An Intrinsic Oscillation in Interneurons of the Rat Lateral Geniculate Nucleus

J. JULIUS ZHU, 1 WILLIAM W. LYTTON, 2 JIN-TANG XUE, 1 AND DANIEL J. UHLRICH 1
1 Department of Anatomy and 2 Department of Neurology, Neuroscience Training Program, Wm. S. Middleton VA Hospital, University of Wisconsin, Madison, Wisconsin 53706

Zhu, J. Julius, William W. Lytton, Jin-Tang Xue, and Daniel J. Uhlrich. Intrinsic oscillation in interneurons of the rat lateral geniculate nucleus. J. Neurophysiol. 81: 702–711, 1999. By using the whole cell patch recording technique in vitro, we examined the voltage-dependent firing patterns of 69 interneurons in the rat dorsal lateral geniculate nucleus (LGN). When held at a hyperpolarized membrane potential, all interneurons responded with a burst of action potentials. In 48 interneurons, larger current pulses produced a bursting oscillation. When relatively depolarized, some interneurons produced a tonic train of action potentials in response to a depolarizing current pulse. However, most interneurons produced only oscillations, regardless of polarization level. The oscillation was insensitive to the bath application of a combination of blockers to excitatory and inhibitory synaptic transmission, including 30 μM 6,7-dinitroquinoxaline-2,3-dione, 100 μM (+)-2-amino-5-phosphonopentanoic acid, 20 μM bicuculline, and 2 mM saclofen, suggesting an intrinsic event. The frequency of the oscillation in interneurons was dependent on the intensity of the injection current. Increasing current intensity increased the oscillation frequency. The maximal frequency of the oscillation was 5–15 Hz for most cells, with some ambiguity caused by the difficulty of precisely defining a transition from oscillatory to regular firing behavior. In contrast, the interneuron oscillation was little affected by preceding depolarizing and hyperpolarizing pulses. In addition to being elicited by depolarizing current injections, the oscillation could also be initiated by electrical stimulation of the optic tract when the interneurons were held at a depolarized membrane potential. This suggests that interneurons may be recruited into thalamic oscillations by synaptic inputs. These results indicate that interneurons may play a larger role in thalamic oscillations than was previously thought.

INTRODUCTION

The brain regularly switches between sleep and wakefulness, and the transition between these two states is accompanied by dramatic changes in synchronized neural activity (Steriade 1997; Steriade et al. 1993, 1994). Electroencephalographic (EEG) studies show that a sleeping brain is dominated by large-amplitude, slow-frequency oscillations, whereas an aroused brain is dominated by low-amplitude, high-frequency oscillations. The thalamus, the primary structure relaying sensory inputs from the periphery to the cortex in mammals, is believed to play a key role in generating and maintaining these rhythms (Steriade et al. 1993). In fact, changes in thalamic rhythms are a dramatic feature of sleep-wake transitions (Steriade et al. 1990, 1993). The thalamus is also implicated in abnormal thalamic rhythms, such as absence seizures (Steriade et al. 1993; Tsakiridou et al. 1995; von Krosigk et al. 1993).

