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Research Paper

Noise exposure alters long-term neural firing rates and synchrony in primary auditory and rostral belt cortices following bimodal stimulation

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ABSTRACT

We previously demonstrated that bimodal stimulation (spinal trigeminal nucleus [Sp5] paired with best frequency tone) altered neural tone-evoked and spontaneous firing rates (SFRs) in primary auditory cortex (A1) 15 min after pairing in guinea pigs with and without noise-induced tinnitus. Neural responses were enhanced (+10 ms) or suppressed (0 ms) based on the bimodal pairing interval. Here we investigated whether bimodal stimulation leads to long-term (up to 2 h) changes in tone-evoked and SFRs and neural synchrony (correlate of tinnitus) and if the long-term bimodal effects are altered following noise exposure. To obviate the effects of permanent hearing loss on the results, firing rates and neural synchrony were measured three weeks following unilateral (left ear) noise exposure and a temporary threshold shift. Simultaneous extra-cellular single-unit recordings were made from contralateral (to noise) A1 and dorsal rostral belt (RB); an associative auditory cortical region thought to influence A1, before and after bimodal stimulation (pairing intervals of 0 ms; simultaneous Sp5-tone and +10 ms; Sp5 precedes tone). Sixty and 120 min after 0 ms pairing tone-evoked and SFRs were suppressed in sham A1; an effect only preserved 120 min following pairing in noise. Stimulation at +10 ms only affected SFRs 120 min after pairing in sham and noise-exposed A1. Within sham RB, pairing at 0 and +10 ms persistently suppressed tone-evoked and SFRs, while 0 ms pairing in noise markedly enhanced tone-evoked and SFRs up to 2 h. Together, these findings suggest that bimodal stimulation has long-lasting effects in A1 that also extend to the associative RB that is altered by noise and may have persistent implications for how noise damaged brains process multi-sensory information. Moreover, prior to bimodal stimulation, noise damage increased neural synchrony in A1, RB and between A1 and RB neurons. Bimodal stimulation led to persistent changes in neural synchrony in intact A1 and RB that were also altered by noise-exposure. Given that increases in neural synchrony following noise may be consistent with tinnitus onset, these data implicate that both A1 and RB may be involved in the etiology of phantom sound perception. These data also suggest that noise alters the effects of bimodal stimulation on neural synchrony in A1 and RB; an effect that may also lead to changes in tinnitus perception.

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1. Introduction

Peripheral noise damage results in adaptive changes throughout central auditory circuits including brainstem (Finlayson and Kaltenbach, 2009; Kaltenbach et al., 2000; Moore, 1994; Moore et al., 1994; Shore, 2005), mid-brain/thalamus (Kamke et al., 2003; Kitzes and Semple, 1985; Mulders and Robertson, 2011, 2013; Shepherd and Hardie, 2001) and primary auditory cortex (A1; Allman et al., 2009; Basura et al., 2012, 2015, 2008; Kral et al., 2000; Meredith and Allman, 2012; Meredith et al., 2012; Norena et al., 2003; Norena and Eggermont, 2005; Rajan et al., 1993; Robertson and Irvine, 1989). Some studies demonstrate that peripheral auditory sensory deprivation leads to significant cross-

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Abbreviations

ABR	Auditory brainstem response
AAF	Anterior auditory field
BF	Best frequency
BI	Bimodal interval
DCN	Dorsal cochlear nucleus
A1	Primary auditory cortex
RB	Rostral belt
SSC	Somatosensory cortex
SFR	Spontaneous firing rate
Sp5	Spinal trigeminal nucleus
TTS	Temporary threshold shift

modal plasticity in A1 (Auer et al., 2007), while others suggest it is modest (Finney et al., 2001) or is absent (Hickok et al., 1997; Kral et al., 2003).

Somatosensory interaction with auditory neurons occurs as early in the central pathway as dorsal cochlear nucleus (DCN; Shore et al., 2000; Kanold and Young, 2001; Shore, 2005; Zhou et al., 2007). Changes in these pathways after cochlear damage reveal plasticity, which is a potential etiology for tinnitus (Koehler et al., 2011; Dehmel et al., 2012; Koehler and Shore, 2013b; Wu et al., 2016). Bimodal somatosensory-auditory stimulation results in changes in spontaneous neural firing rates (SFRs; Dehmel et al., 2012; Koehler and Shore, 2013a) and in neural synchrony (Wu et al., 2016) in DCN, which are accepted neural physiologic correlates of tinnitus. We have shown similar effects in A1 where bimodal stimulation (spinal trigeminal nucleus [Sp5] paired with pure tones at best frequency; BF) altered tone-evoked and SFRs 15 min after pairing (0 and +10 ms bimodal interval; BI; Basura et al., 2015) in animals with and without behavioral evidence of noise-induced tinnitus. While it is evident that somatosensory stimulation can modulate auditory responses in A1 (Allman et al., 2009; Basura et al., 2012, 2015 Ghazanfar et al., 2005, 2008; Lakatos et al., 2007; Meredith and Allman, 2012; Meredith et al., 2012; Schroeder and Foxe, 2002; Schroeder et al., 2001, 2003), it is not known if bimodal stimulation following noise exposure will lead to persistent changes in A1 neurons. Therefore, the first purpose of this study was to evaluate the long-term (up to 2 h) effects of bimodal stimulation on A1 tone-evoked and SFRs and neural synchrony in intact animals and after noise exposure.

