

DIFFERENTIAL RESPONSE DYNAMICS OF CORTICOTHALAMIC GLUTAMATERGIC SYNAPSES IN THE LATERAL GENICULATE NUCLEUS AND THALAMIC RETICULAR NUCLEUS

G. M. ALEXANDER,^a T. L. FISHER^a
AND D. W. GODWIN^{a,b*}

^aThe Neuroscience Program, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157, USA

^bDepartment of Neurobiology and Anatomy, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157, USA

Abstract—The corticothalamic feedback pathway provides excitatory synaptic input to both the thalamic reticular nucleus and the lateral geniculate nucleus. We studied excitatory postsynaptic currents elicited from corticothalamic stimulation in the visual sector of the thalamic reticular nucleus and the lateral geniculate nucleus to compare the response of these neurons to stimulation of their common input pathway. Using whole cell patch clamp recordings in ferret thalamic slices, we compared single excitatory postsynaptic current decay kinetics, presynaptic glutamate release dynamics through paired pulse facilitation and responses to corticothalamic train stimulation. We found that single thalamic reticular nucleus excitatory postsynaptic currents were significantly sharper than lateral geniculate nucleus responses. The mean thalamic reticular nucleus excitatory postsynaptic current decay constant (τ) was 4.9 ± 0.5 ms, while the mean lateral geniculate nucleus excitatory postsynaptic current τ value was 11.8 ± 0.8 ms. Presynaptic release dynamics as measured by responses to paired stimuli were conserved between the thalamic reticular nucleus and lateral geniculate nucleus. However, facilitating responses to train stimulation were markedly different between nuclei. Lateral geniculate nucleus responses showed proportionately larger facilitation (reaching $842.9 \pm 76.4\%$ of excitatory postsynaptic current 1 amplitude) than thalamic reticular nucleus responses (reaching $223.1 \pm 44.0\%$ of excitatory postsynaptic current 1 amplitude). These data indicate that while the corticothalamic pathway produces excitatory postsynaptic currents in both the thalamic reticular nucleus and lateral geniculate nucleus, other factors uniquely affect the functional integration of the inputs in each nucleus. © 2005 Published by Elsevier Ltd on behalf of IBRO.

Key words: EPSC, AMPA, GluR, thalamus, paired pulse facilitation, desensitization.

*Correspondence to: D. W. Godwin, Department of Neurobiology and Anatomy, Wake Forest University School of Medicine, Medical Center Boulevard Winston-Salem, NC 27157, USA. Tel: +1-336-716-9437; fax: +1-336-716-4534.

E-mail address: dgodwin@wfubmc.edu (D. W. Godwin).

Abbreviations: ACSF, artificial cerebrospinal fluid; AMPA, α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; EGTA, ethylene glycol-bis(*b*-aminoethyl ether)-*N,N,N',N'*-tetraacetic acid; EPSC, excitatory postsynaptic current; HEPES, *N*-[2-hydroxyethyl]piperazine-*N'*-[2-ethanesulfonic acid]; LGN, lateral geniculate nucleus; NMDA, *N*-methyl-D-aspartate; PPF, paired pulse facilitation; PPR, paired pulse facilitation ratio; TRN, thalamic reticular nucleus; VP, ventroposterior nucleus.

0306-4522/06/\$30.00+0.00 © 2005 Published by Elsevier Ltd on behalf of IBRO.
doi:10.1016/j.neuroscience.2005.11.012

One of the most compelling issues in thalamic physiology is the role of the massive feedback that thalamic relay neurons receive from their target sensory cortices. In the visual system, synapses derived from the feedback pathway originating in cortical layer VI have been shown to be numerically superior to those provided by retinal ganglion cells (Wilson et al., 1984; Erisir et al., 1997). In addition to dense innervation of lateral geniculate nucleus (LGN) relay neurons, layer VI neurons emit collaterals into the visual sector of the adjacent thalamic reticular nucleus (TRN), which contains only GABAergic neurons that project to the LGN (Montero and Singer, 1984; Robson, 1983). Cortical excitation of TRN cells inhibits LGN cells via inhibitory postsynaptic potentials (IPSPs), with both GABA_A and GABA_B receptor involvement (Sanchez-Vives and McCormick, 1997). This disynaptic inhibitory influence is central to the mechanism of sleep spindles (McCormick and Bal, 1997) and may be selectively involved in active visual processing (Weese et al., 1999; Montero, 2000).

