



Glutamate and GABA_B transmissions in lateral amygdala are involved in startle-like electromyographic (EMG) potentiation caused by activation of auditory thalamus

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Abstract

The lateral amygdala nucleus (LA) receives auditory inputs from both the auditory thalamus (medial geniculate nucleus, MGN) and auditory association cortex (AAC). These auditory inputs are closely linked with glutamate and GABA_B receptors in the LA. The LA has intra-amygdaloid connections with the central amygdala nucleus, which mediates auditory fear potentiation of startle (AFPS) via pathways to the startle circuits. The purpose of the present study was to establish an electromyographic (EMG) model for studying AFPS-related neural transmissions in the LA. Hind-limb startle-like EMG responses to single-pulse electrical stimulation of the trigeminal nucleus (TN) were recorded in anesthetized rats. These EMG responses were enhanced by single-pulse sub-threshold electrical stimulation of the MGN when the MGN stimulus led the TN stimulus at short inter-stimulus intervals (ISI). However, the EMG responses were not affected by single-pulse sub-threshold electrical stimulation of the AAC. Bilateral injection of the glutamate antagonist, kynurenic acid, into the LA decreased both the EMG enhancement caused by MGN stimulation at short ISIs and EMG responses to combined TN and AAC stimulation across various ISIs. Moreover, bilateral injection of the GABA_B antagonist, phaclofen, into the LA increased both EMG responses to combined TN and MGN stimulation across various ISIs, and EMG responses to combined TN and AAC stimulation at short ISIs. These results suggest that the auditory inputs to the LA from the MGN and those from the AAC are affected differently by glutamate and GABA_B receptors in the LA, and play differential roles in modulating startle responses.

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The startle reflex, which has been used as a sensorimotor model for studying emotional learning [5,11], involves rapid contractions of skeletal muscles along the full length of the body immediately following strong and sudden sensory stimuli [1,2,12,43]. Startle-like responses can be reliably elicited by electrical stimulation of sensory nuclei, such as the trigeminal nucleus (TN) [22] or the vestibular nucleus [21].

The lateral nucleus of the amygdala (LA) mediates auditory fear conditioning and auditory fear potentiation of startle (AFPS) [9,13,19,27,38,44]. The LA receives auditory

inputs from both the auditory thalamus (medial geniculate nucleus, MGN) and the auditory association cortex (AAC) [3,20,28,37,39,45,47]. LA principal (projection) neurons are inhibited by local GABAergic interneurons [16,17,25] partially via presynaptic GABA_B receptors [14,42]. Outputs from LA principal neurons are transferred through intra-amygdaloid connections toward the central amygdala [34,35], which modulates startle via both direct [8,15,40] and indirect [6,7,10,30,31,48] neural connections to the startle circuits.

Electrical stimulation of the MGN or the AAC elicits excitatory neural responses in the LA [23,24]. If the electrically elicited responses in the LA can be reflected in the startle re-

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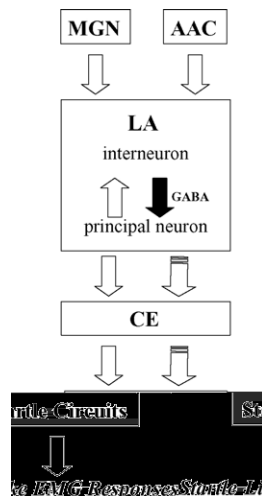


Fig. 1. The hypothesized neural pathways mediating auditory fear potentiation of startle (AFPS). The arrows without broken tails represent direct axonal projections; the arrows with broken tails represent indirect neural connections. MGN: medial geniculate nucleus; AAC: auditory association cortex; LA: lateral amygdala nucleus; CE: central amygdala nucleus.

flex (Fig. 1), the startle reflex would be useful for studying (1) the difference between MGN and AAC inputs in modulating startle, and (2) LA neural transmissions that are involved in modulating startle. In the present study, startle-like electromyographic (EMG) responses were elicited by electrical stimulation of the TN [21,22] in anaesthetized rats. Effects of electrical stimulation of the MGN or AAC on the electrically elicited EMG responses were investigated before and after blocking either glutamate or GABA_B receptors in the LA.

