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Research report

$GABA_A$ and $GABA_B$ receptors mediated inhibition affect the pattern adaptation of relay cells in the dorsal lateral geniculate nucleus (LGNd) of cats

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Abstract

Pattern adaptation is very important for visual function, while the mechanisms that mediate pattern adaptation, especially in the dorsal lateral geniculate nucleus (LGNd), are still unclear. Iontophoresis of the antagonists and agonists of GABA receptors were employed to separately investigate the contribution of $GABA_A$ and $GABA_B$ receptors to pattern adaptation of LGNd cells. When $GABA_A$ receptors were blocked by bicuculline both the response amplitude of LGNd cells and the degree of adaptation increased significantly. Many neurons showing no pattern adaptation under the normal condition became adapted to a prolonged stimulus. Moreover, the proportion of cells showing adaptation doubled (from 40 to 88%). The mean adaptation index (AI, adapted response amplitude/original response amplitude) was 0.82 during bicuculline application, compared with 0.92 under the control condition. In additional, iontophoresis of baclofen, a selective $GABA_B$ receptor agonist, decreased the mean response amplitude to grating stimuli to 53% of normal. Nearly half of the neurons increased their adaptation index following baclofen administration and the mean AI increased from 0.89 to 1.01. Iontophoresis of $GABA_B$ receptor antagonist (CGP35348) could abolish this effect, though it had no significant effect on visual response amplitude and pattern adaptation itself. Iontophoresis of another $GABA_B$ receptor antagonist, 2-OH-saclofen, also had no significant effect on visual response amplitude and pattern adaptation. These results suggest that both $GABA_A$ receptors and $GABA_B$ receptors modulate the pattern adaptation of LGNd cells and are involved in synaptic plasticity.

Theme: Sensory systems

Topic: Subcortical visual pathways

Keywords: $GABA_A$ receptor; $GABA_B$ receptor; Pattern adaptation; Bicuculline; Baclofen; 2-OH-saclofen; CGP35348; Dorsal lateral geniculate nucleus; Cat

1. Introduction

Pattern adaptation is an important property of cells in visual cortex. The response discharge rate of cortical cells diminishes gradually when the cells are repeatedly stimulated by a visual pattern. Since the original work of Maffei et al. in 1973 [16], adaptation in visual cortex has been widely studied [2,8,9,19,20,26]. However, most studies suggest that pattern adaptation is a property of cortical cells, not of subcortical cells. Shou and his colleagues recently demonstrated that about half of the dorsal lateral geniculate nucleus (LGNd) cells in both normal and visually deprived cats exhibit a significant degree of pattern adaptation [21,27]. But the mechanism underlying the adaptation of relay cells in the LGNd is still unclear.

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Neurons in the LGNd send the main excitatory input to the visual cortex. Study of the adaptation mechanism in LGNd may provide clues to that in cortex. The adaptation of LGNd cells may result from: (i) adaptation of firing due to intrinsic properties of LGNd neurons (neuron fatigue), (ii) adaptation of input of retinal ganglion cells; (iii) feedback from cortical neurons with significant adaptation; (iv) inhibitory connections in the LGNd; and (v) other mechanisms, such as excitatory connection, synaptic plasticity, etc.

Neuron fatigue spiking was assumed to induce the adaptation of cortical cells by Sutherland and others [12,22,23]. However, most recent experiments indicate that pattern adaptation does not show significant correlation with the response amplitude in cortical neuron [13]. Moreover, the discharge rate of relay cells can remain at a stable level even under long-time stimuli, which can hardly be explained by the fatigue mechanism.

Light and dark adaptation is a well-known phenomenon in the retinal ganglion cells, but previous work has suggested that very few ganglion cells exhibit significant pattern adaptation [15]. Shou et al. [21] found that the adaptation of relay cells did not change when the visual cortex was frozen, indicating that the cortical feedback did not participate in the adaptation of LGNd neurons.

The purpose of this work was to investigate the contribution of intrageneculate inhibition connections to the pattern adaptation of LGNd cells. A combination of electrophysiological and pharmacological methods was used to study changes in pattern adaptation when GABA receptors were blocked or activated. We administered $GABA_B$ receptor agonists and antagonists to study whether $GABA_B$ receptors mediating inhibition participate in adaptation. The effects of the $GABA_A$ receptor antagonist bicuculline were also studied in LGNd, and the results were compared with previous results in visual cortex.

2. Materials and methods

2.1. Animal preparation

A total of 15 adult cats, weighing 2.0–3.5 kg, were lightly anesthetized with ketamine HCl (20 mg/kg). Lidocaine (1%) was applied to all intended sites of surgical entry. After intravenous and tracheal cannulae were inserted, cats were placed in a stereotaxic apparatus. Pupils were maximally dilated with atropine (1%), and appropriate contact lenses were used to protect the cornea. Neosynephrine (5%) was administered to retract the nictitating membranes. A mixture of urethane (20 mg/h per kg) and gallamine triethiodide (10 mg/h per kg) was infused intravenously to maintain anesthesia and paralysis. Expired pCO_2 was maintained at ~4%. Heart rate (~180–220 beats/min) was recorded continuously to monitor the level of anesthesia.