Since the LGN and TRN receive common inputs from layer VI neurons, at first glance the influence of corticothalamic inputs on LGN and TRN neurons should be similar. However, there are indications that TRN neurons may respond differently to this input. For example, TRN cells have been shown to express higher levels of the α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor subunit, GluR4, and therefore show increased sensitivity to corticothalamic excitation than associated relay nuclei (Mineff and Weinberg, 2000; Golshani et al., 2001). Despite the possibility of differing postsynaptic responses to this common input, the relative functional influence of corticothalamic inputs has not been comparatively assessed. In this study, we characterized aspects of excitatory postsynaptic currents (EPSCs) recorded in the LGN and TRN upon corticothalamic stimulation using whole cell patch clamp recordings. We first measured the decay of individual postsynaptic glutamatergic responses from neurons in each nucleus and found them to possess distinctive kinetics. We then compared presynaptic release by using paired pulse stimulation and found that basic release dynamics in response to paired pulses are conserved between the TRN and LGN. However, on assessing the responses of LGN and TRN neurons to high frequency stimulus trains we observed that the responses to paired pulses did not predict the degree of facilitation to prolonged stimuli. Facilitation at corticogeniculate synapses was more pronounced than that of TRN neurons in response to train stimulation at the same frequency. We conclude that TRN neurons quickly reach maximum facilitation in response to corticothalamic feedback, while LGN neurons show a

greater dynamic range of facilitation that scales more linearly to corticothalamic feedback.

EXPERIMENTAL PROCEDURES

Slice preparation

Brain slice recordings were made from ferrets as reported previously (Alexander and Godwin, 2005). Briefly, young-adult male ferrets (Marshall Farms; North Rose, NY, USA) older than postnatal day 42 (eyes open at postnatal day 35) were anesthetized with halothane and decapitated in accordance with the Wake Forest University Animal Care and Use Committee and in agreement with National Institutes of Health and United States Department of Agriculture guidelines, including measures to eliminate suffering and to reduce animal numbers to a minimum. The brain was rapidly removed and immersed in oxygenated, ice cold sucrose substituted artificial cerebrospinal fluid (ACSF). Sections, at 400 μm thickness, containing the LGN, TRN and optic radiations were then cut and maintained in oxygenated, warm (34 °C) normal ACSF.

Whole cell recordings

Recordings were made from either the A laminae of the LGN or the TRN as previously described (Alexander and Godwin, 2005). Patch pipettes (5–10 M Ω) were filled with internal solution containing (in mM): 100 gluconic acid, 100 cesium hydroxide (CsOH), 10 sodium chloride, 10 HEPES, 20 tetraethylammonium chloride, 1 EGTA, 4 ATP disodium salt, (pH 7.3 with 2 N CsOH, osmolarity 270–290 mOsm). All experiments were run at 34 °C. Cellular activity was acquired with an AxoClamp 2B amplifier (Axon Instruments, Union City, CA, USA), digitized with a Digidata 1322 (Axon Instruments), and analyzed using pCLAMP 9.0 software (Axon Instruments). PicROTOXIN (100 μM ; Sigma-RBI, St. Louis, MO, USA) and DL-amino-5-phosphopentanoic acid (50 μM ; Sigma-RBI) were routinely included in the ACSF to block GABA_A inhibition and N-methyl-D-aspartate (NMDA) excitation, respectively. Cells were held at a hyperpolarized membrane potential, between –75 and –85 mV. A bipolar stimulating electrode, powered by a stimulus isolator (World Precision Instruments, Sarasota, FL, USA), was positioned in the optic radiations. For train experiments, stimulation intensity was kept to a minimum.