Three basic neuron types were described in the thalamus (Jones 1985). Thalamocortical cells are excitatory and project an axon to cortex. Local interneurons, which reside among the thalamocortical cells in the principal relay nuclei, and cells in the adjacent thalamic reticular nucleus (TRN) are GABAergic, and their axons are restricted to the thalamus. Thalamocortical and TRN cells are implicated heavily in oscillations (Steriade et al. 1993). Thalamocortical cells can oscillate intrinsically at low frequencies (1–4 Hz, the frequency range of delta oscillations in the EEG). The interplay of two conductances, the low-threshold calcium conductance, \( I_t \), and the hyperpolarization-activated cation conductance, \( I_h \), is crucial for this oscillation (Destexhe et al. 1993; Leresche et al. 1991; McCormick and Huguenard 1992; McCormick and Pape 1990; Soltetz et al. 1991). The intrinsic oscillation in thalamocortical cells prevails at a hyperpolarized membrane potential where \( I_h \) and \( I_t \) are largely deinactivated and activated, respectively. TRN cells are also endowed with an intrinsic oscillation (Avanzini et al. 1989; Bal and McCormick 1993). When a hyperpolarizing pulse is injected, these cells respond at pulse offset with a dampening oscillatory sequence, which consists of cyclic (7–14 Hz, the frequency range of sleep spindles in the EEG) bursts of action potentials that progressively decrease in frequency to rhythmic single spikes. The cellular mechanisms underlying this dampening spindle-frequency oscillation differ somewhat from those that underlie the delta oscillation in thalamocortical cells. In TRN cells, the interaction of \( I_h \) with a calcium-activated, nonselective cation conductance, \( I_{calc} \), and the calcium-dependent potassium conductance, \( I_{AHP} \), is thought to be the mechanism underlying the oscillation (Bal and McCormick 1993). Spindle-frequency oscillations may also be generated by the reciprocal connection between thalamocortical and TRN cells, with the promotion of the intrinsic properties of these cells (Bal et al. 1995a,b; Huguenard and Prince 1994; Warren et al. 1994).

Although the contributions of thalamocortical and TRN cells to oscillations are well studied, the role of thalamic local interneurons in these rhythms is less clear. Results from in vivo studies suggest indirectly that interneurons can oscillate (Deschenes et al. 1984; Steriade and Deschenes 1984; Steriade et al. 1976), and recent in vitro studies indicate that thalamic interneurons are endowed with critical oscillation-related conductances, such as \( I_h \), \( I_t \), and \( I_{calc} \) (Munch et al. 1997; Pape and McCormick 1995; Pape et al. 1994; Williams et al. 1996; Zhu et al. 1999a,b). However, the identity of the interneurons in the previously mentioned in vivo studies was not confirmed, and the results of recent in vitro studies of identified interneurons led to the conclusion that thalamic interneurons neither generate an intrinsic oscillation nor participate in oscillations that are a dramatic feature of sleep-wake transitions.
occur in the thalamic slice (Bal et al. 1995a; McCormick and Pape 1988). Other in vitro studies described rhythmic behavior in thalamic interneurons (Williams et al. 1996; Zhu et al. 1995).

It is possible that earlier in vitro studies that have not observed oscillations failed to do so because the cells were compromised by the sharp electrode impalements, which can introduce an artificial leak (Staley et al. 1992). In this study, we used the whole cell recording technique, which reportedly produces less leak during recording (Staley et al. 1992). By using this technique in an in vitro preparation, we found and characterized an intrinsic oscillation in interneurons of the thalamic lateral geniculate nucleus (LGN). A preliminary report of this study appeared in abstract form (Zhu et al. 1995).

METHODS

Electrophysiology

The slice preparation was described previously (Zhu and Uhlrich 1997). Briefly, Sprague-Dawley rats (100–300 g) were deeply anesthetized by halothane. After decapitation, the brain was quickly resected. After resection, slices containing LGN, each 500 μm thick, were cut from the tissue blocks with a microslicer. Slices were kept in an oxygenated physiological solution (in mM) containing 126 NaCl, 2.5 KCl, 1.25 NaH2PO4, 26 NaHCO3, 1 MgSO4, 20 dextrose, 2 CaCl2, at pH 7.35. The solution was continuously bubbled with 95% O2-5% CO2. Slices containing LGN, each 500 μm thick, were cut from the tissue blocks with a microslicer. Slices were kept in an oxygenated physiological solution for ≥2 h before recording. During the recording, slices were submerged in a Plexiglas chamber and stabilized with a fine nylon net attached to a platinum ring (Edwards et al. 1989). The chamber was perfused with warmed, oxygenated physiological solution, and the half time for the bath solution exchange was ~7 s. The temperature of the bath solution in the chamber was kept at 34.0 ± 0.5°C. Antagonists were applied with the bath solution.

