Title: Central Nervous System Oxidative Stress interplay with inflammation in a rat model of Type C Hepatic Encephalopathy – brothers in arms?

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Content:

**BACKGROUND**

- Although oxidative-stress (OS) and neuroinflammation play a role in type C hepatic encephalopathy (C HE), their involvement and synergistic action is not well understood.
- Under normal conditions the physiological levels of intracellular reactive oxygen species (ROS) are controlled by the counteracting antioxidant response to maintain redox homeostasis.

**AIMS**

- Longitudinal tracking of CNS OS in a rat model of type C HE using in-vivo-1H-MRS and ex-vivo-ESR spin-probing combined with UV-Vis spectroscopy and histological assessments (IHC).

**METHODS**

- **In-vivo-1H-MRS** indirect OS detection – ascorbate and glutathione concentrations.
- **Ex-vivo ESR** direct and quantitative detection of OS (O2−) with CMH spin-probe.
- **Histology**: BDL rats at 4 and 8-weeks post BDL (n=3 per group) and SHAM rats (n=3).
- **NBT**: histo-enzymatic technique for ROS visualization.

**CONCLUSION**

- For the first time, longitudinal presence of CNS OS together with inflammation in a rat model of type C HE.
- OS increase is not due the declined antioxidants activity but rather a response to ROS increase.
- OS is one of the major pathways driving neurodegeneration. Therefore, CNS OS, together with inflammation, may strongly contribute to HE pathogenesis.

**References**