2.1.1. Recording and iontophoresis

Three-barrel glass electrodes were lowered into layers A and A1 of the LGNd. Action potentials of single neurons were extracellularly recorded through the central barrel of the electrodes which was filled with 3 M NaCl. The recording sites were at least 150 μm apart. In some cases, biocytin (2.5% in 0.05 M Tris in 1 M KCl, pH 7.2) was placed in the recording barrel to mark the recording site. The second electrode barrel was used for iontophoretic drug application and was filled with either bicuculline (Sigma, 0.5 mM, pH 3), baclofen (Sigma, 10 mM, pH 3.5), 2-OH-saclofen (Sigma, 25 mM, pH 3.5), or CGP35348 (Sigma, 45 mM, pH3.5). Drugs were held in the electrode barrel with a small negative retaining current (10–25 nA). Current balancing was provided through the last barrel containing 175 mM NaCl. In some case, four-barrel glass electrodes were used. Recording and balancing electrodes were the same as above, while the other two barrels were filled with baclofen and CGP35348.

Action potentials were amplified and recognized with a waveform discriminator. Data were collected using a CED 1401 computer interface and VS software (Cambridge Electronic Design, Cambridge, UK). The averaged post-stimulus time histograms (PSTHs) of the neuron's response amplitudes to the visual stimulus were compiled.

LGNd cell response amplitudes were collected after 10 min or longer during drug application, which proved to be sufficient for achieving stable responses. The recovery time after drug administration was 10 min.

2.2. Visual stimulus

Visual stimulus patterns were drifting sinusoidal gratings generated with a computer-driven Picasso image synthesizer (Cambridge, USA) and a Tektronix 608 display (Beaverton, USA). The screen of the display was 12.9×10 cm with mean luminance of 19 cd/m^2, and the environment luminance on the cornea was 0.1 lx. The orientation and spatial frequency of gratings used were optimal for each cell, the temporal frequency was kept at 3 Hz and the contrast value was always kept at 0.6. The position of the display could be adjusted in three dimensions in order to keep the cell's receptive field centered on the screen. The distance between the display and the cat's eyes was always kept at 57 cm.

For each cell studied, the receptive field was first plotted on a tangent screen, and then the optimal orientation, spatial frequency was measured using drifting sinusoidal gratings. Repeatedly drifting prolonged gratings (as long as 50 s) were then used to adapt the relay cells. Trials were repeated three to six times to reduce error.

2.3. Data collection and analysis

The responses were analyzed on-line as well as stored in computer for later analyses. The responses to the drifting

sinusoidal gratings were defined as the amplitude of the fundamental Fourier component of PSTHs. To quantify the degree of adaptation, we defined the adaptation index (AI) as the ratio of plateau amplitude to the initial amplitude. The smaller the AI, the more adapted the neuron.

3. Results

3.1. The role of $GABA_A$ receptors in pattern adaptation

GABA is one of the main transmitters in the brain [18] and the $GABA_A$ receptors are thought to be the main target of GABA. $GABA_A$ receptors mediated early Cl^--dependent inhibitory post-synaptic potentials (IPSPs). Since the discharge rate of cells is reduced with prolonged pattern stimuli during pattern adaptation, the GABAergic inhibitory system may be involved. We compared the adaptation of LGNd cells before, during and after applying bicuculline, the $GABA_A$ receptor antagonist. Of 50 cells studied, 19 cells (38%) showed pattern adaptation, consistent with previous results [21]. Stable recording during long-time iontophoresis (more than 50 min) was difficult. Nevertheless, 25 cells were successfully recorded following bicuculline application, including 21 X cells and four Y cells (15 on-center cells and ten off-center cells). There is no significant difference of adaptation rate between these types.

Our results show that spontaneous and visually evoked response amplitude increased significantly during application of bicuculline. This indicates a disinhibitory effect upon LGNd cells. To ensure that the bicuculline effect was stable during the iontophoresis, we recorded the spontaneous response and the visually evoked response amplitude during application. Fig. 1 shows that after an iontophoretic current of 80 nA is applied for 11 min, the cell's response amplitude remains high even after the current is reduced to 20 nA. Both the spontaneous and evoked response amplitudes increased by ~50% during the application of bicuculline.

If the adaptation of LGNd cells was mediated by $GABA_A$ mediated inhibition, it should disappear or at least diminish when $GABA_A$ receptors are blocked. The result, however, is the opposite. Fig. 2A,B shows the response time courses of one cell that exhibited adaptation before and during application of bicuculline, respectively. Although the response in both the early stages and the late stages increased significantly due to bicuculline, the cell's response amplitude quickly diminished and reached a plateau within 10 s. The adaptation index (AI) of the cell decreased from 0.78 (Fig. 2A) to 0.72 (Fig. 2B) indicating no increase in adaptation.

