An Investigation on some Hepatic Enzymes and Haematological Variables among Alcoholic Volunteers in Kadima District of Jos South Local Government Area of Plateau State, Nigeria

Yohanna Denkok[1*], Samuel Y. Gazuwa[1] and Ishaya Y. Longdet[1]

[1]*Department of Biochemistry, Faculty of Basic Medical Sciences, University of Jos, P. M.B. 2084, Jos Plateau State, Nigeria.*

Authors' contributions

Article Information

Original Research Article

ABSTRACT

Aim: This research work investigated the impact of the differences in duration of alcoholic beverage consumption on hepatic and heamatological parameters.
Methods: Fifty (50) healthy male volunteer subjects were recruited for this study from Kadima district of Jos South Local Government Area of Plateau State, Nigeria and were coded according to the years of alcohol consumption: 1-7, 8-14, 15-21, and 22-28 for groups B, C, D and E respectively with each having ten (10) volunteers. Group A was christened as the control having human subjects that have neither drank *Burukutu* nor factory- based lager beer. Full blood count was done using haematology analyser while spectrophometric method was used to assay for the activities of AST, ALT, ALP, GGT as well as various levels/concentrations of TB, DB, TP, urea and creatinine.
Results: Result obtained showed that AST and ALP were raised ($p < 0.05$) in all the groups when compared to the control. ALT was elevated in group E only while GGT was increased ($p < 0.05$) in groups D and E. Total and direct bilirubin were both elevated ($p < 0.05$) in groups B, C and E but

_____

*Corresponding author: E-mail: ydenkok@gmail.com;

equal control in group D. Total protein was higher in groups B and C but lowered in groups D and E relative control. Concentrations of urea was statistically significant ($p < 0.05$) in groups C and E. Creatinine was only statistically significant in group C but equal control in other groups. Results of the haematological parameters showed that RBC and WBC were reduced in all the groups' relative control. Hb concentration was increased ($p < 0.05$) in groups C, D and E. PCV was found to be reduced in groups B, C and D when compared to the control.

Conclusion: This study has shown that prolong ingestion of local brew impacts negatively on hepatic biomarkers as well as some haematological indexes like WBC, RBC and PCV.

1. INTRODUCTION

Beer is the world's oldest and most widely consumed alcoholic beverage and the third most popular drink after water and tea. The name itself was derived from the Latin word *bibere*, which means "to drink" [1]. Brewing is the production of beer by steeping a starch source, commonly cereal grains, in water and fermenting the resulting sweet liquid with yeast [2].

Burukutu is an indigenous locally brewed alcoholic beverage of vinegar-like flavor prepared from sorghum grains [3]. In *burukutu* production, the grains are usually steeped for about 18 – 24 hours at 30°C and germination takes place at 30°C for 5 days. Kilning is carried out under the sun or in the oven at 50°C within 24 hours [4] after which milling of the grain is done and the milled malt flour is mixed with water. This is followed by vigorous boiling for 3-4 hours, here after, it is allowed to cooled. The cooled slurry is filtered and backed sloped followed by another boiling for 3-4 hours again; cooling is also observed and starter from previous brew is then added. The cooled product is then filtered and allowed to ferment for 12-14 hours thereby giving rise to the resultant product that is called burukutu beer [5].

It is widely consumed as food (because it is heavy and thick) in the rural population of northern and central Nigeria as well as poor urban centers due to its affordability compared to commercially brewed beer [6].

Burukutu contains almost all essential amino acids in required proportion except cysteine and tryptophan which are being completely destroyed by heat during boiling [7]. Traditionally, burukutu is brewed with red or white sorghum variety and/or maize malts [8]. It has a sour taste resulting from the action of the lactic acid bacteria (*Lactobacillus spp.*) and opaque colour because of suspended solid and yeast materials

with thin consistency [9]. The production process of burukutu is time consuming, complex and sometimes carried out under unhygienic conditions. Back slopping (the addition of an old fermented batch of a previous brew to serve as an inoculum) is usually done to hasten the fermentation process. Burukutu has a very short shelf-life and is expected to be consumed within five days after back slopping. It can however stay much longer (about a week or two) if not back slopped, and tightly covered [10]. Bukurutu was said to contain alcohol concentration of 3.7-4.9% (v/v) [11].

Contaminants have been reported to be present in foods including local alcoholic drinks. These contaminants could be organic or inorganic elements such as copper (Cu), iron (Fe), manganese (Mn) and zinc (Zn) [12]. Underprivileged and poor natives, mostly women, use water from the mining ponds and wells for domestic and routine preparation of native beers which are sources of some of these heavy metals. Zinc toxicity for example has been reported to impair immune function; inhibit the absorption of copper, calcium, iron and magnesium and promote folate deficiency [13]. The production of burukutu involves the application of some antiquated techniques which could also lead to the generation of undesirable organic metabolites like hexadecanoic acid, ethyl docosanoate and other higher alcohols in the beers which could themselves also impose some level of toxicity to the system [14].

