

Longitudinal Changes of Cerebral Gray Matter Perfusion in Smoking and Non-smoking Abstinent Alcoholics

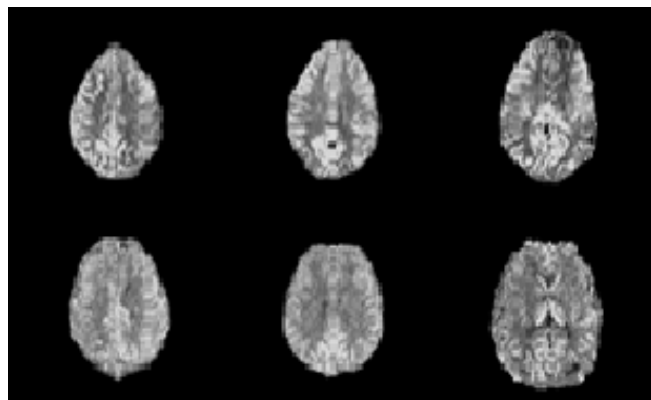
A. Mon¹, T. C. Durazzo¹, S. Gazdzinski², and D. J. Meyerhoff¹

¹Radiology, UCSF, San Francisco, CA, United States, ²CIND, San Francisco, CA, United States

Introduction: Previous PET, SPECT and MRI studies have demonstrated significantly decreased frontal and parietal gray matter perfusion in alcohol dependent individuals compared to controls [1-3]. Cross-sectional SPECT also suggested that these perfusion abnormalities are persistent in long-term abstinent alcoholics [4]. We previously demonstrated in a perfusion MRI study that concomitant cigarette smoking worsens cerebral blood perfusion in alcoholics (3). In the current report, we compared changes in cerebral perfusion in abstinent smoking and non-smoking alcoholics over approximately one month of abstinence from alcohol. We hypothesized that cerebral gray matter perfusion in non-smoking alcoholics will significantly increase with abstinence, whereas smoking will hinder perfusion recovery in smoking alcoholics.

Methods: We measured cerebral perfusion in 40 alcoholics (ALC); 19 were non-smokers (nsALC: 2 females, 17 males), with age 49 ± 8 years and 21 smokers (sALC: all males with age 48 ± 7 years). We used a pulsed arterial spin labeling single-shot EPI sequence at one week of abstinence from alcohol and repeated the experiments after one month of sustained abstinence. The perfusion sequence yielded five 8 mm thick slices (with a 2 mm gap between adjacent slices), placed above the circle of Willis and oriented 5° off the orbital meatal line. Imaging parameters were TR/TE=2500/15 ms, 1500 ms time between labeling pulse and the excitation pulse and an in-plane resolution of $2.3 \times 2.3 \text{ mm}^2$. 3D high resolution T1-weighted images were also acquired and segmented into probabilistic maps of gray matter (GM), white matter (WM) and cerebro-spinal fluid (CSF) in the frontal and parietal lobes. These maps were co-registered to the perfusion images, thus allowing for calculation of mean perfusion in frontal and parietal GM. Regional GM perfusion were calculated from voxels containing at least 80% GM tissue. Data analysis was performed using SPSS 15.

Results: The figure shows characteristic perfusion images of one sALC (bottom) and one age-matched healthy volunteer (top). The poorer gray matter (GM)-to-white matter (WM) contrast in the sALC images suggests lower GM perfusion in the sALC. A 2 (group) x 2 (time point) repeated measures ANOVA showed a significant group x time-point interaction for parietal GM (pGM) perfusion ($p = 0.05$) and a trend for frontal GM (fGM) ($p = 0.08$). Follow-up t-tests indicated nsALC demonstrated significant increases in pGM ($p = 0.02$; +12%) and fGM ($p = 0.05$; +11%) perfusion from week one to week five, while sALC showed no significant changes in pGM (-1 %) and fGM (-2.0%). Main effects for group and time point were not significant for either region. Regression analyses showed no association between measures of drinking/smoking severity on change of perfusion. In the combined group (sALC + nsALC) increases of fGM perfusion related positively to increases of aural working memory ($r = 0.35$; $p = 0.04$).



Discussion: This preliminary study provides evidence that over one month of abstinence from alcohol, cerebral perfusion shows significant recovery in non-smoking alcoholics, whereas there are no significant changes in smoking alcoholics. Our findings also indicate that improved frontal gray matter perfusion is positively correlated with increases of aural working memory. These results are convergent with our longitudinal MRSI findings where nsALC exhibited significantly greater NAA recovery than sALC in the frontal lobes and Cho in the frontal and parietal lobes over one month of abstinence from alcohol [5]. Additional studies that compare changes in cerebral perfusion in smoking and non-smoking alcoholics over extended periods of abstinence are warranted.

Acknowledgement: This research is supported by NIH AA10788.

References:

1. B. Erbas et al, *Clinical Nuclear Medicine*: 1992: 17: 123-127
2. B. Melgaard et al, *Acta Neurol Scand* 1990: 82: 87-93.
3. S. Gazdzinski et al, *Clinical and Experimental Research* 2006: vol. 30, No. 6 947-957.
4. R. M. Dupont et al, *Psychiatry Research: Neuroimaging* 1996: 67: 99-111.
5. T. Durazzo et al., *Alc Clin Exp Res* 2006: 30: 593-51.