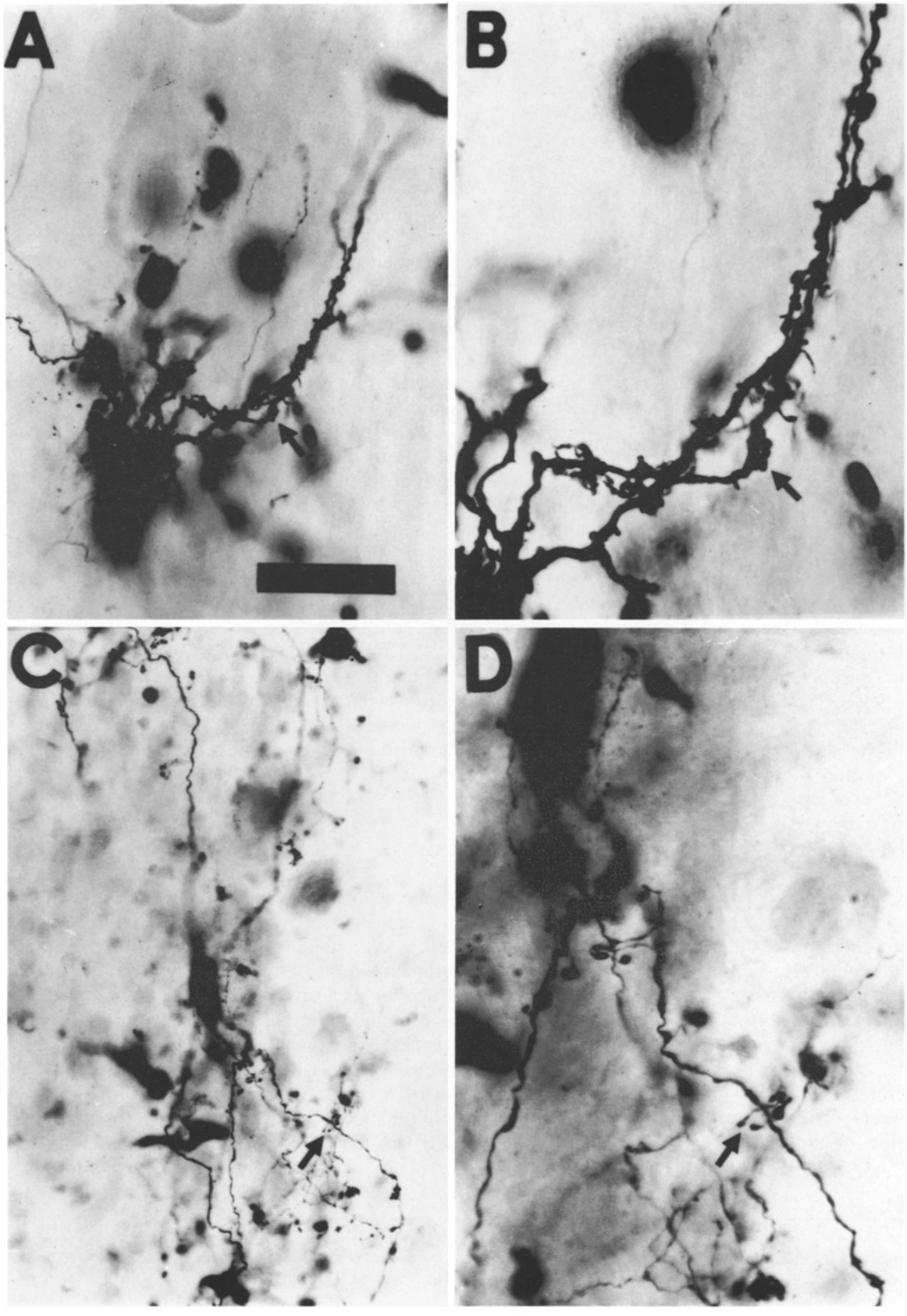

Fig. 2

Morphological features of the interneurons

The labeled interneurons have the following morphological characteristics in common. Their somata are relatively small. In cross-sectional area, the soma size range is 65–180 μm^2 compared to a range for relay cells that extends beyond 900 μm^2 (Friedlander et al. 1981; Stanford et al. 1983; Fitzpatrick et al. 1984; Montero and Zempel 1985). The dendritic arbors are richly branched, having up to 15th order branching compared to the 5th or 6th order branching common to relay cells (Bloomfield et al. 1987; Bloomfield and Sherman 1987). Each interneuron remains completely within the lamina of the neuron's origin. Typically, each of their arbors nearly completely spans the lamina dorsoventrally and is relatively narrow mediolaterally and rostrocaudally. These arbors thus have pronounced long axes that are oriented perpendicular to the laminar borders (see Fig. 1). The dendrites themselves are extremely thin and sinuous, and they give rise to numerous complex appendages (see Fig. 2C, D). These appendages include swellings or boutons that are the sites of the dendritic presynaptic outputs of these interneurons (Famiglietti and Peters 1972; Hamos et al. 1985; Montero 1986). The boutons can occur singly or in complex clusters, and they are attached to one another and to the stem dendrite by long, thin processes (often less than 0.1 μm in diameter and up to 10 μm or more in length). Finally, although at least some of these interneurons have axons (Hamos et al. 1985; Montero 1987), none of these axons can be followed beyond the dendritic arbor, and thus none are translaminar (but see Tömböl 1969).

Physiological features of the interneurons

All of the interneurons exhibited action potentials that seemed completely conventional. Of course, since we first located these cells with extracellular recording, any interneuron that lacked an action potential, such as is the case for many retinal interneurons, might have been bypassed with our methodology (see Discussion). None of these neurons could be activated antidromically by electrical stimulation of