The methods for tight-seal patch recordings follow Hamill et al. (1981), with modification for brain slices (Blanton et al. 1989; Edwards et al. 1989). Patch electrodes were made from borosilicate tubing (1.2 mm OD, 0.9 mm ID) with a horizontal puller (Sutter Instruments). Electrode resistances were 7–9 MΩ. The standard intracellular solution (in mM) was 120 Cs2HPO4, 10 N-2-hydroxymethylpiperazine-N’-2-ethanesulfonic acid, 5 ethylene glycol-bis(β-aminoethyl ether)-N,N,N’,N’-tetraacetic acid, 2 MgCl2, 4 ATP, 0.1 GTP, 0.5 CaCl2, 10 KCl, and biocytin 0.25% at pH 7.25. To obtain whole cell recordings, electrodes were advanced into the slice while applying 0.1-nA current steps lasting 200 ms. When a significant increase in electrode resistance was evident, gentle suction was applied to attain a whole cell configuration.

Current-clamp recordings were performed on an Axoclamp-2A amplifier (Axon Instruments). The bridge balance was continuously monitored and adjusted. The optic tract was stimulated by a concentric bipolar electrode with single voltage pulses (200 μs, ±9 V). A 10-nV liquid junction potential was subtracted from all membrane potentials.

We selected for study only interneurons with resting membrane potentials negative to −50 mV, input resistances >300 MΩ, and overshooting action potentials. Except when noted, recordings were very stable with little change in input resistance and resting membrane potential throughout the entire recording period. The recordings routinely lasted 1–6 h.

Autocorrelograms and second derivative calculations were done with NEURON (Hines and Carnevale 1997).

Histology

After each recording, the slice was fixed by immersion in 4% paraformaldehyde in 0.1 M phosphate buffer and resectioned at 60 μm on a freezing microtome. Sections were processed histologically with the avidin–biotin–peroxidase method to reveal the cell morphology (Horikawa and Armstrong 1988). Cells were subsequently drawn with the aid of a camera lucida system.

Drugs and chemicals

The following compounds were purchased from Research Biochemical International: (±)-2-amino-5-phosphonopentanoic acid (t-APV), β-aminomethyl-4-chloro-benzenethansulfonic acid (saclofen), bicuculline methiodide, 6,7-dinitroquinoxaline-2,3-dione (DNQX), picrotoxin, ATP, biocytin, GTP, and all other chemicals were purchased from Sigma.

RESULTS

We studied the response of 69 interneurons in rat dorsal LGN to current clamp. In correspondence with previous studies (Munch et al. 1997; Pape and McCormick 1995; Williams et al. 1996; Zhu and Uhlrich 1997), we found that LGN interneurons and thalamic cortical cells exhibited very different responses: interneurons had a higher input resistance and longer membrane time constant than thalamic cortical cells (Fig. 1A). These two properties alone were sufficient to distinguish the two cell types (Fig. 1B). The identity of all interneurons was confirmed morphologically (Fig. 1C).

Previously, others have shown that return of interneurons from a hyperpolarizing holding state can generate a calcium-mediated burst (Pape and McCormick 1995; Williams et al. 1996). In our hands, a depolarizing current boost consistently elicited such a burst (Zhu et al. 1999a). Figure 2 shows that a still greater depolarizing current injection generated a more vigorous burst response followed by rhythmic burst activity. Such an oscillation occurred in 48 (70%) interneurons in our sample. This subset of cells will be the focus of the following characterization.