Alcohol is metabolized in the liver. Elevated consumption of alcohol posts a major risk factor for the development of liver fibrosis, alcohol liver disease (ALD), and hepatocellular carcinoma (HCC) [15]. Alcohol-dependent induction of cytochrome P 450 2E1 (CyP2E1) leads to formation of acetaldehyde [16]. CYPE1-dependent alcohol metabolism leads to increased hepatic oxidative stress due to the production of reactive oxygen species (ROS)

including hydroxyethyl radicals [17]. Excess (ROS) inflict injury on the parenchyma cells of the liver, leading to release of enzymes and other biomarkers which can be detected in body fluids especially serum [18].

The aims of this study were to investigate the impacts of alcoholic beverage consumption on hepatic and hematological parameters based on the disparity in duration of consumption of alcoholic beverage among 50 volunteer subjects in Kadima district of Jos South Local Government Area of Plateau State, Nigeria.

2. MATERIALS AND METHODS.

Location: This study was carried out in Kadima district of Jos South Local Government Area of Plateau State, Nigeria.

Subjects: Apparently 50 healthy male volunteers between the ages of 20-65 years were recruited for this research work.

2.1 ETHICAL APPROVAL

The ethical approval for this research work was granted by the ethical committee on research of the Plateau State Specialist Hospital, Jos Plateau State, Nigeria.

2.2 Experimental Design

Fifty (50) healthy male human volunteer subjects between the ages of 20 -65 years were recruited for this study and were coded into 5 groups: A, B, C, D and E. Since the study was solely volunteer in nature, as a result, informed consent form was administered to volunteered subjects to obtain their approval for this study. Questionnaires were also applied to collect the volunteer's bio-data and the history of their drinking habit.

Group 1 10 volunteers who have neither drank burukutu or any other form of alcohol beverage

Group 2 10 volunteers who have been consuming burukutu between 1-7 years

Group 3 10 volunteers who have been consuming burukutu between 8- 14 years

Group 4 10 volunteers who have been consuming burukutu between 15-21 years

Group 5 10 volunteers who have been consuming burukutu between 22-28 years

2.3 Sample Collection

Vacutainer closed system of venous blood collection was used for sample collection. 5ml of blood was carefully collected from each of the subjects at the forearm into a plain tube avoiding venoustasis and was allowed to clot at room temperature. Samples were then placed in a cooler and transported to the laboratory. They were spun at about 3500rpm for 10minutes within one hour of sample collection. The serum was aspirated using Pasteur pipettes into the micro vials for the analysis of analytes of interest.

2.4 Biochemical Analysis

Heamatological Parameters: The haematological parameters were analyzed using haematology auto analyzer.

Total protein: Total protein was estimated using the biuret method.

Measurement of bilirubin (Total and direct bilirubin): Total and Direct bilirubin were determined using spectrophometric method as described by Jendrassic and Grof (1938).

Aspartate Amino Transferase (AST): AST was determined by the method of Reitman and Frankel (1957).

Alanine Amino Transferase (ALT): ALT was determined by the method of Reitman and Frankel (1957).

Alkaline Phosphatase: (ALP): ALP was determined of method Englehart (1970).

Gamma glutaryl (GGT): GGT was determined by the method of Heersink and Hafkenscheid (1980).

2.5 Statistical Analysis

Data was analyzed using instat3 software. One way analysis of variance (ANOVA) was used for comparison of different groups and values were considered significant at $P < 0.05$. Results were presented as the means ± SEM.

3. RESULTS

Effect of Alcoholic Beverage on Human Volunteer Subject in Kadima of Jos south Local Government Area of Plateau State.

The result in table one shows that total bilirubin in groups A, B, C, D and E were found to be 0.55±0.169, 1.13±0.185, 1.41±0.211, 0.58±0.209 and 2.82±1.442, respectively. Total bilirubin was significantly higher (p < 0.05) in group B, C and E when compared to control but equal to control in group D. Direct bilirubin in groups A, B, C, D and E were found to be 1.16±0.188, 2.23±0.786, 1.60±0.305, 1.19±0.189 and 3.63±1.493 respectively. Direct bilirubin was significantly higher (p < 0.05) in groups B, C and E when compared to the control group but equal to control in group D. Total protein in groups A, B,

C, D and E were found to be 66.01±5.242, 66.57±5.367, 68.28±5.113, 61.74±1.931 and 65.50±1.030 respectively. Total Protein was significantly higher (p < 0.05) in groups B, and C but significantly lower (p < 0.05) in groups D and E when compared to control.

The result in table two shows that AST in groups A, B, C, D and E were found to be 11.00±1.224, 22.64±7.602, 23.17±6.248, 34.23±3.477 and 16.41±0.770 respectively. ALP was also found to 30.05±2.925, 42.62±11.544, 66.85±9.639, 184.00±15.182 and 89.24±32.790 for groups A, B, C, D and E respectively. These results show that both AST and ALP were significantly increased (p < 0.05) in all the groups relative control group. ALT in groups A, B, C, D and E were found to 11.13±2.772, 6.66±0.881, 8.66±1.202, 5.33±1.856 and 11.50±2.500

Table 1. Effect of variation in the duration of alcoholic beverage consumption on hepatic function

