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Chronic exposure to broadband noise at moderate sound pressure levels spatially shifts tone-evoked responses in the rat auditory midbrain

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ABSTRACT

Noise-induced hearing disorders are a significant public health concern. One cause of such disorders is exposure to high sound pressure levels (SPLs) above 85 dBA for eight hours/day. High SPL exposures occur in occupational and recreational settings and affect a substantial proportion of the population. However, an even larger proportion is exposed to more moderate SPLs for longer durations. Therefore, there is significant need to better understand the impact of chronic, moderate SPL exposures on auditory processing, especially in the absence of hearing loss. In this study, we applied functional magnetic resonance imaging (fMRI) with tonal acoustic stimulation on an established broadband rat exposure model (65 dB SPL, 30 kHz low-pass, 60 days). The auditory midbrain response of exposed subjects to 7 kHz stimulation (within exposure bandwidth) shifts dorsolaterally to regions that typically respond to lower stimulation frequencies. This shift is quantified by a region of interest analysis that shows that fMRI signals are higher in the dorsolateral midbrain of exposed subjects and in the ventromedial midbrain of control subjects ($p < 0.05$). Also, the center of the responsive region in exposed subjects shifts dorsally relative to that of controls ($p < 0.05$). A similar statistically significant shift ($p < 0.01$) is observed using 40 kHz stimulation (above exposure bandwidth). The results suggest that high frequency midbrain regions above the exposure bandwidth spatially expand due to exposure. This expansion shifts lower frequency regions dorsolaterally. Similar observations have previously been made in the rat auditory cortex. Therefore, moderate SPL exposures affect auditory processing at multiple levels, from the auditory cortex to the midbrain.

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Introduction

Acoustic noise exposure can lead to numerous health disorders. One of the most prominent disorders is noise-induced hearing loss. According to the National Institute on Deafness and Other Communication Disorders of the United States, approximately 15% of Americans between the ages of 20 and 69 have noise exposure related hearing loss (NIDCD, 2014). This may well be an underestimate as not all hearing loss is easily detected using the current gold standard of pure tone audiometry (Plack et al., 2014). Further, 16% of teenagers from 12 to 19 years of age may have noise exposure related hearing loss. This suggests the prevalence of hearing disorders will increase in the coming years. The noise exposures leading to hearing loss can come from occupational and recreational settings. To help protect hearing, the National Institute for Occupational Safety and Health has set a recommended exposure limit of 85 dBA averaged over 8 hr/day (NIOSH, 1998).

Unfortunately, a significant proportion of the population still receives acoustic exposures exceeding the 85 dBA limit. Furthermore, a potentially much larger proportion receives more moderate sound pressure level (SPL) exposures, in the 60–80 dBA range, for a longer duration. Chronic and moderate SPL exposures can come from noise sources such as transportation vehicles, construction equipment, and crowded restaurants. The potential impact of such exposures on auditory health is not well understood. Therefore, there is significant need to better understand the impact, especially in the absence of clinically significant hearing loss. Chronic exposures at moderate SPLs may be related to hearing disorders that affect central auditory processing. Recent human studies have observed auditory abnormalities in noise exposed subjects without hearing loss. For example, Stamper et al. studied subjects with a range of chronic noise exposure backgrounds, 67–83 dBA (Stamper and Johnson, 2015). They observed that the auditory brainstem response wave I amplitude decreased with noise exposure

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background, which could suggest a loss of afferent nerve terminals and cochlear nerve degeneration (Kujawa and Liberman, 2009). Kujala et al. studied subjects with noisy occupations (shipyards, daycare centers) and subjects with quiet occupations (Kujala et al., 2004). The noise levels in a shipyard ranged from 95 to 100 dBA (workers wore ear protection) and in a daycare center ranged from 67 to 75 dB. The investigators found that noisy occupation subjects had impaired speech-sound discrimination compared with quiet occupation subjects. Also, noisy occupation subjects were more easily distracted by irrelevant sounds. Brattico et al. studied subjects from the armchair industry and subjects with quiet occupations (Brattico et al., 2005). The background noise level in the armchair industry was 70–80 dB. The mismatch negativity results to deviant and speech sounds of armchair subjects differed from those of quiet occupation subjects, again suggesting impaired speech–sound discrimination.

Recent animal model studies of the auditory system have begun to investigate behavioral and neuronal changes following chronic exposures at moderate SPLs (Pienkowski and Eggermont, 2010a; Zhou and Merzenich, 2012). These studies have shown considerable functional changes in the auditory cortex. Our group has recently used blood oxygenation level dependent (BOLD) functional magnetic resonance imaging (fMRI) to investigate the impact of acoustic exposure in animal models (Cheung et al., 2012b; Lau et al., 2015). BOLD fMRI exploits the different magnetic properties of oxyhemoglobin and deoxyhemoglobin in blood to indirectly measure neuronal activity (Ogawa et al., 1990). It is well suited to investigating the impact of acoustic exposures because measurements are noninvasive, simultaneously investigate the entire central auditory system, and have relatively good spatial resolution. fMRI has been employed extensively to investigate the central auditory system in both human and animal subjects (Bach et al., 2013; Baumann et al., 2011; Boemio et al., 2005; Boumans et al., 2008; Brown et al., 2013; Maeder et al., 2001; Opitz et al., 2002; Patterson et al., 2002; Perrodin et al., 2011; Van Meir et al., 2005). Our group and others have extended fMRI methods to perform investigations of rodent models, including of the auditory system (Chan et al., 2010; Cheung et al., 2012a,b; Duong et al., 2000; Gao et al., 2014; Hyder et al., 2002; Lau et al., 2011a,b, 2013, 2015; Pawela et al., 2008; Peeters et al., 2001; Shih et al., 2009; Sicard et al., 2003; Silva and Koretsky, 2002; Weber et al., 2006; Yu et al., 2012; Zhang et al., 2013; Zhou et al., 2012).

