



Some Biochemical and Hematological Studies of a Local Indigenous Alcohol Beverage in Male Albino Rats

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Abstract

Goskolo (GSK) is a newly introduced local alcoholic drink on the Plateau with several negative impacts among the youths. Currently, there is paucity of scientific information on the content of GSK and the identity of its content remains unknown to the public. This study is aimed at investigating the Gas Chromatography and Mass Spectrometry, (Chromatographic profiling) of GSK, and its Biochemical, Hematological and Histological effects in adult male Wister Albino rats. Four (4) brands of commercially produced GSK beverages tagged Dark Rum "DR", Alomo Bitters "AB", Swagga Schnnaps "SS" and Swagga Dry Din "SDG" were procured from retail shop (Local suppliers). Sample was analyzed using Gas Chromatography and Mass Spectrometry (GC-MS) method. Fifty (50) adult male Wister Albino rats were randomly divided into groups of ten (10) rats each. Animals were sacrificed by Chloroform inhalation at the end of thirty days. Necroscopy was performed on all the animals. Blood samples were also collected and serum levels of Glucose, Total bilirubin (TB), Alanine Transaminase (AST), Total Protein (TP), Albumin (Alb), Gammaglutamyl Transferase (GGT), Alkaline Phosphatase (ALP), Malondialdehyde (MDA), White Blood Cells (WBC's), Red Blood Cells (RBC's), Packed Cell Volume (PCV), Platelets (Plt), Hemoglobin (Hb), Mean Cell Volume (MCV), Mean Cell Hemoglobin (MCH), Mean Cell Hemoglobin Concentration (MCHC) were all analysed using standard methods. The biochemical parameters revealed marked increased levels of ALT, AST, GGT, and MDA. Particularly among treated animals in groups 4 and 5 when compared to the control (Group 1). However, Creatinine values were significantly increased in group 2, 4 and 5 ($581.18 \pm 1.36\mu\text{mol/L}$; $653.86 \pm 0.59\mu\text{mol/L}$; $560.99 \pm 0.15\mu\text{mol/L}$) as against the control ($531.93 \pm 0.9\mu\text{mol/L}$). Conversely, serum levels of Albumin, Total protein and Glucose were reduced statistically ($p < 0.05$). The hematological variables also revealed significant difference in average values of RBC's, PCV, MCH, MCHC, Hb, Neutrophils and Monocytes which were reduced in various groups. Significantly higher values of WBC ($16.27 \pm 0.09 \times 10^9/\text{L}$; $14.30 \pm 0.06 \times 10^9/\text{L}$) and MCV ($63.70 \pm 0.42\text{FL}$) were seen in animals treated with ("SS") and ("SDG") respectively.

Keywords: Wister Albino Rat; Alomo Bitters; Swagga Schnnaps; Swagga Dry Din; Biochemical; Hematological Parameters

Introduction

Alcohol consumption has occurred for thousands of years. Excessive alcohol use is a global public health problem accounting for about 6% of mortality and 5% of disability adjusted life years lost worldwide [1]. In many parts of the world, an alcoholic beverage is a common feature of social gathering especially in Africa [2]. However, alcohol consumption has health and social consequences via intoxication (drunkenness), dependence (habitual, compulsive and long term drinking), and other biochemical effects. Alcohol Use Disorders Identification Test (AUDIT) score of ≥ 8 is estimated at 4% globally and 3% in Africa, and is generally more prevalent among men than women [1]. In addition to chronic diseases that may affect drinkers after several years of heavy use, Alcohol also contributes to traumatic outcomes that kills or disables one at relatively young age, resulting in the loss of many years of life to death or disability. Alcohol is estimated to cause about 20 to 30% Worldwide, disease of oesophageal Cancer, liver cancer, and cirrhosis of the liver, homicide, epilepsy and motor vehicle accident [3]. Harmful use of alcohol is one of the major factors contributing to premature deaths and avoidable disease burden worldwide and has a major impact on public health. Harmful use of alcohol was estimated to cause about 2.3 Million premature deaths worldwide in 2009 [4]. Alcoholic beverages, and the problems they engender, have been familiar fixtures in human societies. It is very likely that alcohol use and related disorders will increase as a public health problem in Nigeria over the coming years and the global burden of alcohol related illness will continue to rise. In fact, Alcohol consumption has been linked to more than Sixty (60) medical conditions and is presently also linked to categories of disease whose relative impact on the global burden is predicted to continues increases [5].

