Properties of the Pathways From the Lateral Amygdal Nucleus to Basolateral Nucleus and Amygdalostriatral Transition Area

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Wang, Chunsheng, Maeng-Hee Kang-Park, Wilkie A. Wilson, and Scott D. Moore. Properties of the pathways from the lateral amygdal nucleus to basolateral nucleus and amygdalostriatral transition area. J Neurophysiol 87: 2593–2601, 2002; 10.1152/jn.00759.2001. Studies have revealed that the amygdala formation is involved in emotional learning, attention, and autonomic functions. Although intra-amygdala connections have been described anatomically, the functional characteristics of these connections are not well understood. We used a rat brain slice preparation with a voltage-sensitive imaging system to compare the electrophysiological characteristics of intra-amygdala pathways. Electrical stimuli delivered to the lateral nucleus (La) caused the optical signal to propagate to basolateral nucleus (BL) and amygdalostriatral transition area (AStr), but not the central nucleus (Ce), consistent with previous anatomical studies, including the recently characterized projections from La to AStr. The velocity of propagation of the evoked potential along the La-AStr pathway was significantly faster than that along the La-BL pathway. In addition, the efficiency of the signal transmission (determined by the rate of decay) along the La-AStr pathway was higher than that along the La-BL pathway. Also, AStr possessed a distinct property of temporal summation of La signals. On the other hand, the La-BL pathway possessed a significantly higher sensitivity to bicuculline/picrotoxin and a stronger paired-pulse inhibition than the La-AStr pathway. Furthermore, the La-BL pathway expressed a higher D-2-amino-5-phosphonovaleric acid (a NMDA blocker) sensitivity than the La-AStr pathway. These results suggest that the La-AStr pathway, which conducts signals with high velocity and less attenuation, may be involved in rapid reflexive responses during fear-induced behavior, whereas the La-BL pathway facilitates signal integration and learning.

INTRODUCTION

Increasing evidence reveals that the amygdala formation is a convergence for processing emotionally significant information (Davis 1992; LeDoux 1992, 2000; McGaugh et al. 1992), causing autonomic and affective responses involved in pain, aggression, fear, and anxiety. Extensive anatomical studies have demonstrated a complex internuclear connectivity within the amygdala (Cassell et al. 1999; Doron and LeDoux 1999; McDonald 1998; Pitkänen 2000; Turner and Herkenham 1991). Several projections from the lateral nucleus (La) to a variety of intra-amygdala nuclei have been identified, including projections to the basolateral nucleus (BL) (Pitkänen et al. 1995) and a recently characterized projection from La to the amygdalostriatral transition area (AStr) (Jolkkonen et al. 2001).

Because of their complex anatomical organizations, the physiological properties of the intra-amygdala connections are not well established. Unlike the hippocampus, in which the neuronal connections are aligned in lamina, the amygdala does not possess such a cellular organization, making electrophysiological studies more difficult to interpret. To reveal the function of the network and signal propagation in the amygdala, we employed a photodiode array imaging system coupled with a voltage-sensitive dye (JPW1131) (Loew 1999), which allowed us to observe neuronal electrical activities in different nuclei simultaneously during signal propagation. This technique has previously been used to study the spatiotemporal aspects of evoked and spontaneous activity in neocortex (Wu et al. 1999b), olfactory bulb (Keller 1998), thalamocortical pathways (Lairis et al. 2000), piriform cortex (Demir et al. 1999), and the amygdala formation (Wang et al. 2001). Our imaging system covered a hexagonal area (1 mm facets) with each diode covering an area of 80 × 80 μm² (Fig. 1). This range of coverage, combined with the high temporal resolution (0.6 ms) of the photodiodes, allows recording of signal propagation along network pathways connecting intra-amygdala nuclei and identification of circuitry which has previously been inaccessible with standard physiological techniques.

Using the voltage-imaging system, we found that electrical stimuli delivered to La caused the signal to propagate to the BL and AStr but not to the central nucleus (Ce). We then characterized the physiological properties of the signal propagation along the La-AStr and La-BL pathways and found that the signal transmission toward AStr possessed a significantly faster velocity and a higher transmission efficiency than the signal transmission toward BL. In addition, temporal summation of high-frequency stimulation was more efficient in AStr than BL. On the other hand, the La-BL pathway expressed significantly higher sensitivities to bicuculline (Bic)/picrotoxin (PTX) and D-2-amino-5-phosphonovaleric acid (a NMDA blocker) and greater paired-pulse inhibition than the La-AStr pathway, indicating more effects of γ-aminobutyric acid-A (GABA A ), N-methyl-D-aspartate (NMDA), and γ-aminobutyric acid-B (GABA B) in the La-BL pathway. The differences in physiological and pharmacological characteristics between the two pathways suggest the La-AStr pathway may be involved in rapid, reflexive responses during fear-induced behavior, whereas La-BL pathway...
may facilitate signal integration, which in turn may be involved in instrumental behavior and emotional learning (Killcross et al. 1997).

