SALT TASTE SENSATION IN WISTAR RATS CONSUMING SALT SOLUTIONS OF DIFFERENT MOLARITY

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Background: Innate tendency to consume salt to maintain sodium balance is observed in all species of animals. Over-consumption of salt may be lethal. In this study, the function of salty taste sensation in the face of consuming salt concentrations was evaluated. **Methods:** 120 adult Wistar rats, weighing 300±20 g were allocated to six groups. Test groups consumed 0.1, 0.2, 0.5, 1 and 2 M salt concentrations as drinking water and control group used potable water. The rate of water intake and salt concentrations consumption among different groups was calculated during five days and considered as the main parameter for evaluation of the salty taste sensation. **Results:** The rate of 0.1, 0.2 and 0.5 M salt concentrations consumption were increased 68%, 75% and 32% respectively and the groups maintained on 1 and 2 M salt concentrations showed 56 and 88 percent decrease respectively. **Conclusion:** 0.1, 0.2 and 0.5 M salt concentrations did not provoke animal's avoidance and since thirst sensation was not suppressed, animals showed an excessive tendency to drink salt concentrations. But 1 and 2 M salt concentrations stimulated salty taste sensation strongly. The neuralgic protective mechanisms provide an important protective function against drinking these salt concentrations.

Key words: Concentrations, Molar, Salt, Salty taste sensation, Solution,

INTRODUCTION

Sodium is the major extracellular cation in the body fluid.[1] Salt is the main source for sodium level balance, innate tendency to consume salt to maintain sodium balance level is observed in all species of animals.[2] Salt is Sodium Chloride, an inorganic chemical that has no nutritional value, it is nondigestible, irritant, stimulant, and a poisonous substance.[3] Excessive ingestion of salt is a well-recognized cause of hypernatremia in children.[4] Salt toxicity is more common in swine (the most sensitive species), cattle, and poultry. Sheep are relatively resistant. The acute oral lethal dose of salt is 2.2 g/kg in swine and 6.0 g/kg in sheep.[5] Salt poisoning is common in all ages of pig and is related to shortage of water availability. The normal levels of salt in the diet (0.4-0.5%) become toxic in the absence of water.[6] Salt toxicity varies in relation to the availability of drinking water. If water intake is restricted and sodium chloride intake is normal, a relative poisoning occurs. If combined with water deprivation, polioencephalomalacia develops when the water intake returns to normal.[7] The main protective mechanism against dangerous substance found in food and water is related to taste sense function. The tongue is a kind of funny organ, from a physiological point of view. If you place solutions on the tongue that is potentially toxic to any cell, the tongue will give information, in a hopefully reversible manner, about the solution's chemical composition and whether it should be ingested or not.[8] Taste plays an essential role in food selection and consequently overall nutrition. Because salt taste is appetitive, human ingest it more than they need.[9]

The sensory receptors for taste are the taste buds. A taste bud consists of some 50 gustatory receptor cells in association with supporting cells and basal cells.[10] Molecules that can be tested are detected by taste cells clustered in taste buds on the tongue, palate, pharynx, epiglottis, and upper third of the esophagus. The detection of salty taste stimuli is mediated by Na ion influx through Na channels.[11] In mammals where this Na^+ sensing system is functional, passive movement of stimulus Na^+ ions across the taste cell apical membrane causes membrane depolarization and, consequently, release of neurotransmitter onto primary taste afferents.[12] In human, the threshold for stimulation of the salty taste by sodium chloride is 0.01 M; and for the bitter taste by quinine, 0.000008 M. Because this much more sensitive, bitter taste sense provide an important protective function against many harmful toxins in food.[13] A 30% change in the concentration of the substance being tasted is necessary before an intensity difference can be detected.[14] Salty taste sensation provides an important protective mechanism against over-consumption of salt in food and water.

In this study, the function of salty taste sensation in the face of different molar salt concentrations consumption was evaluated.

MATERIAL AND METHODS

This study was conducted at the Department of Physiology, Faculty of Medicine, Zanjan University

