Looking back: corticothalamic feedback and early visual processing

Javier Cudeiro¹ and Adam M. Sillito²

Although once regarded as a simple sensory relay on the way to the cortex, it is increasingly apparent that the thalamus has a role in the ongoing moment-by-moment processing of sensory input and in cognition. This involves extensive corticofugal feedback connections and the interplay of these with the local thalamic circuitry and the other converging inputs. Here, using the feline visual system as the primary model, some of the latest developments in this field are reviewed and placed in the perspective of an integrated view of system function. Cortical feedback mediated by ionotropic and metabotropic glutamate receptors, and effects mediated by the neuromodulator nitric oxide, all have a role in integrating the thalamic mechanism into the cortical circuit. The essential point is that the perspective of higher-level sensory mechanisms shifts into the cortical circuit. The essential point is that the cortical feedback pathway from the cortex and via non-specific modulatory inputs from the brainstem and other areas (Figure 1). With a recent renewal of interest in this pathway, and growth in our knowledge of corticothalamic interactions, it is timely to examine the hypothesis that the thalamic mechanism is in fact integral to formation of the cognitive maps that constitute our perception of the external world. This involves a cascade of time-linked and modulatory interactions that evolve in a dynamic, moment-by-moment fashion where cortical and subcortical circuits are part of a single integrated system. Here, we review the ways in which some elements of these circuits work together to enable this process. We consider the situation for thalamic sensory relay nuclei and not interactions that pertain to nuclei that receive projections from layer 5 of the cortex. The main components of this review are drawn from the feline visual system, which has been the ipso facto model for many studies of synaptic mechanisms and sensory processing in the thalamus [6], but we also use examples from other species as necessary.

Elements of the circuitry

In the mammalian visual system, much of the work on circuitry and sensory processing in the thalamus has drawn on the cat, and for simplicity we shall focus on these studies here. For many years, it has been known that the lateral geniculate nucleus (LGN) of the cat receives major projections from the visual cortex [7]. According to both anatomical and electrophysiological studies, corticogeniculate efferent axons originate exclusively from layer 6 of the visual cortex in cats [8–11] and most other species (e.g. [12,13]), and the cells involved can be regarded as a specific functional and morphological class of layer 6 neurons [14,15].

Two features characterize the cortical feedback: first, the fibres involved largely outnumber any other projection [16]; and second, the pathway exhibits a strong degree of retinotopy and enables the stimulus selectivity of visual cortical neurons to influence thalamic circuitry [7]. In cats, the anatomical spread of an individual corticogeniculate axon arbour can be extensive (~1.5 mm), reaching well beyond the region in which LGN receptive fields matching those of the cortical cell can be recorded [11]. Nevertheless, the projection from a given location in area 17 has a centre of maximum terminal density in the LGN ~400–500 μm across, which within the LGN is in retinotopic
account the number of axons, and their morphology, corticothalamic connectivity (which must take into
Nevertheless, despite its importance, the impact of convergence in the corticothalamic pathway [6,16,22]. These corticothalamic axons exert both an excitatory and an indirect inhibitory influence on LGN relay neurons (Figure 1). The excitatory influence is achieved by monosynaptic connections and involves classic excitatory transmission (mediated by ionotropic receptors) and a modulatory excitatory transmission (mediated by metabotropic receptors) [25–27]. These different receptor classes enable a subtle mixture of fast and slowly influences on the thalamic circuitry. For example, the type 1 and type 2 metabotropic receptors seem to have different roles in the thalamic circuitry. Type 1 metabotropic glutamate receptors (mGlu1 receptors) are found on relay-cell distal dendrites that are postsynaptic to axon terminals from cortical layer 6 [28–30] (Figure 1b) and, as will be discussed later, they have been implicated in the cortex-mediated effects on geniculate relay cells observed in anaesthetised cats [31]. More recently, in experiments using slices of adult ferret CNS containing the LGN, a presynaptic metabotropic receptor belonging to group II (mGlu2) has been claimed to reduce cortical feedback to the thalamus selectively (Figure 1b). This presynaptic inhibition might partially attenuate cortical input and prevent re-entrant excitation from initiating abnormal thalamic rhythms [32]. However, the main inhibitory influence of the cortex is achieved by polysynaptic connections either with intrinsic inhibitory interneurons within the relay nuclei or with GABAergic neurons that have cell bodies located above the LGN in the reticular nucleus of the thalamus (TRN), which is a major contributor to the overall response profile of the relay cells [6] (Figure 1). In the case of the TRN, layer 6 cortical inputs to interneurons seem to activate only ionotropic glutamate receptors [33,34]. In cats, there is immunocytochemical evidence that TRN cells contain both ionotropic and metabotropic glutamate receptors (types I and II/III); however, the pattern of postsynaptic receptors associated with cortical input and its physiological actions are far from clear [6].