**METHODS**

We used male rat brain slices (Sprague-Dawley, 4- to 6-wk-old). Rats were anesthetized with halothane and quickly decapitated. The brains were then removed and coronal slices (500 \( \mu \text{m} \)) were made with a vibratome. Slices containing the mid-caudal amygdala formation were incubated in artificial cerebrospinal fluid (ACSF) composed of the following (in mM): 124 NaCl, 10 dextrose, 26 NaHCO\(_3\), 2 KCl, 1.25 KH\(_2\)PO\(_4\), 2 CaCl\(_2\), and 1 MgSO\(_4\), equilibrated with 95\% O\(_2\)-5\% CO\(_2\). After 1 h of incubation, slices were stained in ACSF with the fast voltage-sensitive dye JPW1131 (also known as RH479) (from Dr. L. Loew, University of Connecticut, Farmington, CT) at 0.02 mg/ml for 45–60 min. The stained slice was then placed in an immersion-type recording chamber and perfused with ACSF at 2–3 ml/min for ≥60 min before recordings. The slices were maintained at a constant temperature of 29°C throughout the recordings.

The optical signal was obtained using an upright microscope (Axioskop 2FS; Carl Zeiss, www.zeiss.de) with a water-immersion lens (10×/0.30w Ph1, Zeiss) and two camera ports. One camera port was equipped with a fixed zoom lens and a fast charge-coupled device (CCD) camera (SensiCam; Cooke Corp., www.cookecorp.com) for taking pictures of slices and calibrating the objective plane. The other camera port had an adjustable zoom lens and a 464-element photodiode array (WuTech Instruments; www.wutech.com) that covered a 1-mm-sided hexagon area. Under these conditions, one pixel of the CCD image covers an area of 3.125 \( \times \) 3.125 \( \mu \text{m}^2 \) and one diode of the photodiode array covers an area of 80 \( \times \) 80 \( \mu \text{m}^2 \). The fastest sampling rate of the photodiode array is 0.613 ms per frame. Both the CCD camera and the photodiode array were calibrated independently. The double calibration allowed us to correlate a specific spot of the slice to a particular diode. The second stage amplifiers utilized 10 kHz low-pass and 0.2 Hz high-pass resistor-capacitor (RC) filters (see Wu et al. 1999a for details). The response of the voltage-sensitive dye has a sub-millisecond kinetics and a linear dependence on voltage change within ±100 mV (Loew 1999). JPW1131 has a specific light absorption at 705 ± 50 nm. Data acquisition and analysis were performed with the program NeuroPlex (RedShirt Imaging, www.redshiraimaging.com) on a Pentium PC. A vibration isolation system (250WS-1; Minus-k Technology, www.minusk.com) was used to minimize the vibration.
noise. Detailed methods have been described previously (Wang et al. 2001).

The evoked signal was triggered by delivering single stimulus (70–100 μA/200 μs) to La every 4–5 min with a unipolar tungsten electrode. A 2-s recording was conducted for each trial with a scan rate of 0.613 ms per frame. With this method of optical recording, we could obtain stable responses for more than 2 h. The paired-pulse stimuli were delivered to La with an interval of 300 ms. This interval was used to allow a clear separation of the second response from the slow phase of the first response (Wang et al. 2001).

The velocity of the signal propagation was obtained by linear fitting the latency-to-peak signal against the distance traveled. We examined two vectors along the La-BL and La-AStr pathways, each containing 10 photodiodes (800 μm total distance). Each pathway was then divided into two halves (400 μm). The first half represents the signal propagation within La, while the second half represents the signal propagation into BL or AStr. The two halves were fitted separately. The attenuation of the signal along the pathways followed an exponential decay. We applied the cable equation to model the signal attenuation (Johnston and Wu 1995). We used the rate of attenuation, or length constant, to evaluate the efficiency of synaptic transmission. To determine the length constant of the signal decay, we constructed a semi-logarithm plot of peak amplitude against the distance (which showed a linear relationship, Fig. 3A) and did a linear fit for the two halves along each pathway. The inverse of the slope times (−1) was the length constant. To estimate temporal summation along the two pathways, a short train (4 stimuli of 100 Hz) was delivered to La. The ratio of peak responses to each stimulus of the train to the first peak response was used to evaluate the level of temporal summation. Independent (unpaired) t-tests were used to compare parameters of the two pathways.

