

Ethanol Suppresses the Effects of Sodium Arsenite in Male Wister Albino Rats

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Abstract

Background: Millions of people around the world get exposed to high levels of heavy metals in drinking-water. Therefore, quality control in drinking-water, food industries and detection of heavy metals is an extremely critical issue in maintaining the human health. Of such heavy metals is arsenic. Water as one of the major ingredient in both traditional and modern beer fermentation process may/may not be contaminated with arsenic due to poor quality control. As a result people are exposed to the end product which constituted arsenic compound. Sodium arsenite and ethanol has been established to be toxic both *in vivo* and *in vitro*.

Objective: This study is primarily designed to investigate the effect of co-administration of sodium arsenite and ethanol in male Wister albino rats.

Methods: Forty five (45) albino rats divided into nine (9) groups of five rats each were administered with distilled water, 2.5mg and 5mg/kg body weight of sodium arsenite, 3% and 6% (v/v) respectively. Treatment was based on single and combined administration. Micronucleated polychromatic erythrocytes, acetylcholine esterase, Aspartate Transaminase (AST), Alkaline Phosphatase (ALP), Alanine Transaminase (ALT) and hematological parameters were determined by standard procedures (Figure 1).

Results: Combined treatment with sodium arsenite and ethanol significantly decreased the number of micro nucleated polychromatic erythrocytes, AST, ALT and ALP activities as against the single treated groups. However, there was stabilization of acetylcholine esterase activity in the brain. The hematological parameters level was also stabilized.

Conclusions: We therefore proposed that the chemical interaction between sodium arsenite and ethanol is what was responsible for the suppression of sodium arsenite – induced clastogenic, hepatotoxic, anti-acetylcholine esterase and anemic effects.

Keywords: Sodium arsenite; Ethanol; Micronucleated polychromatic erythrocyte; Acetylcholine esterase, Hematology

Introduction

Continuous exposure of humans to arsenic through long-term ingestion of contaminated water and its attendant health problems has been widely reported. Inorganic arsenic compounds are widely distributed in nature and a lot of epidemiological evidence exist associating occupational and environmental exposure to them with human carcinogenesis [1,2]. For instance, exposure to trivalent and pentavalent forms of arsenic, which occurs worldwide primarily through occupational and environmental exposure, causes characteristic skin alterations (ulceration), including hyperkeratosis and skin cancer [3]. Epidemiological studies conducted in Taiwan [4], Chile [5] and Japan [6] indicated a connection between arsenic exposures from contaminated drinking water among the inhabitants. It is also known that arsenic interact with other substances, metals inclusive there by potentiating its effects and/or vice versa [7]. There is growing evidences that sodium arsenite intoxication can compromise the integrity of the liver in mouse, rat, fish, and goat [8,9,10]. Recently, some studies suggest the use of antioxidants and antioxidant rich foods and herbal medicinal plant for the management of arsenicosis [11]. Induction of cancer is frequently associated with DNA damage, changes in ploidy of cells, and non-random chromosome aberrations which can result from exposure to arsenic [12]. Association between chronic alcohol abuse and the development of cirrhosis, as well as between cirrhosis

and the development of Hepatocellular Carcinoma (HCC), is well documented [13,14]. Acute and chronic ethanol treatment has been shown to increase the production of reactive oxygen species, lower cellular antioxidant levels, and enhance oxidative stress in many tissues, especially the liver [15]. Ethanol-induced oxidative stress plays a major role in the mechanisms by which ethanol produces liver injury [15]. Water as one of the major ingredient in both traditional and modern beer fermentation process may/may not be contaminated with arsenic due to poor quality control. As a result people are exposed to the end product which constituted arsenic compound. Sodium arsenite and ethanol has been established to be toxic both *in vivo* and *in vitro*. Therefore, this study was undertaken to investigate the effect of co-administration of sodium arsenite and ethanol on male Wister albino rats with a view to establish the facts on whether or not their interaction could be enhance or suppress by one another.

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Materials and Methods

Chemicals

Sodium Arsenite (NaAsO₂, Mol.wt 129.91, 98% prod 30110, BDH chemicals Ltd Poole England) and ethanol (C₂H₅OH, Mol.wt 46.07, 99.7 to 100% v/v pod 10107, BDH chemicals Ltd Poole England) were dissolved in distilled water to prepare 3%, and 6% ethanol (v/v).

