REAL-TIME BIOSENSING OF GLUCOSE, LACTATE AND GLUTAMATE EXTRACELLULAR LEVELS IN NUCLEUS ACCUMBENS OF FREELY MOVING RATS AFTER SYSTEMIC ETHANOL ADMINISTRATION

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Introduction: It has been widely assessed that people who have been drinking high amounts of alcohol for long time, have high probability to develop grave and persistent changes in the brain. In fact, it has been amply demonstrated how ethanol (EtOH) can influence some neurotransmitter systems, including opioids, acetylcholine, dopamine but also glutamate as well as brain energetics. These phenomena help to determine the ethanol-evoked repercussions at brain level, such as behavior, cognition, acute rewarding, but also neuroadaptations from abuse and addiction. In previous papers, the variation of the above-mentioned neurochemicals have been monitored by means of "no-real time" techniques, as microdialysis coupled with HPLC or by means of the 2-deoxy-D-[1-^{14}C] glucose method, some of which also requireing preventive treatment of the sample. The aim of the present study was to evaluate the *in vivo* real-time variations of glucose (Glu), lactate (Lac) and glutamate (Glut) by means of amperometric biosensors implanted in the Nucleus Accumbens (NAc), as this brain region resulted involved in ethanol effects due to the ethanol intake.

Material and methods: In the present study amperometric biosensors were manufactured at Day 0 and calibrated by exposing them to increasing concentration of glucose (ranging from 0 to 180 mM), and lactate and glutamate (ranging from 0 to 100 mM), in order to evaluate their kinetic and analytical performances. All biosensors were evaluated also on the shielding against ascorbic acid (AA), which represents the most interfering species in the extracellular cerebral spaces. On the same Day, after calibrations, the most performing biosensors, in terms of AA shielding and analyte sensitivity, were then implanted in right and left NAc. The next day, after animal recovery, a constant potential of +700 mV was applied to the biosensor 1g/Kg i.p. of EtOH was performed.

Results and discussion: Following EtOH administration, an rising in glucose concentrations were recorded, while lactate and glutamate, after an initial short increase, didn't demonstrate any valuable change if compared with baseline values. As previously demonstrated, the increasing of glucose concentrations would be charged to a decrease in the glucose utilization in the NAc. Also glutamate and lactate results were in agreement with those published in previous studies, where EtOH administration was unable to modify glutamate or lactate concentrations, even if evaluated with different analytical methods.

Conclusions: These preliminary results confirm the switch between neuronal energy substrates following the ethanol administration and can be useful for a better comprehension of cerebral bioenergetics due to ethanol intoxication and also help in developing potential therapeutic strategies aimed at restoring normal neuronal glucose use.