**Functional aspects of thalamocortical interactions**
Owing to the complexity of connections established by corticofugal axons, which involve different types of receptors and different types of cells (relay cells, interneurons and TRN cells), one can also predict difficulties in finding a single clear-cut effect of this input on LGN cells. In the cats, cooling the visual cortex has been reported both to increase and to decrease the response of LGN cells to visual stimulation, affecting the centre–surround balance [35,36]; this can be interpreted as a combination of non-specific excitatory and inhibitory effects [37]. In primates other approaches, using stimuli regarded as effective for studying LGN neurons in addition to activating layer 6 cells, have produced more subtle results [38,39]. These highlight the observation that cortical feedback can change both the spatial (Sillito et al., in
this issue) [40] and the temporal properties of LGN cells—properties that are related to the centre–surround balance of the receptive field [38,39,41]. Furthermore, it has been demonstrated in cats and primates that individual LGN cells receive both facilitatory and inhibitory influences, but that these are driven by areas of cortex representing different regions of visual space [38,39,42]. Together, these results provide an interesting picture in which there are overlying suppressive and facilitatory influences from the cortex, with the suggestion that suppressive influences extend further than the facilitatory ones. However, it needs to be emphasized that the facilitatory influences under physiological conditions are expressed in LGN cells as a modulation of their response to retinal input and that the feedback does not inject extra spikes into the LGN cell response. For this reason, the feedback connections to the sensory thalamic nuclei are considered to be modulators, in contrast to corticofugal axons arising from layer 5 and projecting to other subcortical structures, which are considered to be drivers [6]. A further point to note is that the layer 6 corticofugal axons make excitatory contacts with relay cells through their entire arborization; in this sense, the data suggesting that suppressive effects are seen beyond the limits of the facilitatory effects [38,39,42] must reflect the impact of connections via GABAergic interneurons that either extend beyond or swamp the effects of the facilitatory influence.

It is crucial to select stimuli that effectively drive the visual cortex to engage the corticothalamic loop fully, and so reveal cortical influences in the LGN. A further implication of this idea is that systematically changing the parameters of the stimuli will change cortical responses and, in turn, their influence on LGN cell responses. This has been demonstrated in a series of experiments that aimed to study the spatial properties of the corticofugal effect. These experiments revealed a corticofugal modulation of the centre–surround antagonism, which reflects the orientation-selective properties of layer 6 neurons and enhances the low spatial frequency cut-off in the LGN [18], suggesting that the layer 6 cells providing the feedback might be biased towards low spatial frequencies.

Recently, using a theoretical approach, a model of feedback from V1 has been developed to deal with interactions between the classical receptive field and surrounding regions [43]. This model readily accounts for the aforementioned experimental data, but it also makes some interesting predictions that remain to be tested— for example, that the sensitivity of LGN neurons to orientation discontinuities at low contrast should be twice that at high contrast, and that responses to drifting gratings should be less linear at spatial frequencies where layer 6 cells give robust responses. Other work has shown how a model of visual processing that includes top-down corticogeniculate feedback might contribute to the dynamics of binocular vision [44,45], to perceptual grouping [46], to brightness perception and illusory contours [47], and to the temporal response properties of geniculate relay cells in a way that alters the speed tuning of cortical cells [48].