Analyses

For analysis of single EPSC decay constants, an exponential function with one term was fitted to the EPSC decay from peak amplitude, and a τ value was calculated using pCLAMP 9.0 software. For paired pulse facilitation (PPF) experiments, the ratio of the amplitude of peak 2 to the amplitude of peak 1 was calculated for three to five sweeps for each cell which was then averaged at each interstimulus interval time point to yield paired pulse facilitation ratios (PPFRs) at each interstimulus interval for a single cell. To measure statistical significance of EPSC amplitude facilitation during train stimulation, we performed a log transformation of the stimulus number within the train and plotted the EPSC amplitude versus the transformed axis for each response in the train to linearize the data. Significance was then assessed as a significant difference in the slope of the best fitting lines through these TRN and LGN data. Although the mean % of control values is reported in text, all statistical analyses were performed on raw data.

RESULTS

We recorded glutamatergic EPSCs elicited from stimulation of the optic radiations. Recordings were made from 36 TRN cells with a resting membrane potential of -68.2 ± 1.0 mV and 34 LGN relay cells, with a resting membrane potential of -61.0 ± 1.3 mV. TRN recordings were made

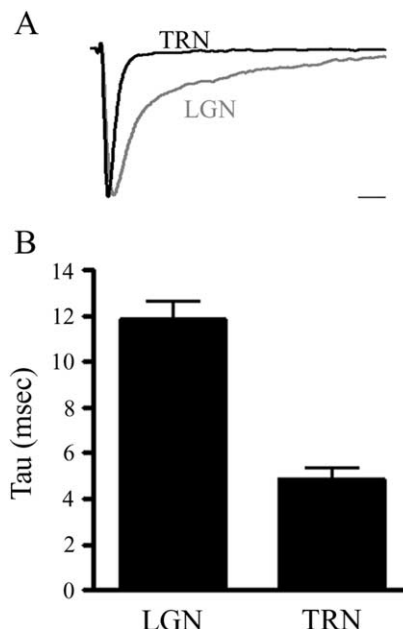


Fig. 1. Cortical stimulation elicits sharper EPSCs in the TRN than the LGN. (A) Scaled EPSCs recorded in TRN (black trace) and LGN (gray trace) upon stimulation of the optic radiations. LGN cell response decay is longer than TRN response decay. Note also the time to peak from the stimulation artifact of the TRN EPSC is faster than the LGN EPSC. (B) Values of τ for a population of TRN and LGN cells upon cortical stimulation. EPSCs were fit with a single exponential decay function to measure τ values. The mean τ value measured from LGN cells ($n=21$) was significantly greater than that measured from TRN cells ($n=22$). Scale bar=10 ms.

from the perigeniculate nucleus, a visual sector of the TRN that is reciprocally connected with the LGN (Fitzgibbon, 2002). Relay cells in the LGN were identified based on the physiological presence of burst firing, which is rare or absent in interneurons due to masking by a transient K⁺ current (I_A) (Pape and McCormick, 1995).

LGN and TRN cell EPSCs show different decay kinetics

EPSCs recorded in the two nuclei yielded responses with distinctive decay kinetics (Fig. 1). EPSCs recorded in the TRN were sharper than those recorded in the LGN. We fit EPSCs recorded in the two nuclei with a single exponential decay function starting from the peak of the response and measured the time constant of decay (τ) value. For all fits, correlations (r values) were greater than 0.96, and sum of squares errors less than 0.01. Values of τ measured from TRN cell EPSCs (4.9 ± 0.5 ms, $n=22$) were significantly less than those measured from LGN cell EPSCs (11.8 ± 0.8 ms, $n=21$; $P < 0.01$).