of Medical Science, Zanjan, Iran from January 2004 to December 2006. Prior to the initiation of experimentation, study protocols were reviewed and approved by the animal research committee of Zanjan University of Medical Science. All work involving experimental animals was performed in full compliance with NIH Guidelines for the Care and Use of Laboratory Animals. The animals were purchased from the Iranian Razi Institute, one of the certified centers of laboratory animals breeding in Iran. Wistar rat is one of the accepted races employed by most of the research centers of animal studies. Wistar Rats were received at 12 weeks of age and were quarantined for one week prior to the administration of test or control articles. Prior to salt concentrations administration, healthy animals were selected following physical examinations and 120 adult Wistar rats, weighing 300±20 g were allocated randomly to six groups. Rats were housed five per cage in a climate-controlled room that was maintained on a 12-hour light/12-hour dark cycle. The temperature of the testing room was kept at 24 °C. At all times, during the quarantine, animals were permitted free access to certified diet, containing 0.5% salt, Zanjan city potable water delivered via a manual bottle watering system. At all times, during salt administration periods, animals were permitted free access to the same diet, different test groups consumed special salt concentration and control group consumed Zanjan city potable water with similar living conditions for all groups. Evaluation of salty taste sensation was performed by administration of 0.1, 0.2, 0.5, 1 and 2 Molar salt concentrations to test groups. By adding 5.85, 11.7, 29.25, 58.5 and 117 g of salt to one liter of distilled water, 0.1, 0.2, 0.5, 1 and 2 Molar salt concentrations were prepared. These concentrations were prepared from NaCl of Merck Brand (Darmstadt, Germany), containing about 100, 200, 500, 1000 and 2000 Mmol Na and the same amount of Cl respectively. The above mentioned salt concentrations osmolarities are about 200, 500, 1000, 2000 and 4000 Mmol respectively.

The amount of water intake and salt concentrations consumption among different groups was calculated during five days. The amount of salt concentrations consumption in test groups was compared with the rate of water intake in control group and changes in salt concentrations consumption was considered as the main parameter for evaluation of the salty taste sensation. The amount of water intake in control group was considered as base, which presented as 100%, and in test groups, changes in salt concentrations consumption was expressed as percentage of decrease and increase fluctuations. Following salt administrations, daily physical examinations were performed with concomitant recording of salt poisoning signs and weight changes. Following the appearance of salt poisoning signs, salt concentrations administration was disrupted in test groups and animals were treated with gradual exposure to tap water. The data were presented as decrease and increase fluctuations and statistical analysis of data was performed using descriptive statistical.

RESULTS

The consumption of 0.1, 0.2 and 0.5 M salt concentrations as compared to the amount of water intake in control group increased 68%, 75% and 32% respectively. In contrast, the amount of salt concentrations consumption in test groups maintained on 1 and 2 M salt concentrations showed a decrease of 56% and 88% respectively. 0.1 and 0.2 M salt concentrations consumption did not stimulate salty taste protective mechanism strongly and the neuralgic protective mechanism could not suppress thirst sensation, hence animal showed an excessive tendency to drink 0.1 M salt concentration. 0.1 M salt concentration consumption did not threat animal's life and could be tolerated without significant complications and signs of salt poisoning, but animal's health could be affected. 0.2 M salt concentration consumption could not be tolerated without significant complications; this concentration could threat animal's life. Signs of salt poisoning appeared in the animal and significantly affected their health. 0.5 M salt concentration consumption stimulated salty taste protective mechanism and up to some extent, the neuralgic protective mechanism limits the function of thirst sensation, hence animal tendency to drink 0.5 M salt concentrations was decreased as compare to 0.1 and 0.2 M salt concentrations. But the amount of 0.5 M salt concentration intake showed 32% increase as compare to control group. Since, salty taste sensation did not provide an important protective mechanism against 0.5 M salt concentration, consumption of 0.5 M salt concentration was higher than control group consuming tap water. Prolonged consumption of 0.5 M salt concentration threatens animal's life and accompany with signs of salt poisoning. 1 and 2 M salt concentrations consumption stimulated salty taste sensation strongly and the neuralgic protective mechanism suppressed thirst sensation, hence animal tendency to drink 1 and 2 M salt concentrations decreased as compared with the control group. Respectively, 56 and 88 percent of decrease in salt concentrations consumption was due to neuralgic protective mechanism. 0.1, 0.2 and 0.5 M salt concentrations consumption did not provoke animal's avoidance of drinking salt concentrations. But,

following 1 and 2 M salt concentrations consumption, salty taste sensation was provoked strongly and the neuralgic protective mechanism provided an important protective function against drinking these salt concentrations. In absence of salty taste sensation response and avoidance of drinking 1 and 2 M salt concentrations, excessive salt load leads to rapid animal death.

DISCUSSION

The present study produced several key findings about the function of salt taste sensation in the face of consuming different Molar (M) of salt solutions.

First, different salt solutions had different effects on the amount of salt solutions consumption. The rate of 0.1, 0.2 and 0.5 M salt solutions consumption in comparison with control group increased 68% , 75% and 32% respectively and groups maintained on 1 and 2 M salt solutions showed respective decrease 56 and 88 percent in salt solution consumption.

