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Ethanol Influences on Rat's Brain Electrical Activity

T.M. El-Sayed

Biophysics Branch, Physics Department,
Faculty of Science (Boys), Al Azhar University, Cairo, Egypt

Abstract: The goal of the current study was to estimate the influences of a moderate dose of ethanol on the electrical activity of the brains of adult rats. 120 seconds of EEG were recorded post 1.5 g/kg of ethanol administration. The righting reflex was assessed to evaluate the sedative consequences of ethanol for each rat. The present findings demonstrated that ethanol raised power in the 1–2 Hz band and reduced the power in the 32–50 Hz band in the rat's visual cortical region.In the frontal cortex of the rats, alcohol reduced the power in the 16–32 Hz frequency band. Ethanol differentially raised power in two slow-wave frequency bands (2–4 Hz and 4–6 Hz) in the visual cortex of the rats. Ethanol-administrated rats revealed shorter sleeping time than control rats. Our findings present additional indications about the influences of ethanol administration the brain electrical activity.

Key words: Ethanol · EEG · Acute Moderate Dose · Righting Reflex

INTRODUCTION

Possible effects of alcohol ingestion on mood-alternation, behavioral effects and neural activity are of great concerns. A number of studies have examined the influences of alcohol on brain electrical activity using electroencephalography [1]. Most of recent studies have declared that, there is an increase in alpha frequencies (10 Hz) as a result of administration of moderate dose of alcohol [2]. Other studies suggested that; I addition to the increases in alpha frequencies, a detectable increases in beta activity as a consequence of alcohol administration [3].

Electroencephalograph (EEG) power, a measure of the amplitude of the EEG as a function of frequency (microvolts squared per Hz), has been shown to be altered due to alcoholism [4]. Many studies reported that; EEG is considered as useful way in evaluating the effects of alcohol administration in humans as well as in rats [5 - 7]. Changes within EEG power (i.e. in particular frequency bands) appear to be a good tool to determine both acute and chronic effects of alcohol. Most of the recent studies categorized the continuous rhythmic EEG activities into Delta (1–4 Hz), Theta (4–8 Hz), Alpha (8–16 Hz), Beta (16–32 Hz) and Gamma (32–50 Hz) frequencies[8]. Other studies used EEG spectral analysis as a good tool to evaluate the effects of alcohol on the functions of the central nervous system [8].

Some studies have shown that; alcohol-induced changes in the stability of the EEG in adult rats previously exposed to alcohol as well as sleep disturbances[9].

The coefficient of variation (CV) has been used as a good measure of EEG stability and is also used as an indicator in the correlations of drug effects and drug-induced behavior [10].

The effects of acute alcohol on EEG responses and its linkage to sedative effects of acute alcohol are not well understood [10].

The goal of the current study was to evaluate further the influences of a single moderate dose (acute dose) of alcohol on cortical EEG and behavioral sedation in adult freely moving rats.

MATERIALS AND METHODS

Male adult albino rats (n=25) Rattus norvegicus (above 150 g in weight) were used in the present study. Animals were picked up from the experimental animal

Corresponding Author: Tamer M. El-Sayed, Biophysics Branch, Department of Physics, Faculty of Science (Boys), Al-Azhar University, Cairo, Egypt. E-mail: tamer_mmahmoud@hotmail.com.

breeding station at Helwan, cairo, Egypt. Animals were housed as a single rat/cage in a standard Plexiglas cage [25 (w) × 20 (h) × 45 cm]with light metal mesh as covering part. Except during the EEG recordings there was free access to balanced standard maintenance of food and tap water.

Animals are then divided into two groups called control group (n=10) and alcohol group (n=15).

Under deep sodium pentobarbital anesthesia (50 mg/kg, intra-peritoneal), rats were implanted bilaterally with epidural stainless steel screw electrodes over the frontal (B: +2 mm, L: 2 mm) and visual cortex (B: 2 mm, L: 3 mm) for EEG chronic recordings. A fifth electrode (reference electrode) was placed in the contra lateral crest of the skull at the interaural line (asterisk in Fig. 1). Electrodes were fixed to the skull via dental acrylic cement.