In this study, we used BOLD fMRI with tonal acoustic stimulation to investigate the central auditory system of adult rats following chronic noise exposure at moderate SPLs. fMRI images were analyzed using conventional methods and custom methods tailored to this study.

Methods

Animal subjects

All aspects of this study were approved by the animal research ethics committees of the City University of Hong Kong and the University of Hong Kong. Ninety day old female Sprague Dawley rats (N = 13) were employed in this study. Female subjects were chosen to reduce size increase during the course of the study. Male rats increase in size considerably faster than females and large size increase can complicate functional magnetic resonance imaging (fMRI) experiments. One subject was used to acquire a template image for image analysis. The final blood oxygenation level dependent (BOLD) fMRI data set consisted of images from six control and six noise exposed subjects. In the remainder of this section, BOLD and fMRI will both refer to BOLD fMRI. All subjects were euthanized after imaging.

Acoustic noise exposure

Subjects entered the study at postnatal day 90 and received either acoustic noise exposure (exposed subjects) or sham exposure (control subjects) for 60 days. All subjects were housed in pairs in standard cages and received food and water ad libitum. Cages with control and exposed subjects were placed in separate rooms with similar layouts and with 12 hour light–dark cycles. The background noise level in the control cages (without subjects present) was less than 40 dB total SPL. Note that total SPL $= 10\log(\sum_{k=1}^{n} 10^{SPL(k)/10})$, where the summation is over all acoustic frequencies measured. Measurements were performed with a calibrated reference microphone that is sensitive up to 50 kHz (M50, Earthworks, USA) and a 192 kHz recorder (FR-2, Fostex, Japan).

Exposed subjects were exposed to a pulsed noise 24 hr/day for 60 days while control subjects received no additional exposure beyond background noise (sham exposure). The pulsed noise was pulsed at 5 Hz with 25% duty cycle (each pulse was 50 ms in duration). Each pulse was low-passed at 30 kHz and had total SPL = 65 dB inside the cage at the level of the subjects' ears. See Fig. 1 for the acoustic power spectrum of the exposure. This and similar exposures have been employed in fMRI, electrophysiological, and behavioral studies of the rat auditory system by our group and others (Lau et al., 2015; Zhou and Merzenich, 2012; Zhou et al., 2011). The noise was generated by broadband speakers (MF1, Tucker-Davis Technologies, USA) placed inside the cages but out of reach of the subjects. This acoustic exposure model does not elevate auditory brainstem response thresholds (Zhou and Merzenich, 2012), i.e., causes no conventional hearing loss, and the exposure SPL was well below the threshold for permanent hearing loss (NIOSH, 1998). Preliminary testing demonstrated that control and exposed subjects gained body weight at similar rates.

Preparation for imaging

After the 60 day acoustic exposure period, subjects were prepared for fMRI as in our earlier studies (Cheung et al., 2012a,b; Gao et al., 2014; Lau et al., 2013, 2015; Zhang et al., 2013). The subject was placed in an induction chamber filled with 3% isoflurane mixed with room air for 5 min. The subject was then transferred to the magnetic resonance imaging (MRI) compatible animal bed in the prone position. Warm water flowed through the animal bed. Anesthesia was maintained with 1% isoflurane delivered through a motion restricting nose cone and maintained for the remainder of the session. A tooth bar was used to further restrict head motion. The ear piece end of a custom sound delivery tube was inserted into the right ear. The left ear canal was blocked with an ear plug. The sound tube enabled delivery of acoustic stimulation from outside the high magnetic field area to the subject's ear drum and also reduced MRI scanner acoustic noise reaching the ear

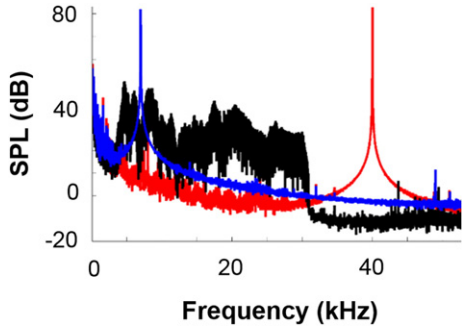

Fig. 1. Acoustic power spectra of the broadband exposure (black) and the tonal stimuli (7 kHz: blue, 40 kHz: red). Measurements were performed with a calibrated reference microphone that is sensitive up to 50 kHz (M50, Earthworks, USA) and a 192 kHz recorder (FR-2, Fostex, Japan). The exposure was measured at the level of the subject's ears inside its cage. The stimuli were measured at the ear piece end of the sound delivery tube that enters the ear canal. The exposure is approximately flat up to 30 kHz while the stimuli are sharply peaked at 7 and 40 kHz. The low frequency components of the stimuli are background noise in the magnetic resonance imaging scanner room. The total sound pressure level (SPL) of the exposure is 65 dB while the peak SPLs of the 7 and 40 kHz stimuli are 82 and 83 dB, respectively. Note that the threshold of normal human hearing at 1 kHz is used to set 0 dB SPL. Therefore, negative values of SPL, in units of dB, are physically possible.

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