To compare the effects of pharmacological agents between BL and AStr during La stimulation, we averaged responses from a small area containing seven diodes within each nucleus at the same distance (500 μm) away from the stimulating electrode. To better evaluate the synaptic response along the two pathways, we integrated the initial 50 ms of the evoked signal from each pathway and used this value to quantify the response level. This value minimized the contamination from nonsynaptic slow kinetic responses (Wang et al. 2001). Based on this quantification, the percentage change due to Bic was calculated by dividing the difference between the responses in control and the responses in the presence of Bic by its control response. The same was done for PTX. The percentage change due to d-APV was determined by dividing the difference between the responses in Bic alone and Bic/d-APV together by its response in the presence of Bic alone. For comparison of paired-pulse inhibition between the two nuclei, we also did integration for the second 50 ms of the signal trace because it had shown a dominant inhibition on the slow phase of the response. The percentage paired-pulse inhibition (the difference between the first and second responses divided by the first response) was compared for each 50-ms period of response. Responses in the second period reflect to a greater extent the voltage-dependent conductances of the membrane rather than synaptic transmission as characterized by a previous study (Wang et al. 2001). Independent (unpaired) t-tests were used to compare responses from the two nuclei and paired t-tests were used to compare responses before and after drugs within each nucleus. The averaged results were expressed as mean ± SE. An asterisk indicates a two-tailed significance at P < 0.05 and two asterisks indicate a two-tailed significance at P < 0.01 level.

R E S U L T S

Physiological properties of signal propagation along the La-BL and La-AStr projections

Different nuclei of the amygdala formation were identified in a CCD image of a rat slice (Fig. 1A). The 464-element photodiode array recorded electrical activity from different areas of the amygdala formation simultaneously (Fig. 1B). As shown by the pseudocolored image, a single stimulus delivered to La triggered a depolarizing signal to propagate to BL and AStr but not to Ce (Fig. 1C). This pattern of propagation was not changed by increasing the intensity of stimulation (n = 4).

To demonstrate the signal propagation to different nuclei, we averaged the responses from a small area (a 7-diode hexagon) in each of La, BL, AStr, and Ce as defined in Fig. 1B. The traces of the depolarizing signal obtained from each amygdaloid nucleus were illustrated in Fig. 1D. Little response was observed in Ce. These data are consistent with anatomical studies, indicating that La projects to BL and AStr, but not to Ce beyond the lateral capsular division (Jolkkonen et al. 2001; Pitkänen et al. 1995; Savander et al. 1997).

To compare the physiological properties of the signal propagation along the La-BL and La-AStr projections, we examined the responses along the vectors of the two pathways, each containing 10 diodes (800 μm total distance) (Fig. 2A). First, we measured the latency-to-peak response and plotted this against the distance of propagation which reflected the velocity of signal propagation. Since the peak response represents the maximum synaptic activation, the velocity thus determined reflects the speed of synaptic transmission. As shown in Fig. 2, B and C, there is an acceleration of propagation velocity in AStr while the velocity in BL is slowed down. Averaged velocity along the first half of the La-BL pathway (which is contained within La as shown in Fig. 2A) was 81.2 ± 3.6 mm/s while the second half (within BL) was 56.6 ± 6.2 mm/s, demonstrating a significant decrease in the velocity in BL (Fig. 2D). In contrast, the averaged velocity along the two halves of the La-AStr pathway was 88.8 ± 4.4 and 141.1 ± 16.5 mm/s, respectively, a significant increase in the velocity in AStr. There was no significant difference between the velocities of the first halves along the two pathways.

