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Feedback of the amygdala globally modulates visual response of primary visual cortex in the cat

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ABSTRACT

The amygdala is an important center for emotional behavior, and it influences other cortical regions. Long feedback projections from the amygdala to the primary visual cortex were recently reported in the cat and monkey, two animal models for vision research. However, the detailed functional roles of these extensive projections still remain largely unknown. In this study, intrinsic signal optical imaging was used to investigate the visually driven responses of the primary visual cortex of cats as focal drugs were injected into the basal nucleus of the amygdala. Both the visually evoked global signals and differential signals in the functional maps of the primary visual cortex were enhanced or reduced by glutamate-induced activation or GABA-induced deactivation of neurons in the amygdala, respectively. This modulation was found to be non-selective, consistent with the gain control mechanism—both the preferred orientation and its mapped orientation tuning width remained unchanged. The single unit recordings showed similar results supporting the above observations. These results suggest that the distal feedback signals of the amygdala enhance the primary sensory information processing in a non-selective, gain-control fashion. This provides direct neurophysiological evidence and insight for previous studies on emotional-cue related psychological studies.

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Introduction

The amygdala is believed to be the emotional center in the brain (Pessoa, 2011; Phelps and LeDoux, 2005; Pourtois et al., 2012; Zald, 2003). In a study using functional magnetic resonance imaging (fMRI), it was found that fear led to the activation of the amygdala in humans (Pessoa et al., 2006). Anatomically, a broad range of sources send signals to the amygdala (Zald, 2003), which integrates the various inputs into representations of abstract affective values (Paton et al., 2006). On the other hand, the amygdala also modulates various brain functions (Amaral and Price, 1984; Amaral et al., 2003; Ben-Ari, 1981; Emery et al., 2001; Pessoa, 2011; Phelps and LeDoux, 2005; Pourtois et al., 2012; Zald, 2003).

With regard to vision, fear cues lead to enhanced performance on visual tasks (Anderson and Phelps, 2001; Fox et al., 2000; Frischen et al., 2008), stronger fMRI signals in various visual cortices (Brooks et al., 2012; Morris et al., 1998; Padmala and Pessoa, 2008; Vuilleumier et al., 2001), as well as increased ERP signals (Pourtois et al., 2004; Rauss et al., 2009). In primates and cats, the amygdala directly sends extensive feedback projections to visual cortices, including high order cortex and V1/primary visual cortex (Amaral et al., 2003; Chen et al., 2009), and modifications of this fundamental sensory cortex may lead to significant changes in visual perception/cognition. However, this functional modulation of the amygdala on V1 still lacks key neurophysiological evidence to link the psychological phenomenon and the anatomical findings. It is thus of interest to directly investigate the effect of amygdala modulation on the visually evoked neuronal responses in the primary visual cortex.

Current literature holds that the primary visual cortex is modulated by feedback from a broad range of sources, resulting in a fairly diversified modulation effect. In certain areas, modulations of the higher visual cortex seem to be feature specific: area 21a, which is the gateway of form pathway in cats, can alter the distribution of preferred orientation in the primary visual cortex. In other areas such as the posteriomedial lateral suprasylvian (PMLS), which is the gateway of motion pathway in cats, direction selectivity but not form-related orientation selectivity can be selectively enhanced (Huang et al., 2004; Liang et al., 2007; Shen et al., 2006, 2008; Tong et al., 2011; Wang et al., 2000). A similar feature-specific feedback effect also exists in the cortico-thalamus projections in the lateral geniculate nucleus (LGN) (Andolina et al., 2007; Cudeiro and Sillito, 1996; Murphy and Sillito, 1987; Sillito et al., 1993). On the other hand, feedback from higher levels, such as visual attention, commonly modulate the neuronal responses in a less specific gain control way—in a proportional increase of firing rates to all stimuli (Herrmann et al., 2012). Emotion, which involves the activation of amygdala, was suggested to modulate perceptual processing in a similar gain control mechanism (Lang and Davis, 2006; Pourtois et al., 2012). It is thus necessary to illuminate the detailed mechanism of amygdala feedback on primary visual cortex in an animal model with clear neurophysiological data.

In this study, we performed experiments on anesthetized and paralyzed animals to investigate the modulation of visually driven neuronal

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responses specifically in the primary visual cortex of cats, when the amygdala was focally activated/de-activated. We examined whether the amygdala could modulate fundamental sensory processing in the primary visual cortex, and whether it modulates in a feature specific fashion or through a general gain control mechanism. Population evaluation based on intrinsic signal optical imaging and single neuron analysis based on single unit recordings was employed to elucidate this issue and double confirm the major findings.

Methods and materials

Animal preparation

Fifteen normal adult cats of either sex, weighing between 2.5 and 3.0 kg, were used in the study. All experiments involving animals conformed to the policy of the Society for Neuroscience on the Use of Animals in Neuroscience Research. All animal use procedures were performed in strict accordance with the Guide for the Care and Use of Laboratory Animals described by Fudan University and U.S. National Institutes of Health, and all experiments were designed to minimize the number of animals used and their suffering. Animals were initially anesthetized with ketamine (20 mg/kg). All pressure points and incised tissues were infiltrated with lidocaine. During the remainder of the experiment, anesthesia was maintained with IV pentobarbital sodium given at a loading dose of 4 mg/kg followed by an infusion of 3 mg/kg/h. After IV and tracheal cannulations were performed, the cat was placed in a stereotaxic apparatus (Jiangwan II type, The Second Military Medical University, Shanghai, China). Gallamine triethiodide (Flaxedil; Shanghai Dongfeng Chemicals Factory, Shanghai, China; 8–10 mg/kg/h) was then used for immobilization and animals were given artificial respiration using a pulmonary pump. The animals' physiological conditions were kept in normal ranges throughout the experiment. Thus, the end-tidal CO_2 was kept at 4% by adjusting the rate and/or stroke volume of the pulmonary pump. The body temperature of animals was monitored and maintained at 38 °C throughout the procedures by an automatic temperature control system. Electroencephalogram (EEG) and electrocardiogram (ECG) were used to continuously monitor the degree of cortical excitation. EEG recording showed that the slow wave and the heart rate were maintained normally throughout the procedures. The pupils of the cat were dilated with atropine (0.5%) and nictitating membranes were retracted with neosynephrine (2%). The eyes were carefully refracted and corrected with contact lenses of appropriate refractive power. To reduce the amount of spherical aberration, artificial pupils (3 mm in diameter) were placed in the front of the eyes.

