

Effect of short-term high-intensity noise exposure on auditory physiology: a functional magnetic resonance imaging study

C Lau*

KEY MESSAGES

1. Short-duration, high-intensity noise exposure changes midbrain auditory processing.
2. Functional magnetic resonance imaging is sensitive to such changes, which are temporary and reversible in weeks and are likely related to more permanent changes in the inner ear and auditory nerve.

Hong Kong Med J 2018;24(Suppl 4):S46-7

HMRP project number: 11122581

C Lau

Department of Physics, City University of Hong Kong

* Principal applicant and corresponding author: condon.lau@cityu.edu.hk

Introduction

The present study investigated changes in the central auditory system following short-duration, high-intensity noise exposure. Normal female Sprague-Dawley rats were exposed to 100-dB sound pressure level noise for 15 minutes. The auditory system of each rat was scanned using functional magnetic resonance imaging (fMRI) to determine the blood-oxygen-level-dependent signal at three timepoints (before exposure and at days 7 and 14 after the exposure). fMRI was sensitive to the resulting functional changes to the central auditory system.

Results

Figure 1 shows the t-value maps at three timepoints (days 0, 7, and 14) with 12- and 20-kHz stimulation. For 12-kHz stimulation, t-values were highest at days 0 and 14 and lowest at day 7 in the inferior colliculus, whereas t-values were highest at day 0, slightly lower at day 14, and lowest at day 7 in the lateral lemniscus. For 20-kHz stimulation, t-values were highest at day 0 and lower at days 7 and 14 in the inferior colliculus and highest at day 0, slightly lower at day 14, and lowest at day 7 in the lateral lemniscus. Thus, there were transient changes in fMRI responses following short-duration, high-intensity noise exposure.

Tukey's honest significant difference test showed that signal amplitudes in the inferior colliculus were significantly greater at day 0 than day 7 for 12-kHz stimulation ($P<0.01$) and 20-kHz stimulation ($P<0.001$), and they were also significantly greater at day 14 than day 7 for 12-kHz and 20-kHz stimulation ($P<0.05$). Overall, fMRI responses were significantly reduced at day 7 and largely recovered at day 14.

Figure 2 shows the blood-oxygen-level-dependent signal amplitudes measured from the

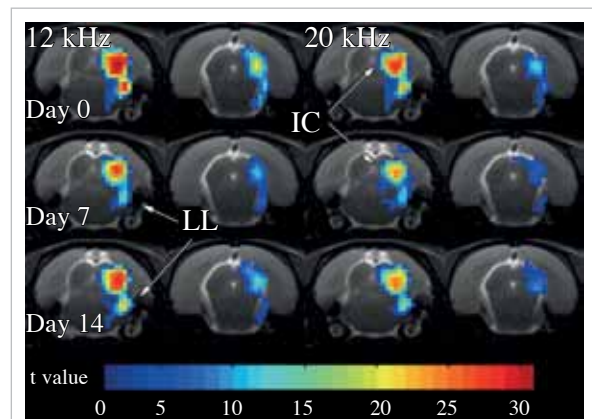


FIG 1. Functional magnetic resonance imaging showing the mean t-value maps immediately after 15 minutes of exposure to 100-dB sound pressure level noise (day 0), 7 days after exposure (day 7), and 14 days after exposure (day 14). Activation maps acquired with 12-kHz and 20-kHz noise stimulation are overlaid on anatomical images. Activation is mainly observed in the inferior colliculus (IC) and lateral lemniscus (LL) in the hemisphere contralateral to the stimulated left ear.

inferior colliculus at the three time-points. The orders of signal amplitudes from highest to lowest were those at days 0, 14, and 7 for 12-kHz stimulation ($P<0.05$), and those at days 0, 14, and 7 for 20-kHz stimulation ($P<0.01$). Noise exposure significantly reduced fMRI response in the inferior colliculus at day 7, but the response had largely recovered at day 14.