Adult male Wistar rats (350–400 g), obtained from the Animal Center of the Health Science Research Center of Peking University, were used in this study. They were housed individually on a 12-h-light/12-h-dark cycle (light from 7.00 a.m. to 7.00 p.m.), with food and water freely available. The rats were allowed at least one week to adapt to the laboratory environment before surgery. All efforts were made to minimize animal suffering (e.g. pain or discomfort) and to use only the number of animals necessary to produce reliable scientific data.

The rats were anesthetized deeply with 10% chloral hydrate (400 mg/kg, i.p.) and placed in a Kopf stereotaxic head holder. A state of areflexia was carefully maintained throughout the experiment by supplemental injection of the same anesthetic. Flexible wire electrodes were implanted into the hindlimb anterior biceps femoris muscles for measuring EMG responses. A midline incision was made in the scalp, and the skin and temporal muscles were retracted laterally. The animal head was positioned with bregma and lambda at the same horizontal plane. Craniotomies were made on the dorsal surface of the skull to permit insertion of stimulation electrodes and injection needles into the brain.

Stainless steel electrodes [22] were aimed at the following three brain structures on the right hemisphere, referenced to

bregma and based on the coordinates provided by Paxinos and Watson (1997) [33]. (1) TN: AP = –10.0 mm, ML = 2.9 mm, DV = –9.4 mm; (2) MGN: AP = –5.6 mm, ML = 3.4 mm, DV = –6.0 mm; (3) cortical area TE3: AP = –5.8 mm, ML = 6.5 mm, DV = –3.5/–4.0 mm. Area TE3 in rats is the major AAC that projects to LA [28,39]. Injection needles were aimed at the LA on both sides: AP = –2.8 mm, ML = ±5.4 mm, DV = –5.5/–6.0 mm.

Electrical stimuli were generated by a Grass S-88F stimulator (Grass, Quincy, MA, USA), which provided monophasic cathodal rectangular pulses (pulse duration = 0.2 ms) via constant-current, photoelectric stimulus-isolation units (model PSIU6).

Single-pulse stimulation of the TN was tested to establish the minimum current required to elicit a reliable startle-like response. The current level for stimulating the TN was set at 100 μ A above the threshold. The average stimulating current in the TN was 300 μ A (ranged from 150 to 550 μ A). Single-pulse stimulation of either the MGN or AAC was also tested. The current level for stimulating the MGN or AAC was set at levels without eliciting startle-like responses (ranged from 450 to 700 μ A).

For each experiment, the glutamate receptor antagonist kynurenic acid (2 mM) and GABA_B receptor antagonist phaclofen (0.5 mM) (Sigma, St. Louis, MO, USA) were dissolved in Locke's solution and saline, respectively. Injection was made by a microinjection pump. The injection volume on each side of the brain was 1 μ l, and the injection time was 1 min.

All the tests were conducted between 9.00 a.m. and 4.00 p.m., during the light part of the light–dark cycle. EMG responses to TN stimulation, combined TN and MGN stimulation, or combined TN and AAC stimulation were amplified through an amplifier (Model EX4-400, Quad differential Amplifier) and recorded through a digital real-time oscilloscope (Tektronix, TDS 220), before and after bilateral injection of saline, kynurenic acid or phaclofen into the LA in a pseudo-random manner. The inter-stimulation interval (ISI) between MGN and TN or between AAC and TN stimulation was 0, 1, 2.5, 5, 10, 20, 30 and 50 ms. Fig. 2 illustrates the arrangement of electrical stimulation and injection for studying the effect of MGN stimulation on startle-like responses to TN stimulation. Statistical tests applied to the data were one-way analyses of variance. The probability level at which the null hypothesis was rejected was 0.05.

The rats were assigned into six groups: (1) MGN-stimulation/saline-injection, (2) MGN-stimulation/kynurenic acid-injection, (3) MGN-stimulation/phaclofen-injection, (4) AAC-stimulation/saline-injection, (5) AAC-stimulation/kynurenic acid-injection, and (6) AAC-stimulation/phaclofen-injection groups. In three groups stimulation electrodes were in the TN and MGN, and injected chemicals were saline, kynurenic acid, and phaclofen, respectively. In the other three groups stimulation electrodes were in the TN and AAC, and injected chemicals were saline, kynurenic acid, and phaclofen, respectively.

