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ACUTE RESTRAINT STRESS ALTERS SOUND-EVOKED NEURAL RESPONSES IN THE RAT AUDITORY CORTEX

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12 **Abstract—Stress is known to elicit various adaptive or maladaptive responses in the nervous system function. Psychophysical studies have revealed that stress exposure induced the changes in auditory response that can be interpreted as a transient, stress-induced hypersensitivity to sounds. However, the underlying neural mechanism remains unresolved. Thus, in this study, we explored the neural activities of the auditory cortex (AC) in response to stress. We elicited stress by physically immobilizing rats and recorded the extracellular single-unit activities through the electrodes chronically implanted in the AC of rats. By comparing the spike activities of the same rat before, during and after immobilization, we found temporal and significant changes in the sound-evoked neural activities. In most cases, acute restraint stress enhanced neural responses evoked by pure-tones and click-trains, but in a minority of neurons, stress suppressed responses. The immobilization-induced enhancement was more frequently found in the neurons that originally had a low responsibility for sound stimuli. The enhancement effects on pure-tone response were reflected by an increase of response magnitude, decrease of response latency, and extension of bandwidth of tuning curve (BW). But the spontaneous firing rate and best frequency (BF) remained unchanged. Stress also increased the ability of neural response to synchronize to click-trains, even in the neurons whose response magnitude was not significantly increased. Taken together, these results provide direct evidence that stress alters the function of auditory system at the level of AC.** **© 2015 Published by Elsevier Ltd. on behalf of IBRO.**

Key words: cerebral cortex, neural response, chronic recording, acute stress, serum corticosterone, acoustic perception.

INTRODUCTION

Stress is a complex biological reaction common to all living organisms that allows them to adapt to their environments. There is extensive evidence that stress exposure elicits adaptive or maladaptive changes of brain functions, particularly learning and memory performance (de Kloet et al., 2005; Roozendaal et al., 2009). Similar impacts of stress appear to exist for auditory system. Clinical reports have demonstrated a well-established relationship between stress and hearing problems. An acute stress could increase auditory sensitivity (hyperacusis) in humans, particularly women with high levels of emotional exhaustion (Hasson et al., 2013). Stress exacerbated the sudden hearing loss or tinnitus (Ban and Jin, 2006; Al-Mana et al., 2008). Clinical data associate tinnitus onset and tinnitus severity with stress (Hinton et al., 2006; Fagelson, 2007; Hébert and Lupien, 2007). A study using event-related potentials in humans also reported that acute stressor is able to alter auditory-selective attention (Elling et al., 2011).

The impacts of stress on auditory function were also demonstrated by the studies using various animal stress models. The mean threshold for the stressed guinea pigs following noise exposure was significantly lower (better) than that of the controlled, sedated, guinea pigs (Muchnik et al., 1992). In mice, the mild physical restraint stress significantly increased cochlear sensitivity (Wang and Liberman, 2002), and distortion product otoacoustic emissions (DPOAEs) were confirmed to be enhanced by heat stress (Murakoshi et al., 2006). In rats, the prenatal stress induced by frequent handling, cage changing and mock injections of the pregnant rats resulted in offspring having a low-frequency hearing loss (Kadner et al., 2006). Sonic stress by 24-h exposure to an acoustic rodent repellent decreased the thresholds and increased the amplitudes of auditory brainstem responses and DPOAEs (Mazurek et al., 2010).

Though stress-induced changes of auditory functions have been well documented as mentioned above, neural correlates of the functional changes remain unclear. Single-unit neuronal studies are critical for shortening the bridge from neural to sensory changes. For this reason, we investigated how behavioral stress triggers the changes of neural spike activities in the auditory cortex (AC). We used the acute restraint stress

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Abbreviations: ABR, auditory brainstem responses; AC, auditory cortex; BF, best frequency; BW, bandwidth of tuning curve; DPOAEs, distortion product otoacoustic emissions; GC, glucocorticoid; GRs, glucocorticoid receptors; HPA, hypothalamus–pituitary–adrenal; IC, inferior colliculus; MI, modulation index; PSTH, peri-stimulus time histogram; RS, Rayleigh statistic; SPL, Sound pressure level; VS, vector strength.

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model induced by physical immobilization of a rat. Physical restraint in rodents has been widely used to investigate neurophysiological and pathological conditions associated with stress exposure (Glavin et al., 1994; Buynitsky and Mostofsky, 2009). In the present study, we recorded the single-unit activities *in vivo* in the AC of awake rats using chronically implanted electrodes, and examined the neural responses to pure-tone and click-train stimuli to evaluate the fundamental characteristics of neuronal response including the latency, magnitude, frequency-tuning and ability to follow temporally repeated sound stimuli. These neuronal characteristics were compared before, during and after immobilization of the same subject. This preparation eliminates the effects of anesthesia on neuronal activity and avoids the possibility of anesthesia altering the perception of the stressor.