Groups	TB(µmol /L)	DB(µmol /L)	TP(g/L)
Group A (control)	0.55±0.17	1.16±0.19	66.01±5.24
GROUP B	1.13±0.19[b]	2.23±0.79[b]	66.57±5.37[b]
GROUP C	1.41±0.21[b]	1.60±0.31[b]	68.28±5.11[b]
GROUP D	0.58±0.21[d]	1.19±0.19[d]	61.74±1.93[a]
GROUP E	2.82±1.44[b]	3.63±1.49[b]	65.50±1.03[a]
p-values	0.06	0.13	0.87

*Values are expressed as mean ± SEM, n = 4. If p value is less than 0.05, there is significant difference in mean values; [a] Values are significantly low when compared with control (p < 0.05); [b] Values are significantly high when compared with control (p < 0.05); [c] Values are equal to control; [d] Values are not significantly different when compared with control (p < 0.05).; *Statistically significant from the control (p<0.05) using one-way ANOVA*
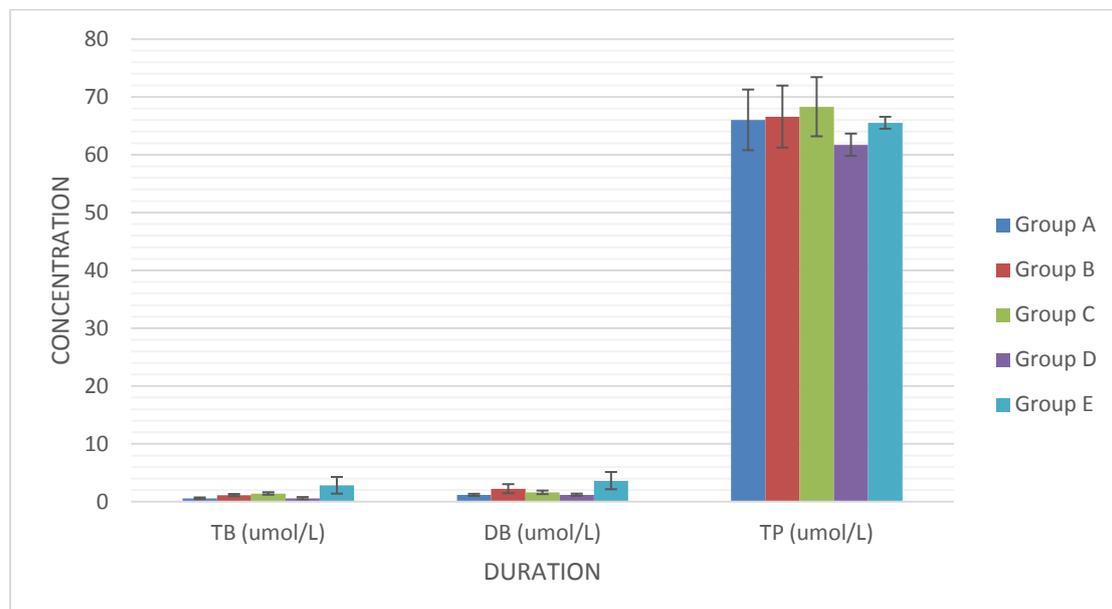
respectively. ALT values are only significantly higher ($p < 0.05$) in group E but lowered in the other groups. GGT in groups A, B, C, D and E were found to 3.18±0.289, 2.99±0.950, 1.68±0.342, 4.53±0.920 and 6.08±3.474 respectively. This shows the GGT values were only elevated in groups D and E but lowered in other groups when compared to the control group.

The result in table four shows that RBC in groups A, B, C, D and E were found to 5.45±0.059, 2.79±0.406, 3.04±0.528, 3.20±0.404 and 2.99±1.390 respectively. Mean WBC concentration was also found to be 5.56±1.598, 2.90±0.606, 4.46±0.483, 3.97±0.624 and

3.52±2.375 respectively. These results show that both RBC and WBC levels were lowered in all the groups relative control.

The result also shows that haemoglobin in groups A, B, C, D and E were found to 127.67±2.728, 121.33±6.984, 174.67±29.672, 147.33±10.088 and 132.50±4.500 respectively. Heamoglobin concentration is elevated in all the groups ($p < 0.05$) but lowered in group B.PCV in groups A, B, C, D and E were found to be 46.03±0.088, 40.00±2.000, 41.33±4.667, 42.33±0.333 and 46.00±3.000 respectively. PCV values were lowered in all the groups but equal to control in group E.

Table 2. Effect of variation in the duration of alcoholic beverage consumption on hepatic enzymes

GROUPS	AST (U/L)	ALT (U/L)	ALP (U/L)	GGT (U/L)
Group A (control)	11.00±1.22	11.13±2.77	30.05±2.93	3.18±0.29
Group B	22.64±7.60[b]	6.66±0.88[a]	42.62±11.54[b]	2.99±0.95[a]
Group C	23.17±6.25[b]	8.66±1.20[a]	66.85±9.64[b]	1.68±0.34[a]
Group D	34.23±3.48[b]	5.33±1.856[a]	184.00±15.182[b]	4.53±0.920[b]
Group E	16.41±0.77[b]	11.50±2.50[b]	89.24±32.79[b]	6.08±3.47[b]
p-values	0.08	0.19	0.01	0.22

