TOPICAL REVIEW

Corticogeniculate feedback and visual processing in the primate

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Corticogeniculate neurones make more synapses in the lateral geniculate nucleus (LGN) than retinal ganglion cells, yet we know relatively little about the functions of corticogeniculate feedback for visual processing. In primates, feedforward projections from the retina to the LGN and from the LGN to primary visual cortex are organized into anatomically and physiologically distinct parallel pathways. Recent work demonstrates a close relationship between these parallel streams of feedforward projections and the corticogeniculate feedback pathway. Here, we review the evidence for stream-specific feedback in the primate and consider the implications of parallel streams of feedback for vision.

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Introduction

A dense network of feedforward and feedback projections interconnects neurones in the lateral geniculate nucleus (LGN) of the thalamus and primary visual cortex (V1). In the feedforward pathway, LGN neurones receive visual signals from the retina and relay these signals to V1. In the feedback pathway, corticogeniculate neurones provide synaptic input to the LGN as well to the overlying cortical layers targeted by LGN projections. As a consequence of this organization, corticogeniculate neurones are in a strategic position to influence the transmission and processing of visual information en route from retina to cortex. While corticogeniculate feedback is ubiquitous across mammals, recent work in the primate is providing new insight into the possible functions of this pathway for vision. In the sections below, we review the anatomical organization and physiological properties of corticogeniculate neurones in the primate followed by a discussion of their potential contributions to visual processing. Where appropriate, results will also be presented from studies examining other animal models.

Parallel processing streams from retina to cortex

There is a striking relationship between the organization of corticogeniculate neurones and their feedback projections and the feedforward parallel processing streams. Parallel processing streams are robust in the primate visual system and are particularly evident in the LGN where three classes of neurones – the magnocellular, parvocellular and koniocellular neurones – are segregated into distinct layers (Fig. 1). These three classes of LGN neurones receive input from separate classes of retinal ganglion cells, give rise to axons that terminate in different cortical laminae, and display distinct visual physiology (reviewed in Schiller & Logothetis, 1990; Shapley, 1992; Merigan & Maunsell, 1993; Casagrande & Kaas, 1994; Casagrande, 1994; Hendry & Calkins, 1998; Hendry & Reid, 2000; Rathbun & Usrey, 2008; Briggs & Usrey, 2009a; Nassi & Callaway, 2009).

The physiology of magnocellular and parvocellular LGN neurones has been studied extensively (partial list: Schiller & Malpeli, 1978; Kaplan & Shapley, 1982, 1986; Derrington & Lennie, 1984; Benardete et al. 1992; Reid & Shapley, 1992; O’Keefe et al. 1998;
Maunsell et al. 1999; Solomon et al. 1999; Usrey & Reid, 2000; Levitt et al. 2001; Movshon et al. 2005; Alitto & Usrey, 2008; Alitto et al. 2010). Compared to parvocellular neurones, magnocellular neurones respond better to low contrast stimuli, are more sensitive to stimuli modulated at high temporal frequencies (but see Spear et al. 1994; Hawken et al. 1996), display greater extracortical surround suppression, and respond with a shorter latency following stimulus presentation. In addition, magnocellular neurones lack colour selectivity, while most parvocellular neurones in old world monkeys have long- (L) and medium- (M) wavelength opponent receptive fields. Less is known about the physiology of koniocellular neurones; however, existing evidence indicates that many are selectively modulated by short- (S) wavelength inputs and have visual responses (e.g. contrast gain, temporal-frequency tuning) that are generally intermediate to those of magnocellular and parvocellular neurones (Hendry & Reid, 2000; White et al. 2001; Chatterjee & Callaway, 2003; Tailby et al. 2008; Roy et al. 2009).