EXPERIMENTAL PROCEDURES

Subjects

Male Wistar rats weighing 250–350 g (8–10 weeks old) at the beginning of the experiments were used. Animals came from our own colony housed in a humidity-controlled (50–55%) and temperature-controlled (22–24 °C) facility on a 12-h-light/dark cycle (lights on at 7:30 A.M.) with access to food and water *ad libitum*. All animal works were carried out in strict accordance with National Institutes of Health Guide for the Care and Use of Laboratory Animals (NIH Publications No. 80-23) revised 1996. The protocol for animal handling and the treatment procedures were approved by the China Medical University Animal Care and Use Committee. All surgery was performed under anesthesia, and all efforts were made to minimize the number of animals used and their suffering.

Surgical preparation and electrode implantation

The animal was anesthetized by an initial injection of chloral hydrate (330–350 mg/kg, 5%, i.p.) supplemented by additional doses (usually 100 mg/kg once per hour). Temperature was monitored rectally and maintained at 37 °C using a feedback-controlled blanket. After placing the animal in a stereotaxic frame (SR-5R, Narishige, Tokyo, Japan), the cranium was exposed, four small holes were drilled over the parietal bone and fine jeweler's screws were inserted to serve as an anchor for a metal head-post holder that was cemented to the skull with dental acrylic. A craniotomy (2 × 1.5 mm) was performed above the left AC according to the coordinates of the Paxinos and Watson brain atlas: 3–7 mm posterior of the bregma and 3–5 mm lateral to the bregma (Paxinos and Watson, 1986). The dura above the AC was removed under binocular control. We then implanted a microwire array consisting of four (2 × 2) formvar-insulated 50-μm-diameter nichrome wires (part #762000; A-M Systems, Carlsborg, WA, USA). The tip impedance of each wire was around 0.5 MΩ at 1 kHz. The distance of two neighbor electrodes was approximately 300 μm. A silver wire, used as ground, was inserted between the temporal bone and the dura mater on the contralateral side. The microwire array was mounted on a custom-built holder that was lowered stepwise with a pulse motor-driven manipulator (SM-20, Narishige, Tokyo, Japan). Wires were inserted into the cortex until the tips of the electrodes were 0.5–0.8 mm below the dura, while viewing through a microscope and listening to an audio monitor of the recorded signal. The craniotomy was then filled with SILASTIC, a silicone elastomer (World Precision Instruments) and sealed using dental acrylic. After the cement had completely hardened, the spare part of wires and the connector were fixed on the skull by dental acrylic. In the end of surgery, an antibiotic (Cefuroxime, Zinacef injection, Glaxosmithkline) was administered systematically (30 mg/kg, i.p.). Animals were then housed in a medal grid cage for 1–2 weeks of postoperative recovery.

Electrophysiological recording and experimental protocol

The physiological recording was conducted in a shielded, soundproof room. Before the recording experiment, the animal within the housing cage was moved to the recording room to acclimatize to the environment for three consecutive days (1 h per day). At the beginning of the experiment, we firstly recorded the neural activities when the rat was free in its housing cage. The microwire output was connected to a multi-channel preamplifier (RA16PA; TDT, Alachua, FL, USA) using a flexible, low-noise cable. The output of the preamplifier was delivered to a digital signal processing module (RZ-2; TDT). Action potentials were detected on-line by threshold crossing, and waveforms were stored to hard disk. During recording, a video camera was used to monitor rat's position and movement. The animal stood fairly motionless some of the time and occasionally moved its limbs, whisked, groomed, etc. Any recording data interrupted by the artifacts of animal's movement were abandoned, and the recordings were repeated as the animal returned to resting state. On average, 1–3 well-isolated single-units were collected in each session. After completing the recordings of all the tested stimuli (usually lasted 20–30 min), the animal was moved out from the housing cage. Its head was fixed through the head-post holder, and the body was put into a half-cut plastic tube (diameter, 5 cm) to restrain the movements. After the animal has been immobilized for 30 min and became quiet, we restarted the recording procedure under the immobilized condition. Then, the animal was released to its housing cage. The recording procedure was repeated at 30 min after the animal returned to the free condition.

Acoustic stimuli

Acoustic stimuli were digitally generated by custom-built programs under MATLAB (Mathworks) environment and delivered via an earphone (NW-STUDIO PRO W; Ninewave), which was attached on the cement platform implanted on the rat's skull during surgery. The placement of the earphone was adjusted to 1 cm from

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