A stainless steel imaging chamber was positioned and fixed with dental cement on the skull, covering a portion of the primary visual cortex. After careful removal of the dura, the chamber was filled with warm (37 °C) silicone oil and sealed with a transparent glass window. The amygdala was reversibly activated by injection of 1.0–1.5 μl 0.2 mM glutamate (Sigma-Aldrich Corporation, USA) and inactivated by injection of 1.0–1.5 μl 100–400 mM GABA (Sigma-Aldrich Corporation, USA). As a control, we also injected 1.5 μl of phosphate-buffered saline (PBS, pH 7.4) or saline at the same site. Solutions were injected slowly (over a period of 4 min) and the needle of the micro-syringe was withdrawn 10 min after the termination of injection. The injection sites were centered at the Horsley–Clarke coordinates A 12, L8.5 and 22.5 mm beneath the pial surface to activate or inactivate neurons in the basal nucleus of the amygdala. According to Chen et al. (2009), only the electrical stimulus at the basal nucleus of the amygdala can evoke strong potentials in the primary visual cortex. We adopted this protocol to locate the basal nucleus of the amygdala. In each experiment, we examined the evoked potentials in the primary visual cortex elicited by the electrical stimulus at the injection site, and adjusted the site accordingly (Supplementary Fig. 1). Previous studies have indicated that 1.0 μl of 100 mM GABA tends to diffuse over a region of 1.5 mm in diameter in the mammalian cortex (Hupé et al., 1999). To reduce extent of the mechanical damage to the injected part of the amygdala, drug injections were limited to no more than three per site. The interval between two trials was longer than 3 h to guarantee a sufficient recovery. Following 2–3 days of experiments, the animal was perfused for histological staining. The animal was deeply anesthetized with pentobarbital sodium (25 mg i.v.) and transcardially perfused with 0.9% saline, followed by 4% paraformaldehyde in 0.1 M sodium phosphate buffer. The location of the centers of the injection sites was assessed histologically in 50 μm sections counterstained for Nissel substance with Cresyl Violet. Only data from animals with the correct injection locations within the amygdala were included in further analysis.

Visual stimulation

The cats were stimulated binocularly with a 20° × 30° of drifting sinusoidal wave gratings (contrast 90%) with a spatial frequency of 0.5 cycles/degree and a temporal frequency of 2 Hz. Stimuli were randomly presented at 8–16 directions (for optical imaging) or 24 directions (for single unit recordings) which are distributed uniformly from 0° to 360°. The moving direction of the gratings was kept orthogonal to the orientation. The visual grating stimulus was presented on the screen of a high resolution monitor (FlexScan F931, Eizo Nanao Corporation, Japan) positioned 57 cm apart from the cat's eyes for 2 s with 10 s blank intervals in between for optical imaging, and 2 s on and 2 s blank for single unit recording, repeatedly. The mean luminance of the blank screen was 15.1 cd/m^2.

Optical data acquisition

A custom-made optical imaging system was employed. As used in our previous studies (Chen et al., 2003; Huang et al., 2004), a slow-scan CCD camera (512 × 512 pixels, 24 × 24 μm/pixel; DALSA, Inc., Waterloo, Ontario, Canada) was used to record the optical images of intrinsic signals from the exposed portion of primary visual cortex. A macroscope tandem-lens arrangement of two coupled 50-mm lenses (f = 1:1.2) was used to achieve a very shallow depth of field (less than 100 μm) in order to alleviate blood vessel artifacts and influence of surface layers in the functional maps. The vessel map on the cortical surface was obtained with green light (540 nm) shining on the surface of the cortex. Intrinsic signals evoked by the grating stimuli were detected under illumination with red light (640 nm) when the camera was focused on the plane of 500 μm below the pial surface. Data acquisition started 3 s before the appearance of the stimulus and a total of seven frames at a frame rate of 1 Hz were recorded. Since the greatest intrinsic signals appear at 3 s after the onset of visual stimulus (Chen et al., 2003; Huang et al., 2004), except in the time–course study, we used the 6th frame in a trial to analyze the data. To allow the relaxation of activity-dependent microvascular changes, the period of stimulus representation was followed by a 10 s blank. The order of stimulus presentation in each trial was randomized. Each stimulus was presented 16–32 times, and the evoked signals were averaged.

Electrophysiological recording

The primary visual cortex at Horsley–Clarke coordinates P 1–8 and L 0–3 was exposed for electrophysiological recording. The action potentials of the cells in the visual cortex in the cat were recorded with a glass micropipette filled with 3 M NaCl, whose impedance ranged from 3 to 5 M. The glass microelectrode was positioned perpendicularly to the surface of the exposed cortex covered with agar. Action potentials of the recorded primary visual cortex neuron were amplified then fed to an audio monitor and a data acquisition system (CED micro 1401, Cambridge Electronic Design Ltd., UK). The raw data was stored for further analysis (Spike 2, version 4; Cambridge Electronic Design Ltd.). The post-stimulus time histogram (PSTH) was repeated 10–20 times and

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