One-week as a recovery and a training period was provided before the effects of alcohol were studied. This method was adopted after Skinner, J. E [11].

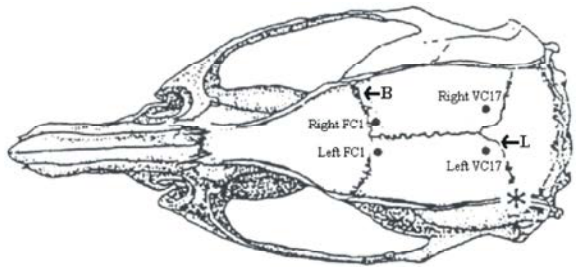

Fig. 1: Locations of the EEG recording electrodes.

Simultaneous EEG records from all 4 electrodes were carried out one week post surgery by means of monopolar 24-channel EEG polygraph (Braintronics). Before the beginning of recording the EEG signals, the animal was transferred into a Faraday cage (30 x 30 x 25 cm). EEG records were stored on computer disk with sampling frequency of 512 sample/s by means of a program called EEG Translation.

EEG free artifact signals (120-s) were obtained from the 4 epidural electrodes; such that from the frontal cortex area 1 (Right FC1 and Left FC1) and the visual cortex area 17 (Right VC17 and Left VC17).

Fast Forier Transformation (FFT) was carried out to give power spectrum density for the 120-s recorded EEG. The obtained spectra were then divided into 7 frequency bands: 1-2 Hz, 2-4 Hz, 4-6 Hz, 6-8 Hz, 8-16 Hz, 16-32 Hz and 32.-50 Hz.

Mean spectral power, variability and coefficient of variation (CV) = standard deviation power/ mean power) in each band of the EEG were calculated [12]. A detailed overview on the electroencephalographic recordings could be seen at El-Gohary et al. [13].

Alcohol (1.5 g/kg) was administrated intraperitoneally (i.p.) into adult (n=15) rats. After administration, rats were placed into their cages and the time to loss of the righting reflex (LORR) was recorded. The duration of LORR was also recorded. Time to LORR was defined as the time post-injection when the rat could no longer right itself onto all 4 paws. Time to regain of righting reflex (RORR) was defined as the time post-injection when the rat could right itself onto all 4 paws two times. Total alcohol-induced sleep time was calculated by subtracting the time to LORR from the time to RORR [14].

The mean of each band and the total spectral power was computed for each rat during 120-s period selected from artifacts free stages in both control and alcohol conditions. These values were tested using paired student's t-test for individual band mean comparisons. paired student's t-test was followed by computing the standard deviation (SD), standard error mean (SEM) and 2-way paired analysis of variance (ANOVA) for repeated measures.

RESULTS

Visual monitoring of the freely moving rats before alcohol treatment revealed normal exploratory and grooming behaviors in both control and alcohol groups. Injection of the 1.5 g/kg alcohol dose reduced motor activity during handling, as well as falling to one side of the animal bodies.

Frontal and Visual Cortical EEG Power – Alcohol treated rats showed significant reduction in high frequency 16–32 and 32–50 Hz bands. (Fig. 2a).

Similar to the motor cortex, the visual cortical power showed significant increases in mean power in multiple bands (i.e, 1–2, 2–4, 4–6 Hz bands) and decreases in the 16–32 and 32–50 Hz bands (Fig. 2b).

Frontal and visual EEG Variability - Analysis of the power CV (cortical EEG variability) in the frontal EEG demonstrated significant increases in power CV through the 32–50 Hz band (Fig. 3a).

Similar to frontal cortex, alcohol produced increases in power CV in the 32–50 Hz band over the visual cortex (Fig. 3b).