Other stimuli that drive both cortical and thalamic cells effectively include moving bars of light. LGN cells (like some cortical cells) are selective for bar length [49]. Neurons responding this way are known as end-stopped or length-tuned neurons. In the LGN, this property is highly dependent on the integrity of the visual cortex and it is considered to be an emergent property of the geniculocortical loop [40]. Cortical feedback seems to contribute to bar-length selectivity in the LGN by an appropriate combination of cortical driving of the receptive-field inhibitory surround and a direct excitation of the centre. A crucial element in this feedback control of thalamic centre–surround antagonism is the mGlu1 receptor (Figure 1b). In adult cats, the cortex uses a synaptic drive mediated by these receptors specifically to enhance the response of the thalamic receptive field to the retinal inputs driving its centre mechanism [31]. Moreover, the effect is maximal in response to stimuli that effectively drive cortical cells and, importantly, it does not affect the spatiotemporal structure of the thalamic receptive field, as determined using stimuli that effectively engage the retinal input (e.g. flashing spots, which are considered not to be an optimal stimulus for layer 6 cortical cells). In this study [31], the visual responses of LGN cells were recorded extracellularly before, during and after the iontophoretic ejection of (+2-methyl-4-carboxyphenylglycine (LY367385), a highly specific mGlu1 antagonist [50,51]. Figure 2(a) shows how the blockade of mGluR1 receptors using iontophoretic application of LY367385 decreases the responses of an LGN cell to a moving visual stimulus (in this case, a drifting grating). However, the tuning curve in Figure 2(b) illustrates the activity of a typical length-tuned LGN cell in response to a bar of light of varying length moving over its receptive field during control conditions and during ejection of LY367385. The key point is that there is a marked difference in the tuning curves with and without cortical feedback (mediated by the mGlu1 receptors). After 6 min of continuous ejection of LY367385, the responses of the cell are reduced. However, it is important to note that the reduction most affects the responses to the ‘preferred’ length bar, which optimally occupies the centre of the field. What happens when static stimuli are used? Blockade of mGlu1 receptors using LY367385 has been found not to affect the first, transient part of the response to flashed spots covering the receptive-field centre [31] (Figure 2c). Instead, only the sustained phase of the response clearly decreased during LY367385 application (Figure 2c). Interestingly, other work has shown that after selective chronic elimination of corticogeniculate feedback, this effect is reversed, with cells showing an unusually enhanced and prolonged tonic visual response; this could be related to a compensatory overexpression of metabotropic glutamate receptors at the corticogeniculate synapse [52].

Further roles for mGlu1 receptors in the control of thalamic activity by the cortex have been suggested. Synaptic activation of mGlu1 receptors might underlie the initiation of a slow oscillation in thalamic neurons [53]. Through this, the level of cortical activity could regulate sleep-related activity of thalamic neurons and actively
control the oscillatory output of thalamocortical neurons during different stages of vigilance [54]. It has also recently been shown in LGN slices that activation of mGlu \(_1\) receptors induces synchronized oscillations at alpha and theta frequencies, which share similarities with thalamic rhythms recorded in vivo. It was proposed that these oscillations are a candidate mechanism by which the thalamus could support the generation of electroencephalogram (EEG) alpha and theta rhythms in the intact brain [55]. For a recent review on glutamate receptor functions and sensory thalamic activity, see Ref. [56].

The main point in this section is that the corticothalamic projection, using push–pull control that selectively enhances centre and/or surround mechanisms, modulates thalamic function; in this way it optimizes integration of the thalamic and cortical processes that extract salient features for abstraction of the external world in higher cortical areas. Similar mechanisms have been suggested to operate in both the somatosensory [57–60] and the auditory systems.