Animals

Forty five (45) male albino rats weighing 151 – 198 g were used for the present investigation. They were reared at the animal house of the Department of Pharmacy, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, and Zaria, Nigeria with the approval of animal rights review committee. They were acclimatized for 1 week on normal diet of pelletized mouse chow, with water given *ad libitum* at room temperature within a 12-h light and dark cycle before the commencement of the experiment.

Experimental design

The animals were divided into nine different groups of five (5) rats each according to their body weight proximity and treated once in a week for a period of four week as shown below:

Group 1: Distilled water

Group 2: 2.5mg/kg body weight of sodium arsenite

Group 3: 5mg/kg body weight Sodium arsenite

Group 4: 3% ethanol (v/v)

Group 5: 6% ethanol (v/v)

Group 6: 2.5mg/kg body weight Sodium arsenite + 3% ethanol (v/v)

Group 7: 5mg/kg body weight Sodium arsenite + 6% ethanol (v/v)

Group 8: 2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)

Group 9: 5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)

Twenty-four (24) hours after the last administration, animals were sacrificed by using 60mg/kg body weight of sodium pentothal. Liver, Brain, Femur and Blood samples were collected, part of the blood samples were placed in Ethylene Diamine Tetra acetic Acid (EDTA)

bottles for hematological analysis and the remaining were centrifuged at 4,000 rpm for 5 minutes to obtain the serum for analyses. The liver and brain weight were immediately taken and brain was quickly placed on an inverted Petri dish on ice. The fore brain was dissected, weighed and homogenized in 10ml of a medium containing 10mM Tris – HCl buffer (pH 7.2) and 160mM sucrose. The total homogenate was centrifuged at 3500g at - 4°C in a refrigerated centrifuge for 5 minute. The supernatant was used for Acetylcholine Esterase (AChE) activity. The femurs were preserved for micronucleus assay.

Determination of biochemical and hematological parameters

The Alkaline Phosphatase (ALP), Alanine Aminotransferase (ALT), and Aspartate Aminotransferase (AST) were determined by using Auto Analyzer Hitachi Roche 7020 (902), Japan Inc. according to manufacturer's protocols. Complete blood counts were also determined using Coulter HmX Hematology Analyzer Beckman Coulter Inc. according to manufacturer's protocols.

Determination of acetylcholine esterase activity

The activity of AChE in the brain was determined by the method described by Ellman et al. [18], as modified by Srikumar et al. [19] The mixture was prepared by mixing 0.4mL aliquot of the homogenate and added to 2.6ml phosphate buffer (0.1M, pH8.0) and 100µL of DTNB (270µM). This was pre-incubated for 2 minute at 30°C and the reaction was started with the addition of 20µL ATC (30mM). The product of thiocholine reaction with DTNB was determined at 412nm for a period of 10 minute at 2 minute intervals. The absorbance per minute was determined.

Micronucleus assay

Clastogenic effects were evaluated in the rat bone marrow using the micronucleus assay as described by Heddle and Salmone, [20] and modified by Heddle, et al, [21]. Two hours prior to sacrifice, the animals were injected (i.p.) with 0.04 % colchicine (1ml/100g body weight). Bone marrow cells from both femurs were used for preparing slides. The slides were fixed, air-dried and pretreated with May-Grünwald solution. They were then stained with 5% Giemsa solution. The slides were scored for the presence of micronucleated polychromatic erythrocytes (mPCEs) in 1000 cells according to standard procedure.