The majority of interneurons studied produced oscillations regardless of whether the prior holding potential was a hyperpolarization or a depolarization. The sole factor required to produce an oscillation in most cases was a slightly depolarized potential. This result differed from earlier reports that show tonic firing at depolarized membrane potentials (McCormick and Pape 1988; Pape and McCormick 1995; Williams et al. 1996). A few cells showed evidence of tonic firing (Fig. 3A) coexisting with an oscillatory mode (Fig. 3B) that could be obtained with slightly different holding potentials or depolarizing plateaus. In most interneurons, however, no tonic firing was seen. In three oscillating interneurons, a large variety of protocols was used in an attempt to elicit tonic spiking. The protocols included the use of successive depolarizing current steps (e.g., Fig. 9), slow depolarizing ramps, depolarizing the cells to the point of sodium spike inactivation and then slowly hyperpolarizing. These maneuvers were designed to inactivate Ih and other channels that might be responsible for repetitive bursting. In all of these cases, the cell response was consistently oscillatory.

Oscillation is intrinsic

To determine whether the oscillation observed was intrinsic or due to circuitry, we tried blocking axonal conduction with tetrodotoxin (TTX). Bath application of TTX blocked the oscillation (n = 3; Fig. 4B), leaving only an initial calcium
spike at depolarization onset (cf. Zhu et al. 1999a). This result demonstrated either that the oscillation resulted from circuitry or that it was dependent on the sodium channel. To distinguish between these possibilities, we blocked both excitatory and inhibitory synaptic transmission with 30 μM DNQX, 100 μM di-APV, 20 μM bicuculline, and 2 mM saclofen. This combined blockade of non-N-methyl-D-aspartate (non-NMDA) glutamatergic, NMDA, γ-aminobutyric acid-A (GABA_A) and GABA_B receptors did not eliminate the oscillation (n = 4; Fig. 4B). In two of the four cells, 10 μM picrotoxin, a more potent GABA_A receptor blocker, was also included with no effect. These results suggest that the bursting oscillation in thalamic interneurons was intrinsic and dependent on sodium channels.

Although the oscillation was generated intrinsically, it could be initiated through synaptic activation of the optic tract in cells held just below firing threshold (Fig. 5B). At a slightly depolarized membrane potential, optic tract stimulation evoked a large IPSP (cf. Zhu et al. 1999a), followed by an oscillation similar to that elicited by current injection (Fig. 5A).

**Basic properties of the bursting oscillation in interneurons**

Figure 6 illustrates the oscillatory responses of four representative interneurons to a series of depolarizing current pulses. In general, the firing pattern showed two components: a robust initial burst comparable to that described previously (cf. Zhu et al. 1999a), followed by repetitive bursting. Sometimes the initial response was not a single burst but was instead two or more bursts in quick succession (Fig. 6B, middle, and D, top). The sustained oscillation generally showed regularly recurring multispike bursts with two to five action potentials at a 25- to 150-Hz intraburst frequency. In two cells repetitive bursts had 10–16 action potentials (not shown). In some cells the definition of the firing pattern to large depolarizing currents was ambiguous, with only a small difference between the intraburst interspike intervals and the interburst interspike intervals (compare Fig. 6A, top and bottom). In other cases, the firing pattern was quite regular (Fig. 6B, middle and top).

The character of the oscillation depended on the intensity of
the sustaining depolarizing current. Increasing the depolarizing current increased the duration of the initial burst and decreased the subsequent interburst intervals, resulting in higher-frequency oscillations. We used the first peak of the autocorrelogram to determine its frequency at different current injections (e.g., Fig. 6D, right). Results of this analysis are summarized for seven cells in Fig. 7. Interneurons that showed clear burst oscillations at all current injections (Fig. 6D) yielded a relatively shallow current-frequency slope. In contrast, interneurons that displayed nonburst firing at depolarized levels yielded a steeper current-frequency relationship.

Inspection of the nonburst firing seen at high current injections suggested the possibility of two different firing patterns. In some instances, the hyperpolarization trajectory of the interspike intervals was similar to the sharp intraburst trajectory at lower current injections. In other cases, these trajectories seemed more similar to the dull interburst trajectory seen with lower depolarizing current. We therefore hypothesized that the former, sharp trajectories were associated with increased burst duration such that the burst was continued throughout the period of depolarization, whereas the latter, dull trajectories represented a reduction in burst duration to the point where each “burst” was only a single spike. This latter situation would then represent a continuation of burst oscillation dynamics, which may be denoted “single-spike oscillation” (Fig. 6B, middle and top).