**Temporal influences on thalamic processing**

Visual responses of LGN neurons depend on the context of the stimulus, not only in spatial terms (as already discussed) but also temporally. In a visual system designed to work continuously and seamlessly during prolonged observation of the visual world, temporal changes in visual responses should also be considered. By temporal changes, we mean not only variability of the response of a cell but also, and as importantly, the effect of the visual image preceding the scene currently under analysis. The amount of information a sensory neuron carries about a stimulus is directly related to response reliability; therefore, to understand the coding of information by neurons, it is important to quantify the variability in their responses. It has been shown that, at least in the anaesthetized condition, the LGN can respond to visual stimuli with remarkable temporal precision and low variability [61–64]. Interestingly, it has been suggested both by computer simulations and experimental work that the temporal dispersion of neural events found in the LGN is small (even smaller than that of the retinal afferents) as long as the cortex is active [65]. Whereas cortical inactivation leads to widening of the distributions of interspike intervals, cortical stimulation can make them sharper [65]. It was concluded that the corticofugal feedback can reduce the temporal dispersion of spike events at the level of the LGN. Paradoxically, this ‘sculpting’ effect mediated by the cortex will in turn have a powerful effect on visual cortical responses, because the convergence of several nearly synchronous spikes is a highly effective input [66]. This phenomenon can also be seen at work in the earlier study of Sillito and co-workers, who reported that the corticofugal feedback induces correlated firing in LGN relay cells when driven by moving oriented visual stimuli [67]. This synchronization among neurons covered by a coherent stimulus disappears in the absence of feedback. We suggest that this follows from loss of the modulatory influence on the processes that create centre and surround mechanisms in the LGN receptive fields, and hence a loss of spatial resolution of the receptive fields. Consequently, the synchronous excitatory postsynaptic potentials (EPSPs) elicited in their target cortical cells will be lost, and the effective drive to the cortex will be diminished. Using different approaches, including slice preparations of the visual thalamus and computational models, it has also been shown that the cortical feedback can induce highly synchronous oscillations in the LGN and control their frequency [68].

Another temporally mediated effect linked to the corticothalamic feedback can be formulated: the firing

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**Figure 2.** Cortical influence mediated by mGlu\(_1\) receptors in anaesthetised cats. (a) Peristimulus time histograms (PSTHs) illustrate the response of the cell to a full-field sinusoidal drifting grating (illustrated above the PSTHs) of optimal characteristics under control conditions (i), in the presence of the specific mGlu\(_1\) antagonist LY367385 (ejected iontophoretically using 60 nA for 6 min; ii), and after a recovery period of 15 min (iii). (b) Length-tuning curves constructed for a bar of varying length moving in both directions (illustrated above the PSTHs) over the receptive field. The blue curve is the control response; the red curve is the response during LY367385 application (6 min, 60 nA). PSTHs are shown for each bar length. It is clear that the response decreases when the mGlu\(_1\) antagonist is applied. The strongest effect is seen for the optimal length (3° in this example). (c) Transient responses are not affected by mGlu\(_1\) blockade. PSTHs show the response of an LGN cell to flashed spots (represented by the circle above the PSTHs) restricted to receptive-field centre, before (i), during (ii) and after (iii) application of the specific mGlu\(_1\) antagonist (ejection: 80 nA, 3 min). Only the sustained part of the response was reduced by the antagonist. Modified, with permission, from Ref. [31] © (2002) the Society for Neuroscience.
rate of many LGN cells is related to the intensity of their firing up to several minutes before any point of measurement. Basically, a period of heightened driven activity (presentation of a stimulus that has elevated contrast or electrical stimulation of the optic chiasm for 1 min) results in higher background and visually evoked firing, which lasts for several minutes longer than the period of heightened activity. Such an effect has been observed both in cats and monkeys [69] (Figure 3). Prolonged continuous visual stimulation using a high-contrast version of the test stimulus (Figure 3a,ii) could induce a response augmentation following the stimulation period, and this effect lasted 6–12 min (Figure 3a,iii). Removal of the visual cortex resulted in a near complete loss of the observed high-contrast-induced potentiation. This is illustrated in Figure 3(b), which compares the responses of two cells recorded in the same experiment, one before and one after the cortex was removed. The cell receiving intact corticofugal feedback showed a clear augmented response when it was tested again with a low-contrast grating (test stimulus) 3 min after high-contrast stimulation. Figure 3(c) summarizes the data from the population of neurons studied. There is an obvious and highly significant reduction in the percentage of cells showing augmentation in the absence of corticofugal input. Similar results were obtained in monkeys, in neurons including both parvocellular and magnocellular cells. The basic phenomenon is again illustrated in Figure 3(d): following control low-contrast stimulation, a period of high-intensity stimulation (arrow) can evoke an augmentation that is significant and reproducible, on a relatively short timescale [69] (but see the following section). Interestingly, a similar effect has been described when a non-specific glutamate receptor agonist was used to induce the necessary period of heightened activity [70]. In summary, what we are describing here might be a mechanism whereby activity in the visual cortex opens a window in time and retinotopic space in the LGN (possibly via metabotropic mGlu1 receptors on LGN cells), in which the gain of the highlighted relay cells is enhanced, thus focussing the corticothalamic mechanism as a whole onto this temporal–spatial window.

