

Electrolytic Evaluation of Nicotine Treated Rats

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Abstract

This work assessed the effects of electrolyte in rats treated with nicotine diet. A total of thirty (30) rats weighing 80-190g were randomly assigned into two groups of fifteen (15) each. Group A was fed with normal rat chow and acted as the control group while group B received 0.5ml of concentrated nicotine dissolved in 200ml of distilled water mixed with 100g of animal feed. They were fed for 28 days and had free access to drinking water. The results revealed that Sodium (Na^+), Potassium (K^+) and Chloride (Cl^-) were significantly higher ($P < 0.001$) when compared with the control group, while the bio-carbonate (HCO_3^-) was lower. This result is suggestive that nicotine consumption is capable of changing the ionic composition of the body.

Key words: Wistar rat, Nicotine, Electrolytic

Introduction

Nicotine is highly addictive. People who regularly consume nicotine and then suddenly stop experience withdrawal symptoms, which may include cravings, a sense of emptiness, anxiety, depression, moodiness, irritability, and inattentiveness. The American Heart Association says that nicotine (from smoking tobacco) is one of the hardest substances to quit - at least as hard as heroin. According to a report published by the Massachusetts Dept of Public Health, tobacco companies steadily increased the nicotine content of their cigarettes from 1998 to 2004, by approximately 10%. The higher the nicotine dose in each cigarette, the harder it is for the regular smoker to quit. The Department accused the tobacco companies of deliberately making their customers more addicted, so that they could secure sales. Doctors complain that this business strategy of getting customers more hooked undermines the success rates of smoking cessation therapies. (Medical News today, 2012) nicotine is also an antiherbivore chemical, specifically for the elimination of insects. When humans, mammals and most other types of animals are exposed to nicotine, it increases their heart rate, heart muscle oxygen consumption rate, and heart stroke volume. The consumption of nicotine is also linked to raised alertness, euphoria, and a sensation of being relaxed. Nicotine at low doses, directly stimulates the CNS especially the brainstem resulting in sympathetic neural discharge, which increase blood pressure and heart rate among other behavioural stimulations (Comroe 1960; Su 1982).

One of the effects of nicotine is development of tolerance to its own actions; a likely mechanism by which it produces addictive drugs. After repeated use of nicotine, the responsiveness to the drug becomes decreased and increasingly larger doses will be required to produce the same effect (USNIH, 2008). Although nicotine is a major chemical constituent of tobacco, which affects neurobehavioural activity, other alkaloids are also present. These smaller quantities of chemicals although absorbed in small quantities may also affect behavior and effect of nicotine (Stahlandske T & Slanina P, 1982). The other alkaloids include; nornicotine, anabasine, myosmine, nicotyrine and anatabine. These make up 8 to 13 percent of the total alkaloid content of tobacco products. Nornicotine and anabasine have pharmacological activity qualitatively similar to nicotine, with potencies of 20 to 75 percent compared with that of nicotine, and depending on the test system and animal model (Clark *et al.*, 1965). Some of the alkaloids apart from having a direct effect may influence the effect of nicotine e.g. nicotyrine inhibits metabolism of nicotine in animals (Stahlandske T & Slanina P, 1982) thereby prolong the effect.

Nicotine is a potent parasympathommetric alkaloid found in the night-shad family of plants (*Solanaceae*) and a stimulant drug. It is a nicotinic acetylcholine receptor agonist. It is made in the roots and accumulates in the leaves of the plants. It constitutes approximately 0.6-3.0% of the dry weight of tobacco (USNIH, 2008) and is present in the range of 2-7 $\mu\text{g}/\text{kg}$ of various edible plants (Research health, 2008). It functions as an antiherbivore chemical; consequently, nicotine was widely used as an insecticide in the past (Johnson, Fung & Squier, 1989) and nicotine analysis such as imidacloprid are currently widely used. Research in 2011 found that nicotine inhibits chromatin-modifying enzymes (class I and II histone deacetylases); this inhibition has been shown to increase the ability to cocaine to cause an addiction (Stein, 1998). Research has shown that nicotine is very well absorbed from tobacco; it is very well distributed rapidly and in biologically active concentration to body organ especially the brain. Nicotine has also in many research works been implicated as it register the major cause of the predominant behavioural effects of tobacco and some of its physiologic consequences. It induces a dose-dependent increase in neuronal activity in a distributed system of brain regions, including the nucleus accumbens, amygdala, cingulate, and frontal lobes (Stein, 1998). The

effects of nicotine in rats are dose dependent, with low doses having anxiolytic and high doses anxiogenic effects. The effects are also time dependent between injection and testing (Irvine *et al*, 1999).

