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CHAPTER 7

Dynamic properties of thalamic neurons for vision

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Abstract: A striking property of neurons in the lateral geniculate nucleus (LGN) of the thalamus is the ability to dynamically filter and transform the temporal structure of their retinal spike input. In particular, LGN neurons respond to visual stimuli with either burst spike responses or tonic spike responses. While much is known from in vitro studies about the cellular mechanisms that underlie burst and tonic spikes, relatively little is known about the sensory stimuli that evoke these two categories of spikes. This review examines recent progress that has been made towards understanding the spatiotemporal properties of visual stimuli that evoke burst and tonic spikes. Using white-noise stimuli and reverse-correlation analysis, results show that burst and tonic spikes carry similar, but distinct, information to cortex. Compared to tonic spikes, burst spikes (1) occur with a shorter latency between stimulus and response, (2) have a greater dependence on stimuli with transitions from suppressive to preferred states, and (3) prefer stimuli that provide increased drive to the receptive field center and even greater increased drive to the receptive field surround. These results are discussed with an emphasis placed on relating the cellular constraints for burst and tonic activity with the functional properties of the early visual pathway during sensory processing.

Introduction

The lateral geniculate nucleus (LGN) of the thalamus is a major bottleneck for visual information traveling from retina to cortex. As a result, LGN neurons are in a strategic position to influence visual processing. Although once viewed as a simple relay in the visual pathway, several studies now show that the LGN is able to dynamically filter and transform visual input arriving from the retina (Usrey, 2002). A striking example of this property is the ability of LGN neurons to respond to excitatory input with spikes that belong to either burst responses or tonic responses (Jahnsen and Llinás, 1984a,b; Guido et al., 1992; Lu et al., 1992; Sherman, 1996, 2001, 2005).

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Whether or not an LGN neuron produces burst or tonic spikes depends critically on the membrane potential history of individual neurons and the activation state of their low-threshold, T-type Ca^{2+} channels (Jahnsen and Llinás, 1984a,b; Huguenard and McCormick, 1992; McCormick and Huguenard, 1992; Zhou et al., 1997; Destexhe et al., 1998). T-type Ca^{2+} channels have the special property that they cannot be opened unless they have been sufficiently hyperpolarized for an appropriate amount of time (typically more than 50 msec). As a result, if the resting potential of a neuron is not hyperpolarized to a level that de-inactivates T-type channels, then LGN neurons will respond to afferent excitation with a train of tonic Na^{+} spikes whose frequency is proportional to the strength of the afferent stimulus. In contrast, if the resting potential of a neuron is hyperpolarized to a greater extent and for a sufficient

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amount of time to de-inactivate T-type Ca^{2+} channels, then LGN neurons will respond to afferent excitation with a suprathreshold Ca^{2+} current that evokes a burst of Na^{+} spikes whose frequency is not related to the strength of the stimulus (Sherman, 1996, 2001, 2005).

Before discussing a possible role for burst and tonic spikes during sensory processing, it is important to acknowledge first the well-documented involvement of thalamic bursts during periods of low arousal and slow-wave sleep when information processing along the thalamocortical pathway is at a minimum (Steriade and Llinas, 1988; McCormick and Feeser, 1990; Steriade et al., 1990; McCormick and Bal, 1994; Steriade, 2001). During slow-wave sleep, excitatory inputs from the brainstem to the thalamus are withdrawn and thalamocortical neurons hyperpolarize. Feedback loops between neurons in relay nuclei (e.g., the LGN) and the reticular nucleus then serve to synchronize large numbers of thalamocortical neurons causing them to oscillate together at low frequencies and produce bursts of spikes en masse. Because bursts occur simultaneously among large numbers of thalamic neurons projecting to the cortex and do not reflect sensory input, information transmission along the thalamocortical pathway is severely disrupted.

An important distinction between bursts that occur during slow-wave sleep and bursts that occur during sensory processing lies in their timing. While bursts are synchronized across large numbers of neurons during sleep, bursts are presumed to be unique to individual neurons or small ensembles of neurons during sensory processing. Nevertheless, it is important to keep in mind that both categories of bursts (those that occur during slow-wave sleep and those that occur during sensory processing) are believed to rely heavily on the same cellular mechanism: a hyperpolarization-dependent, de-inactivation of T-type Ca^{2+} channels.