In Nigeria, psychoactive substance misuse, especially alcohol, which has for many years, been an issue of increasing health and social importance. This is specially so for the critical adolescent period marked by several changes including the psychological phenomenon of experimentation [6].

Research from the United State of America in 2003 has shown that about 500 young people under the age of twenty-one (21) die from alcohol-related injuries each year [7]. An estimated number of 1600 (32%), of these deaths, are fuelled by alcohol through homicide. In 2005, yet another American study, established evidence showed that about 700,000 University students are assaulted each

year by other students who have been under the influence of alcohol [8].

Studies linking youth violence and harmful alcohol use have been conducted in several countries. In Australia, a report released by the government in 2011 stated that young people aged 10-14 years, who had engaged in binge drinking in the previous two weeks were five times more likely to have been violent than non-binge drinkers [9].

Over the years, there has been a proliferation of the Nigerian alcoholic industries with several adulterated brands of beer with its antecedent consequences among productive youths. However, of great concern is that of a locally made gin popularly known as "Goskolo" (GSK). The world over, alcohol consumption continues to be one of the risky behaviours engaged in by adolescent [10]. And therefore, one of the common habits among peer groups that cause physiological and social problems of phenomenal proportions [11].

Jos the capital city of Plateau and its environs, have witnessed in recent times, a rise in indulgence of youths in the consumption of a locally brewed alcohol product popularly called Goskolo (GSK). This has culminated into the premature death of this productive class of its citizen besides the burden of family disruption, violence, and high increase in Crime rate the state has been struggling with over the years [12]. Besides this, it has been speculated that dangerous substances which are injurious to health and whose nature in not known to the general public forms part of GSK drink. This is to say, GSK is suspected to be adulterated with several substances which might be injurious to the health of humans. In view of the aforementioned, and many Medical complications exhibited by the victims of GSK such as hematesis, jaundice, chronic Liver disease; hepato-Splenomegaly; and chronic dehydration; besides the lack of limited official scientific data regarding the true biochemical and hematological effects of GSK on body organs, makes this study imperative.

Materials and Methods

Study area

The study was carried out in Jos. Samples were collected from Tudun Wada area within Jos North Local Government area of Plateau State, Nigeria.

Jos has a land area of about 26,899 Square Kilometers (Km²). It is the most densely populated area in Plateau State with about 900,000 inhabitants [13].

Jos city (latitude 9° 56' North and Longitude 8° 53') is at an elevation of about 1238 meters above sea level and the designated sampling areas are known for high presence and activities of both Guskolo (GSK) retail shops and alcoholics.

Collection of Guskolo (GSK) alcoholic brands

Four (4) different brands of commercially produced Guskolo (GSK) beverage (Alomo bitters-AB; Dark Rum-DR; Swagga Schnnaps-SS; and Swaga Dry Gin-SDG) were procured from retail shops (local suppliers) in the amount of 200mls each as it is easily available in the local market. Samples were stored under refrigeration between 2-8°C and later sent to the laboratory at low temperature for Gas Chromatography and Mass Spectrometry (GC-MS) analysis (data not provided here).

Experimental animals

Fifty (50) healthy adult male Wister albino rats were bred in an animal house of the Department of Pharmacology, University of Jos. The average weight of each animal was 200 ± 30g. They were housed under Standard Laboratory conditions with a 12 hour day light cycle and free access to feed and water; they were made to acclimatize to Laboratory conditions for two weeks before the commencement of the experiment. All experiments were carried out in compliance with the recommendations of Helsinki's declaration on guiding principles of care and use of animals. Ethical clearance for the experiment was also obtained from the University of Jos.

Study Design (Experimental Design)

The Fifty (50) Adult male Wister albino rats were randomly divided into five (5) groups of ten rats each. The five groups of rats were then subjected to the following oral treatments (using a graduated syringe and stainless intubation canula) once a day for thirty days.

- **Group I:** Included ten adult male Wister albino rats.
- They were given a daily oral dose of only distilled water at a dose level of 0.31mls/kg for a period of 30 days.
- **Group II:** Included ten adult male Wister albino rats that were given a daily oral dose of GSK 'AB' at a dose level of 0.31mls/kg of body weight for a period of 30 days.