Second, different salt solutions had different effects on the function of salt taste sensation and thirst sensation. 0.1, 0.2 and 0.5 M salt solutions did not provoke animal's avoidance of drinking salt solutions, because thirst sensation did not suppress by the neuralgic protective mechanism, animal showed an excessive tendency to drink salt solutions. Therefore we could propose that salt taste sensation did not provide an important protective function against drinking 0.1, 0.2, and 0.5 M salt solutions and animal consumed 68%, 75% and 32% salt solutions higher than tap water in control respectively. 1 and 2 M salt solutions provoked salt taste sensation and the neuralgic protective mechanism suppressed thirst sensation, hence animal tendency to drink 1 and 2 M salt solutions decreased 56 and 88 percent respectively. 0.1, 0.2, and 0.5 M salt solutions did not lead to animal's avoidance of drinking salt solutions, but 1 and 2 M salt solutions led to significant animal's avoidance of drinking salt solutions.

Third, the rate of salt load entrance to the animal body via 0.1 and 0.2 M salt solutions did not exceed the maximal kidney's ability to excrete salt, hence the signs of salt poisoning did not appear in the animal, but its health affected significantly. The rate of salt load entrance to the animal body via 0.5 M salt solution exceed the maximal kidney's ability to excrete salt, as a result different signs of salt poisoning appeared in the animal. 1 and 2 M salt solutions osmolarity are about 2000 and 3000 Moms respectively and these salt solutions could not be used as the only water source to compensate the body fluid loss. These salt solutions provoked neuralgic protective mechanism more powerful and animal tendency to drink these salt solutions decreased significantly. Excessive salt load and animal's avoidance of drinking 1 and 2 M salt solutions resulted in rapid animal death. Detection threshold of both strains (Fischer 344 (F344) and Wistar strains) was similar, lying between 0.001 and 0.002 M NaCl (14). A 30% change in the concentration of the substance being tasted is necessary before an intensity difference can be detected.[15]

As glucose and sodium are necessary substances for body of living organism, hence the threshold for stimulation of salty taste sensation by sodium chloride and sweet taste sensation by glucose are identical (0.01 M).[13] Because salt taste sensation is not much more sensitive to sodium chloride, therefore it is probable that in some circumstances salt taste sensation could not protect our body against eating and drinking excessive amount of salt found in food and water. The rate of salt in 0.001 M salt solution is about 0.06 g/L, however, detection threshold for stimulation of the salty taste by sodium chloride in Wistar strains lying between 0.001 and 0.002 M NaCl, but 0.1 , 0.2 and 0.5 M salt solutions did not provoke animal's avoidance of drinking salt solutions. Only 1 and 2 M salt solutions provoked salt taste sensation and the neuralgic protective mechanism more powerful and animal tendency to drink salt solutions decreased.

When salty water was consumed as the only source of drinking fluid, the body of living organism had to select a perfect selection. Water is fundamental to existence, total body water is tightly regulated within ±0.2% of body weight each day.[16] Thirst is important for maintaining body fluid homeostasis.[17] Normally, when the serum sodium rises, thirst develops and ADH is secreted.[18] The stimulation of thirst, which promotes fluid intake, and the actions of vasopressin to reduce water excretion, are synergistic in returning plasma osmolality to normal.[19] Although ADH release may occur earlier, it is thirst that provides the ultimate protection against hypernatremia.[20] Hypernatremia will not occur in individuals with an intact thirst mechanism and free access to water, due to a potent osmolar stimulus to drink.[18] While the rate of salt in water is low, salty water could be used as a water source to compensate for body fluid loss by living organism. But, high salt solutions provoked neuralgic protective mechanism more powerful and the animal had no tendency to drink salt solution. If the neuralgic protective mechanism did not counteract with thirst sensation, the animal would drink excessive amount of concentrated salty water, put a positive feedback loop into action and cause a progressive body water loss leading to enhanced osmolarity and thirst induction and animal consumed excessive amount of salty water and became massively dehydrated and die in a

short time. Salt taste sense provide an important protective mechanism, and the animal's refusal from drinking highly concentrated salt concentrations will keep it alive for some days and if it can get fresh water at this time it will escape death.

CONCLUSION

0.1, 0.2 and 0.5 M salt concentrations did not provoke animal's avoidance and since thirst sensation was not suppressed, animals showed an excessive tendency to drink salt concentrations. But 1 and 2 M salt concentrations stimulated salty taste sensation strongly and the neuralgic protective mechanism provide an important protective function against drinking these salt concentrations.

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