Short term synaptic plasticity is similar between LGN and TRN cells

Analysis of EPSC decay kinetics suggested a difference in postsynaptic glutamatergic receptors contributing to differences in the time course of EPSCs. We also compared presynaptic glutamate release dynamics by measuring the

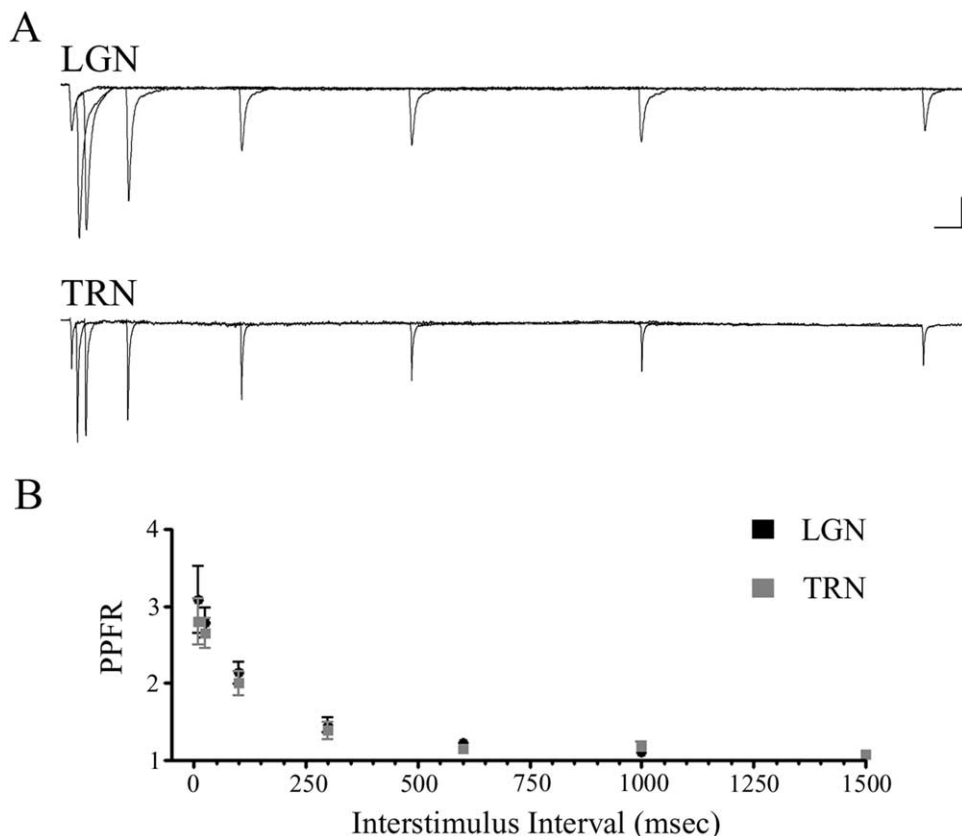


Fig. 2. PPFR in TRN and LGN cells upon stimulation of the optic radiations as a function of interstimulus interval. (A) Representative EPSC recorded in the LGN (top) and TRN (bottom) with interstimulus intervals of 10, 25, 100, 300, 600, 1000 and 1500 ms. (B) Plot of facilitation ratio for a population of LGN (black; $n=17$) and TRN cells (gray; $n=12$) as a function of the interstimulus interval. At all time points, the facilitation ratio measured between EPSC₁ and EPSC₂ did not differ between LGN and TRN cells. Values were fit with single exponential decay functions with τ values of 106.9 ms (LGN) and 99.8 ms (TRN). Scale bar=100 pA, 50 ms.

degree of PPF at the two synapses (Fig. 2). Two stimuli were delivered to the optic radiations at interstimulus intervals of 10, 25, 100, 300, 600, 1000 and 1500 ms. For both cell types the peak PPFR occurred at the shortest interstimulus interval of 10 ms. Peak PPFR for TRN cells was 2.8 ± 0.3 ($n=12$), and the peak PPFR for LGN cells was 3.1 ± 0.4 ($n=17$). As shown in Fig. 2B, we plotted the PPFR values at each interstimulus interval for each cell type. These data points were fit with single exponential decay functions with decay constants of 106.9 ms for LGN cells and 99.8 ms for TRN cells, and these values did not significantly differ ($P>0.05$). There was no significant difference between PPFR values obtained from the LGN versus the TRN at any interstimulus interval ($P>0.05$), and even at the longest interstimulus interval of 1500 ms facilitation was found in both nuclei. These data suggest that while differences exist between postsynaptic non-NMDA receptor function in the two nuclei, there is no apparent difference in release dynamics from corticothalamic axon terminals in response to paired stimuli.