the visual cortex. While this is hardly surprising and is a primary feature of interneurons, we have evidence that many relay cells also could not be so activated from cortex with our techniques (i.e., these were neurons labeled with HRP that were identified subsequently as relay cells due to their soma sizes and/or possession of axons entering the optic radiations; see Introduction). The interneurons could be transsynaptically activated from the optic chiasm at latencies ranging from 1.6 ms to 2.9 ms. This is essentially the same latency range within which relay X cells are monosynaptically activated by electrical stimulation at the optic chiasm of their retinal afferents (Stone et al. 1979; Sherman and Spear 1982; Sherman 1985), although disynaptic activation via faster conducting retinogeniculate Y axons cannot be ruled out with our stimulation methods.

All of the tested receptive field properties of every interneuron were qualitatively indistinguishable from those of relay X cells (Stone et al. 1979; Sherman and Spear 1982; Sherman 1985; Shapley and Lennie 1985). These properties include linear summation in response to visual stimuli, relatively small receptive field centers, fairly tonic responses to sustained visual stimulation of the receptive field center, and poor responses to rapidly moving visual targets. These receptive field properties support the notion that these cells receive monosynaptic innervation from retinogeniculate X axons. Indeed, until we were able to visualize the morphological properties of these cells, we could not distinguish them from relay X cells.

Discussion

Our sample of intracellularly labeled interneurons from laminae A and A1 of the cat's lateral geniculate nucleus, accumulated over the course of different experiments in our laboratories, suggests that these cells may be exclusively part of the X pathway. All have response properties that are indistinguishable in our hands from those of relay X cells. While we found no evidence for interneurons within the Y pathway, it is not yet possible to use these negative data ro rule out some representation of interneuronal circuitry for the Y pathway.