**Nitric oxide, modulation and more diffuse circuits**

Another modulatory influence in this system is mediated by nitric oxide (NO). This is a ubiquitous neuromodulator that has been implicated in multiple functions in the visual system, including the responsiveness of thalamic and visual cortical neurons [71]. The important point here is that it is a diffuse modulator; it effects both thalamic and cortical processes and by this token might affect their interaction in ways that need to be considered. At the level of the cat LGN, the transmission of visual information is enhanced by the release of both ACh and NO from brainstem terminals (arising from the parabrachial region; Figure 1); NO actively cooperates with ACh, greatly increasing the activation of the visual pathway [71]. This is best illustrated by the example in Figure 4(a),

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**Figure 3.** Cortical feedback influences LGN activity in the temporal domain. (a) The effect of high-contrast stimulation on subsequent visual responses. PSTHs were obtained under control conditions (i.e. stimulation using a low-contrast stimulus) and following visual stimulation using high-contrast stimulus (ii). Responses were then measured again using low-contrast stimulation 1, 3, 6 and 12 min after the end of high-contrast stimulation (iii). Responses were then measured again using low-contrast stimulation 1, 3, 6 and 12 min after the end of high-contrast stimulation (iii). (b) PSTHs from two cells recorded during the same experiment before (i) and after (ii) decortication. (c) Summary of potentiation responses for the population of cat cells studied with and without corticofugal input. (d) Single-cell data from a magnocell in the macaque LGN. The augmentation effect obtained by 1 min exposure to a high-contrast drifting grating (arrow) was reproducible in a second period of stimulation following recovery. Modified, with permission, from Ref. [69] © (2000) Blackwell Publishing Group.
in which NO significantly affects the activity of visually driven cells at the thalamus. A similar scheme can be found in the cortex (Figure 4b), where basal forebrain (BF) axons that contain ACh and NO project to the entire neocortex give rise to phasic activation during waking states [72,73]. It must be borne in mind that activation of the cortex will enhance thalamic inputs, and so it is in keeping with our earlier remarks to concentrate here on modulation of cortical activity. BF neurons participate in the regulation of cortical activity; throughout the sleep–wake cycle, the function of the BF–cortical pathway is complex, and sleep-promoting mechanisms appear to coexist with mechanisms of arousal or activation [72]. Interestingly, it has been known for some time that during periods of inactivity, such as sleep, the deep layers of the cortex (therefore including corticogeniculate feedback to the LGN) are the most suppressed [74]. The activation of the BF-to-cortex pathway promotes tonic firing in cortical neurons and generates EEG patterns characteristic of activated behavioural states, switching sleep-like activity (high-amplitude slow oscillations) to an awake-like pattern (low-amplitude fast oscillations). Evidence also indicates that NO is involved in cortical activation, disrupting slow oscillations and promoting tonic firing of neurons [75,76]. Therefore, another control loop involving the visual cortex and the LGN can be drawn. The BF activates the cortex using ACh and NO; in turn, and in concert with other modulatory pathways, this affects the LGN (in this scenario ‘waking up’ the LGN through feedback from layer 6). This cortical activation also affects other subcortical structures, including the superior colliculus, the pons, and higher-order thalamic regions such as the pulvinar nuclei (here by feedback from layer 5) [77–79] (Figure 4b).