Auditory cortices in many mammalian species are comprised of primary and adjacent associative regions the latter of which are thought to have the capacity to modulate primary centers and auditory processing (Carrasco and Lomber, 2009). One associative auditory region in guinea pig is the dorsal rostral belt (RB), a narrow strip of non-tonotopically organized cortex comprised of neurons with broad tuning curves and preferential responses to noise, longer response latencies and elevated stimulus thresholds (Redies et al., 1988). This region is adjacent to somatosensory cortex (SSC) and whisker barrel fields in many mammals (Wallace et al., 2002). The RB flanks a small anterior region that contains sub-region area S ("small field"). Area S extends into the Sylvian fissure and is readily responsive to limited pure tones. Following cochlear damage in cat, somatosensory and visual systems modulate A1 and the anterior auditory field (AAF; touted equivalent region to guinea pig RB), suggesting that plasticity after hearing loss may also extend outside of A1 to associative auditory cortices (Carrasco and Lomber, 2009). The finding that AAF neurons also modulate A1 firing (Carrasco and Lomber, 2009), implicates this associated cortical region as a separate potential mediator of A1 activity. Given the known projections between SSC, RB and A1 (Wallace et al., 2002), a second hypothesis of this study was that noise exposure may also modify the effects of somatosensory-auditory stimulation that in the intact RB, may also lead to long-term changes in tone-evoked and SFRs and in neural synchrony. To test the two hypotheses for this study we used extra-cellular recording probes to measure single-unit responses simultaneously from A1 and RB neurons. We measured pre- and post-bimodal changes in tone-evoked and SFRs and in neural synchrony 60 and 120 min after Sp5-auditory stimulation only at the two BIs found previously to have the greatest suppression (0 ms) and enhancement (+10 ms) on neural firing (Basura et al., 2015). We then investigated if the long-term bimodal effects are altered following noise exposure. To obviate the effects of permanent hearing loss on the results, tone-evoked, SFRs and neural synchrony were measured three weeks following unilateral (left ear) noise exposure and a temporary threshold shift (TTS). We chose 3 weeks after noise exposure to perform these studies as we have shown that interval period after a noise-induced TTS is adequate for normalization of auditory thresholds and the generation of tinnitus perception to be detected behaviorally and therefore may be present in these animals (Basura et al., 2015).

2. Materials and methods

2.1. Animals/experimental design/noise exposures

Experiments were performed on mature, female, pigmented guinea pigs (250–350g; Elm Hill colony) divided into sham-controls (n = 10) and noise-exposed (n = 12) groups. All procedures were performed in accordance with the National Institutes of Health *Guidelines for the Use and Care of Laboratory Animals* and approved by the University of Michigan Committee on the Use and Care of Animals (UCUCA).

The purpose of this study was to evaluate the long-term (up to 2 h) effects of bimodal stimulation on A1 and RB tone-evoked and SFRs and neural synchrony after noise exposure. To measure the effects of hearing loss on A1 and RB recordings, auditory brainstem response (ABR) thresholds were obtained at three time points during the study to determine normal pre-noise exposure hearing thresholds, immediately following noise to confirm a TTS (4, 8, 12 and 16 kHz) and three weeks later to measure threshold recovery to pre-noise baseline levels immediately prior to physiology recordings. Animals were anesthetized with ketamine (40 mg/kg) and xylazine (10 mg/kg) during noise exposure as ketamine has been shown to have no obvious impact on A1 neural frequency tuning (Huang et al., 2013). Following a 2-h unilateral (left ear only) noise exposure (97 dB noise with ¼ octave band centered at 7 kHz near the tinnitus frequency) and recovery guinea pigs were re-housed in the animal facility for three weeks until physiology recordings began. The remaining animals were grouped as sham-controls and were only subjected to anesthesia alone with no noise exposure. Three weeks following noise exposure or sham anesthesia, single-unit extra-cellular recordings were concurrently measured before and after bimodal stimulation in the contralateral (right hemisphere) A1 and RB.

2.2. Surgical approach and neural recordings

Following adequate anesthesia, animals were placed in a stereotaxic device (Kopf) with hollow ear bars for sound delivery. Rectal temperature was monitored and core temperature was maintained at 38 ± 0.5 °C with a thermostatically controlled heating pad. Supplemental anesthesia (0.5 mls of initial anesthetic concentration; IM) was given approximately every 30 min after

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