*Values are expressed as mean ± SEM, n = 4. If p value is less than 0.05, there is significant difference in mean values; [a] Values are significantly low when compared with control ($p < 0.05$); [b] Values are significantly high when compared with control ($p < 0.05$); *Statistically significant from the control ($p<0.05$) using one-way ANOVA*
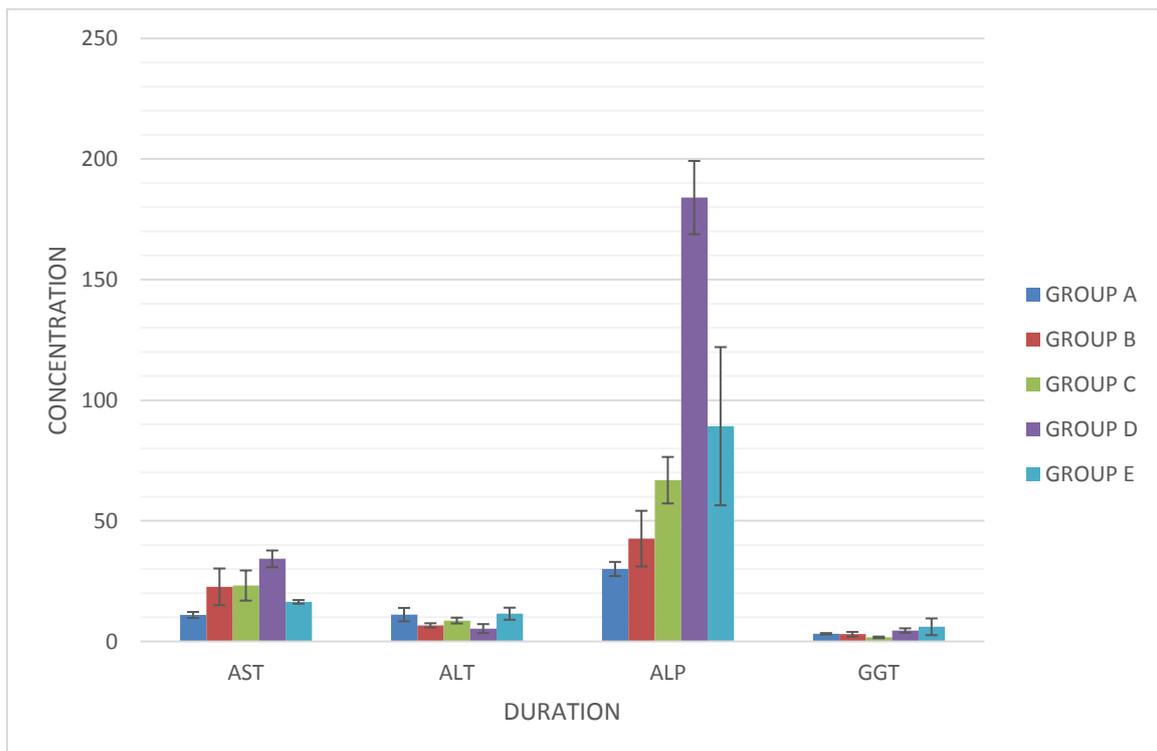
Table 3. Effect of variation in the duration of alcoholic beverage consumption on haematological parameters

GROUPS	LYMPH	MID	GRAN	LYMPH%	MID%	GRAN%
Group A (Control)	2.85±0.80	0.53±0.26	2.00±0.60	50.90±1.23	12.00±3.36	37.06±3.931
Group B	0.72±0.16[a]	0.37±0.09[a]	1.81±0.36[a]	24.60±0.95[a]	12.56±0.52[b]	62.83±1.27[b]
Group C	2.41±0.45[a]	0.42±0.19[a]	1.62±0.13[a]	54.16±5.44[b]	8.96±0.85[a]	36.86±6.26[a]
Group D	1.88±0.26[a]	0.49±0.04[a]	1.59±0.32[a]	54.79±3.10[b]	12.78±2.70[b]	27.75±3.58[a]
Group E	1.39±1.02[a]	0.88±0.73[b]	1.25±0.63[a]	45.10±13.20[a]	8.35±5.05[b]	46.55±8.15[b]
p-values	0.13	0.77	0.81	0.01	0.66	0.003