On the other hand, the cell shown in Fig. 2C, which did not exhibit pattern adaptation in the normal condition became significantly adapted by the bicuculline application (AI 0.75, Fig. 2D). In addition, we found two cells that exhibited facilitation. Interestingly, their firing rates were relatively low, but increased markedly when stimulated by a prolonged grating. The degree of facilitation of the two cells was reduced during bicuculline application, with one of the cells displaying pattern adaptation following drug administration. The AI of this cell decreased from 1.49 to 0.84 (Fig. 2E,F) following bicuculline injection.

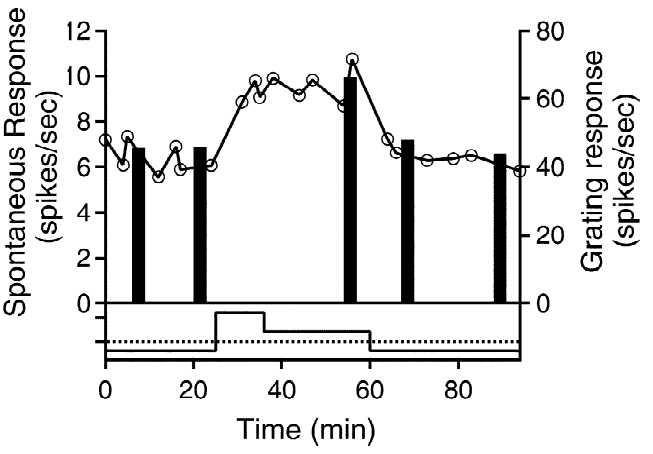

Fig. 1. Effects of iontophoresis of bicuculline on the spontaneous and visually evoked response of an LGNd cell. The left ordinate shows the spontaneous response (circles with lines), while the right shows the visually evoked response amplitude (fundamental Fourier components, columns) to the optimal drifting sinusoidal grating. The abscissa shows time, while the lines above it indicate the iontophoresis time and the current used.

To quantify our results, the adaptation index (AI) distributions of the LGN cells studied before and during bicuculline administration are shown in Fig. 3A. Adaptation was stronger in the bicuculline condition. The proportion of cells that exhibited adaptation increased from 40% in the normal condition to 88% during bicuculline administration. Accordingly, the mean AI decreased from 0.92 to 0.82. These differences were highly significant (*t*-test, $P<0.001$, $P<0.0001$). Fig. 3B shows the overall changes in AI for all the cells during application of bicuculline. The abscissa and ordinate are for the AI of the cells in control and during bicuculline application, respectively. The points on the diagonal line of slope 1 indicate no effect on adaptation. The points under the diagonal indicate that cells, during bicuculline application, were adapted more significantly than those in the normal condition. Almost all the points are under the diagonal line. The extent of the influence on adaptation could be estimated using the ratio between AI in the ordinate and abscissa. The mean of the ratio of AI was 0.87. This is significantly less than 1 (*t*-test, $P<0.0001$), and demonstrates that, overall, bicuculline increased the LGNd cells' adaptation.

3.2. The role of $GABA_B$ receptors in pattern adaptation

Baclofen is a $GABA_B$ receptor agonist, while 2-OH-saclofen and CGP35348 are $GABA_B$ receptor antagonists.