The three major classes of LGN neurones provide stream-specific input to V1 with magnocellular axons targeting layer 4Cα, parvocellular axons targeting layer 4Cβ, and koniocellular axons targeting the cytochrome-oxidase rich blobs, layer 1 and, in a subset of species including the macaque monkey, layer 4A (Fig. 2). In addition to providing input to layers 4Cα and 4Cβ, magnocellular and parvocellular LGN axons also provide input to layer 6 (described below). As a consequence of these projection patterns, corticogeniculate neurones in the primate have the opportunity to receive direct geniculate input onto both their basal dendrites in layer 6 as well as their apical dendrites in the overlying cortical layers. Recent results from the cat, however, indicate that the majority of synapses from the LGN are made onto the basal dendrites (da Costa & Martin, 2009).

### Evidence for parallel streams of corticogeniculate feedback

Corticogeniculate neurones have a pyramidal morphology and use glutamate for synaptic transmission (reviewed in Briggs & Usrey, 2009b). Their cell bodies are located exclusively in layer 6 of visual cortex and their axons branch to innervate the LGN, the reticular nucleus, and the overlying cortical layers (predominantly specific divisions of layer 4). In addition, a subset of corticogeniculate neurones in the very bottom of layer 6 probably provides weak input to the pulvinar nucleus (Conley & Raczkowski, 1990; Bourassa & Deschenes, 1995; Usrey & Fitzpatrick, 1996; Van Horn & Sherman, 2004). Although corticogeniculate neurones typically make up less than 50% of layer 6 neurones (~14% in the macaque monkey; Fitzpatrick et al. 1994), their connections are anatomically robust. Indeed, individual LGN neurones receive more synaptic input from corticogeniculate feedback axons than from retinal axons (Guillery, 1969; Erisir et al. 1997a,b). Similarly, individual layer 4 neurones receive more synaptic input from layer 6 axons than from LGN axons (Ahmed et al. 1994).

In the macaque monkey, layer 6 can be divided into three tiers. The cell bodies of corticogeniculate neurones are restricted to the upper and lower tiers; the middle tier is void of corticogeniculate neurones (Fitzpatrick et al. 1994; see also Lund et al. 1975; Hendrickson et al. 1978). Importantly, neurones in the upper and lower tiers...
tiers provide anatomically distinct patterns of input to the LGN that follow the organization established by the feedforward parallel processing streams. In particular, corticogeniculate neurones in the upper tier of layer 6 have axons that target the parvocellular layers of the LGN, while neurones in the bottom tier have axons that target the magnocellular layers (Fig. 2; Conley & Raczkowski, 1990; Fitzpatrick et al. 1994). In addition, a small percentage of corticogeniculate neurones in the lower tier of layer 6 have axons that probably provide input selective to the koniocellular layers of the LGN or to the koniocellular layers in combination with either the magnocellular or parvocellular layers (Fitzpatrick et al. 1994; Usrey & Fitzpatrick, 1996; Ichida & Casagrande, 2002).

Several lines of evidence indicate that corticogeniculate neurones in the upper and lower tiers of layer 6 are members of distinct processing streams (Fig. 2). First, neurones in the upper and lower tiers of layer 6 receive different patterns of afferent LGN input. Parvocellular LGN axons that project to layer 4C_β often give rise to collaterals that terminate in the upper part of layer 6. In contrast, magnocellular LGN axons that project to layer 4C_α often give rise to collaterals that terminate in the lower part of layer 6 (Lund, 1988). Second, the axons of layer 6 neurones not only target the LGN, but also branch and terminate locally in layer 4C of visual cortex. Neurones in the upper tier of layer 6 have axons that terminate primarily in layers 4C_β and 4A (parvocellular targets); neurones in the lower tier of layer 6 have axons that terminate primarily in layer 4C_α (magnocellular targets; Lund & Boothe, 1975; Wiser & Callaway, 1996). Third, while some layer 6 neurones receive similar input from layers 4C_α and 4C_β, other layer 6 neurones receive a disproportionate amount of input from either layer 4C_α or 4C_β (Briggs & Callaway, 2001).