A number of excellent reviews are available that address the role of burst and tonic spikes during sensory processing (Sherman, 1996, 2001, 2005; Sherman and Guillery, 2002). In general, these reviews regard burst and tonic spikes as representing two distinct activity modes, where mode is determined by non-retinal, modulatory, inputs to the LGN. In the present study, the main focus is on considering what role the visual stimulus plays in directly driving burst and tonic spikes. It is important to emphasize that these two views concerning burst activity during sensory processing are not exclusive of each other. Rather, retinal and non-retinal inputs almost certainly interact with each other in a dynamic fashion to determine whether or not LGN neurons produce burst or tonic spikes. With that in mind, the temporal and spatial properties of visual stimuli that evoke burst and tonic spikes as well as the dynamic relationship between retinal drive and the low-threshold currents that underlie bursts are examined in the following sections. Finally, the functional consequences of burst and tonic activity for thalamocortical communication during sensory processing are discussed in the concluding section.

Temporal properties of visual stimuli that evoke burst and tonic spikes

Several studies have investigated the spatiotemporal organization of LGN receptive fields (Citron et al., 1981; Cai et al., 1997; Reid et al., 1997; Wolfe and Palmer, 1998; Usrey et al., 1999). Very few, however, have explicitly examined the temporal properties of visual stimuli that evoke burst and tonic spikes in LGN neurons. Using an m-sequence modulated, contrast reversing, sine-wave stimulus to excite LGN neurons, Alitto et al. (2005) identified spikes as either burst or tonic (same section), and performed reverse-correlation analysis on each category of spikes to generate spike-triggered averages. Results of this analysis show that the average stimulus to drive burst spikes is similar to, but significantly different from, the average stimulus to drive tonic spikes.

In the temporal domain, both burst and tonic spikes prefer visual stimuli that undergo transition from a suppressive to a preferred state (Fig. 1). Because suppressive stimuli can hyperpolarize LGN neurons (Singer et al., 1972; Martinez et al., 2003), it seems reasonable to suggest that these stimuli might also be capable of de-inactivating the T-type Ca^{2+} channels necessary for bursts. If so, then one would predict that the suppressive phase of the spike-triggered average preceding burst spikes should be greater than that for tonic spikes. As shown in Figs. 1 and 2, the magnitude (integral) of the suppressive

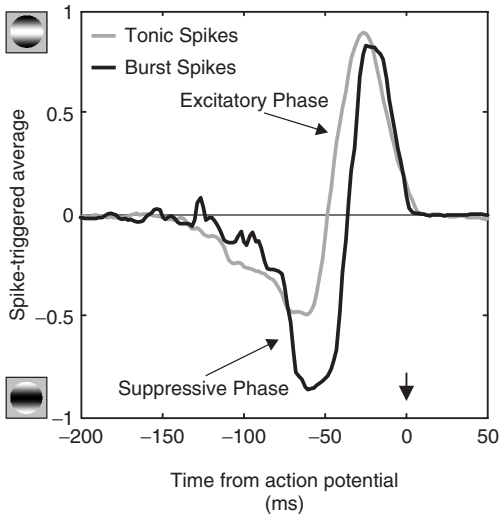

Fig. 1. Spike-triggered averages are similar, but distinct, for burst and tonic spikes. Using reverse-correlation analysis, spike-triggered averages were made from LGN responses ($n = 35$ neurons) to an m-sequence modulated, contrast reversing, sine-wave stimulus. The spike-triggered average shows the temporal sequence of the average stimulus to precede either a burst or tonic spike. Both burst spike (black trace) and tonic spike (gray trace) spike-triggered averages are composed of two phases: an initial suppressive phase followed by an excitatory phase. The suppressive phase is notably larger for burst spikes compared to tonic spikes.

phase of the spike-triggered average is indeed greater for burst spikes than for tonic spikes (0.056 ± 0.003 vs. 0.016 ± 0.001, respectively; $p < 0.00001$). This increase is due to an increase in both the suppressive phase maximum (Fig. 2; burst spikes $= 0.97 \pm 0.02$; tonic spikes $= 0.41 \pm 0.03$; $p < 0.00001$) and the suppressive phase duration (Fig. 2; burst spikes $= 81.9 \pm 2.4$ msec; tonic spikes $= 70.5 \pm 2.4$ msec; $p < 0.01$). These results are consistent with the view that bursts are triggered from a more hyperpolarized state than tonic spikes (see Spartial properties of visual stimuli that evoke burst and tonic spikes).