LGN and TRN cells show different patterns of facilitation in response to corticothalamic train stimulation

In addition to studying short term synaptic plasticity at the corticothalamic synapse in the LGN and TRN, we studied

facilitation of EPSCs in response to train stimulation. We stimulated the optic radiations with 5 s trains at 10 Hz using low stimulus intensity ($<80 \mu\text{A}$ for LGN recordings and $<30 \mu\text{A}$ for TRN recordings). The stimulus intensity required to elicit minimal synaptic responses in the TRN ($13.4 \pm 3.2 \mu\text{A}$) was significantly lower than that required to elicit minimal synaptic responses in the LGN ($51.8 \pm 5.8 \mu\text{A}$; $P<0.01$).

The first EPSC response amplitude for LGN cells ($12.0 \pm 0.9 \text{ pA}$) was significantly less than the amplitude of the first response for TRN cells ($54.6 \pm 17.4 \text{ pA}$, $P<0.01$). This finding is consistent with previous reports of quantal EPSC amplitude recorded in TRN being three to four times greater than that for relay nuclei (Golshani et al., 2001). As previously described, train responses from LGN neurons grew very rapidly in initial synaptic facilitation and then continued to grow at a slightly lower rate beyond the initial rise (Granseth, 2004; Alexander and Godwin, 2005).

Paired pulse responses recorded in the TRN were not predictive of the responses to prolonged trains. Train stimulation resulted in an initial facilitation during the first few responses that either quickly reached a plateau or slightly depressed (Fig. 3). We assessed the percent increase from EPSC₁ of the EPSC amplitude for each stimulus in the train. For the population of cells, the third response in