Statistical analysis

The results were expressed as mean ± Standard error. Differences

Group	Treatment	IBW	FBW	%WT CHANGE	Liver	Brain	RLW	RBW
1	Distilled Water	198.60 ± 12.67	246.40 ± 15.66	24.13 ± 1.94 ^{d,h,i}	7.16 ± 0.54	1.67 ± 0.04	2.99 ± 0.39	0.69 ± 0.04
2	2.5mg/kg Body weight SA	182.44 ± 15.89	225.00 ± 21.86	25.59 ± 4.41 ^{d,h}	7.17 ± 0.56	1.54 ± 0.07	3.36 ± 0.55	0.71 ± 0.08
3	5mg/kg Body Weight SA	195.00 ± 7.52	235.40 ± 14.74	21.57 ± 3.74 ^{g,h,i}	6.81 ± 0.15	1.54 ± 0.03	2.95 ± 0.26	0.67 ± 0.04
4	3% Ethanol	184.56 ± 9.23	229.80 ± 7.33	34.69 ± 3.50 ^{a,b,e}	7.58 ± 0.35	1.63 ± 0.04	3.25 ± 0.25	0.70 ± 0.02
5	6%Ethanol	161.20 ± 7.57	204.60 ± 8.25	27.15 ± 2.20 ^{d,f,h,i}	6.72 ± 0.29	1.60 ± 0.05	3.29 ± 0.13	0.79 ± 0.05
6	2.5mg/kg Body Weight SA+3% Ethanol	169.00 ± 6.37	219.80 ± 3.82	30.53 ± 3.34	6.74 ± 0.21	1.67 ± 0.05	3.01 ± 0.09	0.76 ± 0.03
7	5mg/kg Body Weight SA+6%Ethanol	151.20 ± 4.68	200.00 ± 2.32	32.74 ± 4.00 ^{c,e}	6.27 ± 0.19	1.65 ± 0.05	3.14 ± 0.12	0.82 ± 0.03
8	2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)	153.40 ± 5.36	212.20 ± 2.90	38.77 ± 3.28 ^{a,b,c,e}	7.01 ± 0.29	1.55 ± 0.06	3.31 ± 0.15	0.73 ± 0.04
9	5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)	155.00 ± 9.05	207.80 ± 11.20	34.28 ± 2.00 ^{a,c,e}	5.52 ± 0.17	1.66 ± 0.08	2.67 ± 0.22	0.80 ± 0.03

a= statistically significant (p<0.05) when compared with group 1 b= statistically significant (p<0.05) when compared with group 2 c= statistically significant (p<0.05) when compared with group 3 d= statistically significant (p<0.05) when compared with group 4 e= statistically significant (p<0.05) when compared with group 5 f= statistically significant (p<0.05) when compared with group 6 g= statistically significant (p<0.05) when compared with group 7 h= statistically significant (p<0.05) when compared with group 8 i= statistically significant (p<0.05) when compared with group 9 SA= Sodium Arsenite IBW= Initial Body weight FBW= Final Body weight %WT = % weight RLW = relative liver weight RBW = relative brain weight

Table 1: Body and Organ weight (g) for n=5, (mean ± SE) of the experimental animals before and after exposure to sodium arsenite and ethanol.

Group	Treatment	ALP	ALT	AST
1	Distilled Water	4.97 ± 1.35 ^{d,e,g}	23.00 ± 4.81 ^{b,c,d,e,f,g,i}	108.50 ± 8.50 ^f
2	2.5mg/kg Body Weight SA	17.94 ± 1.38 ^{d,e,g}	37.00 ± 3.99 ^a	110.00 ± 5.00 ^f
3	5mg/kg Body Weight SA	41.40 ± 9.60 ^{e,g}	43.80 ± 0.80 ^a	115.00 ± 5.77 ^f
4	3% Ethanol	73.02 ± 22.32 ^{a,b}	46.60 ± 6.50 ^{a,h}	142.40 ± 18.35
5	6%Ethanol	105.43 ± 23.01 ^{a,b,c,f,h,i}	49.50 ± 0.50 ^{a,h}	150.00 ± 5.00
6	2.5mg/kg Body Weight SA + 3% Ethanol	44.28 ± 15.41 ^e	39.00 ± 4.50 ^a	159.50 ± 9.50 ^{a,b,c}
7	5mg/kg body weight SA + 6%Ethanol	87.40 ± 13.55 ^{a,b,c,g,h,i}	44.60 ± 4.27 ^a	130.40 ± 8.16
8	2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)	31.74 ± 1.38 ^{e,g}	32.33 ± 3.67 ^{d,e}	130.20 ± 7.11
9	5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)	34.04 ± 9.34 ^e	41.60 ± 2.16 ^a	123.67 ± 7.54

a= statistically significant (p<0.05) when compared with group 1 b= statistically significant (p<0.05) when compared with group 2 c= statistically significant (p<0.05) when compared with group 3 d= statistically significant (p<0.05) when compared with group 4 e= statistically significant (p<0.05) when compared with group 5 f= statistically significant (p<0.05) when compared with group 6 g= statistically significant (p<0.05) when compared with group 7 h= statistically significant (p<0.05) when compared with group 8 i= statistically significant (p<0.05) when compared with group 9 SA= Sodium Arsenite ALP=alkaline phosphatase ALT= alanine amino transferase AST= aspartate amino transferase

Table 2: Serum level mean ± S.E (U/L) for n=5, of aspartate amino transferase, alanine amino transferase and alkaline phosphatase in sera of rats.