Fig. 2A–D. Photomicrographs of class 3 cells intracellularly labeled with HRP; coronal view. **A** Lower power view of a class 3 relay X cell. **B** Higher power view of the cell illustrated in **A**. **C** Lower power view of a class 3 interneuron. **D** Higher power view of the cell illustrated in **C**. The scale in **A** represents 50 μm for **A** and **C** and 20 μm for **B** and **D**. The arrows identify the same cluster of dendritic appendates for each pair of lower and higher power photomicrographs. See text for details of the morphological differences between class 3 interneurons and relay cells

Identification of interneurons

A problem with all attempts to study interneurons electrophysiologically is the present inability to identify them unambiguously during recording with positive criteria. In Introduction and Results we have already expressed our concern with the electrophysiological criterion, which is a negative one, of failing to activate the neuron under study antidromically from visual cortex. Our strategy was to use morphological criteria for a subsequent identification of interneurons after studying their response properties, and thus it is reasonable for us to justify these criteria.

Are all interneurons class 3 cells? There is ample evidence that the geniculate interneurons within lamina A and A1 and the cells there that contain GABA are isomorphic. For instance, if large amounts of HRP are placed into visual cortex, cells in the lateral geniculate nucleus can be identified either as relay cells, by virtue of labeling with retrogradely transported HRP, or as interneurons, by virtue of no such transported label. When such retrograde HRP labeling is combined with suitable immunocytochemical techniques, every interneuron and no relay cell can be labeled for GABA or its synthetic enzyme, glutamic acid decarboxylase (Fitzpatrick et al. 1984; Montero and Zempel 1985). Each of these GABA containing cells has distinctive morphological features that are similar to those described in the present paper (Fitzpatrick et al. 1984; Montero and Zempel 1985; Montero 1986, 1987). They have small somata, thin, sinuous dendrites, and complex, stalked appendages with numerous bouton-like swellings. All of these features are characteristics of the class 3 neuron described by Guillery (1966). Therefore, the available evidence indicates that all interneurons have morphological characteristics of the class 3 cells as described by Guillery (1966).

Are all class 3 cells interneurons? While one can thus conclude with reasonable certainty that all interneurons are class 3 cells, are all class 3 cells interneurons? This is a more difficult question to answer. It is clear from retrograde tracing studies that some relay cells in the A-laminae have somata as small as those of interneurons (Fitzpatrick et al. 1984; Montero and Zempel 1985), and relay X cells often have sinuous dendrites with appendages (Friedlander et al. 1981). Because they identified as class 3 all neurons with small somata, sinuous dendrites, and appendages all along their dendritic arbor (cf. Guillery 1966), Friedlander et al. (1981) claimed that at least some of their sample of labeled class 3 cells were relay X cells.

However, it has become apparent to us in recent years that a morphological distinction can be drawn between class 3 cells that are relay cells and those that are interneurons, although such relay cells seem rare since our sample of these is limited to 4. This is best shown by Fig. 2, which compares a "class 3" relay X cell (Fig. 2A, B) with a "class 3" X interneuron (Fig. 2C, D). The relay cell was identified as class 3 because of its relatively small soma, thin sinuous dendrites, and appendages all along the dendrites. However, compared to the interneuron, the relay cell has thicker, less sinuous dendrites and shorter, stubbier appendages; also, the relay cell's appendages lack the prominent swellings that represent the presynaptic dendritic terminals of the interneuron. The differences illustrated in Fig. 2 are representative for our sample of class 3 relay cells and interneurons. Each of the 15 neurons that form the focus of the present paper has the morphology we would now exclusively attribute to interneurons.

X versus Y interneurons

The response properties of every one of our sample of interneurons are essentially the same as those of relay X cells. This firmly places these interneurons functionally within the X pathway, which is consistent with other morphological evidence that interneurons predominantly or exclusively innervate relay X cells rather than Y cells (Wilson et al. 1984; Hamos et al. 1985). Other evidence also suggests that interneuronal input provides major inhibition to the X pathway, while the Y pathway receives its major inhibition via perigeniculate cells (Eysel et al. 1987). However, some indirect electrophysiological evidence exists for the presence of interneurons within the Y pathway (Singer and Bedworth 1973; Lindström 1982). While it is certainly possible that these electrophysiological data may have been misinterpreted with respect to the presence of Y interneurons, we shall instead focus on reasons why our failure to find evidence of such interneurons should be interpreted with caution.