Recent data from cats support this view [80]. After blocking NO synthase (NOS) activity, the capacity of BF stimulation to induce cortical activation was strongly impaired. Furthermore, voltammetric measurements of cortical NO levels revealed an increase after BF stimulation, and this was also blocked by systemic NOS inhibition. Figure 4(c) shows the effect of BF stimulation on spontaneous electrocorticogram (ECoG) activity simultaneously recorded through intracortical electrodes in the primary visual cortex. This ECoG activation was mediated
(at least in part) by NO because systemic application of the neuronal NOS inhibitor 7-nitroindazole (7-NI) strongly diminished the capacity of the BF to modulate cortical activity (Figure 4c, ii). The recording of NO cortical levels indicated an increase in the release of NO after BF stimulation under control conditions. Blockade of NOS by 7-NI eliminated this effect and also reduced baseline levels of NO (Figure 4d). In addition, low frequencies of cortical activation are reduced and high frequencies are enhanced following BF stimulation, with the strongest effect detected in the gamma range (not shown in Figure 4). These effects are strongly diminished in the presence on 7-NI [80].

The BF cholinergic system has been implicated in the transition from sleep to wake, but recently an active role in the promotion and maintenance of sleep has also been proposed, as has involvement in attention, motivation and learning [81–84]. Evidence now suggests that the BF-activating system might work by bringing together the function of ACh and NO, as also occurs in the brainstem [71]. In this scenario, NO production after BF stimulation (either produced experimentally or, for instance, during arousal) would lead to a dual effect: ACh release, both directly and via an NO-mediated pathway, and the straightforward action of NO on the cGMP secondary messenger system. Such cGMP modulation has been widely reported in several brain systems, including the cat visual cortex [85]. Because of its gaseous nature, NO can diffuse locally and affect the activity of a relatively large neuronal population and an extensive cortical volume, and so can affect the thalamus by means of extensive corticofugal connections. Interestingly, corticothalamic feedback has been suggested to be involved in attentional modulation of both the LGN and the visual sector of the TRN in rats and humans [86–89]. Whether NO is involved in these processes, and how general cortical activation can be transformed into focal activity that engages specific components of the corticothalamic loop, are interesting unanswered questions.

Summary and beyond

For sensory input to be rapidly assessed and to guide the behaviour of an organism, it needs to be continually placed in the context of hypotheses formulated by higher brain centres. Recurrent projections are a universal feature of cerebral organization, and in the visual system it seems clear that the cortical feedback to the thalamus can modify how LGN neurons behave under visual stimulation. The feedback thus affects what, when and how visual signals are transferred to the cortex (by altering their nature, temporal properties and state dependency, respectively), and also affects their subsequent transfer through the cortex (probably by similar means). Knowledge of the physiology of the cortex is a key that might unlock our understanding of the thalamus; however, our understanding of thalamus and cortex together is another question.

