

Acoustic Interference on working memory in HIV patients

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INTRODUCTION: HIV infection may decrease neural capacity for attention processing and alter brain activation patterns¹⁻³. Acoustic noise is a major problem associated with the use of fMRI because it may also alter brain activation. Therefore, this study aims to evaluate the effect of fMRI-acoustic noise on brain activation during working memory in HIV patients; the sound pressure level of the fMRI scan was changed by 12 dB (four-fold) by taking advantage of the mechanical resonances of the z-gradient coil (see Fig1)⁴.

METHODS: Fifteen healthy men (age: 34±8 years, education: 16±3 years) and ten HIV-positive patients (AIDS dementia stage 1; CD4 <500/mm³; age: 37±8 years, education: 15±3 years) were recruited. Subjects performed the 0-, 1- and 2-back (sequential letters) verbal working memory tasks with block design (period=1min). The entire battery was performed twice under two different sound pressure levels: 92 dB for “quiet” (Q) and 104 dB for “Loud” (L), with random starting order (Q or L first) to minimize practice effects. After a brief training session outside of the scanner, subjects underwent functional MRI in a 4 T whole-body MRI scanner, using a single-shot gradient-echo EPI sequence (TE/TR 25/3000, 4 mm slice thickness, 1 mm gap, typically 33 coronal slices, 48x64 matrix size, 4.1x3.1 mm in-plane resolution, 84 time points, bandwidth = 200kHz (Q) and 219kHz (L)). Task performance and subject motion were monitored in real-time during fMRI, to assure accuracy > 80% and motion < 1mm-translations and < 1°-rotations. After motion correction, spatial normalization to the Talairach frame (3x3x3mm³ voxel size), and spatial smoothing (8mm-Gaussian), activation maps for Q and L sessions were calculated for each subject using SPM99. Individual activation maps were combined in random-effects analyses (repeated measures ANOVA; Figs 2 and 3). Region of interest (ROI) analyses (cubic, 27 voxels, 0.729 cc) were conducted at the cluster centers of brain activation to extract the average BOLD signal from these regions

RESULTS: The tasks produced a load-dependent bilateral activation in a working memory network that comprises posterior and anterior parietal lobes, the dorsolateral prefrontal cortex, cerebellum, and thalamus (see Fig2) as reported previously¹⁻². For control subjects, increased acoustic noise (12dB) produced increased brain activation in the left cerebellum, right occipital and prefrontal cortices (Fig. 3). For HIV patients, however, noise-related increases were observed in the right cerebellum, left occipital, and frontal cortices (Fig. 3). Differential BOLD signals due to scanner noise and working memory load correlated negatively in all ROIs in the working memory network.

CONCLUSIONS: Louder acoustic noise led to increased brain activation in both healthy and HIV subjects; this finding likely reflects the need for increased attention-network requirement to compensate for the interference of acoustic noise. The different noise-related activation pattern in HIV patients suggests that during louder scans, the HIV injured brain required additional usage of reserve neural networks (adjacent and contralateral regions) to compensate for the increased attention requirements.

REFERENCES:

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