To quantify the difference between these firing patterns, we measured the sharpness of interspike hyperpolarizations by
Role of input resistance

In some cells, we observed that the burst oscillation was lost over the course of the recording (n = 3, Fig. 10). In these cases, the transition appeared to be associated with a substantial loss of input resistance and a depolarization of 7–12 mV in RMP. Despite these signs of injury, we were able to see consistent single-spike responses for another 30–60 min. Hypothesizing that the reduction in resistance resulted from a partial loss of the gigaseal between the patch pipette and cell membrane, we broke the gigaseal and produced a comparable drop in input resistance in two other cells by lightly vibrating the electrode manipulator. These two cells also lost the capacity to oscillate and subsequently exhibited single-firing behavior.

The previous result suggests that the ability to oscillate depends on the input resistance of the cell. Thus we compared the input resistances of the 48 cells that could sustain a burst oscillation with 21 cells that could not. The average input resistance in the former group (643.1 ± 216.0 MΩ) was higher than that in the latter group (553.5 ± 163.8 MΩ; t-test, P < 0.05). However, input resistance is not a strong predictor of firing behavior because we observed robust oscillations in cells whose input resistances spanned the entire range in our sample.

FIG. 4. Effect of synaptic transmission blockade on the bursting oscillation. A: depolarizing current injection induced a bursting oscillation in an interneuron. B: bath application of 4 μM tetrodotoxin blocked the oscillation. RMP = −68 mV. C: depolarizing current injection induced a bursting oscillation in another interneuron. D: bath application of 30 μM 6,7-dinitroquinoxaline-2,3-dione, 100 μM (−)-2-amino-5-phosphonopentanoic acid, 20 μM bicuculline, and 2 mM saclofen, which blocked both excitatory and inhibitory synaptic transmissions, had little effect on the oscillation. RMP = −64 mV. Inset: antagonists block postsynaptic potential recorded in a different cell after optic tract stimulation (arrow). Four trials are superimposed in each condition. Inset scale bar is 30 mV and 100 ms.

calculating the average positive second derivative (Fig. 8). Sharp intervals have relatively large second derivatives compared with rounded intervals, which have relatively small second derivatives. At low depolarizations (Fig. 8, bottom inset traces), the characteristic bursting oscillation generally showed a bimodal distribution of intervals, dull long trajectories from interbursts, and sharp, brief trajectories from the intraburst intervals (Fig. 8, B–F). In some cases, the distribution appeared to be trimodal, with evidence of longer- and shorter-duration interspike intervals within the burst, with corresponding dull and sharp trajectories (Fig. 8A). When a higher depolarizing current was injected into the cell (top inset traces), the interburst interval decreased in duration. For most cells (e.g., Fig. 8, A, B, D, and E), the distribution of intervals within the burst remained similar in duration and sharpness to the distribution produced at lower current injections. However, in several cases, higher current levels gave interspike trajectories that were uniformly dull, with second derivatives comparable to those of interburst intervals at the lower activation levels (Fig. 8, C and F). The small second derivative values of these interspike intervals, similar to those of an interburst interval, allows us to define these firing patterns as single spike oscillations.

The pattern of oscillation depended on the intensity of the depolarization during the oscillation, not on the holding potential before initiating the oscillation (Fig. 9). Varying the conditioning pulse did alter the frequency of action potentials in the initial burst, as shown previously (Zhu et al. 1999a). However, the conditioning pulse had little effect on the sustained oscillation. The pattern of oscillation was generally the same even when preceded by a depolarizing pulse (Fig. 9, top 2 traces). Varying the duration of the preceding conditioning pulse also had no effect on the sustained oscillation (not shown).