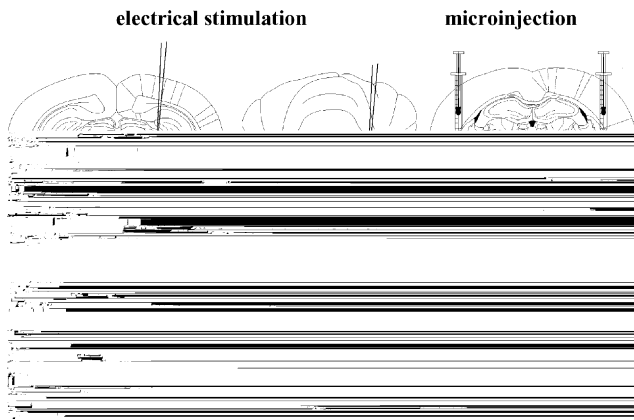


Fig. 2. Diagram indicating the manipulation made in the animal groups with stimulating electrodes in the MGN. Electrical stimulation of the MGN was used to modulated startle-like EMG responses to electrical stimulation of the trigeminal nucleus (TN). The influence of glutamate or GABA_B transmissions in the LA was investigated using pharmacological blocking methods.

At the end of testing, the rats were sacrificed with an overdose of sodium pentobarbital. Lesion marks were made via the stimulating electrodes by an anodal DC current (500 μ A for 10 s). The brains were removed, stored in 10% formalin with 30% sucrose until they sank, and then sectioned at 40 μ m in the frontal plane in a cryostat (-20°C). Sections were stained with cresyl violet to determine electrode and injection needle locations.

Totally 56 rats were used in the present study after successful experiments were achieved in 6 rats for each group. Here only the results from the successful experiments in 36 rats were presented. The locations of injection needle aimed at the LA and stimulation electrode tips aimed at the TN, MGN and AAC in the 36 rats are indicated in Fig. 3. They were within the correspondent target areas.

In chloral hydrate-anesthetized rats, unilateral single-pulse electrical stimulation of the TN with moderate currents (150–550 μ A) produced EMG activity recorded from the hindlimb anterior biceps femoris muscles. The hindlimb EMG activity was always accompanied by bilateral, whole-body, startle-like responses that were similar to those reported previously [21,22]. Thresholds of hindlimb twitches induced by electrical stimulation were higher than those for pinna, neck, and back twitches.

For the three groups with stimulating electrodes in the TN and MGN, EMG response amplitudes to combined TN stimulation and MGN stimulation were largely determined by the ISI (Fig. 4, MGN panels). For all the three groups, EMG responses were enhanced when the MGN stimulus led the TN stimulus by 1–10 ms ($p < 0.05$), and slightly suppressed ($p > 0.05$) at ISIs longer than 30 ms. Following bilateral injection of saline into the LA, EMG amplitudes did not change significantly at various ISIs, except the ISI of 2.5 ms, at which the amplitude was significantly higher than that before injection ($p < 0.05$) (Fig. 4, MGN/saline). Following bilateral injection of kynurenic acid into the LA, EMG amplitudes