Second, the transmission efficacy was studied using the rate of attenuation of the propagated response along the two pathways (the length constant, see METHODS) as an indicator. As shown in Fig. 3A, the semilog plot of peak amplitude versus the distance showed linear decays of peak responses along the two pathways, indicating an exponential attenuation. However, a significantly reduced decay rate inside AStr was observed. The length constant of the decay (which reflects the rate of decay) along the two pathways was determined by linear-fitting of the semilog plot. Each vector was divided into two halves for separate linear-fitting with the first halves of both vectors reflecting the properties within La. The averaged length constant along the first and second halves of the La-BL pathway was 511.9 ± 74.4 and 680.4 ± 150.2 μm, respectively, with no significant difference detected (Fig. 3B). The averaged length constant along the two halves of the La-AStr pathway was 565.1 ± 84.9 and 1717.8 ± 320.3 μm, respectively, with a significantly longer length constant in AStr. That is, the decay rate of signal propagation in AStr was significantly reduced. Meanwhile, no statistical difference in signal attenuation rate was found between the first halves of the two pathways. Since the peak signal reflects the maximum synaptic activation of the evoked response, the lower rate of attenuation of the peak response implies a higher efficiency of synaptic transmission in AStr.

Third, we also studied the characteristics of temporal summation along the two pathways by delivering a short burst of stimulation (4 pulses at 100 Hz) to La. Our results showed that
**FIG. 2.** Propagation velocity of the evoked signal along the La-BL and La-AStr pathways. 

A: a photograph of the brain slice illustrating the 2 vectors along the La-BL and La-AStr pathways along which we examined the propagation velocity and attenuation of the evoked signal from La. Each vector represents 800 μm or 10 photodiodes. 

B: traces of the evoked responses obtained from the 10 diodes along each vector. The traces represent an average of 7 trials from 1 slice. 

C: latency to peak is plotted against distance from La along each pathway. An increased velocity can be observed along the AStr pathway. The bars represent SE. 

D: averaged velocities of the first and second halves of the 2 vectors are demonstrated.

**FIG. 3.** Attenuation of propagation of the evoked signal along La-BL and La-AStr pathways. 

A: a semilog plot of amplitude of peak responses versus the distance along each vector. The linearity of the attenuation of the peak responses in this plot indicates an exponential decay. The rate of signal decay (length constant) along the AStr pathway is significantly reduced. The bars represent SE. 

B: averaged length constants of the propagation are demonstrated separately for the first and second halves of the 2 vectors.
the level of temporal summation for the second and third stimuli of the burst was significantly higher in AStr than in BL (Fig. 4, A and B). The averaged ratio response of the second peak to the first peak was 1.60 ± 0.03 in AStr and 1.21 ± 0.03 in BL (n = 5). The ratio response of the third peak to the first was 1.85 ± 0.03 in AStr and 1.67 ± 0.10 in BL. These data reflect a significantly higher level of temporal summation for the second and third peaks in AStr. The ratio response of the fourth peak to the first was 2.13 ± 0.09 in AStr and 2.09 ± 0.12 in BL; no significant difference was detected. Given that a majority of La projection neurons are capable of generating spike doublets with a intra-doublet frequency of 25–80 Hz (Driesang and Pape 2000), the difference in the level of temporal summation may facilitate a preferential distribution of transmission of the spike doublets toward AStr. On the other hand, there is no preferential transmission of a longer burst of spikes (the 4th spike) along the two pathways. These data suggest that differential temporal summation properties in the amygdala be an important mechanism for signal integration.

Effects of Bic/PTX, D-APV, and paired-pulse inhibition on the synaptic transmission along the two pathways

In control conditions, the response level (the integration of the initial 50 ms of the optical response) in BL and AStr following La stimulation were quite similar, with an averaged value of 18.5 ± 2.8 and 19.5 ± 2.7% ΔI/I (n = 12), respectively; no significant difference was detected. In the presence of Bic (20 μM), however, the response in BL was increased to a significantly greater extent than in AStr (Fig. 5, A and B). The percentage increase caused by Bic was 188 ± 21% in BL and 100 ± 16% in AStr, a significantly higher Bic sensitivity in BL. Since Bic was reported to block Ca2+-dependent K+ channels (Khwaled et al. 1999), we also applied PTX (100 μM), and a similar differential effect was observed (Figs. 5, C and D). The percentage increase with PTX in BL and AStr was 126 ± 7 and 92 ± 8%, respectively, showing a significantly higher sensitivity in BL.

We then examined the D-APV sensitive signal between the two nuclei. Under normal conditions, stimulus delivered to La activated a small fraction of D-APV sensitive signal, which was found mainly within La. To maximally activate the NMDA response in BL and AStr, we used 20 μM Bic as the control condition. The results showed that D-APV (50 μM) inhibited the response to a greater extent in BL (Fig. 6, A and B). The percentage inhibition of D-APV was 31 ± 4% in BL and 20 ± 2% in AStr; significantly more D-APV sensitive signal was detected in BL than in AStr.

