High pulse rate acoustic stimulation reduces fMRI responses in the auditory thalamus and cortex of chronic noise exposed rats

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Target Audience: Audiology, hearing, occupational health, environmental health, fMRI, and animal imaging.

Purpose: Exposure to loud sounds can lead to permanent hearing loss, i.e., the elevation of hearing thresholds. Long-term exposure at more moderate sound pressure levels (SPLs) (non-traumatic and within occupational safety limits) may not elevate thresholds, but could be detrimental to speech intelligibility by altering its spectral and temporal representation in the central auditory system. The evidence for this includes recent observations of electrophysiological and behavioral changes in animals following long-term and passive exposure at moderate SPLs[1,2]. To assess the potential effects of moderately loud noise on the entire central auditory system, we employ fMRI to study noise-exposed adult rats receiving two rates of acoustic stimulation.

Methods: Three months old Sprague Dawley rats (N = 24) were passively exposed for two months to pulsed noise at 65dB total SPL. Following the cessation of exposure, control (unexposed) and exposed subjects underwent fMRI with right ear acoustic stimulation pulsed at two different rates (5 and 10 Hz). The stimulation total SPL was 85dB. The fMRI methods employed in this study largely followed those in our earlier rat auditory fMRI studies[3-7]. The stimulation rates were presented in an interleaved block-design paradigm. SPM8 was employed to compute the activation maps. Region of interest analysis was performed on fMRI signals measured from the contralateral superior olivary complex (SOC), lateral lemniscus (LL), inferior colliculus (IC), medial geniculate body (MGB), and auditory cortex (AC) of the central auditory system. The amplitudes of signals from the different brain structures, subject groups, and stimulation rates were compared. Standard t-tests were employed for statistical comparisons.

Results and Discussion: Figure 1 shows that during 5 Hz acoustic stimulation, the primary structures in the central auditory system are active, including the contralateral (left) LL, IC, MGB, and AC. The ipsilateral (right) AC is also active. This is in good agreement with earlier fMRI studies of the rat auditory system[3,6,7]. The response in the AC of exposed subjects is reduced in both brain hemispheres compared to that of control subjects. Figure 2 shows that during 10 Hz stimulation, the responses in the contralateral MGB and both AC hemispheres of exposed subjects are significantly reduced compared to those in control subjects. Comparing Figs. 1 (5 Hz) and 2 (10 Hz), we see that acoustic exposure reduces responses in the MGB and AC, particularly at 10 Hz stimulation. These observations are backed up by the fMRI signal amplitudes shown in table 1. In the MGB, the signal amplitude from exposed subjects is significantly lower than that from controls during 10 Hz stimulation. During 5 Hz stimulation, no statistically significant differences are observed. In the AC, the signal amplitude from exposed subjects is also significantly lower than that from controls during 10 Hz stimulation. During 5 Hz stimulation, no statistically significant differences are observed. In summary, acoustic exposure reduces fMRI signal amplitudes in the MGB and AC. The reduction is greater during 10 than 5 Hz stimulation. The results of our study likely reflect homeostatic plasticity during long-term acoustic exposure[8]. In homeostatic plasticity, the brain attempts to stabilize neural firing rates within a prescribed long-term range (hours or days). Long-term acoustic exposure at moderate SPLs leads to a prolonged increase of auditory nerve and central auditory system activity. Over time, frequently active MGB and AC neurons compensate by reducing their synaptic gains. This would result in reduced fMRI signals during post-exposure acoustic stimulation.

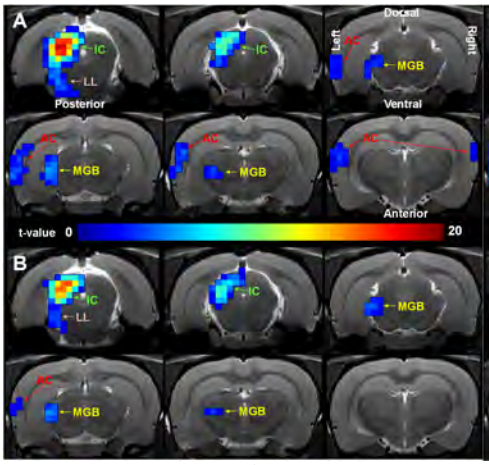

Figure 1: Activation maps of control (A) and exposed (B) subjects receiving 5 Hz acoustic stimulation.

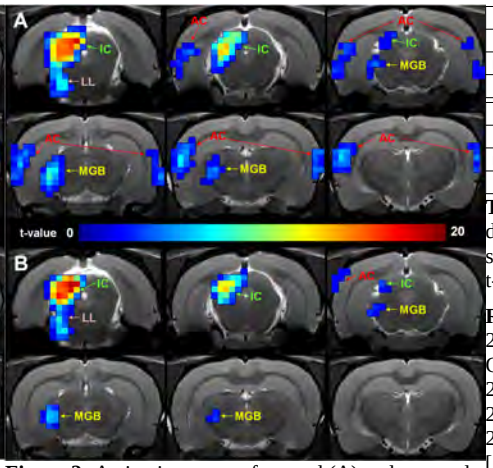

Figure 2: Activation maps of control (A) and exposed (B) subjects receiving 10 Hz acoustic stimulation.

5 Hz	SOC	LL	IC	MGB	AC
Controls	0.24 ± 0.16	0.23 ± 0.16	0.30 ± 0.11	0.09 ± 0.10	0.07 ± 0.11
Exposures	0.26 ± 0.14	0.15 ± 0.09	0.37 ± 0.15	0.05 ± 0.08	0.05 ± 0.07
p	-	-	-	-	-
10 Hz	**SOC**	**LL**	**IC**	**MGB**	**AC**
Controls	0.49 ± 0.13	0.45 ± 0.14	0.53 ± 0.17	0.14 ± 0.10	0.14 ± 0.06
Exposures	0.42 ± 0.17	0.37 ± 0.13	0.63 ± 0.18	0.06 ± 0.07	-0.02 ± 0.09
p	-	-	-	< 0.05	$< 10^{-4}$

Table 1: fMRI signal amplitudes (mean ± standard deviation) obtained during 5 and 10 Hz acoustic stimulation. P-values were obtained by performing the t-test between subject groups.

References: [1] Pienkowski et al. Hear Res 2010;261:30-5. [2] Zhou et al. Nature Comm;2012;3:843. [3] Cheung et al. Neuroimage 2012;60:1205-11. [4] Cheung et al. Neuroimage 2012;61:978-86. [5] Gao et al. Neuroimage 2014;91:220-7. [6] Lau et al. PLoS One 2013;8:e70706. [7] Zhang et al. Neuroimage 2013;65:119-26. [8] Gourévitch et al. Nat Rev Neurosci 2014;15:483-91.

Conclusion: fMRI with pulsed acoustic stimulation was performed on adult rats which were passively exposed for two months to broadband noise at moderate SPL. During 10 Hz (but not 5 Hz) stimulation, the contralateral MGB and AC of exposed subjects showed significantly lower signal amplitudes compared to controls. This indicates that noise exposure has a greater effect on the processing of higher pulse rate sounds. These findings are important for speech processing, which depends on accurate processing of sounds with a wide spectrum of pulse rates. This study also adds evidence that noise pollution is a significant health concern.