Alitto et al. (2005) used two criteria to identify the cardinal spikes of bursts: (1) a preceding interspike interval (ISI) greater than 50 msec, and (2) a subsequent ISI less than 4 msec. When these criteria are applied to spike trains in vivo, Sherman and colleagues have shown that they are highly effective at identifying bursts that rely on low threshold Ca^{2+} currents (Lu et al., 1992; Guido et al., 1992). With this in mind, one could nevertheless argue that the measured differences between the suppressive phases preceding burst and tonic spikes simply reflect differences between spikes that are preceded, on average, by long and short interspike intervals. If so, then one would expect the suppressive phase for tonic spikes to be statistically indistinguishable from that of burst spikes when the two categories of spikes are matched for preceding interspike interval (i.e., a subset of tonic spikes are examined that meet the first criterion for a burst). On the other hand, if a visually induced hyperpolarization is the variable that determines whether or not an LGN neuron will produce a burst, then one would expect the suppressive phase of the spike-triggered average to be greater for burst spikes than for tonic spikes matched for preceding interspike interval. Alitto et al. (2005) tested these possibilities and found that all of the reported differences between the suppressive phases that precede burst and tonic spikes are similarly significantly different for the suppressive phases that precede burst spikes and tonic spikes matched for initial interspike interval (data not shown, see Alitto et al., 2005). These results are consistent with the idea that suppressive stimuli are capable of hyperpolarizing LGN neurons and de-inactivating T-type Ca^{2+} channels that underlie bursts.

The excitatory phase of the spike-triggered average also differs significantly for burst and tonic spikes (Figs. 1 and 2). In particular, the latency between stimulus and response is significantly less for burst spikes than for tonic spikes (Fig. 2; 29.6 ± 1.0 ms vs. 33.8 ± 1.1 ms, respectively; $p < 0.01$). In addition, the magnitude (integral) of the excitatory phase is significantly less for burst spikes than for tonic spikes (0.024 ± 0.001 vs. 0.030 ± 0.001, respectively; $p < 0.00001$); an effect that reflects a decrease in the duration of the excitatory phase (burst spikes $= 32.7 \pm 0.5$ ms; tonic spikes $= 52.3 \pm 1.1$ ms; $p < 0.00001$; Fig. 2), but not a decrease in the maximum of the excitatory phase (burst spikes $= 0.95 \pm 0.02$; tonic spikes $= 0.95 \pm 0.02$; $p < 0.8$; Fig. 2). The finding that both the latency and duration of the excitatory phase are decreased for burst spikes compared to tonic spikes is consistent with results from a previous study examining latency and timing variability of burst and tonic spikes using drifting sine-wave gratings to drive LGN responses (Guido and Sherman, 1998).

