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The Diuretic Effects Of Ethanol Ingestion In Wistar Rats

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ABSTRACT

Of all potential substances of abuse, alcohol is one of the most readily available. Most adults regularly or occasionally consume it. The habitual urination immediately following ingestion was experimentally studied in the ethanol fed-Wistar rats.

Experimental equivalent concentrations of ethanol: 10% 20% and 30% v/v in the respective alcoholic beverages: lager beer, wine and dry gin were administered separately to three groups of Wistar rats orally and the quantity and durations of urinations carefully monitored.

The shortest time lag and time interval respectively, 20 minutes and 9 minutes, and highest volume of urine 7.56ml, were recorded in the 30% ethanol-fed rats.

The anti-diuretic action of vasopressin is actively suppressed by ethanol direct inhibition of the hormone secretion in supra-optic nuclei; this inhibitory potency of ethanol is concentration dependent.

Keywords: Diuresis; Wistar rats; Ethanol

Alcoholism the chronic compulsive use of and loss of control over alcohol intake, its direct and indirect public health costs are estimated to be in the range of hundreds of billions of dollars yearly in the United States alone, and probably the same or even much more in other parts of the world. Currently, there is no cure, and the neurobiology of the disease is not completely understood. Despite its widespread use and reputation as "social lubricant", alcohol extracts a heavy toll on society. Alcoholics do not feel empathy for others or have a sense of justice. They can be entirely "unattached", feeling nothing, even for the people who raise them. Because of these deficits, they have no internalized sense of right and wrong. A person with no morals, empathy, values or feelings can be a danger to themselves and to society. Drunk driving, for instance, is a major scourge; about a third of the total traffic fatalities every year involve drunk drivers. Tens of thousands more die each year from alcohol poisoning, and from alcohol-related degenerative conditions such as gastritis, cardio-myopathy, and liver disease. Fetal alcohol syndrome, caused by alcohol consumption by pregnant women, is the leading cause of mental retardation worldwide. It is estimated that perhaps as many as one out of every 1,000 babies born each year suffers from fetal alcohol syndrome, (Friggard, 2003; Zhiguo et al., 2004).

Some of the multiple pathological effects of ethanol ingestion ranging from loss of body fluid, nausea, emaciation, fluid and electrolyte imbalance had been well documented (Rao, 1988; Schapira, 1990; Adebisi, 2003; Akunyili and Igwe, 2005). These mostly result in gastro-intestino-urinary disorders, common in the heavy alcoholics. One of the cardinal characteristics symptoms of this phenomenon is the frequent or incessant urination 'bladder emptying' immediately following drinking, probably in response to the inhibition of the antidiuretic functions of vasopressin, a hormone released from the anterior lobe of the pituitary gland (Rix and Rix, 1983).

This paper experimentally investigates the diuretic responses in rats to ingestion of different corresponding concentrations of ethanol in some commonly ingested alcoholic beverages. Wistar rats were chosen due to their common mammalian biological similarities and wide acceptance as experimental animal model, (Drous, 1981).

MATERIALS AND METHODS

Forty healthy adult Wistar rats (20 males and 20 females) weighing between 200g and 250g were procured for experiment. The animals were kept in the tidy animals holdings of the Department of Human Anatomy, A. B. U., Zaria; and fed adequately on animal mash. On the eve of the experiment, the animals were fasted from noon through the night. They were

grouped into four: a, b, c, and D, with each group consisting of 10 rats (5 males and 5 females). Each animal in groups A, B, C received 0.79g/kg of 10%, 20%, 30% v/v ethanol (BDH Chemials, Poole, England, Analar R grade) respectively as their main drinks for one day, 8.00am 8.00pm. Intubations were avoided to prevent stress or any sorts of nervousness, which could possibly err the aim or twat the results. Ethanol concentrations administered are experimental equivalents in the alcoholic beverages of lager beer, whisky and dry gin for groups A, B, C respectively (Rix and Rix, 1983); and the dosage used is the calculated normal equivalent g/ml weight of ethanol (Adebisi, 1995). Group D rats served as control and received normal saline.

Throughout the ethanol-feeding hours (day) and experiment, each rat was caged separately, in a relaxed state and under close watch. The floor of each cage was laid with clean white water-proof cellophane slighted and channeled towards one corner of the floor ending in a cup-like form for easy drainage and pooling of the animal urine. The urine was each time carefully and wholly sucked-up with 2ml syringe attached needle and immediately gauged in it and stored in 10ml glass measuring cylinder.