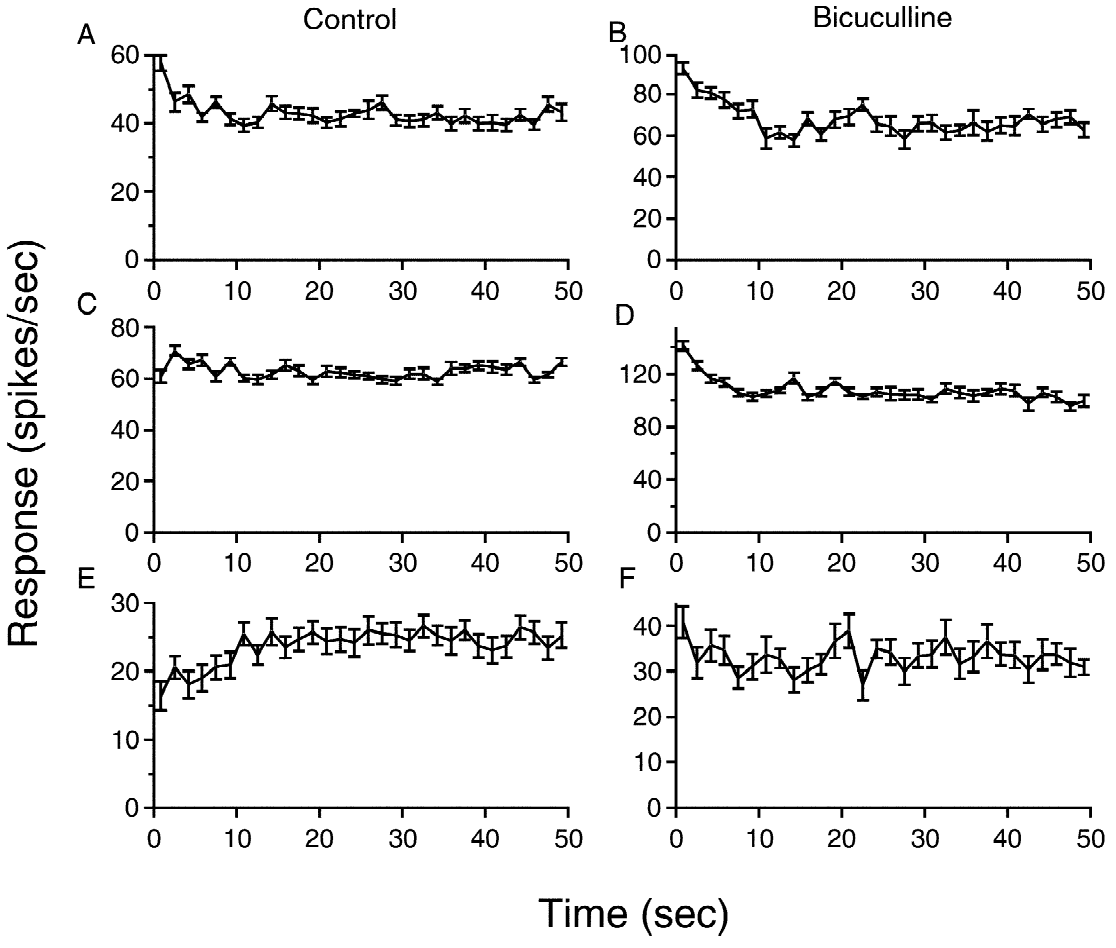

Fig. 2. Changes in adaptation of three typical cells before and during iontophoresis of bicuculline. In control, the cells show adaptation (A), stable response (C) and facilitation (E). However, during iontophoresis, the degree of adaptation increases in all three cells. Notice the different scales of the ordinate in the left and right columns.

As a member of the GABA receptor family, $GABA_B$ receptors are widely distributed in LGN [5]. Their relative ratio to $GABA_A$ receptors ranges from 1:3 to 1:1 due to different measures [10,14]. $GABA_B$ receptors participated in the synchronous and oscillating firing of LGN and contributed to habituation in some areas of brain, such as the superior colliculus [3]. We studied whether the inhibition mediated by $GABA_B$ receptors influenced the pattern adaptation by iontophoresis of baclofen, 2-OH-saclofen and CGP35348 in LGN.

3.2.1. 2-OH-saclofen and CGP35348 have no effect on pattern adaptation

Although 2-OH-saclofen and bicuculline are antagonists of GABA receptors, unlike bicuculline, 2-OH-saclofen did not significantly increase the response amplitude of the relay cells in the LGNd, as shown in Fig. 4A. Spots near the diagonal line of slope 1 indicate that 2-OH-saclofen had no effect on the response amplitude of the cells, while points above or below the line indicate that the firing rate increased or decreased. Obviously, the response amplitude of most cells changed slightly. The mean slope for the lines through all the points was 0.92 ± 0.04, not significantly different from 1 (*t*-test, $P > 0.10$).

Considering that $GABA_B$ receptors may only function with long-term stimulation, we measured the effect of 2-OH-saclofen on pattern adaptation. The results are summarized in Fig. 4B. The abscissa represents the AI in control and the ordinate represents that during application of 2-OH-saclofen. The adaptation characteristics of nearly all cell studies did not shift significantly. Neither previously adapted nor facilitated cells showed remarkable change. The mean slope for the lines through all the points was 1.05, not significantly different from 1 (*t*-test, $P = 0.33$).

Since 2-OH-saclofen has no effect on response amplitude or pattern adaptation, we used CGP35348 to do the same experiments. Like 2-OH-saclofen, CGP35348 also did not affect response amplitude or the pattern adaptation in LGNd cells. The results are summarized in Fig. 4C,D.

3.2.2. Baclofen can decrease pattern adaptation and CGP35348 can partly remove the effect

The results of 2-OH-saclofen administration were not consistent with the histological result that there were a