Sampling biases. It is possible that Y interneurons exist, but that we simply have failed to sample them. This could occur for two reasons: first, our limited sample of 15 interneurons is insufficient to observe a substantial minority of Y interneurons that actually exist; and/or second, strong biases may operate against sampling Y interneurons that are present. We shall consider each of these in turn.

If all we sampled were interneurons, a compelling argument could be made that, with only 15 neurons,

a minority population could be missed altogether. However, in addition to these interneurons, we have 83 relay X cells and 88 relay Y cells among our sample of physiologically identified and intracellularly labeled neurons. It is particularly interesting to consider the population of Y cells. These are all identified as relay cells, because: their morphological features are clearly not those of class 3 cells (see above); their somata are too large for interneurons (cf. Fitzpatrick et al. 1984; Montero and Zempel 1985); most have axons that can be followed into the optic radiations; and, in experiments for which stimulating electrodes were placed in the visual cortex, most were antidromically activated from electrical stimulation through these electrodes. While 17% of our X cells are interneurons, none of our Y cells are, and this difference is statistically significant ($p < 0.001$ on a Chi-square test). Therefore, sample size alone does not seem to be a strong reason to believe that we missed Y interneurons that are present.

However, there may be many reasons why uncontrolled electrode sampling biases operate against recording from, and thus labeling, Y interneurons. Such biases are poorly understood and are always a potential source of artifact in studies based on single cell recording (for a discussion of this, see Friedlander et al. 1981; Friedlander and Stanford 1984). One plausible bias has to do with the fact that cells in the central nervous system can operate as interneurons without requiring an axon or the ability to discharge or propagate action potentials. Examples of this are horizontal and bipolar cells in the retina. Our experimental protocol involves first detecting the presence of single cell activity via extracellular recording; the cell is then electrophysiologically analyzed and its membrane is penetrated for HRP labeling. A cell that does not fire action potentials (or even one that did so infrequently) might never be detected with our methodology. Therefore, if Y interneurons exist but do not discharge action potentials (or do so rarely), we might miss them.

One would expect action potentials to be associated with the presence of axons. At least some, and perhaps all, X interneurons possess locally ramifying axons (Hamos et al. 1985; Montero 1987). However, interneurons without axons have been described for the cat's ventral posterolateral nucleus (Ralston et al. 1988), the somatosensory equivalent of the lateral geniculate nucleus, so their presence in laminae A and A1 is not implausible. Perhaps such cells exist in the lateral geniculate nucleus and function as Y interneurons. If so, they might not generate action potentials, and we might well have missed them during our recording sessions. Such non-spiking cells could still produce inhibition in relay cells of the type that Lindström (1982) argued was due to interneurons receiving retinogeniculate input from Y axons.

Cable properties. A final proviso to the notion that interneurons do not exist within the Y pathway has to do with the cable properties of geniculate neurons. These properties are a major determinant of a neuron's integrative characteristics (i.e., how the neuron responds to various synaptic inputs). Relay X and Y cells display similar cable properties: their dendritic arbors are electrically compact, which implies that any synaptic input should have significant effects at the soma (and axon) and throughout all the intervening dendritic arbor (Bloomfield et al. 1987). In contrast, interneurons seem not to have electrically compact dendritic arbors (Bloomfield and Sherman 1987). Because of their unusual dendritic branching patterns, many localized areas of the dendritic arbor are electrically isolated from one another and from the soma; only synaptic inputs on the most proximal dendrites and soma are likely to have significant influence on the soma and axon. Bloomfield and Sherman (1987) suggest that this permits the interneuron to perform several integrative functions simultaneously: one or more for the dendritic outputs and another for the axonal output.

