

## Assessment of neurochemical alterations in rats exposed to long-term alcohol treatment

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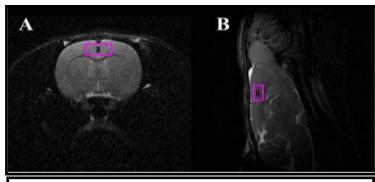


Fig. 1. T1-weighted images (A: axial, B: sagittal) of the rat brain. The rectangular boxes indicate the position of VOI (4 x 1.6 x 3 mm).

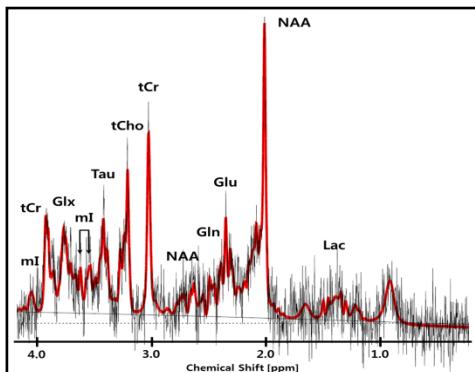


Fig. 2. Representative *in vivo* 4.7 T <sup>1</sup>H MRS spectrum (200 MHz) of the ethanol treated group, in the region of the frontal cortex. The original spectrum and the fitted LCModel spectrum are represented by the black and red line, respectively.

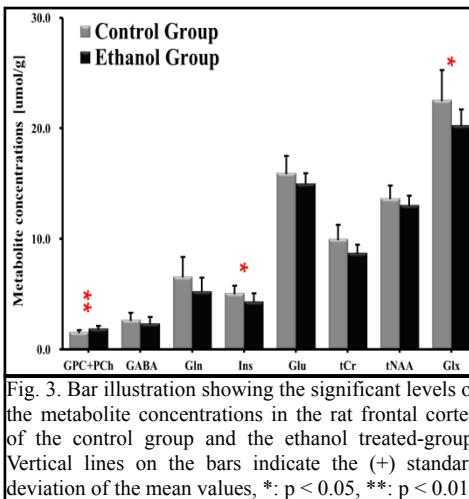


Fig. 3. Bar illustration showing the significant levels of the metabolite concentrations in the rat frontal cortex of the control group and the ethanol treated-group. Vertical lines on the bars indicate the (+) standard deviation of the mean values, \*: p < 0.05, \*\*: p < 0.01

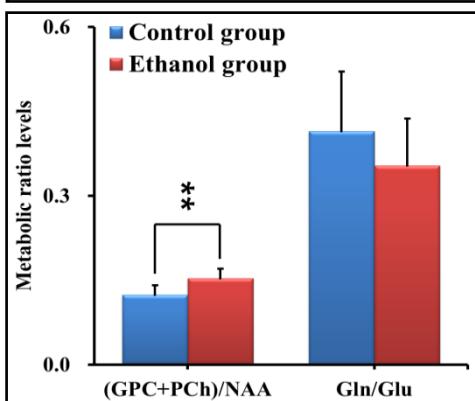


Fig. 4. Bar illustration shows that the (GPC+PCh)/NAA and Gln/Glu ratio levels were significant different between the control group and the ethanol group. Vertical lines on the bars indicate the (+) standard deviation of the mean values, \*\*: p < 0.01

mechanism in the frontal cortex of the long-term alcohol exposure adolescent rats [5]. Therefore, increased GPC+PCh concentrations and (GPC+PCh)/NAA ratio levels of the frontal cortex might be utilized as the key marker in chronic adolescent alcohol intoxication.

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