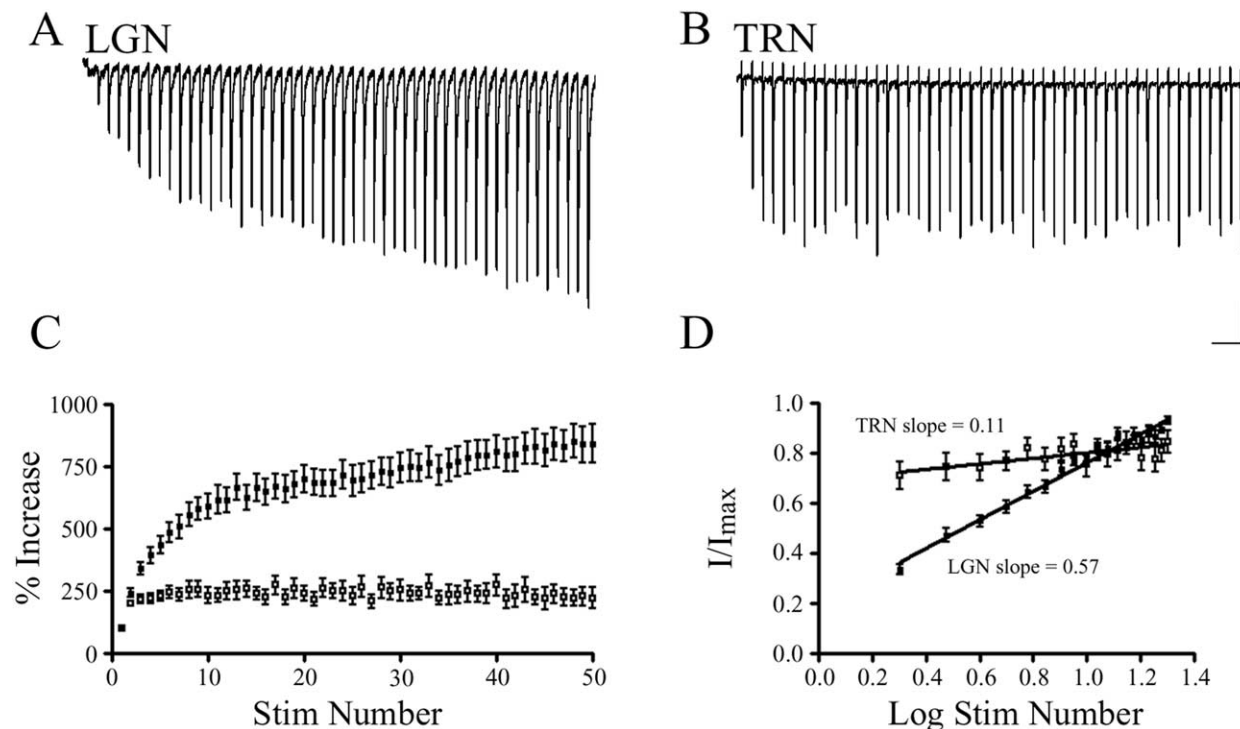


Fig. 3. The response of LGN and TRN cells to 10 Hz train stimulation 5 s in duration. Optic radiation train stimulation recorded in LGN (A) cells resulted in successively larger EPSCs without reaching plateau, while train stimulation recorded in TRN (B) cells resulted in an initial increase in EPSC amplitude which plateaued early in the train. Initial EPSCs from train stimulation recorded in the TRN were larger than initial responses recorded in the LGN. (C) Percent increase in EPSC amplitude from EPSC₁ in the train plotted as a function of the stimulus number within the 10 Hz train 5 s in duration. LGN cell responses (filled squares; $n=22$ cells) increased from stimulus 1 significantly more than TRN cell responses (open squares; $n=12$ cells). (D) I/I_{\max} values were calculated from EPSC amplitudes for stimulus numbers 2 through 20 in the train and plotted as a function of the log of the stimulus number in order to characterize the growth in EPSC amplitudes following the initial facilitation from EPSC₁ to EPSC₂. The slopes describing the rate of EPSC facilitation following the initial facilitation from EPSC₁ to EPSC₂ are shown on the graph. Scale bar=20 pA, 300 ms.

the train was the first to differ significantly in the rate of facilitation between the LGN and the TRN, consistent with our findings of no significant difference between LGN and TRN cells in PPFR (Fig. 2). LGN cells reached an EPSC amplitude of $842.9 \pm 76.4\%$ of EPSC₁ amplitude ($n=22$) on the fiftieth stimulus of the train. In TRN neurons, EPSC growth was significantly smaller, with the 50th stimulus eliciting an EPSC amplitude of $223.1 \pm 44.0\%$ of EPSC₁ amplitude ($n=12$). However, since the initial EPSC amplitude recorded in the TRN was significantly greater than that recorded in the LGN, the resulting EPSC amplitude at the end of the train was not significantly different. The amplitude of the last response was 153.0 ± 51.4 pA for LGN cells, which was not significantly different from the amplitude of the last response for TRN cells (165.9 ± 66.1 pA, $P>0.05$). Train responses in each nucleus were fit with exponential decay functions. LGN cell responses were fit with a double exponential with τ values of 75 and 1350 ms. TRN cell responses were fit with a single exponential decay function with a τ value of 101 ms.

In order to characterize the EPSC facilitation following the initial facilitation from EPSC₁ to EPSC₂ we calculated the I/I_{\max} values from EPSC amplitudes for stimulus numbers 2 through 20 in the train and plotted I/I_{\max} as a function of the log of the stimulus number. These values were fit with a linear regression to calculate the slope,

which describes the rate of EPSC facilitation following the initial facilitation from EPSC₁ to EPSC₂. As shown in Fig. 3D, the slope describing the rate of facilitation for neurons in each nucleus is linear on the log scale, but the LGN slope (0.57) was significantly larger than the slope describing the rate of facilitation in the TRN (slope=0.11, $P<0.01$). In both the LGN and the TRN, the slope differed significantly from zero ($P<0.01$). The correlation between facilitation and stimulus order within the train was significant for both LGN responses ($r=0.99$, $P<0.01$) and TRN responses ($r=0.71$, $P<0.01$).