The axonal output is carried by action potentials, and this may be the only functional unit of the interneuron we have observed with our electrophysiological techniques. That is, because the soma and mechanisms for generating action potentials seem electrically isolated from much of the dendritic arbor, we might not detect evidence of synaptic inputs that are distally located on dendrites. These may nonetheless control the dendritic outputs. When we say that all of our interneurons display response properties of X cells, we must limit this to proximally located synaptic inputs and the axonal output. Perhaps significant inputs from retinogeniculate Y axons contact the distal dendrites of these interneurons, which would place them squarely in the Y pathway.

While this is possible and worth investigating further, all of the available evidence indicates otherwise. Much and perhaps virtually all of the retinal input that has been described onto distal dendrites of interneurons seems to derive from X axons (Wilson et al. 1984; Hamos et al. 1985, 1987). Even if significant input from Y axons did exist, the dendritic terminals of interneurons almost exclusively innervate relay X cells, with very little innervation of relay Y cells (Wilson et al. 1984; Hamos et al. 1985). Thus, while the unusual and interesting cable properties of

interneurons do limit the conclusions that can be drawn from our electrophysiological data, particularly those regarding events underlying the dendritic outputs of these cells, the available evidence does not support the notion that these outputs are a major component of the Y pathway.

Conclusions

Our major conclusion is that practically every interneuron that can be recorded with conventional electrophysiological techniques in laminae A and A1 of the cat's lateral geniculate nucleus is part of the X pathway. Limitations in our methodology prevent us from applying these data to conclude that Y interneurons do not exist. While many qualifications could be raised, most seem not to counter the notion that the Y pathway lacks a significant contribution from interneurons. The major exception to this is the possibility that substantial numbers of interneurons exist, but that they lack action potentials and would thus be invisible to conventional electrophysiological methodology. This possibility bears further investigation.

The conclusion that interneurons in the A-laminae of the cat's lateral geniculate nucleus are limited to the X pathway has some fairly obvious functional implications. As noted recently by Sherman and Koch (1986), geniculate circuitry is largely disposed to gate transmission along the retino-geniculo-cortical pathways. In general, this implies that, compared to the Y pathway, the X pathway has an extra, or at least different, means of effecting local gating. It seems plausible that selective means of gating the Y pathway at the level of the lateral geniculate nucleus also exist, although we presently lack specific evidence for this. Given the likelihood that the X and Y pathways are organized to perform somewhat different visual functions (cf. Ikeda and Wright 1972; Stone et al. 1979; Lennie 1980; Sherman 1985), this suggests the possibility that the visual system can take advantage of geniculate circuitry to alter the balance of activity between the two pathways. What this means for vision, of course, depends on the distinctive functional roles played by the X and Y pathways. However, our data suggest the possibility that much of the switching between these roles can be achieved through geniculate circuitry.

Acknowledgement. This research was supported by USPHS grants EY03038 and EY05116.

References

Bloomfield SA, Hamos JE, Sherman SM (1987) Passive cable properties and morphological correlates of neurones in the lateral geniculate nucleus of the cat. J Physiol (Lond) 383: 653–692

Bloomfield SA, Sherman SM (1987) Dendritic current flow in X cells in the cat's lateral geniculate nucleus: a comparison of relay cells and interneurons. Soc Neurosci Abstr 13: 203

Dubin MW, Cleland BG (1977) Organization of visual inputs to interneurons of lateral geniculate nucleus of the cat. J Neurophysiol 40: 410–427

Eysel UT, Papa H-C, Van Schayck R (1987) Contributions of inhibitory mechanisms to the shift responses of X and Y cells in the cat lateral geniculate nucleus. J Physiol (Lond) 388: 199–212

Famiglietti EV, 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

Fitzpatrick D, Penny GR, Schmechel DE (1984) Glutamic acid decarboxylase-immunoreactive neurons and terminals in the lateral geniculate nucleus of the cat. J Neurosci 4: 1809–1829

Friedlander MJ, Lin C-S, Stanford LR, Sherman SM (1981) Morphology of functionally identified neurons in the lateral geniculate nucleus of the cat. J Neurophysiol 46: 80–129