Materials and methods

Thirty (30) rats weighing 80-190g were procured from the animal house, Department of physiology, University of Calabar, Calabar, Nigeria. The rats were maintained under normal laboratory conditions of temperature, humidity and light for a period 2 weeks in the animal holdings of the Department of Human Anatomy, University of Calabar, Calabar, Nigeria, before commencement of experiment.

Liquid (concentrated) Nicotine was obtained from the Department of Pharmacology, University of Calabar, Calabar in Cross River State of Nigeria. 0.5ml of concentrated nicotine was dissolved in 200ml of distilled water, this was stored in a sealed tube at a normal room temperature and was administered orally once daily by means of feeding for a period of 28 days.

Experimental protocol

Albino rats of the wistar strain were animals of choice for this study, because they are tough, easy to obtain, cheap to maintain and even easier to get closely inbred colonies to avoid variation in and outcome as a result of difference in species. The rats weighed between 80-190g at start of experiments and were randomly chosen from both sexes. They were all kept in plastic cages with wire net covers. The ethics for the use of experimental animals were strictly adhered to.

Discussion

In this study, it was observed that chronic consumption of nicotine mixed diet reduced sodium (Na^+) and Bicarbonate (HCO_3^-) concentration while Potassium K^+ was significantly raised. This may be due to one of the constituent of nicotine that is suppressing the absorption of K^+ , Na^+ and Cl^- , hence the outcome.

Therefore, chronic intake of nicotine should be discouraged since the results show that ionic concentration are altered in nicotine fed rats.

Result

Effect of sodium ion (Na^+) concentration

The mean Na^+ concentration in the control, nicotine group was 3.96 ± 0.09 (mmol/L). the result showed that the Na^+ concentration in nicotine group was significantly lower ($P < 0.01$) when compared to control group. This is shown in figure 1.

Effect of potassium ion (K^+) concentration

The potassium ion (K^+) concentration in nicotine group (2.74 ± 0.18 mmol/L) was significantly higher ($P < 0.05$) when compared with the control group. This result is indicated in figure 2.

Effect of chloride ion (Cl^-) concentration

The mean Cl^- concentration in the control and nicotine groups were 10.00 ± 0.00 and 15.60 ± 2.31 (mmol/L) respectively. The results showed that the Cl^- concentration in the nicotine group had no significant difference when compared to the control group (Figure 3).

Effect of bicarbonate ion (HCO_3^-) concentration

The mean bicarbonate ion concentration in the control and nicotine groups were 11.60 ± 0.24 mmol/L and 10.40 ± 0.24 mmol/L respectively. The HCO_3^- concentration in the nicotine group was significantly lower ($P < 0.01$) when compared with control group (Fig 4).