The cells are illustrated oscillating for a short-duration, stopping after the termination of the depolarizing current. We tested other interneurons with longer depolarizing pulses (2 min, n = 3; 20 min, n = 1; not shown) and found that the oscillation lasted as long as the depolarizing current was present.

FIG. 5. Bursting oscillations can be elicited synaptically. A: interneuron oscillates in response to depolarizing current injections. B: electrical stimulation of the optic tract (●) induced a large inhibitory postsynaptic potential in the same cell, followed by several cycles of rhythmic bursts and then single spike firing (cell membrane potential held at −47 mV, just below the threshold of sodium spikes). RMP = −70 mV.
DISCUSSION

Oscillations in LGN interneurons

We have shown that local interneurons in the LGN can intrinsically generate a low-frequency oscillation. The idea that interneurons can oscillate is consistent with early studies by Steriade and colleagues (Deschenes et al. 1984; Steriade and Deschenes 1984). In these previous in vivo studies, putative thalamic interneurons were identified by their prolonged, slow frequency burst firing after synaptic stimulation, a unique response that was not observed in the larger sample of putative thalamocortical cells (Burke and Sefton 1966; Deschenes et al. 1984). The authors also showed that interneurons were involved in both spontaneous and stimulus-evoked spindle oscillations in vivo (Deschenes et al. 1984; Steriade and Deschenes 1984). In the previous studies, the frequency of action potentials in each burst of the oscillation was lower than that in thalamocortical cells, which is consistent with the low intraburst frequency reported in this study (see also Zhu et al. 1999a). In addition, previous work also demonstrated that the bursting phase of the oscillation in interneurons was opposite of that in thalamocortical cells (Deschenes et al. 1984), consistent with the interaction between inhibitory and excitatory cell types (Andersen and Andersson 1968). Finally, both in vivo and in vitro studies reported spontaneous oscillatory inhibitory postsynaptic potential (IPSP) sequences at spindle frequency in thalamocortical cells (Pape and McCormick 1995; Steriade et al. 1985). These IPSP sequences persist after the TRN was disconnected anatomically from the thalamocortical cells, suggesting that these events are generated by the local interneurons (Steriade et al. 1985).

Previous studies (Bal et al. 1995a; McCormick and Pape 1988), concluded that geniculate interneurons are unable to generate oscillations. A few factors may contribute to the difference with our results. First, we used the whole cell technique, whereas the previous studies used sharp electrodes. Impalement by sharp electrodes typically results in a decrease in input resistance, especially in small neurons (Staley et al. 1992). After impalement, the currents that originally were large enough to support a bursting oscillation may no longer be sufficient because of a decrease in input resistance. Furthermore, the leak introduced by sharp electrodes will make it more difficult to see dendritic activity. It is possible that the oscillations that we recorded are

![Figure 6](http://jn.physiology.org/DownloadedFrom/10.220.33.6)  
**FIG. 6.** Varying oscillation patterns with increasing current injection in 4 interneurons. Traces in each row are plotted relative to the membrane potential shown on the left. Right inset: autocorrelation function for each trace in D.

![Figure 7](http://jn.physiology.org/DownloadedFrom/10.220.33.6)  
**FIG. 7.** Oscillation increased in frequency with increasing depolarizing steps in a representative sample of cells. The 4 indicated curves (A–D) refer to the 4 cells illustrated in Fig. 6.
being generated out in the dendrites and were thereby inaccessible to sharp electrode recording. In this context, we note the recent study of Williams et al. (1996), who also reported an intrinsic 10-Hz oscillation in LGN interneurons. Although they used sharp electrodes and reported input resistances below those of the current study, their resistances were considerably higher than those of other studies that used sharp electrodes.