Paired-pulse inhibition was studied in the two pathways by delivering two stimuli to La with a 300-ms inter-pulse interval. As shown in Fig. 7A, the dominant inhibition appeared in the late phase of the signals. Therefore we compared the paired-pulse inhibition for both the early phase (1st 50 ms) and the late phase (2nd 50 ms) of the signal in BL and AStr. First, paired-pulse stimulation produced significant inhibition of the signals within each nucleus regardless of the phases of the response (P < 0.05 for early phase and P < 0.01 for late phase of the signal, n = 8, paired t-test). Second, the inhibition of the late phase (2nd 50 ms) of the signal was significantly stronger than the inhibition of the early phase (1st 50 ms) of the signal in both nuclei (P < 0.001, n = 8, paired t-test). Third, there was no significant difference in paired-pulse inhibition of the early phase of the signal between BL and AStr, with a percentage inhibition of 20 ± 4 and 18 ± 4%, respectively (Fig. 7B). Last, a significantly stronger inhibition of the late phase of the signal was found (P < 0.05, n = 8) in BL with a percentage inhibition of 54 ± 2% in BL and 43 ± 4% in AStr (Fig. 7C). Different phases of the signal have been characterized in a previous study, which showed that the early phase was most sensitive to 6,7-dinitroquinoxaline-2,3-dione (DNQX), whereas the slow phase was sensitive to D-APV and calcium channel blockers (Wang et al. 2001). Therefore we speculate that paired-pulse inhibition exerts a greater inhibitory effect on the slow kinetic voltage-gated calcium and NMDA conductances (to a lesser extent) than on the fast α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor-mediated postsynaptic response.

Finally, application of 20 μM DNQX blocked the signal propagation to BL and AStr (Fig. 7D), for little response was observed in the last five traces (or beyond La) of either pathway (n = 5). This result indicated that the signal propagation toward BL and AStr was dependent on AMPA receptor-mediated synaptic transmission. The residual signal within La under DNQX (the first 5 traces of each pathway) was generated from the voltage-dependent sodium and calcium conductances, as calcium channel inhibitors block the slow component of the signal while TTX blocks both the fast and the slow components (Wang et al. 2001).
DISCUSSION

Extensive anatomical studies have demonstrated that La is a major recipient of cortical and subcortical sensory information (Doron and LeDoux 1999; McDonald 1998). La sends projections to BL, the accessory basal nucleus, the capsular division of the central nucleus, and AStr (Jolkkonen et al. 2001; Pitkänen et al. 1995; Savander et al. 1997). Because La serves as one of the main entry points for sensory information to the amygdala formation, it is important to understand the physiological properties of the intra-amygdaloid projections originating from La.

In this study, we characterized the signal propagation along

![Figure 5](image)

**FIG. 5.** Comparison of the effects of bicuculline (Bic; 20 μM)/picrotoxin (PTX; 100 μM) on BL and AStr responses to La stimulation. A: superimposed responses of the control and after Bic from both BL and AStr show that Bic remarkably increases the evoked response in both nuclei. B: averaged percentage increases in evoked responses in BL and AStr show a significantly higher sensitivity to Bic in BL. C: superimposed responses from both BL and AStr show increased responses after PTX. D: averaged results in BL and AStr reveal a significantly higher sensitivity to PTX in BL.

![Figure 6](image)

**FIG. 6.** Comparison of D-2-amino-5-phosphonovaleric acid (D-APV; 50 μM) sensitivity in BL and AStr in response to La stimulation. A: superimposed responses of the control (in the presence of Bic) and after D-APV in BL and AStr show decreased responses in both nuclei. B: averaged results of D-APV inhibition display a higher D-APV sensitivity in BL than in AStr.
the La-AStr and La-BL pathways following La stimulation. The finding of evoked signal propagation to both BL and AStr is consistent with previous anatomical studies which demonstrate that La sends major projections toward BL and AStr (Jolkkonen et al. 2001; Pitkänen et al. 1995). The finding of a faster velocity of the signal transmission in AStr than in La and BL may have significance for emotional information processing. Second, a lower signal attenuation rate (higher propagation efficiency) in AStr may serve to keep the signal accurate and subject to less error or failure rate. Third, less GABA A-sensitive component in AStr may indicate less GABAergic tone in this nucleus. Fourth, most La projection neurons are capable of generating spike doublets on excitation (Driesang and Pape 2000) and our temporal summation data shows that the La-AStr pathway should preferentially facilitate the transmission of very short bursts of spikes such as these doublets. Given the highly topographical organization of La-AStr projection (Jolkkonen et al. 2001) and the above characteristics, the La-AStr pathway may be responsible for fast and accurate transmission of information for initial reflexive emotional and behavioral responses.