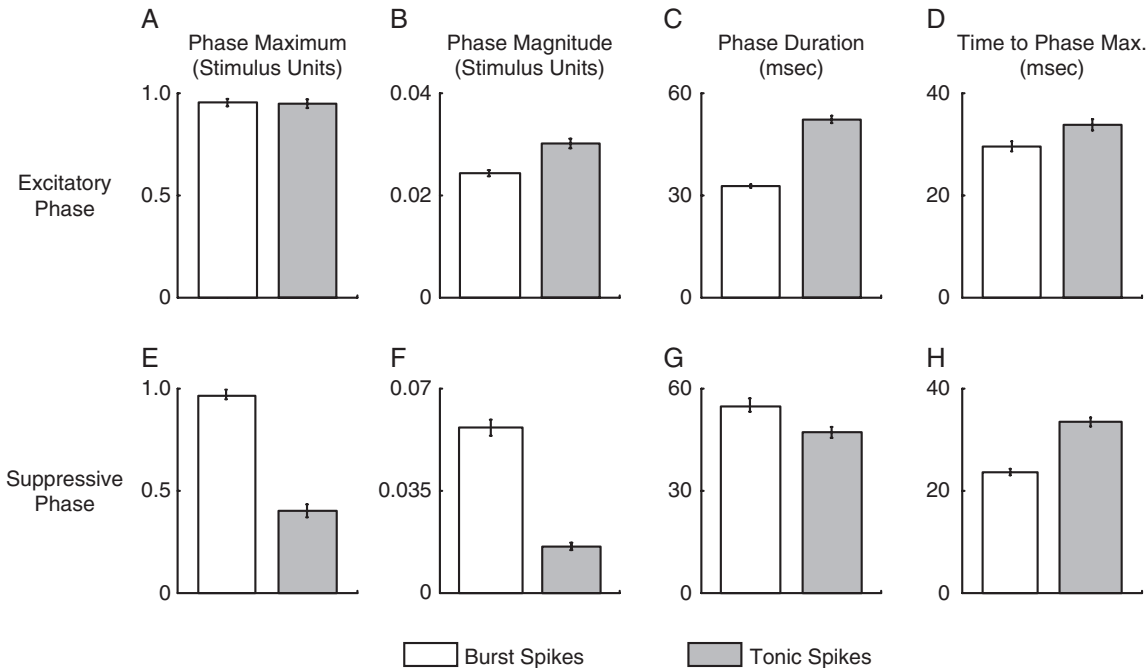

Fig. 2. Comparison of spike-triggered averages made from burst and tonic spikes. A–E, histograms comparing several features of the spike-triggered average excitatory phase (phase maximum, phase magnitude, phase duration, and time to phase maximum). E–H, histograms comparing the same features of the spike-triggered average suppressive phase.

Spatial properties of visual stimuli that evoke bursts and tonic spikes

Using an m-sequence modulated, white-noise stimulus (Reid et al., 1997) to evoke responses from LGN neurons, Alitto et al. (2005) used reverse-correlation analysis to examine the spatial properties of visual stimuli that drive burst and tonic spikes in LGN neurons. Similar to results from their temporal analysis, the average stimulus to drive burst spikes is similar to, but significantly different from, the average stimulus to drive tonic spikes (Rivadulla et al., 2003).

In the spatial domain, burst and tonic receptive fields are always centered at the same spatial location and always share the same center/surround organization (on/off or off/on) (Fig. 3A and B). Closer examination of the center and surround subregions, however, reveals several significant differences between burst and tonic receptive fields that can be quantified by fitting receptive fields to difference of Gaussians (DOG) equations. A particularly notable difference between burst and tonic receptive fields is a greater surround to center ratio for burst spikes compared to tonic spikes (Fig. 3C; burst spikes: mean ratio $= 0.27 \pm 0.02$, tonic spikes: mean ratio $= 0.23 \pm 0.01$; $p < 0.05$; $n = 32$). The larger surround to center ratio for burst spike receptive fields is not due to changes in the spatial extent of the surround and center subregions (Fig. 3D; mean Δ surround $\sigma_{(\text{burst vs. tonic})} = -0.025 \pm 0.037$, $p = 0.49$; mean Δ center $\sigma_{(\text{burst vs. tonic})} = 0.013 \pm 0.014$, $p = 0.36$), but rather, is due to a disproportionate increase in the amplitude of the surround subregion compared to the center subregion (Fig. 3D; mean Δ surround amplitude$_{(\text{burst vs. tonic})} = 0.155 \pm 0.044$, $p = 0.01$; mean Δ center amplitude$_{(\text{burst vs. tonic})} = 0.053 \pm 0.016$, $p = 0.01$).

The finding that burst spikes, compared to tonic spikes, require a disproportionate increase in stimulation to the surround and center subregions of the receptive field may reflect an increase in the spike