From the beginning of the ingestion, the time lag (tl) for the first urination; and time-interval (ti) for each subsequent urination were recorded. The quantity of urine (qu) collected each time was recorded. The water diuresis produced by drinking large amounts of fluids begins about 15 minutes after ingestion of water load reaches its maximum in about 40 minutes. The delay represents the time required for the water to be absorbed, the vasopressin secretory mechanism inhibited, and the previously circulating vasopressin metabolized.

RESULTS

At the first taste, animals were initially reluctant to drink from the ethanol preparations since they were not used to such drinks. However, they later gradually began to drink from the bottle probably in response to thirst and the absence of water. The animals however gradually stopped drinking towards the close of the experiment possibly due to drowsiness. The mean of the recorded values are presented as follows: table 1a indicates the time lag (tl), that is, duration between the periods of first sipping and first urination with 40 minutes for the control; 35 min, 30 min and 20 min respectively for the treated groups A, B, C rats. Time interval (ti), that is the duration between urinations was 50 min for control, while it was 30 min, 20 min, and 9 min for the treated groups A, B, C respectively. (Table 1b). The quantity of urine (qu1) obtained for the 12hours were 3.37ml for the control, and 3.50ml, 5.64ml and 7.07ml respectively for the treated groups A, B, C. (Table 2a). The number and quantity (qu 2) of urinates per rat per group in 4hurs are as shown in Table 2b.

DISCUSSION

The time lag (tl) and time intervals (ti) were lesser in the 30% rats than in the 20% and 10% ethanol treated rats, indicating the promptness of vasopressin inhibition during alcohol intakes; the direct inhibition of vasopressin secretion from the supra-optic nuclei of hypothalamus by ethanol is well established, Ganong, (1975), however, it presently appears that ethanol action could be dose dependent. The frequency and amount of urinates reached maximum in 30% ethanol rats and by the 8th hour of continuous ingestion. But decreased towards the last quartile, suggestive of the fading effects of ethanol. Alcohol is usually

Table 1a: Showing the time lag (tl) between the first sipping and first urination

	A	B	C	D
Range (min)	30 – 38	28 – 35	20 – 25	35 – 43
Mean	35	30	20	40
SD	3.75	3.24	2.74	3.10

Table 1b: Showing the time interval (ti) between urinations

	A	B	C	D
Range (min)	28 – 33	18 – 23	7 – 12	45 – 52
Mean	30	20	9	50
SD	2.52	2.63	3.54	2.61

Table 2a: Showing the quantity of urinates (qu1) in 12 hours

	A	B	C	D
Range (min)	2.50 – 3.72	3.42 - 5.70	6.75 – 7.56	2.45 – 4.22
Mean	3.50	5.64	7.07	3.37
SD	0.57	1.23	1.56	0.19

Table 2b: Showing the mean quantity (qu2) and number of urinates (in brackets)

	A	B	C	D
First 4 hours (ml)	2.60 (5)	3.53 (6)	6.80 (10)	2.50 (17)
Second 4 hours (ml)	3.72 (5)	5.70 (12)	7.56 (14)	4.20 (18)
Third 4 hours (ml)	3.14 (3)	4.76 (6)	5.18 (8)	2.95 (10

eliminated from the body within 12 hours, though that can vary depending on how much was drunk, (Adelman and Bozian, 2005). The present results however, serve as indicator to the sole effects ethanol, unlike the mimicked alcoholic beverages, which contain a number of non-alcoholic ingredients, which are probably more osmotically effective and could produce more diuresis; since the ultimate effect of alcoholic beverages is the results of interplay of the effects of ethanol itself and the non-alcoholic contents (Andreas, 2005). Compared with control, ethanol treatments resulted in prompt and more frequent urinations, which could possibly be more complicated with alcoholic beverages intakes (Chiari, et al., 1993).

The presence of large quantities of un-reabsorbed molecules such as those of ethanol in the renal tubules causes an increase in urine volume called osmotic diuresis. Such substances that are not re-absorbed in the proximal tubules exert an appreciable osmotic effect as the volume of tubular fluid decreases and their concentration rises. They therefore 'hold water in the tubule'. In addition; there is a limit to the concentration gradient against which Na^+ can be pumped out of the proximal tubules. Normally, the movement of water out of the proximal tubules prevents any appreciable gradient from developing; but Na+ concentration in the fluid falls when water re-absorption is decreased because of the presence in the tubular fluid falls when water re-absorption is decreased because of the presence in the tubular fluid of increased amounts of such un-re-absorbable substances. It is important to recognize the difference between osmotic diuresis and water diuresis. In water diuresis, the amount of water re-absorbed in the proximal part of nephron is normal, and the maximal urine flow that can be produced is about 16ml/min, if water is ingested at a more rapid rate than this for any length of time, the swelling of the cells due to up-take of water from the hypotonic ECF becomes severe and the symptoms of water intoxiction and diuresis develop. In osmotic diuresis, increased urine flow is due to decreased water re-absorption in the proximal tubules and loops, hence very large urine flow can be produced. This is usually the case in alcoholism when vasopressin is suppressed, (Ganong, 1975).