DISCUSSION

The amplitude of corticothalamic EPSCs can be influenced by several presynaptic and postsynaptic factors, including dendritic filtering (Destexhe et al., 1996), presynaptic autoreceptors (Alexander and Godwin, 2005), and postsynaptic GluR subunit composition (Golshani et al., 2001). Dendritic filtering differs between LGN and TRN neurons, but LGN neurons are more electrically compact and solely on this basis we would expect to observe larger EPSCs in LGN cells, which is opposite to our results. While our results could also be attributed to differences in presynaptic distribution of mGluR2 autoreceptors, no differences in

protein density for these receptors have been observed between the LGN and TRN (Ohishi et al., 1998).

The density of GluRs and their composition may account for most of our results. Cortico-TRN synapses have been shown to possess three to four times greater GluR4 expression than cortico-ventroposterior nucleus (VP) synapses, which may explain the increased quantal response amplitude in the TRN compared with the VP (Mineff and Weinberg, 2000; Golshani et al., 2001). We have similarly observed larger EPSC amplitudes in TRN neurons in response to minimal stimulation relative to the LGN, consistent with an increased GluR density in the TRN.

Differences in the rate of AMPA receptor desensitization result in sharper EPSCs and reduced EPSC enhancement during train stimulation and therefore may account for the dramatic differences in the profiles of single AMPA receptor-mediated EPSCs and responses to train stimulation between the LGN and TRN (Jones and Westbrook, 1996). Variations in specific AMPA GluR subunit composition (GluRs 1–4, along with flip/flop splice variants) at a given synapse result in differing AMPA receptor desensitization rates (Dingledine et al., 1999; Schlesinger et al., 2005).

LGN cells express higher levels of GluR1 and GluR3 than TRN cells, and these subunits possess lower rates of desensitization (Dingledine et al., 1999; Schlesinger et al., 2005; Beneyto and Meador-Woodruff, 2004; Jones et al., 1998). Additionally, although our results of increased minimal EPSC amplitude in the TRN are consistent with the increased quantal EPSC amplitude in TRN relative to VP, no difference was observed in EPSC decay kinetics between the TRN and VP (Golshani et al., 2001) while we found significant differences between LGN and TRN. This may be explained by the increased expression of GluR1 and GluR3 in the LGN relative to the VP (Beneyto and Meador-Woodruff, 2004; Jones et al., 1998). Additionally, flip splice variants of GluRs have been shown to promote assemblies of slowly desensitizing AMPA receptors, while flop variants rapidly desensitize (Dingledine et al., 1999; Koike-Tani et al., 2005), producing sharper EPSCs and plateau of train responses.

The short term plasticity that is intrinsic to glutamatergic synapses enriches their computational repertoire (reviewed in Abbott and Regehr, 2004). Synapses that possess a low probability of release, such as corticothalamic feedback synapses (Granseth et al., 2002; Granseth, 2004; Alexander and Godwin, 2005), often show facilitation that may be important in the integration of inputs. In the context of corticothalamic feedback such integration may be important in proposed mechanisms of coincidence detection (Koch, 1987; Sillito and Jones, 2002). TRN neurons quickly reach their maximum facilitation, but facilitation in LGN cells scales more directly to the activity of layer VI feedback inputs. This greater dynamic range may promote more finely tuned responses of thalamocortical neurons in response to excitatory feedback signals.

Acknowledgments—This work was supported by EY11695; AA013246; AA011997; NS046222; Grant-In-Aid of Research from

the National Academy of Sciences, administered by Sigma Xi, the Scientific Research Society.