Group	Treatment	Micronuclei/1000 PCE
1	Distilled Water	3.67 ± 1.67 ^{b,c,g}
2	2.5mg/kg Body Weight SA	15.00 ± 2.02 ^{a,c,d,e,f,h,i}
3	5mg/kg Body Weight SA	22.80 ± 3.50 ^{a,b,d,e,f,g,h,i}
4	3% Ethanol	6.00 ± 1.05 ^{b,c,g}
5	6%Ethanol	7.60 ± 1.70 ^{b,c}
6	2.5mg/kg Body Weight SA + 3% Ethanol	6.20 ± 1.53 ^{b,c,g}
7	5mg/kg Body Weight SA + 6%Ethanol	11.80 ± 1.62 ^{a,c,d,f}
8	2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)	8.20 ± 1.32 ^{b,c}
9	5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)	8.80 ± 1.24 ^{b,c}

a= statistically significant (p<0.05) when compared with group 1 b= statistically significant (p<0.05) when compared with group 2 c= statistically significant (p<0.05) when compared with group 3 d= statistically significant (p<0.05) when compared with group 4 e= statistically significant (p<0.05) when compared with group 5 f= statistically significant (p<0.05) when compared with group 6 g= statistically significant (p<0.05) when compared with group 7 h= statistically significant (p<0.05) when compared with group 8 i= statistically significant (p<0.05) when compared with group 9 SA= Sodium Arsenite PCE= Polychromatic Erythrocyte

Table 3: Induction of micronucleated polychromatic erythrocytes (mPCEs) in rat bone marrow cells after exposure to sodium arsenite and ethanol mean ± SE for n=5.

Group	Treatment	Activity (µmole of substrate hydrolyzed/min/g of tissue)×10 ⁻²
1	Distilled Water	6.44 ± 0.78 ^{b,c}
2	2.5mg/kg Body Weight SA	1.77 ± 0.85 ^{a,c,d,g}
3	5mg/kg Body Weight SA	0.93 ± 0.38 ^{a,c,d,g}
4	3% Ethanol	12.63 ± 5.97 ^{a,b,c}
5	6%Ethanol	6.95 ± 2.88
6	2.5mg/kg Body Weight SA + 3% Ethanol	7.78 ± 4.21
7	5mg/kg Body Weight SA + 6%Ethanol	13.73 ± 4.09 ^{a,b,c}
8	2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)	4.96 ± 1.45
9	5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)	7.88 ± 2.91

a= statistically significant (p<0.05) when compared with group 1 b= statistically significant (p<0.05) when compared with group 2 c= statistically significant (p<0.05) when compared with group 3 d= statistically significant (p<0.05) when compared with group 4 e= statistically significant (p<0.05) when compared with group 5 f= statistically significant (p<0.05) when compared with group 6 g= statistically significant (p<0.05) when compared with group 7 h= statistically significant (p<0.05) when compared with group 8 i= statistically significant (p<0.05) when compared with group 9 SA= Sodium Arsenite PCE= Polychromatic Erythrocyte

Table 4: Acetylcholine esterase activity mean ± SE for n=5, in the brain of the rats after exposure to sodium arsenite and ethanol.

between the groups were analyzed by one-way analysis of variance (ANOVA) with the aid of Statistical Package for Social Sciences (SPSS) software, SPSS Inc., Chicago, Standard version 10.0.1. P-values < 0.05 were considered statistically significant for differences in mean using the Least of Significance Difference (Lsd).

Results

The body weight results from table 1, shows a significant (p<0.05) decrease in % weight change in a concentration dependent manner with concomitant significant (p<0.05) increase in the groups treated with sodium arsenite and ethanol together. From table 1 there are no significant (p>0.05) difference on the organs and relative organs weight.

Based on the activity of the liver enzymes (ALP, ALT and AST) from

table 2, the interaction of ethanol and sodium arsenite significantly (p<0.05) decrease the activity of the liver enzymes as compared with the groups treated with sodium arsenite, ethanol alone. This might suggest the fact that ethanol is suppressing the effect of sodium arsenite.

As depicted in table 3, it was observed that administration of ethanol and sodium arsenite significantly (p<0.05) decreased the number of micronuclei/1000PCE as compared with the groups treated with sodium arsenite, ethanol respectively, indicating the fact that the ethanol administration suppressed the clastogenic effect of sodium arsenite. This might further suggest an interaction between sodium arsenite and ethanol which led to the observed anticlastogenic activity as sodium arsenite is a known clastogen.