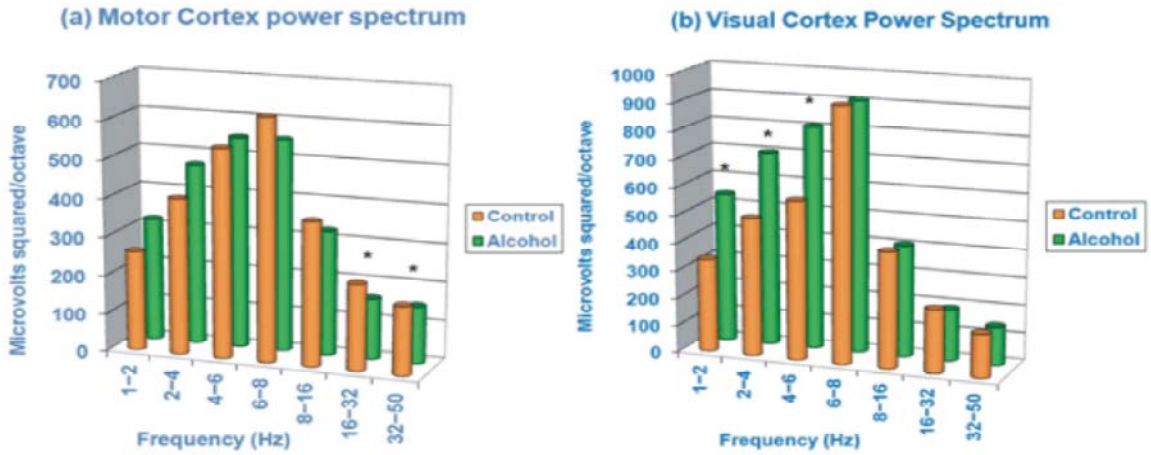

Fig. 2(a&b): Mean EEG power in (a) frontal and (b) visual cortices of control and alcohol treated rats after injection of 1.5 g/kg of alcohol. Data are the mean ± standard error of the mean. *p<0.01 indicates significant different from vehicle.

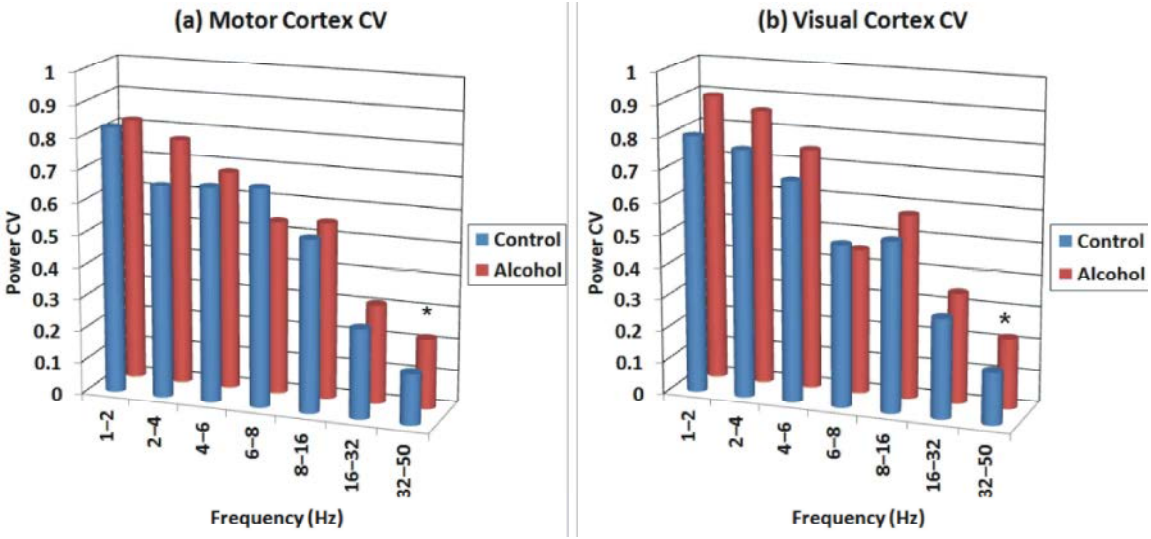

Fig. 3(a&b): EEG power CV in (a) frontal and (b) visual cortices of control and alcohol treated rats. Data are the mean ± standard error of the mean. *p<0.01 indicates significant different from vehicle.

