RAPID COMMUNICATION

ACUTE ETHANOL INCREASES TAURINE BUT NEITHER GLUTAMATE NOR GABA IN THE NUCLEUS ACCUMBENS OF MALE RATS: A MICRODIALYSIS STUDY

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Abstract — The effects of acute intraperitoneal administration of ethanol (1–3 g/kg body wt) on the extracellular concentrations of glutamate (GLU), taurine (TAU) and γ-aminobutyric acid (GABA) in rat brain nucleus accumbens were studied by the microdialysis technique coupled to HPLC-EC detection. Levels of GLU and GABA were not affected by ethanol, whereas those of TAU were increased by doses of the drug of 2–3 g/kg.

INTRODUCTION

The major advantage of the technique of intracerebral microdialysis is that it allows concurrent evaluation of the regional intracerebral neurochemical changes over time, using unanaesthetized animals maintained under in vivo conditions.

After stereotaxic insertion of a microprobe on which the dialysis membrane is mounted, the neurochemical compounds bathing in the intracerebral extracellular fluid are made to diffuse across the membrane. Among the various neurotransmitters affected by alcohol, we chose to study the effect of alcohol on glutamate, an excitatory amino acid, and taurine and γ-aminobutyric acid, GABA, two inhibitory amino acids, in the nucleus accumbens of male adult Wistar rats. This nucleus was chosen because it is an actual interface between the cortical areas which control wakefulness and the limbic areas which regulate motivations and emotions. As such, this nucleus is increasingly involved in the origin and maintenance of drug misuse.

MATERIALS AND METHODS

The microdialysis probe, equipped with a 3 mm long dialysis membrane, was inserted into a cannula and directed to the nucleus accumbens (1.2 mm anterioposteriorly; 1.2 mm laterally and −5.7 mm ventrally to bregma). The probe was continuously perfused with Ringer’s solution (189 mM NaCl; 3.9 mM KCl; 3.4 mM CaCl2; pH 7.2) using a micro-injection pump delivering 1 μl/min. Every 20 min, 20 μl dialysate was collected after addition of 10 μl 0.1 mM EDTA.

After 120 min, the animals received an injection of either 15% ethanol (1, 2 or 3 g/kg) or 0.9% saline. The derivatization reagent was prepared by dissolving 27 mg O-phthalaldehyde (OPA) in 1 ml ethanol (Donzanti and Yamamoto, 1988). Then, 5 μl β-mercaptoethanol (BME) and 9 ml of 0.1 M sodium tetraborate (pH 9.3) were added. The work solution was prepared each day 24 hr before use by diluting 1 ml of the above solution in 3 ml 0.1 M sodium tetraborate. This derivatization reagent was then used by mixing 25 μl of dialysate with 12.5 μl OPA/BME for 2 min in complete darkness before
injection into the column.

The HPLC system consisted of a pump delivering 1 ml/min at a pressure of 5300 psi, a guard cell, a column (ODS 3 µm; 100 × 3.2 mm, reversed phase-II), an analytical cell, a 0.2 µm carbon filter upstream to the guard cell and the analytical cell, and an electrochemical detector consisting of two electrodes (Coulochem Detector ESA). The work potentials were: −0.4 V (detector 1), +0.6 V (detector 2) and +0.4 (guard cell).

Fig. 1. Extracellular level of glutamate (GLU), taurine (TAU) and γ-aminobutyric acid (GABA) after ip injection of alcohol (1, 2 and 3 g/kg) or saline at 120 min after inserting the microdialysis probe into the nucleus accumbens. Results are presented as percentage of baseline level, which represents the mean of the dialysate concentrations at time 80, 100 and 120 min. *Statistical differences using Anova I followed by Neuman–Keuls (P < 0.05).
The mobile phase consisted of 7.520 g/l NaH₂PO₄, 28% HPLC methanol and 720 ml Milli-Q purified water. The mobile phase was adjusted to pH 5.7 with 6 N NaOH and filtered through 0.2 μm nylon filters.

RESULTS AND DISCUSSION

The variation in extracellular concentrations of neuro-exciting and neuro-inhibiting amino acids was not significantly affected by ethanol injection with the exception of taurine. Taurine levels increased significantly within 40 min following intraperitoneal injection of 2 and 3 g/kg ethanol.

The presence of a sulfonic group confers on taurine the capacity of increasing the alcohol metabolism (Iida and Hikichi, 1976; Sprince, 1988). Taurine would thus enhance the oxidation of the first alcohol metabolite, acetaldehyde, by activating its degradation enzyme, aldehyde dehydrogenase (Aragon et al., 1992; Watanabe et al., 1985).

An increased taurine level could be considered as a physiological adaptation to acute alcohol inducing a fluidification of the membrane structure by stabilizing the plasmic membrane (Sturman et al., 1978) and by maintaining the brain osmotic regulation (Baxter et al., 1986) when alcohol induced a loss of water cells produced by the inhibition of antidiuretic hormone. The question of whether the taurine measured by HPCC-EC represents neuronal or glial taurine remains open.

REFERENCES