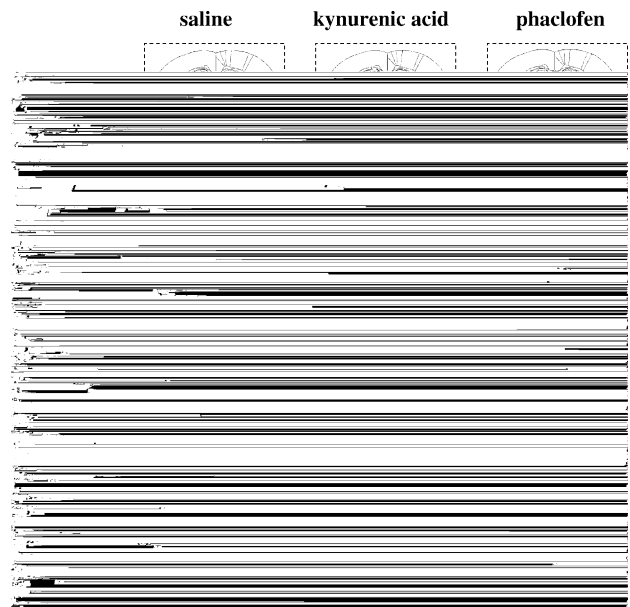


Fig. 3. Histological reconstruction of the injection needle and electrode placements for the six animal groups. In all the six groups, injecting needles were in the LA bilaterally. In three groups, electrodes were in both the MGN and TN; in the other three groups electrodes were in the AAC and TN.

decreased significantly at the short ISIs of 1, 2.5 and 5 ms ($p < 0.05$). However, at the long ISIs (10, 20, 30 and 50 ms), no changes were observed (Fig. 4, MGN/kynurenic acid). Following bilateral injection of phaclofen into the LA, EMG amplitudes increased significantly at all of the ISIs ($p < 0.05$), except the ISIs of 0 and 10 ms (Fig. 4, MGN/phaclofen).

For the three groups with stimulating electrodes in the TN and AAC, EMG response amplitudes to combined TN stimulation and AAC stimulation were not influenced by the ISI (Fig. 4, AAC panels). Following bilateral injection of saline into the LA, EMG amplitudes did not change at all of the ISIs (Fig. 4, AAC/saline). Following bilateral injection of kynurenic acid into the LA, EMG amplitudes decreased at all of the ISIs ($p < 0.05$) (Fig. 4, AAC/kynurenic acid). Following bilateral injection of phaclofen into the LA, EMG amplitudes increased significantly only at the short ISIs of 0, 1, 2.5, and 5 ms and a long ISI of 20 ms ($p < 0.05$) (Fig. 4, AAC/phaclofen).

The excitatory glutamate receptor types in the LA include *N*-methyl-D-aspartate (NMDA), and α -amino-3-hydroxy-5-methyl-4-isoxazolepropionate (AMPA) receptors [4,18]. Previous studies have reported that the LA contains two main types of neurons, principal (projection) neurons and interneurons [17,25,29,32,36,41,46]. The two types of neurons receive excitatory glutamate axonal projections from both the MGN and AAC [4,17,24,26,42,47].

The results of the present study indicate that electrical stimulation of the MGN with moderate currents has different effects from that of the AAC on startle-like EMG responses elicited by electrical stimulation of the TN. MGN stimulation markedly enhanced the startle-like EMG responses at a short