The thalamus and cortex work closely together and can be regarded as a single circuit in functional terms. All aspects of the cortical basis of cognition, such as attention, contour discrimination and movement detection, will be reflected in the activity of the corticothalamic loop that operates through both fast ionotropic and slow metabotropic glutamate receptors and is modulated by several extra-retinal inputs, including various typical neurotransmitters and the unorthodox neuromodulator NO. From this perspective, activity in the visual cortex opens a window in time and retinotopic space in the LGN, in which the gain of the highlighted LGN relay cells is enhanced, and the reinforced message is sent back to the cortex. This can be seen as a mechanism set up to extract the salient features of our perceived visual world. Further dissection of the components of this system requires new approaches. For example, it is known that dynamic receptive-field properties of neurons in the visual system change within milliseconds as a function of time post-stimulus, and several computational models have suggested that feedback circuitry might underlie the time-varying properties of cortical and thalamic neurons in many sensory systems [57]. Therefore, a tool that would enable us to study the function of visual corticothalamic feedback over a wide temporal range, from sub-seconds to minutes, in both a reliable and a reproducible way would be useful. In this sense, techniques such as repetitive transcranial magnetic stimulation might permit disruption of visual cortex activity in the timescale we believe to be important for the relationship between cortex and thalamus [90].

Acknowledgements

Supported by Spanish grants MCYT (BF2002–3200) and MEC (BFU2005–06502). We thank Dr Kenneth Grieve for helpful comments on the manuscript.

References

1 Bullier, J. et al. (2001) The role of feedback connections in shaping the responses of visual cortical neurons. Prog. Brain Res. 134, 193–204
7 Updyke, B.V. (1975) The patterns of projection of cortical areas 17, 18, and 19 onto the laminae of the dorsal lateral geniculate nucleus in the cat. J. Comp. Neurol. 163, 377–395
12 Lund, J.S. et al. (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


Vidnyanszky, Z. et al. (1996) Immunocytochemical visualization of the mGluR1a metabotropic glutamate receptor at synapses of corticothalamic terminals originating from area 17 of the rat. Eur. J. Neurosci. 8, 1061–1071

Godwin, D.W. et al. (1996) Ultrastructural localization suggests that retinal and cortical inputs access different metabotropic glutamate receptors in the lateral geniculate nucleus. J. Neurosci. 16, 8181–8192


Tsuzuto, T. et al. (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


Hughes, S.W. et al. (2003) Cellular mechanisms of the slow (<1 Hz) oscillation in thalamocortical neurons in vitro. Neuron 33, 947–958


Ghazanfar, A.A. et al. (2001) Role of cortical feedback in the receptive field structure and nonlinear response properties of somatosensory thalamic neurons. Exp. Brain Res. 141, 88–100


Physiogenic and pathogenic oscillations: beauty and the beast

Neuronal networks generate a variety of oscillations and patterns that are involved in sensory integrative processes as well as memory processes. Thus, sensory binding is thought to involve the generation of oscillations in different parts of the brain that are related to various sensory modalities and somehow help in reconstructing the image. Oscillations are also involved in a wide range of pathogenic patterns and provide a signature of the neurological disorder. For example, in Parkinson’s disease, abnormal patterns are likely to be involved in akinetic disorders, and epilepsies are associated with high-frequency oscillations that also transform a naive structure to one that generate seizures, on the principle that ‘seizures beget seizure’.

Using a wide range of recordings and preparations, imaging techniques and mathematical models, we are beginning to understand how the brain generates these patterns and which neuronal population is dominant in the generation of a given pattern. What is now needed is to confront the various results and concepts and to generate an organised model of the physiogenic and pathogenic patterns: what are the similarities and differences in terms of mechanisms, of generators, etc? We shall invite to the meeting experts in the clinical, physiological, imaging and theoretical aspects of this question. We expect attendees to include students and senior researchers in neurology and neurobiology, as well as in mathematics and modelling.

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