From table 4, the activity of acetylcholine esterase in the brain was significantly ($p < 0.05$) inhibited by sodium arsenite in a concentration dependent manner. At low concentration ethanol did not significantly ($p > 0.05$) inhibit the activity of the enzyme but did at a higher concentration. However, the co-administration of sodium arsenite and ethanol led to an increased activity of acetylcholine esterase suggesting that the interaction might have caused the enzyme induction.

However, from table 5, most of the haematological parameters PCV, Hb and RBC were significantly ($p < 0.05$) decreased with increased sodium arsenite and ethanol concentration but these were observed to start increasing from group 6 to 9. Furthermore, in terms of WBC it was only group 8 that showed a significant ($p < 0.05$) decrease while others showed no remarkable increase in WBC. This signifies that the simultaneous administration of the toxicants in question has a counteracting effect, thus suggesting that the interaction is possibly having anti-anaemic effect.

Discussion

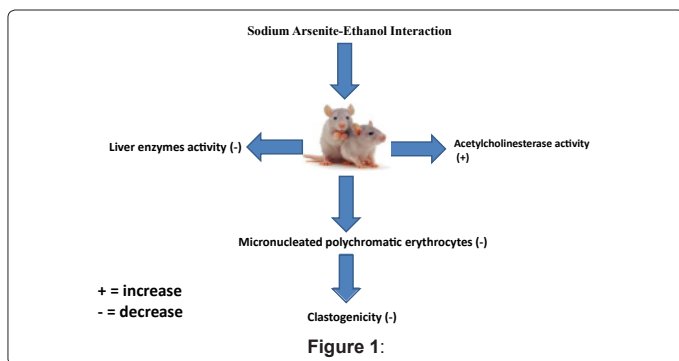
Exposure to arsenite has been linked to diverse defects in both experimental animals and in humans [22-26]. The liver is an important target organ for arsenic toxicity [27]. Arsenic has been claimed to be of clinical utility in the treatment of syphilis, amoebiasis, and certain other tropical diseases [24] and also has been used in Fowler solution in the treatment of arthritis [24], but recently arsenic intoxication in experimental animals has been associated with hepatic tumours [25], the inhibition of testicular steroid genic function [28], and spermatogenesis [26], as well as with severe metabolic disorders such as diabetes in humans [22,23]. It is known that (SA) can act as comutagen due to its ability to inhibit the activities of thiol containing enzymes [27], such as DNA ligase [28] resulting in defective DNA replication,

repair, recombination and joining of single- and double-stranded DNA breaks [29].

Ethanol-induced oxidative stress is as a result of the combined impairment of antioxidant defences and the production of reactive oxygen species by the mitochondrial electron transport chain, the alcohol-inducible Cytochrome P450 (CYP) 2E1 and activated phagocytes [15]. Furthermore, Hydroxyethyl Radicals (HERs) are also generated during ethanol metabolism by CYP2E1. The available evidence indicates that, by favouring mitochondrial permeability transition, oxidative stress promotes hepatocyte necrosis and/or apoptosis and is implicated in the alcohol-induced sensitization of hepatocytes to the pro-apoptotic action of TNF- α . Moreover, oxidative mechanisms can contribute to liver fibrosis, by triggering the release of pro-fibrotic cytokines and collagen gene expression in hepatic stellate cells [30].

This study examines the effect of co-exposure to sodium arsenite and ethanol on male Wister albino rats. The interaction between sodium arsenite and ethanol seem to reverse the effect of decreased body weight. This might be attributed to the fact that ethanol induces fatty liver with enhanced lipogenesis that ultimately lead to an increase in weight [31]. The results of the present study clearly demonstrate that administration of sodium arsenite and ethanol respectively, significantly ($P < 0.05$) induced the formation of micronuclei in the Polychromatic Erythrocytes (PCEs) of the rat bone marrow cells. However, the reversal of that happened when both toxicants were co-administration. This is may be due to the fact that arsenite generates free radicals that can attack DNA leading to chromosomal breakage. In addition, acetaldehyde the end product of ethanol metabolism can form DNA adducts which might also explain why groups treated with sodium arsenite and ethanol respectively were able to induced clastogenicity. The results obtained from the assessment of the serum activities of ALP, ALT and AST shows that their activities increased in a concentration dependent manner. Interestingly, the activities were decreased in the groups of co-administration providing a clue that the chemical interaction between sodium arsenite and ethanol is having a reversal effect. Exposure to sodium arsenite had been shown to induce ALP, AST and ALT activity [32], which is clearly an indication of induction of hepatotoxicity and oxidative stress in the hepatocytes.