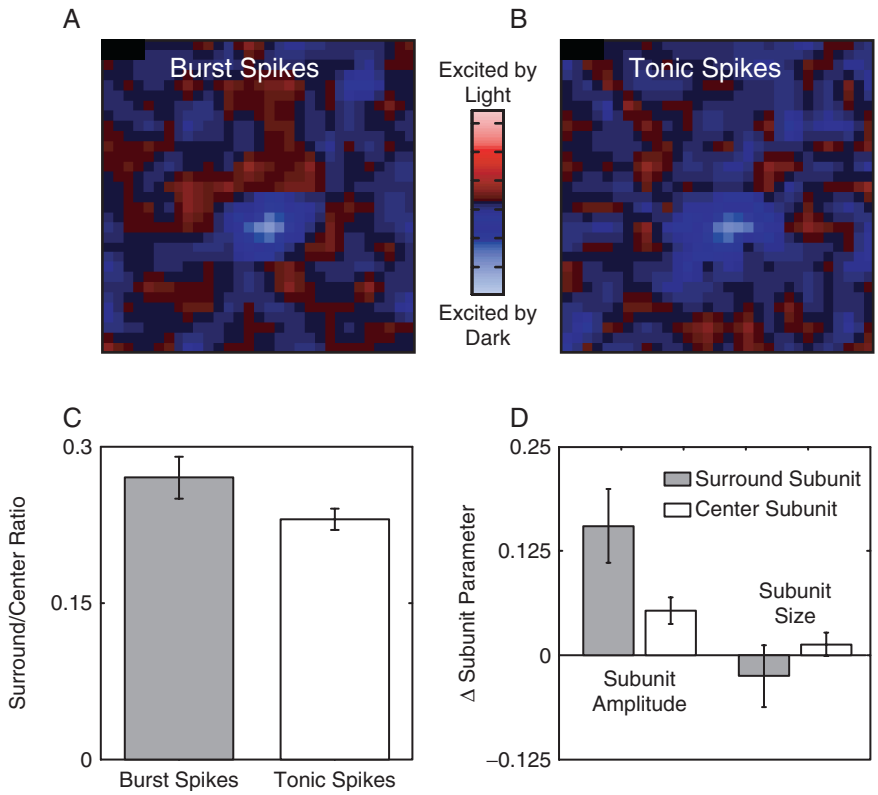

Fig. 3. Spatial properties of burst and tonic receptive fields. Using reverse-correlation analysis, receptive field maps were made from LGN responses to an m-sequence modulated, 16×16 checkerboard pattern of pixels. A and B, receptive field maps showing the average spatial stimulus to evoke burst and tonic spikes from a representative neuron. C, histogram comparing the surround to center ratio calculated from the receptive fields (burst and tonic) of 32 LGN neurons. D, histogram showing that the increase in surround to center ratio for burst spikes is due to a disproportionate increase in the strength of surround subregion in the burst receptive field and not due to a change in the spatial extent of subregions.

threshold of burst spikes compared to tonic spikes. This possibility can be illustrated by convolving a model LGN receptive field with the same white-noise stimulus used to map receptive fields. By performing the convolution twice (Fig. 4), once using a low spike threshold and again using a high spike threshold, one can qualitatively mimic the increase in the surround to center ratio of burst spikes (high threshold) compared to tonic spikes (low threshold).

While it may seem reasonable that LGN neurons should require more excitation to reach spike threshold when they are more hyperpolarized (i.e., prior to a burst event), in vitro studies have shown that an otherwise subthreshold current injection can evoke a burst from LGN neurons when T-type Ca^{2+} channels are de-inactivated (Lo et al., 1991). Based on this finding, one might expect bursts to require *less* excitatory drive from a visual stimulus than tonic spikes and certainly not *more* excitatory drive, as reported by Alitto et al. (2005). One possible explanation for this paradoxical set of findings rests on considering the dynamic relationship between low threshold currents in the LGN and retinal drive during visual stimulation. Because LGN neurons receive input from retinal ganglion cells with very similar receptive fields (Levick et al., 1972; Mastronarde, 1987; Usrey et al., 1999), suppressive stimuli that hyperpolarize LGN neurons (via a withdrawal of excitation and/or polysynaptic inhibition) should also hyperpolarize the retinal ganglion cells that provide their

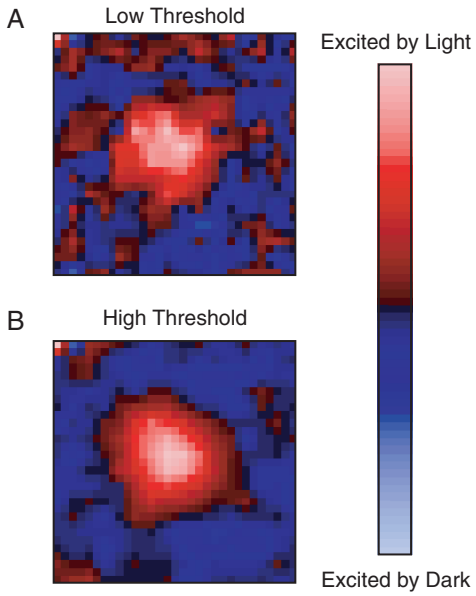

Fig. 4. A variable spike threshold predicts a change in the surround to center ratio under low and high thresholds. A, under low threshold conditions, a linear model of a difference of Gaussians (DOG) receptive field yields a low surround to center ratio. B, under high threshold conditions, the surround to center ratio is increased.

input. However, if LGN neurons hyperpolarize to a greater extent than the retinal ganglion cells that provide their input, then it may be possible for a visual stimulus to de-inactivate T-type Ca^{2+} channels in the LGN, but not T-type Ca^{2+} channels in the retina. As a result, retinal ganglion cells would not have access to low threshold Ca^{2+} currents and would require a stronger visual stimulus to reach spike threshold. In other words, a visual stimulus that decreases spike threshold in an LGN neuron would also decrease the retinal drive necessary for the LGN neuron to reach spike threshold. As a result, a stronger visual stimulus would be needed to drive the retina to a level sufficient for the LGN to reach threshold.