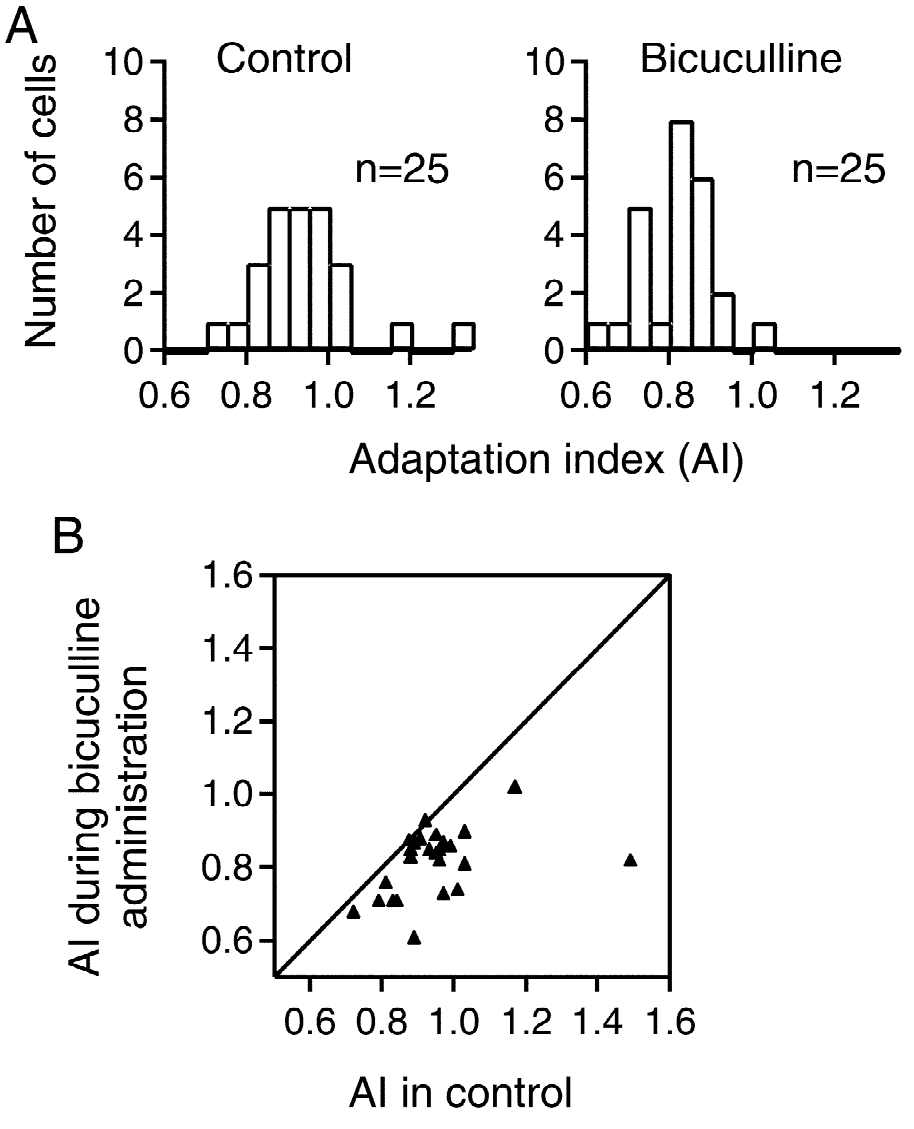

Fig. 3. Bicuculline induced potentiation of the pattern adaptation of LGNd cells. (A) Bar histograms showing the AI distribution of LGNd cells in control and bicuculline administration. The mean AI in control (0.92) is higher than the mean AI in bicuculline application (0.82) (*t*-test, $P<0.001$), indicating that bicuculline potentiates the pattern adaptation of LGNd cells. Both mean AIs are significantly less than 1 (*t*-test, $P<0.001$). (B) Comparison of the AIs of LGNd cells before and after application of bicuculline. The abscissa and ordinate are for the AIs of the cells in control and during bicuculline application, respectively. The points on the diagonal line of slope 1 indicate no effect on adaptation rate. Almost all the points are under the diagonal line, which indicates that, during bicuculline application, cells were adapted more significantly than under the normal condition.

large number of $GABA_B$ receptors in LGN [5]. Therefore, we further administered baclofen, the $GABA_B$ receptor agonist, to 23 relay cells (including 18 X and five Y cells), of which 15 cells exhibited pattern adaptation and one cell showed facilitation before drug application.

When baclofen was applied both spontaneous and visual evoked response amplitude of the LGNd cells reduced. In one cell, a small iontophoresis current (20 nA) ceased cell firing to visual stimulation completely. We selected a specific current for every cell in order to maintain its firing rate at a controlled level. Fig. 5A shows the changes of response amplitude to visual stimuli before and during the iontophoresis of baclofen as in Fig. 4A. All the points in the figure were under the diagonal line of slope 1, indicating that the response amplitude was reduced significantly by baclofen. The mean reduction in amplitude was 47%, significantly different from 1 (*t*-test, $P<0.0001$).

The effect of baclofen on the pattern adaptation index (AI) is shown in Fig. 5B. Baclofen caused more facilitation for cells that had not exhibited adaptation in the normal condition and attenuated the extent of AI for cells that had exhibited adaptation. The average AI increased from 0.89 to 1.01. The extent of the influence of baclofen on AI could be estimated by using the ratio between AIs in the ordinate and the abscissa. The mean of the ratio was 1.16, remarkably larger than 1 (*t*-test, $P<0.001$), suggesting that baclofen may modulate the pattern adaptation in LGNd cells.