Acetylcholine (ACh) is a neurotransmitter that functions in conveying nerve impulses across synaptic clefts within the CNS [33]. Following the transmission of an impulse across the synapse by the release of ACh, AChE is released into the synaptic cleft [34]. This



Group	Treatment	PCV (%)	Hb(g/dL)	RBC(million/ μ L)	WBC $\times 10^9$ /L
1	Distilled Water	37.50 \pm 2.50	12.50 \pm 0.80	7.34 \pm 0.27 ^b	2.56 \pm 0.31
2	2.5mg/kg Body Weight SA	36.20 \pm 2.13	11.35 \pm 0.35	5.62 \pm 0.29 ^{a,c}	3.00 \pm 0.35
3	5mg/kg Body Weight SA	35.00 \pm 1.64	11.00 \pm 0.70	6.72 \pm 0.31 ^b	3.20 \pm 0.54
4	3% Ethanol	31.40 \pm 2.42	10.05 \pm 1.95	5.64 \pm 0.24 ^{a,c}	2.50 \pm 0.35
5	6% Ethanol	28.00 \pm 1.73 ^b	9.23 \pm 0.52 ^a	5.62 \pm 0.33 ^{a,c}	2.90 \pm 0.58
6	2.5mg/kg Body Weight SA+3% Ethanol	34.50 \pm 1.50	11.00 \pm 1.00	6 \pm 0.34 ^a	2.30 \pm 0.25
7	5mg/kg Body Weight SA+6% Ethanol	39.00 \pm 3.39	14.05 \pm 0.95 ^c	6.46 \pm 0.2 ^{a,b}	2.75 \pm 0.25
8	2.5mg/kg body weight Sodium arsenite using 3% Ethanol as solvent (v/v)	34.50 \pm 2.50	13.85 \pm 0.85	6.96 \pm 0.31 ¹	1.30 \pm 0.25 ^{a,b,c}
9	5mg/kg body weight Sodium arsenite using 6% Ethanol as solvent (v/v)	28.20 \pm 2.35 ^{a,b,c}	7.80 \pm 0.70 ^{a,b,c}	5.2 \pm 0.51 ^{a,c}	2.25 \pm 0.25

a= statistically significant ($p < 0.05$) when compared with group 1 b= statistically significant ($p < 0.05$) when compared with group 2 c= statistically significant ($p < 0.05$) when compared with group 3 d= statistically significant ($p < 0.05$) when compared with group 4 e= statistically significant ($p < 0.05$) when compared with group 5 f= statistically significant ($p < 0.05$) when compared with group 6 g= statistically significant ($p < 0.05$) when compared with group 7 h= statistically significant ($p < 0.05$) when compared with group 8 i= statistically significant ($p < 0.05$) when compared with group 9 SA= Sodium Arsenite WBC= White Blood Count PCV= Packed cell volume Hb= Haemoglobin RBC=Red blood cells

Table 5: Results of Heamatological parameters mean \pm SE for n=5, of rats after exposure to sodium arsenite and ethanol.

enzyme hydrolyzes ACh to choline and acetate and transmission of the nerve impulse is terminated [35]. The same scenario surfaced on acetylcholine esterase activity being suppressed as concentration of the toxicants increased and elevated in the co-administered groups; probably the interaction might have initiated an enzyme induction as the activity of AChE is vital to neurological functions. Similarly, based on the hematological parameters single and co-administered groups were depicting an antagonistic effect. This suggests the possibility of an interaction between sodium arsenite and ethanol to have a stabilizing effect on the levels of hematological parameters as the toxic compounds like CCl_4 , arsenic and ethanol has been found to negatively affect the levels of these parameters [36]. It is against this background we proposed that the reaction between sodium arsenite and ethanol in the presence of water generates dimethyl hydroxyl arsenous acid which ultimately make it more polar and as such easily excreted without necessarily causing harm to the system.

Conclusion

We therefore conclude that the chemical interaction between sodium arsenite and ethanol is what is responsible for the suppression of sodium arsenite – induced clastogenic, hepatotoxic, anti-acetylcholine esterase and anemic effects by ethanol. This might be reason why most people that are addicted to alcoholics don't easily come down with arsenic poisoning even though they are exposed to contaminated drinking water. We recommend further molecular studies in this regard.

Potential Conflicts of Interest

There are no conflicts of interest.

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