Further evidence for a significant influence of alcohol consumption on frontal choline measures in social drinkers

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Introduction

In our previous MSSI study of the cerebellum and the frontal lobe in recently detoxified alcoholics we found a significant positive correlation of frontal WM choline containing compounds (Ch) (left and right) and anterior cingulate Ch with alcohol consumption over the last 90 days (timeline followback, tlfb) in the group of healthy controls but not in the alcoholic patients(1). The aim of this work was to corroborate these significant correlations of Ch with the amount of alcohol consumption in the frontal lobe in another control group of light social drinkers with a more widespread alcohol consumption than the previous controls and a different MRSI method yielding more S/N than the previously used MSSI sequence.

Methods

All MRS studies were performed on a 1.5 T Siemens Vision system. For localization, 2D FLASH images in coronal, sagittal, and oblique transverse orientation were acquired. The transverse images were angulated parallel to the border of the corpus callosum posterior to its knee. A 2D MRSI sequence with PRESS volume selection was used with the volume angulated parallel to the transverse images above the corpus callosum enclosing parts of the anterior cingulate gyrus, medial prefrontal and medial frontal gray matter. A MRSI field of view (FOV) of 210×210 mm was used with circular k-space sampling equivalent to a maximum of 24×24 phase encoding steps. Other measurement parameters included TR = 1.5 s and TE = 135 ms, resulting in a measurement time of 11 minutes. Postprocessing of the MRSI data was done with an automated spectral fitting program (2). With use of an automated image co-registration and segmentation program (3) all MRSI voxels were corrected for the CSF content as well as the individual point spread function. Data of 15 healthy controls (9 male, 6 female, aged 26-51, mean 37.7 SD 7.9 years) are presented. All subjects were non-smokers. We looked at two time intervals of drinking history: the last three months and the last 30 days. Subjects consumed between 0 and 2069 g alcohol over the last three months (mean 931 SD 578 g alcohol). If a drink is defined by 12 g alcohol the mean consumption per day in the 90 and 30 days previous to the MRSI examination was 0.85 and 0.86 drinks, respectively.

Results

We found a significant positive correlation of Ch in frontal WM with the tlfb for alcohol (divided by the subjects body weight) over the last three months (r = 0.52, p = 0.05) and the last 30 days (r = 0.53 p =0.04), respectively . The ratio of Ch/Cr reflects the same correlation and also reaches significance for the tlfb of the last 30 days (r = 0.58 p = 0.03) (see Figures). A trend in the same direction is found for Ch values from the anterior cingulate GM and medial frontal GM. We also see a slight trend for a decreasing signal of creatine and phosphocreatine (Cr) but with less slope than the Ch increase.

Discussion

These results corroborate our previous finding of a positive correlation of alcohol consumption and frontal Ch values (1). They further support the assumption that Ch changes might reflect an adaptive mechanism of the brain to alcohol consumption. They are in good agreement with the spectroscopic finding of an initial Ch increase in chronically alcohol exposed rats that is followed by a significant Ch decrease with increasing duration of alcohol exposure by Lee et al. (4). The authors interpret their findings with an initially increased turnover of phosphatidylcholine and other phospholipids reflecting an adaptive mechanism of the brain. Our and other groups have reported significantly decreased Ch signals in detoxifying alcoholic patients (5-7). Therefore, the question remains whether a decreased Ch signal is due to the increased amount and duration of alcohol consumption or a consequence of abstinence and detoxification. The decreased Ch is found in studies reporting on detoxified alcoholics, whereas the study by Meyerhoff et al. (8) did not find frontal Ch differences between light and heavy drinkers but found an increased Ch signal in the thalamus of alcohol depended subjects compared to light drinkers.

The main conclusion from the repeated finding of a positive correlation of alcohol consumption and frontal Ch signals is that monitoring for alcohol consumption (and potentially nicotine and other drugs) is mandatory in MRS studies where pathology depended Ch changes are hypothesized.

References

(1) Ende G, Walter S et al., Proc. Intl. Soc. Mag. Reson. Med. Kyoto, 291 (2004).
(2) Soher BJ, Young K, et al., Magn Reson Med 40:822-31 (1998).
(3) Weber-Fahr W, Ende G et al., Neuroimage 16:49-60 (2002).
(4) Lee H, Holburn GH et al., J. Magn. Reson. Imaging 18:147-151 (2003).
(5) Ende G, Walter S et al., Proc. Intl. Soc. Mag. Reson. Med. Toronto, 439 (2003).
(6) Parks MH, Dawant BM et al., Alcohol Clin. Exp. Res. 26:1368-80 (2002).
(7) Bendszus M, Weijers HG et al., Am. J. Neuroradiol. 22:1926-32 (2001).
(8) Meyerhoff DJ, Blumenfeld R et al., Alcohol Clin. Exp. Res. 28:650-61(2004).