Friedlander MJ, Stanford LR (1984) Effects of monocular deprivation on the distribution of cell types in the LGN_d: a sampling study with fine-tipped micropipettes. Exp Brain Res 53: 451–461

Guillery RW (1966) A study of Golgi preparations from the dorsal lateral geniculate nucleus of the adult cat. J Comp Neurol 128: 21–50

Hamos JE, Van Horn SC, Raczkowski D, Sherman SM (1987) Synaptic circuits involving an individual retinogeniculate axon in the cat. J Comp Neurol 259: 165–192

Hamos JE, Van Horn SC, Raczkowski D, Uhlrich DJ, Sherman SM (1985) Synaptic connectivity of a local circuit neurone in the lateral geniculate nucleus of the cat. Nature 317: 618–621

Ikeda H, Wright MJ (1972) Receptive field organization of sustained and transient retinal ganglion cells which subserve different functional roles. J Physiol (Lond) 227: 769–800

Lennie P (1980) Parallel visual pathways. Vision Res 20: 561–594

LeVay S, Ferster D (1979) Proportions of interneurons in the cat's lateral geniculate nucleus. Brain Res 164: 304–308

Lindström S (1982) Synaptic organization of inhibitory pathways to principal cells in the lateral geniculate nucleus of the cat. Brain Res 234: 447–453

Montero VM (1986) Localization of γ-aminobutyric acid (GABA) in type 3 cells and demonstration of their source to F2 terminals in the cat lateral geniculate nucleus: a Golgi-electron microscopic GABA-immunocytochemical study. J Comp Neurol 254: 228–245

Montero VM (1987) Ultrastructural identification of synaptic terminals from the axon of type 3 interneurons in the cat lateral geniculate nucleus. J Comp Neurol 264: 268–283

Montero VM, Zempel J (1985) Evidence for two types of GABA-containing interneurons in the A-laminae of the cat lateral geniculate nucleus: a double-label HRP and GABA-immunocytochemical study. Exp Brain Res 60: 603–609

Ralston HJ III, Ohara PT, Ralston DD, Chazal G (1988) The neuronal and synaptic organization of the primate somatosensory thalamus. In: Macchi G, Bentivoglio M, Spreafico R (eds) Cellular thalamic mechanisms. Elsevier, North Holland

Shapley R, Lennie P (1985) Spatial frequency analysis in the visual system. Ann Rev Neurosci 8: 547–583

Sherman SM (1985) Functional organization of the W- X- and Y-cell pathways in the cat: a review and hypothesis. In: Progress

in psychobiology and physiological psychology, Vol 11. Academic Press, New York, pp 233–314

Sherman SM, Koch C (1986) The control of retinogeniculate transmission in the mammalian lateral geniculate nucleus. Exp Brain Res 63: 1–20

Sherman SM, Spear PD (1982) Organization of the visual pathways in normal and visually deprived cats. Physiol Rev 62: 738–855

Singer W (1977) Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system. Physiol Rev 57: 386–420

Singer W, Bedworth N (1973) Inhibitory interaction between X and Y units in the cat lateral geniculate nucleus. Brain Res 49: 291–307

Stanford LR, Friedlander MJ, Sherman SM (1983) Morphological and physiological properties of geniculate W-cells of the cat: a comparison with X- and Y-cells. J Neurophysiol 50: 582–608

Stone J, Dreher B, Leventhal A (1979) Hierarchical and parallel mechanisms in the organization of visual cortex. Brain Res Revs 1: 345–394

Tömböl T (1969) Two types of short axon (Golgi 2nd) interneurons in the specific thalamic nuclei. Acta Morphol Acad Sci Hung 17: 285–297

Wilson JR, Friedlander MJ, Sherman SM (1984) Fine structural morphology of identified X- and Y-cells in the cat's lateral geniculate nucleus. Proc R Soc Lond B 221: 411–436

Received March 1, 1988 / Accepted May 31, 1988