*Values are expressed as mean ± SEM, n = 4. If p value is less than 0.05, there is significant difference in mean values; [a]Values are significantly low when compared with control (p < 0.05); [b]Values are significantly high when compared with control (p < 0.05); *Statistically significant from the control (p<0.05) using one-way ANOVA*
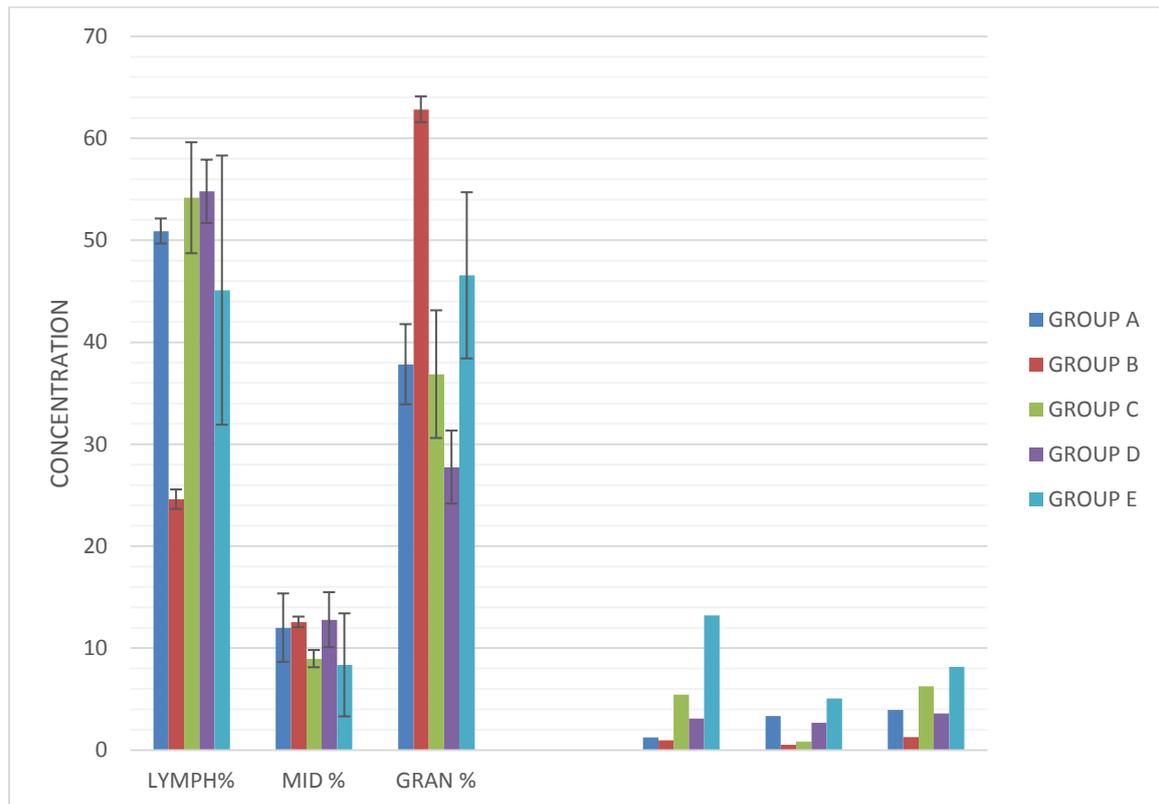

Table 4. Effect of variation in the duration of alcoholic beverage consumption on haematological parameters

Groups	RBC(/mm^3)	Hb(g/dl)	PCV(%)	WBC(mm^3)X10^3
Group A (Control)	5.45±0.06	127.67±2.73	46.03±0.09	5.56±1.60
Group B	2.79±0.41[a]	121.33±6.98[a]	40.00±2.00[a]	2.90±0.61[a]
Group C	3.04±0.53[a]	174.67±29.67[b]	41.33±4.67[a]	4.46±0.48[a]
Group D	3.20±0.40[a]	147.33±10.09[b]	42.33±0.33[a]	3.97±0.62[a]
Group E	2.99±1.39[a]	132.50±4.50[b]	46.00±3.00[d]	3.52±2.38[a]
p-values	0.03	0.20	0.43	0.54

*Values are expressed as mean ± SEM, n = 4. If p value is less than 0.05, there is significant difference in mean values; [a]Values are significantly low when compared with control (p < 0.05); [b]Values are significantly high when compared with control (p < 0.05); [d]Values are not significantly different when compared with control (p < 0.05); *Statistically significant from the control (p<0.05) using one-way ANOVA*

Table 5. Frequency and concurrent ingestion of burukutu with other factory based lager beer

Groups	Consumers of Burukutu only	Consumers of Burukutu with other beer(s)	Frequency of consumption of Burukutu	Quantity consumed per day(litres)	Nutritional factors among volunteers	History of peptic ulcer among volunteers
GROUP A	Not Applicable	Not Applicable	Not Applicable	Not Applicable	Good	Nil
GROUP B	4	6	Daily	5 litres a day	Moderate	4
GROUP C	6	4	Twice a day	7 litres a day	Poor	2
GROUP D	7	3	Once a day	3 litres a day	Poor	4
GROUP E	2	8	Once a day	3 litres a day	Moderate	1
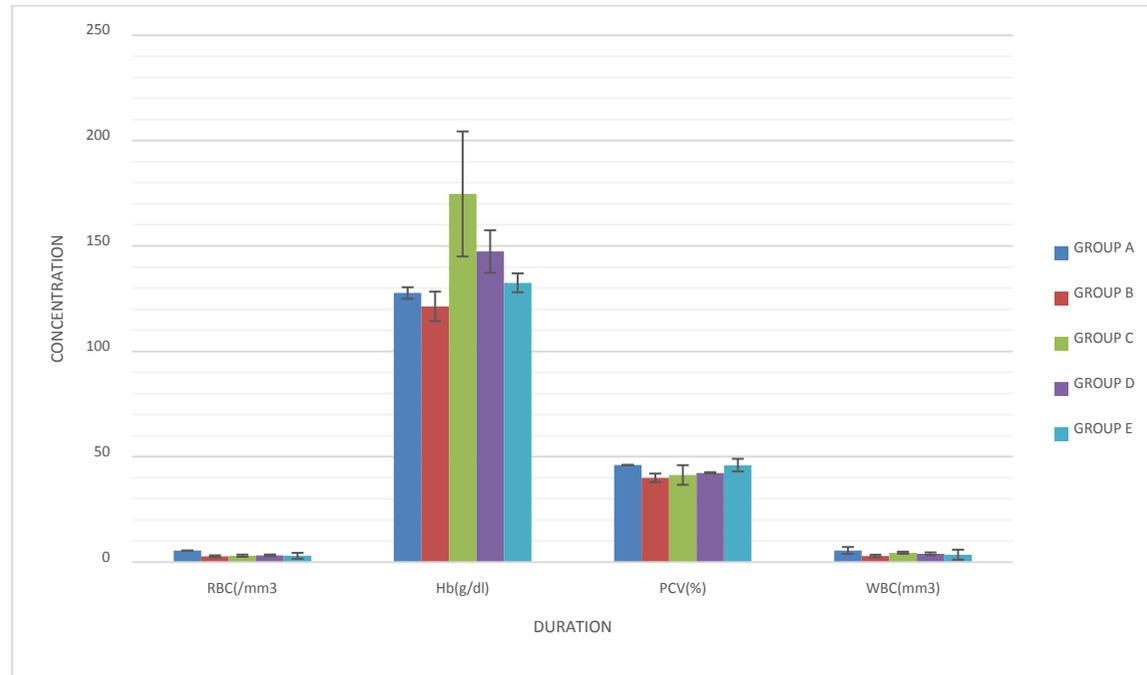
4. DISCUSSION