REFERENCES

- Abbott LF, Regehr WG (2004) Synaptic computation. *Nature* 431:796–803.
- Alexander GM, Godwin DW (2005) Presynaptic inhibition of corticothalamic feedback by metabotropic glutamate receptors. *J Neurophysiol* 94:163–175.
- Beneyto M, Meador-Woodruff JH (2004) Expression of transcripts encoding AMPA receptor subunits and associated postsynaptic proteins in the macaque brain. *J Comp Neurol* 468:530–554.
- Destexhe A, Contreras D, Steriade M, Sejnowski TJ, Huguenard JR (1996) In vivo, in vitro, and computational analysis of dendritic calcium currents in thalamic reticular neurons. *J Neurosci* 16:169–185.
- Dingledine R, Borges K, Bowie D, Traynelis SF (1999) The glutamate receptor ion channels. *Pharmacol Rev* 51:7–61.
- Erisir A, Van Horn SC, Sherman SM (1997) Relative numbers of cortical and brainstem inputs to the lateral geniculate nucleus. *Proc Natl Acad Sci U S A* 94:1517–1520.
- Fitzgibbon T (2002) Organization of reciprocal connections between the perigeniculate nucleus and dorsal lateral geniculate nucleus in the cat: a transneuronal transport study. *Vis Neurosci* 19:511–520.
- Golshani P, Liu XB, Jones EG (2001) Differences in quantal amplitude reflect GluR4-subunit number at corticothalamic synapses on two populations of thalamic neurons. *Proc Natl Acad Sci U S A* 98:4172–4177.
- Granseth B (2004) Dynamic properties of corticogeniculate excitatory transmission in the rat dorsal lateral geniculate nucleus in vitro. *J Physiol* 556:135–146.
- Granseth B, Ahlstrand E, Lindstrom S (2002) Paired pulse facilitation of corticogeniculate EPSCs in the dorsal lateral geniculate nucleus of the rat investigated in vitro. *J Physiol* 544:477–486.
- Jones EG, Tighilet B, Tran VB, Huntsman MM (1998) Nucleus- and cell-specific expression of NMDA and non-NMDA receptor subunits in monkey thalamus. *J Comp Neurol* 397:371–393.
- Jones MV, Westbrook GL (1996) The impact of receptor desensitization on fast synaptic transmission. *Trends Neurosci* 19:96–101.
- Koch C (1987) The action of the corticofugal pathway on sensory thalamic nuclei: a hypothesis. *Neuroscience* 23:399–406.
- Koike-Tani M, Saitoh N, Takahashi T (2005) Mechanisms underlying developmental speeding in AMPA-EPSC decay time at the calyx of Held. *J Neurosci* 25:199–207.
- McCormick DA, Bal T (1997) Sleep and arousal: thalamocortical mechanisms. *Ann Rev Neurosci* 20:185–215.
- Mineff EM, Weinberg RJ (2000) Differential synaptic distribution of AMPA receptor subunits in the ventral posterior and reticular thalamic nuclei of the rat. *Neuroscience* 101:969–982.
- Montero VM (2000) Attentional activation of the visual thalamic reticular nucleus depends on ‘top-down’ inputs from the primary visual cortex via corticogeniculate pathways. *Brain Res* 864:95–104.
- Montero VM, Singer W (1984) Ultrastructure and synaptic relations of neural elements containing glutamic acid decarboxylase (GAD) in the perigeniculate nucleus of the cat. A light and electron microscopic immunocytochemical study. *Exp Brain Res* 56:115–125.
- Ohishi H, Neki A, Mizuno N (1998) Distribution of a metabotropic glutamate receptor, mGluR2, in the central nervous system of the rat and mouse: an immunohistochemical study with a monoclonal antibody. *Neurosci Res* 30:65–82.
- Pape HC, McCormick DA (1995) Electrophysiological and pharmacological properties of interneurons in the cat dorsal lateral geniculate nucleus. *Neuroscience* 68:1105–1125.
- Robson JA (1983) The morphology of corticofugal axons to the dorsal lateral geniculate nucleus in the cat. *J Comp Neurol* 216:89–103.
- Sanchez-Vives MV, McCormick DA (1997) Functional properties of perigeniculate inhibition of dorsal lateral geniculate nucleus thalamocortical neurons in vitro. *J Neurosci* 17:8880–8893.

Schlesinger F, Tammema D, Krampfl K, Bufler J (2005) Desensitization and resensitization are independently regulated in human recombinant GluR subunit coassemblies. *Synapse* 55:176–182.

Sillito AM, Jones HE (2002) Corticothalamic interactions in the transfer of visual information. *Philos Trans R Soc Lond B Biol Sci* 357: 1739–1752.

Weese GD, Phillips JM, Brown VJ (1999) Attentional orienting is impaired by unilateral lesions of the thalamic reticular nucleus in the rat. *J Neurosci* 15:10135–10139.

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

(Accepted 8 November 2005)
(Available online 19 December 2005)