Table 1: Values of mean (S.E.M.) time to loss of righting reflex (LORR), to regain righting reflex (RORR) and total sleeping time.

	Time (min)		
	LORR	RORR	Tslp
Control	-	-	-
Alcohol	5	161	154

Loss of Righting Reflex - Significant elevation was detected in the latency of the onset of the loss of righting reflex (LORR) between both alcohol and control group. Also there was significant elevation in the duration of the LORR and total sleeping time (Tslp) (Table 1).

DISCUSSIONS

Administration of moderate dose of ethanol has multiple effects on the brain electrical activity, EEG power, as well as behavior of freely moving adult rats.

Behavioral sedation following alcohol administration was assessed in the present study using right reflex. Inconsistent with previous behavioral studies [15], it was found that alcohol-treated rats regained their voluntary motor activity after delayed period [16]. The present findings shows that, ethanol administration was associated with a decreased values of the fast bands [17]. Also the present results show that, no significant

differences were found in the low frequencies of the motor cortex [18].

The current study showed that administration of alcohol raised the power in all of the slow wave frequency bands (1-6 Hz) in the visual cortex.

Also the present results propose that there were decrement in the ã frequency band. Many studies mentioned that, the ã frequency band (32–50 Hz) has been associated with the integration of sensory and cognitive processes [19]. Previous studies revealed that lower evoked ã band activity could be a marker helps in detection of alcoholism [20].

Moderate alcohol dose produced considerable inhibition on visual cortical power in â frequency band. The increased sensitivity of â frequency in adult rats is consistent with previous behavioral studies [21, 22].

In the current study we detected both higher frontal and visual cortical power in a broad range of EEG frequencies of the used adult rats. Higher EEG power could theoretically be due to the age-related synaptic pruning process that occurs from adolescence to early adulthood [23, 24].

Our data also revealed that cortical EEG variability is higher in control rats than in alcohol-treated rats across the slow-wave frequency bands in both frontal and visual cortices. We found that following alcohol administration cortical EEG variability was reduced in the 4–8 Hz bands in the frontal and 2–8 Hz bands in visual cortices of control rats. Alcohol-induced action of cortical EEG variability in adult rats could be due to mechanisms in postsynaptic excitatory and inhibitory neural systems[25 - 27].

Cortical EEG variability provides a good measurement for behavioral state of the animal and it was shown that the decreased cortical EEG variability could serve as an index of decreased sedation after administration of alcohol [9]. Some studies have shown that alcohol exposure caused long-term disruption of spatial memory and paradoxical sleep[27, 28]. Slawecki[7] reported that enhanced intoxication scores were not observed in alcohol-exposed rats after acute moderate dose of alcohol exposure. As the results, the EEG variability could be not just an index of decreased sedation, it is also could be a marker for alcohol's effects on cognition. So that, the administration of alcohol moderate dose to rats caused changes in the electrical activities of brain (EEG) and may offer an additional evidence in differential responses to alcohol.

CONCLUSION

The current study suggests that cortical EEG power (2-6 Hz) raised post alcohol administration. These findings may sustain the assumption that alcohol's sedative effects are connected with an increase in visual EEG power in the slow-wave frequency bands in adult rats.. Our findings may offer an additional evidence in differential responses to alcohol.

Abbreviations:

EEG: Electroencephalogram
CV: The coefficient of variation
FC: Frontal cortex
VC: Visual cortex
LORR: Loss of the righting reflex
RORR: Regain of the righting reflex

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