This research work presents an experimental study on the effects of the differences in duration of alcoholic beverage consumption among fifty (50) different human subjects on hepatic and hematological parameters in Kadima district of Jos South Local Government Area of Plateau State, Nigeria. From the result obtained in table (1), total and direct bilirubin were significantly elevated ($p < 0.05$) with an increase in the number of years the volunteer subjects were involved in alcoholic beverage consumption. Total bilirubin abnormalities in chronic alcoholics may result from isolated or combined increase in the non-conjugated and conjugated fractions. An increase in the conjugated fraction always denotes hepatocyte damage [19]. This research work is in tandem with the findings of [20] which also reports that the total and direct bilirubbin increase is proportion to the length or duration in years of alcohol consumption.

Total protein was also found to be statistically elevated in groups B and C but lowered in groups D and E from our investigation. The reduction in total protein was investigated in individuals that have prolonged history of alcoholic beverage consumption. This strongly agrees with the findings of [21] who had earlier reported that heavy alcoholic beverage consumption affect protein synthesis as well as that of [22] who also found out that a progressive decrease in protein could be attributed to prolonged alcohol consumption.

ALP levels were statistically higher ($p < 0.05$) in all the groups' relative control group. The significant increase in ALP among volunteer subjects was traceable to the impact of alcohol concentration in burukutu on the liver hepatocytes since serum ALP activities was mainly from the liver with 50% contribution by the bones [23]. This is because alcohol is known to inflict injury on the liver through oxidative stress resulting from the breakdown of its metabolic products especially acetaldehyde [24]. The effect of chronic consumption of alcohol has been reported to have direct impact on bone cells by decreasing the number of osteoblasts, osteoid formation and osteoblasts proliferation as well as indirect effects through its action on mineral regulatory hormones. This development affects osteoblastic activity and bone remodeling resulting in low generation of bone isoform of ALP which is a biomarker of bone formation [25]. This finding is in line with the investigation of [26]

who also reported an increase in the activities of ALP among incessant burukutu consumers.

AST was significantly elevated in chronic burukutu consumer's groups B, C, D and E as against the control subjects. The significant AST increase observed in burukutu subjects in this study, points to the capacity of chronic alcohol consumption to induce enzyme leakage from the hepatocytes especially in alcoholic hepatitis as reported by [27]. Alcohol increases mitochondrial AST activity in plasma, whereas other causes of hepatitis typically do not [28]. It does so by inducing the release of mitochondrial AST from cells without visible cell damage [29].

From the result in table 1, the values of ALT were reduced in groups B, C and D but increased in group E relative control. This trend shows that alcohol impact more on the body's system with a commensurate increase in the duration and years of consumption. This work is in line with the finding of [30] which also reports an increase in ALT values with increase in timeline of burukutu consumption. This enzyme, ALT is one of the hepatic biochemical markers which are released into blood circulation as a result of injury to the liver by one or more factors. One factor that can lead to elevated level of these enzymes is excessive and continuous alcohol intake [31].

GGT is extremely sensitive to alcohol use and serum GGT is one of the best markers for chronic alcohol consumption, which has a relatively high sensitivity and specificity [32]. The serum GGT is markedly elevated in patients with alcoholic liver disease and primary biliary cirrhosis whereas mean hepatic GGT is significantly elevated only in the alcoholic liver disease group [33].

From the result of our investigation, the values of GGT were lowered in groups B and C but increased ($p < 0.05$) in groups D and E relative control. This increase in values of GGT with increase in consumption of alcohol actually synchronizes with the investigations of [34]. The trend and progression in the values of GGT shows clearly that prolong alcohol consumption impact on the liver. Serum GGT is the most sensitive, most widely employed marker of alcohol consumption. It is more likely to be elevated in regular rather than episodic drinkers [35]. An isolated rise in serum GGT is seen in patients with alcohol abuse, even without liver

disease perhaps because of microsomal enzyme induction [36].