Bursts and thalamocortical processing

While the experiments described above demonstrate that burst and tonic spikes carry distinct spatiotemporal information to the cortex (Reinagel et al., 1999), an important question is whether or not there exists a cortical mechanism for distinguishing these two categories of spikes. A potential readout for burst spikes is based on the dynamic properties of thalamocortical synapses. Several studies examining synaptic transmission at the thalamocortical synapse report that these synapses experience synaptic depression (Stratford et al., 1996; Gil et al., 1999; Chung et al., 2002). If so, then the long interspike interval preceding the cardinal spike of a burst would allow thalamocortical synapses to recover from depression and thereby increase thalamocortical burst efficacy. Even if thalamocortical synapses experience little or no depression (Boudreau and Ferster, 2003), the rapid train of spikes within a burst should experience temporal summation (Usrey et al., 2000; Roy and Alloway, 2001) and thereby lead to a similar increase in thalamocortical burst efficacy. Either way, the temporal structure of a thalamic burst seems ideal for increasing the efficacy of LGN neurons in driving cortical responses.

Using cross-correlation techniques to study thalamocortical transmission in the somatosensory pathway of the awake rabbit, a recent study reports that burst are indeed more effective than tonic spikes at driving cortical responses (Swadlow and Gusev, 2001). Consistent with the notion that thalamocortical synapses experience synaptic depression, this study also reports that the cardinal spike of a burst is always the most effective spike. Following the cardinal spike, subsequent burst spikes are similar to tonic spikes in their ability to drive cortical responses. Although a direct study of burst efficacy has yet to be performed for neurons in the visual pathway, if one assumes that LGN bursts are similarly more effective than tonic spikes at driving cortical responses, then LGN bursts would seem to have all of the necessary ingredients to represent a distinct mode for processing and conveying visual information to the cortex.

Conclusions and future directions

LGN neurons, like all thalamic neurons, produce two distinct categories of spikes — burst spikes and tonic spikes. While most efforts at understanding burst and tonic activity at a systems level have emphasized a role for extra-retinal inputs and their

influence on the membrane properties of thalamic neurons (Steriade and Llinas, 1988; Steriade et al., 1990; Sherman, 1996, 2001), this review has examined recent progress made toward understanding what role the visual stimulus (and presumably the retinogeniculate pathway) plays in directly evoking burst and tonic activity. Compared to tonic spikes, results show that burst spikes (1) occur with a shorter latency between stimulus and response, (2) have a greater dependence on stimuli with transitions from suppressive to preferred states, and (3) prefer stimuli that provide increased drive to the receptive field center and even greater increased drive to the receptive field surround.

While results indicate that burst and tonic spikes follow distinct spatiotemporal patterns of visual stimuli, a number of important questions concerning the influence of sensory stimuli on burst and tonic activity remain unanswered. Perhaps most important, more data needs to be obtained from awake animals. Indeed, all of the data presented in this review come from the anesthetized cat. Given the justified concern that burst activity is diminished in awake animals (Guido and Weyand, 1995; Ramcharan et al., 2000; Weyand et al., 2001; Royal et al., 2003) and therefore may not contribute significantly to sensory processing outside of the anesthetized state, more experiments need to be performed in awake animals to determine the extent to which burst activity contributes to sensory processing. Along these lines, we know little or nothing about what effects do behavioral state, attention, statistics of the visual stimulus, or eye-movement history have on burst and tonic activity. Similarly, we know very little about what effect do cortical feedback and other sources of nonretinal input have on burst activity in awake animals. Given the evidence that bursts are more effective than tonic spikes at driving cortical responses (Swadlow and Gusev, 2001), these are important questions to be answered and the answers will likely change the way we perceive sensory processing and the retinogeniculcortical pathway.

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