It is possible that we may have recorded from a different population of interneurons from those in previous studies because at least two populations of interneurons are present in the thalamus (Bal et al. 1995a; Famiglietti 1970; Gabbott and Bacon 1994). However, we evaluated a large number of interneurons and found nothing that would permit us to dichotomize them physiologically or anatomically. Although the finding of lower input resistance in cells that did not oscillate might suggest that these belong to a different category, the appearance of this firing pattern late in the recording of cells that previously oscillated makes this unlikely. It is also possible that the oscillation we observed might have resulted from the washout effect by patch electrodes (Pusch and Neher 1988). However, this also seems unlikely: in many interneurons, the oscillation was found immediately after the whole cell configuration was formed, before the washout could occur. In some of these recordings, the access resistance could be as high as 100 MΩ, although for most cells it was <20 MΩ.

**Mechanism of oscillation in LGN interneurons**

The nature of the interneuron oscillation appears to be quite distinct from that of thalamocortical neurons. Although thalamocortical cells oscillate at relatively hyperpolarized levels, a depolarizing boost is necessary for the interneuron oscillation. Such a depolarization would largely inactivate I\(_t\) and would preclude activation of I\(_h\), the two channels critical to the thalamocortical oscillation (McCormick and Pape 1990).

Instead we suggest that the interneuron oscillation is more like that seen in thalamic reticular neurons, which also oscillate at relatively depolarized levels. Indeed, we recently showed that interneurons possess I\(_{can}\) (Zhu et al. 1999a), which is believed important in the TRN oscillation (Destexhe et al. 1994). The long-lasting inward current of I\(_{can}\) may augment the
depolarized state on which the bursts occur. Alternatively, the ability of TTX to eliminate the oscillation suggests that this sustaining inward current might be a persistent sodium channel. Other interpretations are possible and will need to be explored further.

**Functional significance of the intrinsic oscillation in interneurons**

Thalamic oscillations appear to be involved in both normal thalamic rhythms, such as those associated with sleep, and abnormal rhythms, such as absence seizures (Steriade et al. 1993; Tsakiridou et al. 1995; von Krosigk et al. 1993). Recent studies demonstrated that both thalamocortical and TRN cells are endowed with intrinsic rhythms that oscillate at delta and spindle frequencies, respectively (Bal and McCormick 1993; Leresche et al. 1991; McCormick and Pape 1990; Soltesz et al. 1991). Synaptic connections between thalamocortical and TRN cells also support a delta or spindle rhythm (Bal et al. 1995a; 1995b; Huguenard and Prince 1994; Warren et al. 1994). It was therefore suggested that these two cell types are responsible for generating and promoting these thalamic rhythms (Steriade et al. 1993; von Krosigk et al. 1993).

We found that thalamic interneurons also generate an intrinsic oscillation across a frequency range that includes the delta and spindle rhythms. Because interneurons are connected synaptically to both thalamocortical cells and reticular cells (Ahlström et al. 1985; Crunelli et al. 1988; Liu et al. 1995; Paré et al. 1991) and inhibitory potentials can readily entrain postsynaptic firing (Andersen and Andersen 1968; Cobb et al. 1995; Lytton and Sejnowski 1991; Whittington et al. 1995), we suggest that interneurons also play a role in promoting and synchronizing thalamic oscillations. This idea is consistent with in vivo studies that showed bursting oscillations in thalamic interneurons (Deschenes et al. 1984; Steriade and Deschenes 1984). Further study will be needed to fully explore the complex
interactions of interneurons with thalamocortical and TRN cells during thalamic oscillations.

This research was supported by the National Eye Institute (D. J. Uhlrich), the National Institute of Neurological Disease and Stroke (W. W. Lytton), and by the Office of Research and Development, Medical Research Service of the Department of Veterans Affairs (W. W. Lytton).

Present address of J. J. Zhu: Dept. of Cell Physiology, Max-Planck-Institute for Medical Research, Jahnstr. 29, Heidelberg D-69120, Germany.

Address for reprint requests: D. J. Uhlrich, Dept. of Anatomy, University of Wisconsin, 1300 University Ave., Madison, WI 53706.

Received 12 December 1997; accepted in final form 6 October 1998.

REFERENCES