Discussion

Short-duration, high-intensity noise exposure results in transient reduction in fMRI responses in the auditory midbrain of rats. The reductions

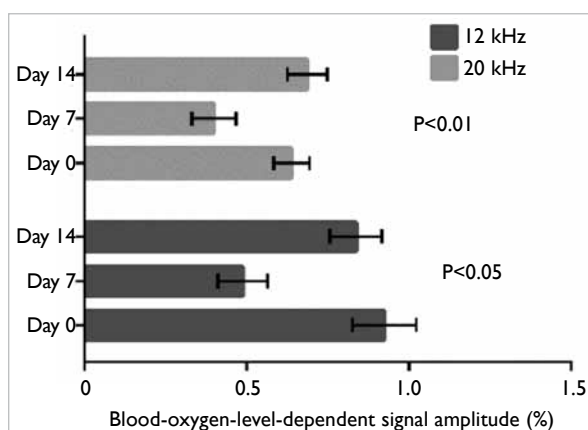


FIG 2. Blood-oxygen-level-dependent signal amplitude (%) in the inferior colliculus (n=10) before exposure and 7 and 14 days after exposure to 12-kHz and 20-kHz stimulation. The signal amplitude is significantly reduced at day 7 after exposure and largely recovers at day 14.

were observed 7 days after noise exposure. These reductions occurred even in the absence of auditory brainstem response threshold elevations, suggesting that the changes were of central origin. The midbrain responses had largely recovered to pre-exposure levels at day 14.

Exposure to high sound pressure level can cause permanent hearing loss and tinnitus. Hearing loss involves an increase in the minimum detectible sound pressure level threshold. Occupational health standards have documented exposure limits. Exposures that do not permanently shift thresholds may still contribute to difficulty comprehending speech and hyperacusis. Noisy environments contribute to sleep loss, cardiovascular disease, and stress. Noise exposure (or the absence thereof) also affects auditory physiology. For example, cochlear implantation alters auditory cortical function.^{1,2} Hearing loss at certain frequencies causes neurons normally sensitive to those frequencies to adjust to neighbouring frequencies.³⁻⁵ The results of this study provide insight on how central auditory processing is affected by exposure to high sound pressure level.

Exposure to high sound pressure level also leads to long-term loss of afferent nerve terminals on inner hair cells and degeneration of the cochlear nerve.⁶ In mice exposed to 100-dB sound pressure level noise for 2 hours, their auditory brainstem response and compound action potential thresholds returned to pre-exposure levels 2 weeks later.⁶ However, suprathreshold responses decreased at high frequencies. There was no loss of hair cells, but there was degeneration of presynaptic and

postsynaptic elements around inner hair cells in the high-frequency portion of the cochlea within 24 hours. At 1-year post-exposure, there was significant reduction in the spiral ganglion cell count in the high-frequency region. This reduction was not significant at 2 weeks post-exposure.

Conclusions

Central structures of the auditory system, such as the lateral lemniscus and inferior colliculus, and possibly even higher auditory structures, are related to degeneration of the cochlea and surrounding structures. This relationship is observed 1 to 2 weeks following noise exposure. fMRI can detect transient changes in inferior collicular responses long before significant spiral ganglion cell reduction in the cochlea has set in. The fMRI signal reduction may be related to decreased auditory brainstem suprathreshold responses.⁶ Suprathreshold decreases primarily occur at much higher frequencies (32 kHz) and are permanent. Therefore, the recovery observed may indicate the central auditory system's ability to compensate for the peripheral damage.

Acknowledgements

This study was supported by the Health and Medical Research Fund, Food and Health Bureau, Hong Kong SAR Government (#11122581), the Hong Kong General Research Fund (#661313), and start-up funding from the City University of Hong Kong and the Hong Kong University of Science and Technology.

References

- Pantev C, Dinnesen A, Ross B, Wollbrink A, Knief A. Dynamics of auditory plasticity after cochlear implantation: a longitudinal study. *Cereb Cortex* 2006;16:31-6.
- Seghier ML, Boex C, Lazeyras F, Sigrist A, Pelizzone M. fMRI evidence for activation of multiple cortical regions in the primary auditory cortex of deaf subjects users of multichannel cochlear implants. *Cereb Cortex* 2005;15:40-8.
- Izquierdo MA, Gutierrez-Conde PM, Merchan MA, Malmierca MS. Non-plastic reorganization of frequency coding in the inferior colliculus of the rat following noise-induced hearing loss. *Neuroscience* 2008;154:355-69.
- Kamke MR, Brown M, Irvine DR. Plasticity in the tonotopic organization of the medial geniculate body in adult cats following restricted unilateral cochlear lesions. *J Comp Neurol* 2003;459:355-67.
- Robertson D, Irvine DR. Plasticity of frequency organization in auditory-cortex of guinea pigs with partial unilateral deafness. *J Comp Neurol* 1989;282:456-71.
- Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. *J Neurosci* 2009;29:14077-85.