The results of this study have shown that ethanol and its derivatives have the capacity to induce hematotoxic effects. The changes in haematological parameters and red blood cell indices provide useful information on the general state of blood after such exposure to exogenous insult. Result obtained from table (4) shows that RBC was significantly reduced in all the groups when compared to the control. This reduction in RBC may be due to proportionate decreased in haemoglobin as obtained from the outcome of our findings. This investigation agrees with the findings of [37].

The result obtained in table (4) shows a decreased in WBC counts among alcoholic volunteered subject relative control. The outcome of our investigations is line with report of [38]. The values of the packed cell volumes were also reduced in the alcoholic beverage consumers. This investigation is not in tandem with the report of [39] which reported that there was no statistical significance in PCV among consumers of alcohol which they suggested that stratifying the study subjects according to the frequency and duration of alcohol consumption may reveal a clearer picture of the effect of excessive and prolonged alcohol intake on haematology.

5. CONCLUSION

This study has shown that intake of alcoholic beverage like *burukutu* over a long period of time impacts negatively on the hepatic functions. Liver biomarkers enzymes like AST, ALT, ALP and GGT were found to be elevated with prolonged consumption of this alcoholic beverage. Haematological parameters were decreased from this study; a reduction in RBC, Hb and percentage PCV are indicators of anaemia. Concentration of WBC was also decreased among volunteer subjects which might drastically impair on their ability to fight infections. Consumption of *burukutu* for a prolong period, especially as observed in our study area should be discouraged or done with some level of control, as this sociocultural practice can cause a deleterious effect on the liver as well as leading to anaemic condition.

CONSENT

As per international standard or university standard, Participants' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

The ethical approval for this research work was granted by the ethical committee on research of the Plateau State Specialist Hospital, Jos Plateau State, Nigeria.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1.   Okafor N. Modern Industrial Microbiology Biotechnology. Enfield (NH): Science Publishers; 2007.
2.   Evans E. The oxford companion to beer. Oxford University Press. 2011;236.
3.   Kolawole OM, Kayode MO, Akinduyo B. Proximate and microbial analyses of burukut and pito produced in Ilorin, Nigeria. Africa Journal of Biotechnology. 2007;6:587-590.
4.   Kingsley CE, Victor E. EA kinetic study of burukutu fermentation. Journal of Engineering and Applied Sciences. 2007;2(7):1193-1198.
5.   Ezeonu Chukwuma Stephen, Nwokwu Chukwumaobim Daniel U, Kadiri Bashir. Comparative physico-chemical analysis of locally brewed beer (Burukutu) from Corn, Millet and Sorghum. American Journal of Science and Technology. 2017;4(3):43-48.
6.   Egemba KC, Etuk VEA. Kinetic study of Burukutu fermentation. Journal of England Apple Science. 2007;2:1193-1198.
7.   Odetokun SM. Practice guideline committee of the american association for the study of liver diseases and practice parameters committee of the American College of Gastroenterology. Mol. Nutr. Food Res. J. 41(6):375-377.
8.   Lyumugabe F, Gros J, Nzungize J, Bajyana E, Thonart P. Characteristics of African traditional beers brewed with sorghum malt: a review. Biotech. Agro. Soc. Environ. 2012;16(4): 509- 530.
9.   Ogunbanwo ST, Adewara, AO, Fowoyo PT. Effect of fermentation by pure cultures of *Lactobacillus fermentum* and *Saccharomyces cerevisiae* as starter cultures in the production of 'burukutu'. New York Sci. J. 2013;6(1):73–81.
10. Adewara AO, Ogunbanwo ST. Effects of processing variables on the production of

'burukutu', a Nigerian fermented beverage. Nature Sci. 2013;11(1):16–28.

11. Gazuwa SY, Denkok Y. Organic Metabolites, Alcohol Content, and Microbial Contaminants in Sorghum-based Native Beers Consumed in Barkin Ladi Local Government Area, Nigeria. Journal of Scientific Research & Reports. 2017;17(4):1-8.

12. Gazuwa SY, Dabak JD, Ubom GA. Iron contamination of two local alcoholic drinks. Journal of Medicine in the Tropics. . 2006;8(1):32-38.

13. Bennet DR, Baird CJ, Chan K, Crookes PF, Bremmer CG, Gottlieb MM, Narikotu W. Zinc Toxicity following Massive Coin Ingestion. American Journal of Medicine and Pathology. 1997;18(2):143-153.

14. Halzelwood LA, Vanmaris AJ. Enrlich pathway for fused alcohol production. Appl Environ. Microbial. 2008;74:2259-2266.

15. Hassan MM, Hwang LY, Hatten CJ, Swaim M, Li D. Risk factors for hepatocellular carcinoma: Synergism of alcohol with viral hepatitis and diabetes mellitus. Hepatology. 2002;36:206-1213.

16. Purohit V, Gao B; Song BJ. Molecular mechanisms of alcoholic fatty liver. Alcohol. Clin. Exp. Res. 2009;33:191-205.

17. Pari L, Karthikesan K. Protective role of caffeic acid against alcohol-induced biochemical changes in rats. Fundaclin. Pharmacol. 2007;21:315-361.