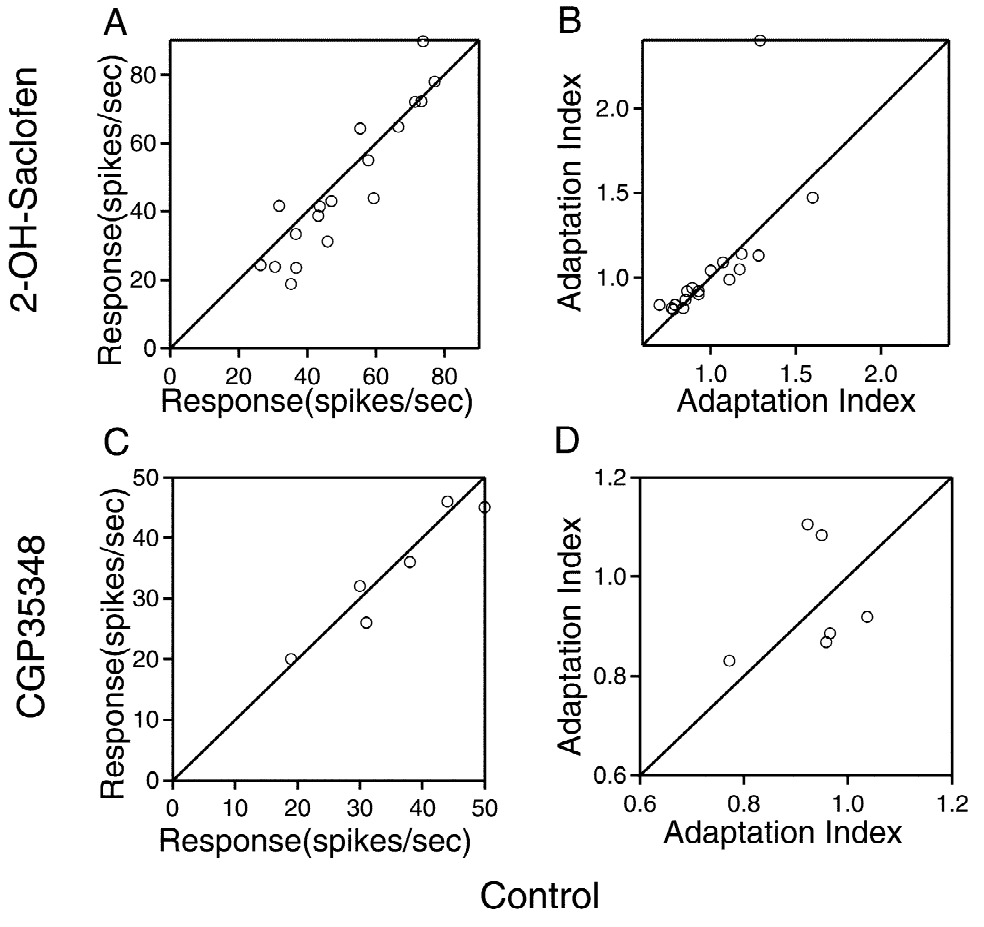

Fig. 4. The effects of 2-OH-saclofen and CGP35348 on visual response amplitude and adaptation rate of LGNd cells. Scatter plot showing the visual response amplitude (A) and adaptation index (B) of each cell during 2-OH-saclofen ($n=18$) injection. The abscissa and ordinate are for the AIs of the cells in control and during 2-OH-saclofen application, respectively. In A and B, almost all scatter plots are placed near the oblique line of slope 1, which means that iontophoresis of 2-OH-saclofen affects neither visual response amplitude nor adaptation rate of LGNd cells. In C and D, the plots are similar to those in A and B, indicating that only application of 2-OH-saclofen or CGP35348 has no effect on visual response amplitude or adaptation of LGNd cells.

If we administered CGP35348 after baclofen application, the effect of baclofen on $GABA_B$ receptors was remarkably eliminated. The results are shown in Fig. 5C,D. The response amplitude and pattern adaptation in LGNd cells all recovered to control levels. The mean response amplitude recovered to 95% of control after application of CGP35348, and the average AI decreased from 1.01 to 0.87.

4. Discussion

In this study we provide insight into the mechanisms that mediate the pattern adaptation of LGNd relay cells in cats using electrophysiological recording and iontophoretic methods in vivo. The results indicated that blockade of $GABA_A$ receptors potentiates the pattern adaptation of LGNd neurons. Increased adaptation is accompanied by an elevation of discharge. Baclofen, a selective agonist of $GABA_B$ receptor, inhibits LGNd cells' response amplitude significantly and depresses the pattern adaptation of LGNd cells. Therefore, both $GABA_A$ and $GABA_B$ receptors appear to be involved in pattern adaptation in the LGNd.

To determine whether cell response amplitude can influence pattern adaptation, we analyzed the data as shown in Fig. 6. In this figure, pattern adaptation has a slight relationship to cell response amplitude. So the changes in pattern adaptation of LGNd cells due to drug application may be mainly affected by the modifying function of GABA receptors.

4.1. Consideration of the effects of baclofen, 2-OH-saclofen and CGP35348

4.1.1. Baclofen

Baclofen is a member of the GABA family and has been widely used to activate the inhibitory current or potential mediated by $GABA_B$ receptors [4,11,24]. Although the current activated by baclofen may also influence the $GABA_B$ current indirectly evoked by visual stimulus, this could reflect the change in physiological properties of relay cells when $GABA_B$ receptors are activated in LGNd.