**Physiological properties of corticogeniculate neurones**

An examination of the visual physiology of corticogeniculate neurones in the macaque monkey distinguishes three major groups of neurones (Briggs & Usrey, 2007, 2009c). The first group is composed of...
complex cells with fast conducting axons (antidromic activation latency: <7 ms) and visual physiology indicative of a strong influence from the magnocellular stream. These neurones respond well to low contrast stimuli and stimuli modulated at high temporal frequencies. (Briggs & Usrey, 2009; see also Hawken et al. 1988) These neurones also exhibit strong surround suppression and have the greatest selectivity for the direction of stimulus motion. Interestingly, many corticogeniculate neurones in this group are also distinct in receiving direct input from the LGN capable of driving suprathreshold spikes (Briggs & Usrey, 2007). As a consequence, there is a fast, disynaptic circuit from the LGN to V1 and back to the LGN that appears specific to the magnocellular stream.

The second group of corticogeniculate neurones is composed of simple cells with moderate conducting axons (antidromic activation latency: 7–15 ms) and visual physiology indicative of strong parvocellular stream input (Briggs & Usrey, 2009c). Compared to the first group of corticogeniculate neurones, these neurones have linear contrast response functions, prefer stimuli drifting at lower temporal frequencies, prefer stimuli with higher spatial frequencies, display less surround suppression, and have lower stimulus-evoked firing rates. Importantly, neurones in this second group are located more superficially in layer 6 compared to those in the first group, consistent with the sublaminar segregation of corticogeniculate neurones described above.

The third group of corticogeniculate neurones is composed of complex cells with slow conducting axons (antidromic activation latency: >15 ms; Briggs & Usrey, 2009c). Neurones in this group are similar to those in the first group in terms of responding well to low contrast stimuli and stimuli modulated at high temporal frequencies. However, these neurones are uniformly not orientation or direction selective. Consistent with the view that these neurones share a relationship with the koniocellular stream, the responses of neurones in this group are more strongly modulated by stimuli selective for S-cone modulation. Although less is known about the visual physiology of corticogeniculate neurones in non-primate species, a comparison of axon conduction latency and simple vs. complex response profiles supports a classification scheme with three groups of neurones (Harvey, 1978; Tsumoto & Suda, 1980; Swadlow & Weyand, 1987; Grieve & Sillito, 1995; Briggs & Usrey, 2005).

Based on an increasing amount of anatomical and physiological data, it appears clear that the feedforward and feedback pathways interconnecting the LGN and V1 are organized with a specificity that aligns well along the axes of the magnocellular, parvocellular and koniocellular streams. As a consequence of this organization, corticogeniculate feedback is well suited to influence feedforward processing of visual information in a stream-specific fashion.

**Functional influence of corticogeniculate feedback on visual processing**

The majority of corticogeniculate synapses in the LGN are made onto the distal dendrites of neurones. These synapses are smaller and contain fewer vesicles compared to retinogeniculate synapses. For these reasons and others, including the fact that LGN receptive fields resemble those of their retinal inputs and not their cortical inputs, the corticogeniculate pathway is thought to be modulatory rather than driving in nature (Sherman & Guillery, 1998). This modulatory input is likely to be complex, though, as corticogeniculate neurones may differentially activate neurones in the reticular nucleus leading to a variety of combined excitatory/inhibitory effects in the LGN (Landsisman & Connors, 2007; Cruikshank et al. 2010; Lam & Sherman, 2010). Moreover, the balance of excitation and inhibition is likely to change with firing rate, as the synapses that provide direct excitation and disynaptic inhibition may experience varying amounts of rate-dependent facilitation (Cudeiro et al. 2000; Granseth et al. 2002; Li et al. 2003; Alexander & Godwin, 2005).