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FIGURES

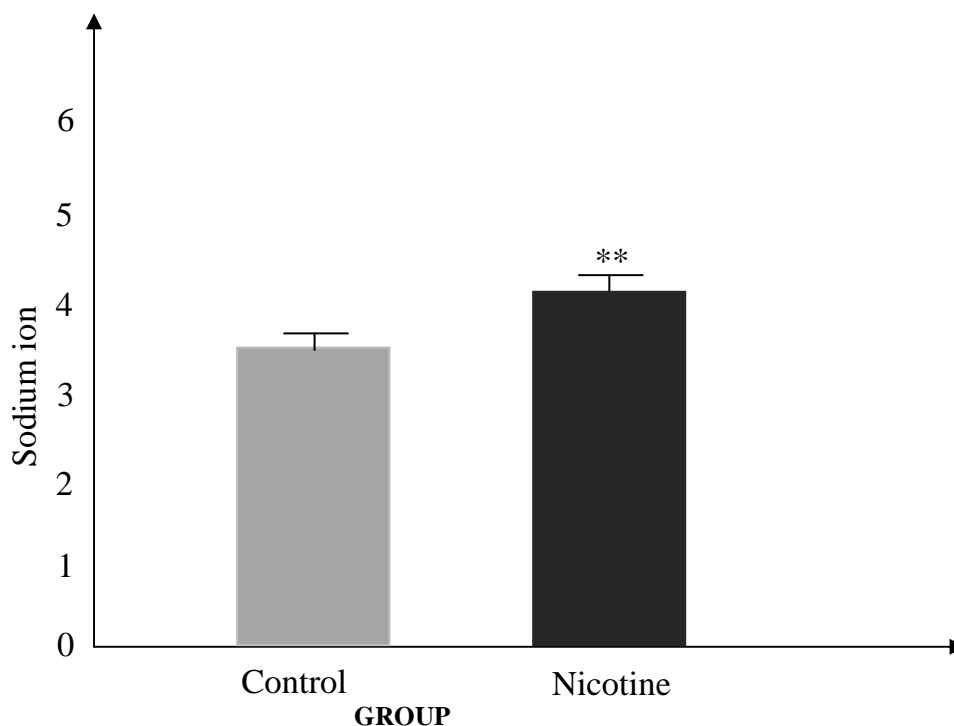


Figure 1: comparison of mean sodium ion levels in the experimental groups. Values are mean \pm SEM, n=5.
**P<0.01 vs control

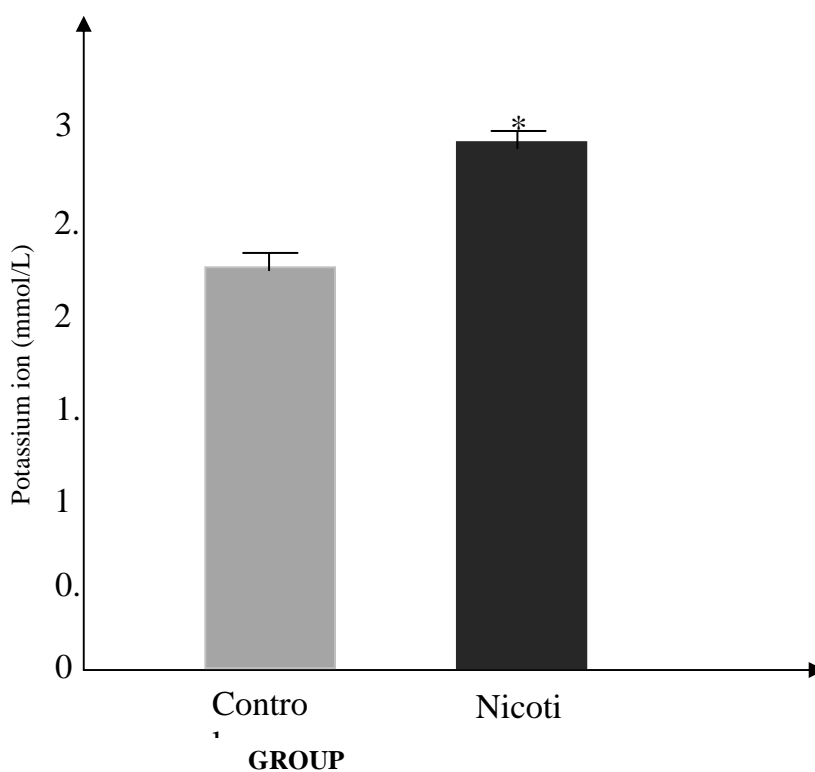


Figure 2: Comparison of mean potassium ion levels in the different experimental groups. Values are mean \pm SEM, n=5
*<0.05 vs control