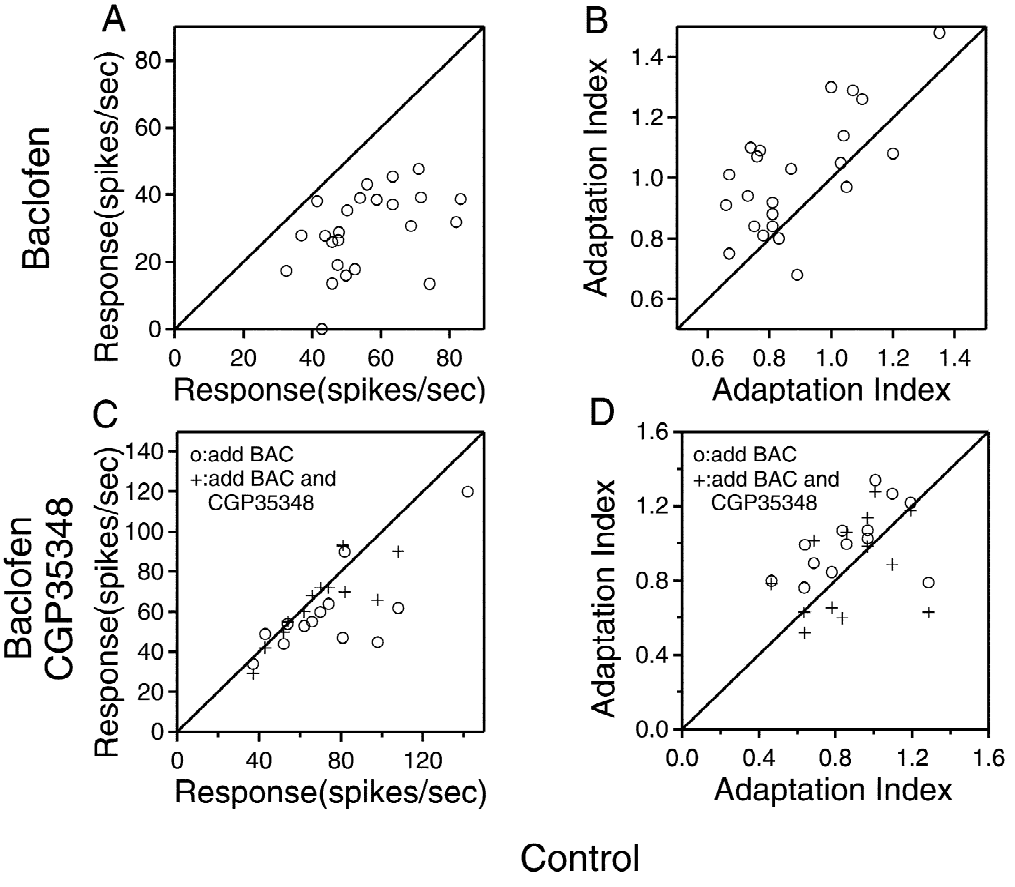

Fig. 5. The effects of baclofen on visual response amplitude and adaptation rate of LGNd cells and CGP35348's effects to it. Scatter circle plots showing the visual response amplitude (A, C) and adaptation index (B, D) of each cell during baclofen (top, $n=24$; bottom, $n=13$) injection. In A, scatter plots are placed below the oblique line, indicating that the iontophoresis of baclofen decreases the visual response amplitude of all cells recorded. In B, AIs increase after iontophoresis of baclofen, indicating that the degree of adaptation of LGNd cells is decreased by the $GABA_B$ receptor agonist baclofen. In C, circle plots showing application of baclofen, almost all points are placed below the oblique line. Almost all '+' plots showing application of baclofen and CGP35348, are placed near the oblique line of slope 1, indicating that the effect of baclofen to cell's response amplitude can be removed by CGP35348. In D, AIs increase after iontophoresis of baclofen, and AIs decrease to control after application of CGP35348, indicating that the effects of baclofen on adaptation of LGNd cells can be recovered by $GABA_B$ receptor antagonist CGP35348.

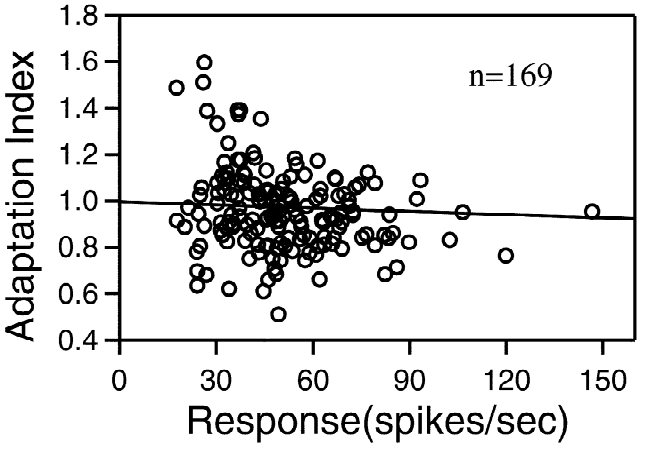

Fig. 6. The effects of response amplitude on adaptation of LGNd cells. The abscissa shows the visual responses amplitude under control condition (no drug used). The ordinate shows the adaptation index (AI). Using line fitting, the oblique line slope is 0.00047 ± 0.00003, and is slightly different from slope 0 line. This result indicates that the response amplitude has a slight effect on pattern adaptation.