Vasopressin is secreted in the supra-optic nuclei of hypothalamus and stored in the posterior pituitary and released into the blood stream by nerve impulse from the hypothalamus. It acts by increasing intercellular cyclic AMP in the cells of the collecting ducts. The c-AMP then acts in some undefined way to increase the permeability of the renal epithelium, so that water enters the hypertonic interstitium of the

renal pyramids. The urine becomes concentrated at its volume decreases. The overall effect is the retention of water in excess of solutes, thus the effective osmotic pressure of the body fluids is decreased. ECF volume also affects vasopressin secretion. Vasopressin secretion is increased when ECF is low and decreased when ECF is high. (Ganong, 1975).

Although urine alcohol testing will indicate the presence of alcohol in a person's body, it will not indicate an individual's current condition. Once consumed, alcohol enters the blood through the stomach within 15 minutes, causing immediate impairment. It is then metabolized by the body and, after 1½ to 2 hours, will begin to show up in the urine. Therefore, urine alcohol does not measure a true present condition of the person; it rather indicates the person's condition several hours before. Additionally, urine alcohol concentration does not directly correspond to blood alcohol concentration. Urine alcohol concentration will vary depending on the person's metabolism and the amount of fluid in his system. For instance, a person who is slightly dehydrated will tend to have a higher alcohol concentration in his urine than someone who has a normal level of fluid in his system. At least one study has indicated that a false positive for urine alcohol can occur; in this case, high levels of sugar and acetone in the body caused fermentation in the urine, creating a false positive for urine alchol. All things considered, the urine alcohol test is the least preferred or perhaps accurate test available for alcohol testing, (Sidneya nd Uduardo, 2005).

REFERENCES

Adebisi S S (1995). Teratogenic effects of ethanol on the intra uterine development of bones in Wistar rats. M.Sc. Thesis. Obafemi Awolowo University, Ile-Ife, Nigeria.

Adebisi S S (2003). Foetal Alcohol Syndrome: An Osteometric evaluation in the Wistar rat animal model Nig. J Surg. Res. 5(3 4): 160-165.

Andreas F, Inaam A N and Alexander S et al., (2005). The effects ethanol and alcoholic beverages on gastric emptying of solid meal in humans. Alcohol & Alcoholism 40(3): 187-193.

Adelman S and Bozian C R (2005). How long does alcohol remain in my body? The Substance Abuse Forum. http://www.medhelp.org/forums/Addiction/faq.html.

Adunyili D N and Igwe S A (2004). Purgative Potentials of Hydroalcoholic extracts of Daldina concentrica. Ebonyi Med. J. 3(1 & 2): 28-31.

Chiari S T, Teyssen S and Singer M V (1993). What controls should be used in studies of acute effects of alcohol and alcoholic beverages on the stomach and the pancreas? Scandinavian J Gastroentorology 28: 289 295.

Friggard L (2003). Fetal Alcohol: The Basics. http://www.educarer/com/alcoholbasics.htm#bio

Ganong W F (1995). Control of Posterior Pituitary Secretion: Neural Centres Regulating Visceral Functions. In: Review of Medical Physiology 7th ed. Lange Med. Pub. Los Altos P. 163-164, 527.

Krous H F, (1981). Foetal Alcohol Syndrome, a dilemma of maternal alcoholism. Pathology Ann 16(1): 295-311.

Rao F H and Larkin E C (1988). Role of nutrition causing the effects attributed to chronic alcohol consumption. Med. Sci. Res. 2: 53-57.

Rix K JB and Rix E L (1983). A nomogram for calculating the content of alcoholic beverages in 'units of alcohol'. In Alcohol Problems: A Guide for Nurses and other health Professionals. Wright Pub. London. P 163.

Schapira D (1990). Alcohol abuse and osteoporosis. Seminars in Arthritis and Rheumatism. 19 (6): 371-376.

Sidney K and Uduardo C (2005). Converting a urine alcohol value into a blood alcohol level. Am J Clin. Path 65(3) 231-235.

Zhiguo N, Paul S, Amanda J R et al. (2004). Scripps scientists describe dangerous cocktail of alcohol, brain peptides and neurotransmitters. Http://www.scripps.edu/html. Http://www.sciencemag/org/html.