Signal propagation in BL displayed a significantly lower velocity and higher attenuation rate (lower efficiency). These differences may not be explained in terms of differences in sensory modality involved with the two pathways because in vivo studies suggest that the two nuclei respond to the same auditory, visual, and somatosensory stimuli (Bordi and LeDoux 1992; Bordi et al. 1993; Uwano et al. 1995). Therefore the difference in signal transmission in the two pathways may reflect different ways of information processing. For example, the slow propagation and high attenuation rate in the La-BL pathway may facilitate modulation of the autonomic functions by hormonal influences. Studies have indicated a number of hormones including adrenaline and corticosterone modulate information processing in BL (for review, see McGaugh et al. 2001). In addition, higher sensitivity to GABA A blockers in BL, together with higher sensitivity to d-APV, and greater paired-pulse inhibition, an indicator of GABA B effect (Lambert and Wilson 1994), indicate that the La-BL pathway may be involved in complex signal integration and emotional learning during fear conditioning (Gewirtz and Davis 1997; Klcross et al. 1997; Stutzmann and LeDoux 1999).

The paired-pulse inhibition effect was complex. Although the paired-pulse inhibition was significant in both the La-BL
and the La-AStr pathways, the inhibitory effect was dominant in the late phase of the signal. Our previous study demonstrated that the optical signal of the evoked response in the amygdala had an early phase and a late phase with different pharmacological characteristics (Wang et al. 2001). The fact that early phase was more sensitive to DNQX block indicates a significant AMPA contribution to this phase of the signal. The late phase contained predominantly voltage-dependent calcium signals and to a lesser extent the NMDA receptor-mediated signal because of its sensitivity to nickel, agatoxin, conotoxin, and 2-APV (Wang et al. 2001). Therefore the early phase of the optical signal reflects synaptic transmission (AMPA receptor-mediated activation) and the late phase reflects to a greater extent the membrane conductances. Previous studies indicate that these membrane conductances in BL, including P/Q- and L-type calcium channels, modulate synaptic plasticity (Huang et al. 1996; Weisskopf and LeDoux 1999). Our finding of stronger paired-pulse inhibition in the late phase of the signal in BL may imply that GABA affects synaptic plasticity through modulation of the voltage-dependent membrane conductances, possibly through activation of GABA<sub>B</sub>-mediated potassium conductance (Lambert and Wilson 1994).

Extensive electrophysiology studies have been conducted in La and BL, and both excitatory and inhibitory synaptic transmission have been characterized (Chapman et al. 1990; Davis et al. 1994; Gean and Chang 1992; Li et al. 1998; Mahanty and Sah 1998; Rainnie et al. 1991a,b; Weisskopf and LeDoux 1999). A variety of glutamate and GABA receptors have been identified which may be involved in synaptic plasticity and emotional learning (Farb et al. 1995; Fritschy and Mohler 1995; Mahanty and Sah 1998). However, the differences in electrophysiological properties between the La-BL and La-AStr pathways have not been previously reported. Comparing the La-BL with the La-AStr pathway, we found more Bic and 2-APV sensitive responses as well as a stronger paired-pulse inhibition in BL, suggesting stronger influences of GABA and NMDA in BL. These findings suggest that BL may be more likely involved in synaptic plasticity and signal modulation than AStr.

As yet, we do not know all the factors which contribute to the differences we observed in the network properties and signal propagation between these pathways. Variables which may influence these properties include pre-/postsynaptic composition, receptor density and subcellular localization, receptor subtypes, cellular populations, pathway organization, fiber myelination, and membrane excitability. Further anatomical and intracellular electrophysiological studies are necessary to address these questions. Nevertheless, identification of the differences in physiological properties between the two intramygdala pathways was greatly facilitated by application of a new technique, the photodiode array coupled with a voltage-sensitive dye. The optical signal obtained over the amygdala allowed us to compare the signal propagation simultaneously along specific pathways. Because many subcortical structures have complex anatomical connections, using fast imaging may also be helpful to sort out functional connectivity for other subcortical systems, particularly those with complex anatomical connectivities.

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