Baclofen causes some LGNd cells to fire less and decreases adaptation. A similar result in the visual cortex has been reported [25]. Thus, inhibitory pathways mediated by $GABA_B$ receptors in both the LGNd and visual cortex appear to mediate adaptation.

4.1.2. 2-OH-saclofen and CGP35348

Immunohistological study has demonstrated that a large number of $GABA_B$ receptors exist in LGNd [5]. However, our results show that iontophoresis of 2-OH-saclofen and CGP35348 had almost no effect on the response properties of LGNd cells in vivo. How to explain these results?

First, although 2-OH-saclofen is widely used as a $GABA_B$ receptor antagonist for study in vivo, such as in the striate cortex and superior colliculus, it is reported that 2-OH-saclofen antagonizes not only the postsynaptic inhibitory current but also the presynaptic calcium current evoked by baclofen [4,5,7]. Furthermore, Emri et al. [11] demonstrated that in the LGNd 2-OH-saclofen partially antagonized the postsynaptic inhibitory current activated

by baclofen, but also had the same effect as baclofen, which hyperpolarized the membrane potential of cells and reduced the input resistance. In this study, the partial agonist effect of 2-OH-saclofen may hide its antagonistic effect. Second, it was found that endogenous GABA activated few $GABA_B$ receptors under normal physiological conditions in vivo [6]. Then although only little inhibitory current through $GABA_B$ is blocked by the administration of 2-OH-saclofen, the effect can be insignificant. This is quite consistent with our observation that baclofen, the agonist of $GABA_B$ receptors, induced significant inhibition of relay cells in the LGNd.

The function of CGP35348, an antagonist of $GABA_B$ receptor, is similar to that of 2-OH-saclofen, but $GABA_B$ receptor is antagonized more by CGP35348 than by 2-OH-saclofen.

4.2. Discussion of results in LGNd cells

The study indicates that the inhibition induced by $GABA_A$ receptors did not contribute to pattern adaptation in the cat LGNd because bicuculline increases the adaptation of LGNd cells and the proportion of cells adapted. It suggests that most LGN cells in the cat may mediate pattern adaptation rather than through $GABA_A$ receptors. However, the mechanism of $GABA_A$ receptor pathways modulates adaptation by reducing the effect.

On the other hand, baclofen causes some adapted cells to lose their pattern adaptation accompanied by decreased firing. A similar result in visual cortex was reported. Many neurons lost their adaptation due to baclofen application when the firing rate decreased significantly. But when CGP35348 was administered following baclofen application, the effect of baclofen can be removed. The depressed response can recover, and a weakened pattern adaptation also appears. This suggests that the inhibitory pathway via $GABA_B$ receptors in LGNd also modulates adaptation. Since CGP35348 can affect baclofen's results, the iontophoresis of CGP35348 is effective. Therefore CGP35348 had no effect on visual response amplitude and adaptation rate was not by the reason that iontophoresis failed.

The work of McLean and Palmer [17] implied that pattern adaptation might be induced through a presynaptic mechanism in the visual cortex. Applying mGlutamate receptor antagonist decreased the adaptation of cortical neurons, while iGlutamate receptor antagonist did not. This is consistent with the activation of $GABA_B$ receptors. Unlike the $GABA_A$ receptors, $GABA_B$ receptors existed in both presynaptic and postsynaptic membranes. However, 2-OH-saclofen could not block the postsynaptic $GABA_B$ receptors completely, compared with the presynaptic $GABA_B$ receptors [11]. In additional, the endogenous GABA only activated the presynaptic $GABA_B$ receptors in normal conditions [10,11]. Presynaptic $GABA_B$ receptors could control the release of transmitter via Ca^{2+} channel, i.e. changing the synaptic efficiency [20]. Thus baclofen may affect adaptation in the LGNd through synaptic plasticity.

Short-term synaptic plasticity can be divided into short-term facilitation (STF) and short-term depression (STD). STD could control the balance of excitation and inhibition and modulate the excitatory level of neurons in the visual cortex [1]. The STD was more prominent in excitatory synapses than in inhibitory synapses and was more easily induced by higher frequency stimuli [1,25]. As the process and duration of STD were similar to that of adaptation, Abbott et al. [1] suggested that STD maybe underlie the mechanism of pattern adaptation. The report that baclofen could depress the STD of synapses [25] agrees with our observation that baclofen depressed pattern adaptation in LGNd cells.

Acknowledgements

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