Results from a variety of species support two prominent roles for feedback: (1) feedback sharpens the receptive fields of LGN neurones, and (2) feedback enhances the transmission of signals relayed through the LGN (reviewed in Briggs & Usrey, 2008). Given the evidence for parallel streams of feedback in the primate, it is worth evaluating these roles for feedback in terms of the magnocellular, parvocellular and koniocellular processing streams. Moreover, because feedback projections display a retinotopic and ocular specificity, the effects of corticogeniculate feedback should be spatially restricted to LGN neurones located within the projection field of feedback axons (Murphy & Sillito, 1996; Angelucci & Sainsbury, 2006; Wang et al. 2006; see also Usrey & Fitzpatrick, 1996; Murphy et al. 2000). With respect to the first role for feedback, corticogeniculate projections are believed to sharpen the receptive fields of LGN neurones by contributing to the strength of their extraclassical (suppressive) surround (Murphy & Sillito, 1987; Jones et al. 2000). This role, however, may be restricted to carnivores as recent work demonstrates that extraclassical suppression in the LGN of primates relies on mechanisms established in the retina (Alitto & Usrey, 2008; but see Webb et al. 2002).

The second major role for corticogeniculate feedback – enhancing signal transmission through the LGN – has been suggested to occur by increasing the gain of LGN responses to visual stimuli, improving the reliability of LGN responses, and/or adjusting the temporal patterns.
of activity among individual neurones and neuronal ensembles. For instance, results from the macaque monkey demonstrate that corticogeniculate feedback can multiplicatively increase the responses of LGN neurones in a contrast-independent fashion (Przybyszewski et al. 2000). With respect to the parallel processing streams, this effect occurs over a much wider range of contrasts for parvocellular neurones than for magnocellular neurones. Corticogeniculate feedback has also been shown to increase the magnitude and high-velocity cutoff of LGN responses to moving patterns and textures (Gulyas et al. 1990; but see Marrocco et al. 1996), as well as increase the reliability and temporal precision of responses among individual LGN neurones and ensembles of neurones (McClurkin et al. 1994; Funke et al. 1996; Sillito & Jones, 2002; Andolina et al. 2007; de Labre et al. 2007).

The augmenting effect that feedback can have on LGN responses has been proposed to increase with directed attention. Attentional modulation of neuronal activity is well documented in V1 (Motter, 1993; Luck et al. 1997; Watanabe et al. 1998; Brefczynski & DeYoe, 1999; Ito & Gilbert, 1999; Somers et al. 1999; Ress et al. 2000; Marcus & Van Essen, 2002; McAdams & Reid, 2005; Chen et al. 2008). Although the effects of attention have not been measured among identified corticogeniculate neurones, it seems likely that these neurones also experience attentional modulation. If so, then the corticogeniculate feedback pathway could provide a route for attention to influence LGN activity. Consistent with this view, attentional modulation of LGN activity has been reported for both human and non-human primates (O’Connor et al. 2002; McAlonan et al. 2008). Moreover, there is evidence that directed attention may be able to selectively modulate LGN activity in a stream-specific fashion (Vanduffel et al. 2000).

Given the influence of activity levels on the strength and efficacy of corticogeniculate synapses (described above), it seems likely that the influence of feedback on LGN responses should be most robust in the alert animal. Along these lines, results from our laboratory indicate a major difference in the visual responsiveness of corticogeniculate neurones in the alert and anaesthetized monkey (Briggs & Usrey, 2007, 2009c). To date, however, most studies of corticogeniculate feedback have relied on measurements collected from animals in the anaesthetized state. While these measurements have certainly advanced our understanding of the functions of corticogeniculate feedback, it seems likely that we will see acceleration in our understanding of the feedback pathway as more studies are performed with alert animals. In addition, while past methods for assessing the function of feedback projections have relied on large-scale inactivation methods (e.g. cortical cooling, cortical aspiration) which are likely to obscure effects dependent on the fine topography of feedback connections, recent and ongoing advances in the development of molecular and optical methods for targeted inactivation of specific cells and cell types will certainly open new doors for determining the functions of this important pathway for vision.

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