18. McKillop IH, Schrum LW. Role of alcohol in liver carcinogenesis. Seminars in Liver Disease. 29: 222-232.

19. Becker U, Deis A, Sørensen TI, Grønbaek M, Borch-Johnsen K, Müller CF, Schnohr P, Jensen G. Prediction of risk of liver disease by alcohol intake, sex, and age: a prospective population study. Hepatology. 1996;23:1025-9.

20. Elanchezhian, Yoganandh T, Mayilsamy S, Shankar Radhakrishnan. Comparison of haematological parameters between alcoholics and non-alcoholics. International Journal of Research in Medical Sciences. 2017;5(11):5041-5047.

21. Osarentin A, Taiwo E, Chioma LA. Gender and alcohol consumption affect human serum enzymes, protein, bilirubin. Asian Journal of Biochemistry. 2007;2:330-336.

22. Stanley PC. Alcohol: A silent Killer. University of Port Harcourt Press Limited; 2003.

23. Mauro P, Renze B, Wouter P. In: Tietz Textbook of Clinical Chemistry and Molecular Diagnosis.(4th Edition).Carl AB, Edward R, David EB, editors. Elsevier, 2006, Enzymes; 604-616.

24. Alexandre L, Philippe M. Alcohol liver Disease: Mechanism of injury and targeted treatment, Nature Reviews Gastroenterology and Hepatology. 2015;12:231-242.

25. Klein RF, Fausti KA, Carlos AS. Ethanol inhibits human osteoblastic cell proliferation. Alcohol Clin Exp Res. 1996;20:572-578.

26. Uchendu Ikenna Kingsley, Chuntan Pius Jibrin, Agu Chidozie Elochukwu, Orji Oliver Chukwuma, Chukwu John-Paul Ikechukwu, Nnedu Ebuka Bitrus, Okongwu Uzoma Chinenye. Comparative Impact of Chronic Consumption of Burukutu and Beer on Liver Biomarkers in Male Volunteers in Akwanga Lga Narasawa State. Annals of Medical and Health Sciences Research. 2017;7(Special Issue 1);84-91.

27. Uchida T, Koa H, Quispe-Sjogren M, Peters RL. Alcoholic foamy degeneration: A pattern of acute alcoholic injury of the liver. Gastroenterology. 1983;84:683-692.

28. Nalpas B, Vassault A, Le Guillou A, Lesgourgues B, Ferry N, Lacour B. Serum activity of mitochondrial aspartate aminotransferase: A sensitive marker of alcoholism with or without alcoholic hepatitis. Hepatology. 1984;4:893-896.

29. Zhou SL, Gordon RE, Bradbury M, Stump D, Kiang CL. Ethanol up-regulates fatty acid uptake and plasma membrane expression and export of mitochondrial aspartate aminotransferase in Hep G 2 cells. Hepatology. 1998;27:1064-1074.

30. Gazuwa SY, Dabak JD and Ubom GA. Contaminants in local alcoholic beverages: zinc and manganese contamination. Int. J. Biol. Chem. Sci. 2008;2(4):411-416.

31. Asma S, Kamel K, MedSaleh B. Ethanol exposure induces liver and kidney injury in adult male rabbits. Annals of Biological Research. 2014;5(4):23-26.

32. Bosma PJ, Chowdhury JR, Bakker C, Gantla S, Boer A, Oostra BA, et al. The genetic basis of the reduced expression of bilirubin UDP glucuronosyltransferase 1 in Gilbert's syndrome. N Engl J Med. 1995;333:1171.

33. Selinger MJ, et al. GGT activity in liver disease. Clin Chim. Acta. 1982;125(3):283-287.

34. Imo C, Uhegbu FO, Ifeanacho NG, Egbeigwe O, Ezekwe AS. Biochemical and histological changes associated with methanolic leaf extract of *Gongronema latifolium* in acetaminophen-induced hepatic toxicity in wistar albino rats. Int. J. Biomol. Biomed. 2014;4(2):1-7.

35. Rosalki S. Identifying the alcoholic. In clinical Biochem of Alcoholism, (Ed. Rosalki S) Churchill, Livingstone, Edinburgh. 1984;65-92.

36. Ireland A, Hartley L, Ryley N. Raised Gamma-Glutamyl-transferase activity and the need for liver biopsy. Br. Med. J. 1991;302:388.

37. Okoroafor C, Ogbo I, Azi S. The effect of alcohol intoxications on hematological parameters of adult albino wistar rats. International Journal of Community Research. 2013;2(3):46-49.

38. Esmeralda Thoma, Sonila Bitri, Klodeta Mucaj, Andrin Tahiri, Irida Pano. Changes of Some Blood Count Variables in Correlation with the Time of Alcohol Abuse. J Addict Res Ther. 2015; 6:2.

39. Moses D. Lugos, Bitrus N. Lekshak, Changjul L. Singnap, Umanka Y. Polit, Benedict H. Mangs. Investigating the impact of alcohol consumption on some haematological variables among alcohol consumers in Jos Metropolis, Nigeria. International Journal of Haematology and Blood Disorder. 2017;3(3):1-6.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle4.com/review-history/70427