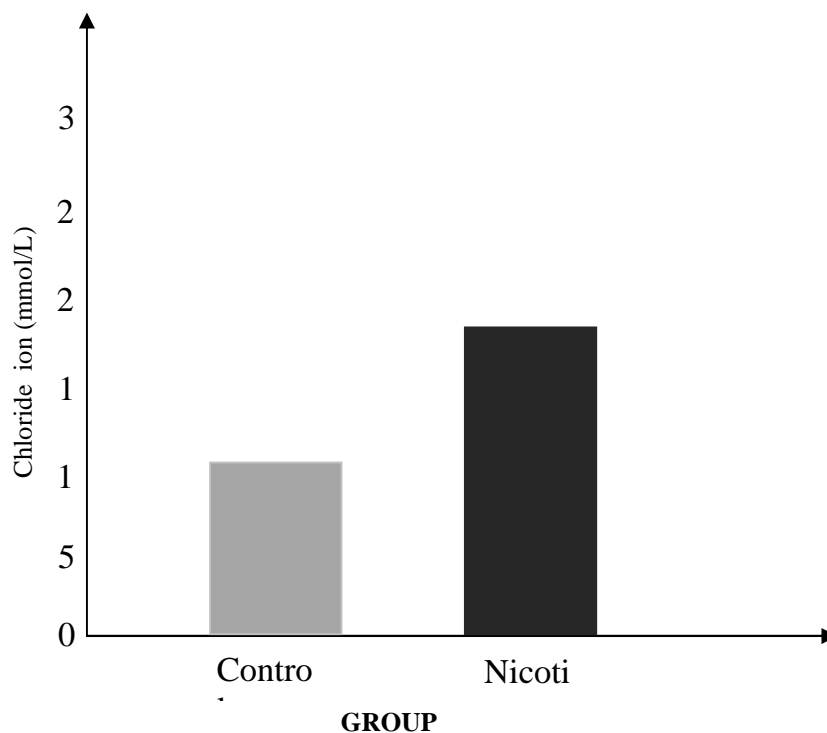


Figure 3: Comparison of mean chloride ion levels in the different experimental groups. Values are mean \pm SEM, n=5

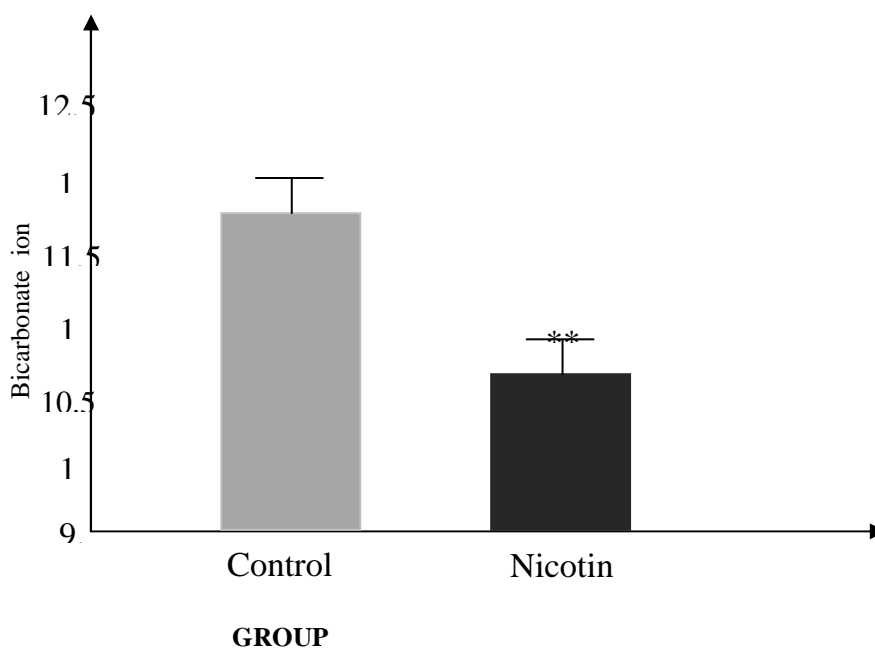


Figure 4: Comparison of mean bicarbonate ion levels in the different experimental groups. Values are mean \pm SEM, n=5
**p<0.01 vs control