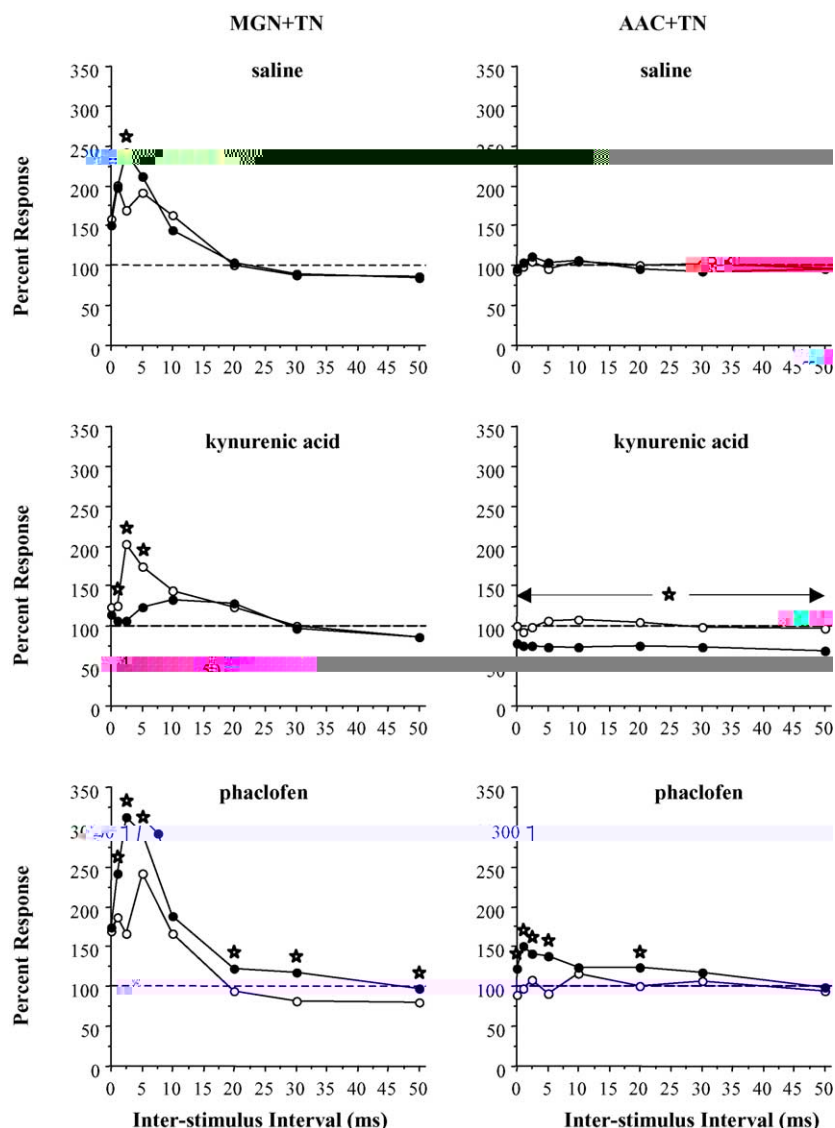


Fig. 4. Normalized EMG responses to combined stimulation of MGN and TN or combined stimulation of AAC and TN. The baseline response (100%) was the one to stimulation of the TN alone before injection. The inter-stimulus interval was the leading time of MGN or AAC stimulation relative to TN stimulation. Open circles indicate responses before injection; filled circles indicate responses following injection. Stars above the data points indicate that at these inter-stimulus intervals EMG responses after injection were significantly different from those before injection.

leading time (no larger than 10 ms) of MGN stimulation relative to TN stimulation. Following injection of kynurenic acid, a broad-spectrum blocker of glutamate receptor, into the LA, the fast enhancement of EMG responses was partially eliminated, suggesting that the fast enhancement of the startle-like EMG activity was mediated, to some extent, through fast excitatory glutamate transmissions in the LA.

Principal neurons in the LA are inhibited by GABAergic interneurons, via both feedforward and feedback inhibition [16,17,24,42]. Studies with paired-pulse- or primed-pulse-stimulation paradigms have shown that inhibition of principal neurons can be reduced by application of antagonists of GABA_B receptors [14,24,42], suggesting that in addition to postsynaptic GABA_A receptors, presynaptic GABA_B receptors are involved in feedforward and/or feedback inhibi-

tion of principal neurons. Previous studies have suggested that excitatory AAC inputs to the LA are stronger onto inhibitory interneurons than onto principal neurons. For example, electrical stimulation of the perirhinal and entorhinal cortical regions in anaesthetized cats mainly produces both short-latency excitatory responses of interneurons and longer latency inhibitory responses of principal neurons, and the excitatory response profile of interneurons corresponds with the inhibitory response profile of principal neurons [17]. In the present study, AAC stimulation had no obvious effects on the EMG responses when either glutamate or GABA_B receptors were not blocked, supporting the notion that the major direct impact of AAC afferents to the LA is on interneurons.

In the present study, blockade of GABA_B receptors in the LA by phaclofen generally enhanced the electrically elicited

EMG activity. Thus, the electrically induced inputs to the LA from the MGN or AAC may be under prolonged suppression mediated by GABA_B receptors in the LA.

The results of the present study indicate that electrical stimulation of the MGN, but not the AAC, in these conditions enhances startle-like EMG responses to TN stimulation, suggesting that the MGN and AAC play differential roles in mediating AFPS. Also, the mediations of AFPS are associated with both glutamate and GABA_B transmissions in the LA. Therefore, the EMG startle model established in the present study can be used for studying the functions of the LA neural transmissions in modulating fear activated by auditory inputs.

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