- **Group III:** Included ten adult male Wister albino rats that were given a daily dose of GSK 'DR' at a dose level of 0.31mls/kg of body weight for a period of 30 days.
- **Group IV:** Included ten adult male Wister albino rats that were given a daily dose of GSK 'SS' at a dose level of 0.31mls/kg of body weight for a period of 30 days.
- **Group V:** Included ten adult male Wister albino rats that were given a daily oral dose of GSK 'SDG' at a dose level of 0.31mls/kg of body weight for a period of 30 days.

Where 'AB', 'DR', 'SDG' and 'SS' were as determined via the LD₅₀ estimated via the up and down method.

All the animals were observed daily for any mortality up to the day 30th for all the groups. Also, the animals were observed for any clinical signs; at least twice daily in order to record any symptoms of ill-health or behavioural changes. Such clinical observations included-changes in skin and fur, in the eyes and mucosa membrane, in the respiratory, circulatory, central nervous and autonomous system and behaviour and were graded as

- 0-No clinical sign
- +-moderate
- +++-high
- ++++-severe (Data not provided)

Also, the body weight of each rat was recorded before the start of experiment and after every week up to the end of the experiment. The mean body weights of different groups were calculated from the individual weights.

Collection of blood samples

Blood samples were collected twenty four hours (i.e. at day 31st) after the last dosing of all the groups).

Blood samples were collected through cardiac puncture in dry tubes for biochemical analysis and in Ethylene Diamine tetra Acetic Acid (EDTA) bottles for Hematological Parameters.

For the Biochemical the blood samples were allowed to clot for 20 minutes before it was centrifuged for 20 minutes using standard bench top centrifuge at 3000g and the supernatant serum was used for Glucose, Total Bilirubin (TB), Alanine Transaminase

(ALT), Aspartate Transaminase (AST), Gamma-Glutamyltransferase (γ -GGT), Total Protein (TP), Albumin (Alb.), Alkaline Phosphatase (ALP), Serum Creatinine (SCr.) and Malondialdehyde (MDA) or separated and kept frozen at -20°C pending analysis. Whereas for Hematological Parameters -Total White Blood Counts (WBC'S), Red Blood cells (RBC'S), Packed Cell Volume (PCV), Platelets, Hemoglobin (HB), Mean Cell Volume (MCV), Mean Cell Hemoglobin (MCH) and Mean Cell Hemoglobin Concentration (MCHC), samples were run immediately to avoid deterioration of samples.

Termination of studies

At the end of the study, the animals were sacrificed by Chloroform Inhalation. Necropsy was performed on them and this included examinations of the external surface of the body orifices, thoracic and abdominal cavities and their content. The organ weight of the kidneys and the liver of each rat were weighted on day 31st for all groups using a metler electronic weighing machine.

Laboratory procedures

All reagents were commercially purchased and the manufacturers Standard Operating Procedures (SOP) strictly adhered to.

Determination of Hematological Parameters

WBC, RBC, HCT, Hb and PLT and the red cell indices were carried out using the MYTHIC 22 CT Haematology auto analyser (5- part differential auto-analyser).

Determination of Biochemical Parameters (Liver and Kidney parameters)

(Transaminases (AST and ALT) Alkaline phosphatase (ALP), Bilirubin (Bil.), Creatinine, (Cr), and Urea. This was done using COBAS CIII chemistry auto analyzer.

Statistical analysis

Data was analysed using one way analysis of variance (ANOVA) followed by Duncan's multiple range test (DMRT). Results were expressed as mean \pm S.D; $P < 0.5$ was taken as accepted level of significance using the Statistical Package for Social Sciences (SPSS) version 21.0.

Results

Table 1 shows a mean and standard deviation value of haematological indices of the various groups of treated male Wister Albino Rat. The table shows significantly ($P < 0.05$) raised levels of Lym-

phocytes and Neutrophils in groups III (DR), IV (SS) and V (SDG) when compared to the control (group I). Mean cell Volume (MCV) was also significantly raised in groups IV (SS). Decrease levels of Neutrophils and Monocytes were recorded in groups IV (SS) and V (SDG) as well as III (DR), IV (SS) and V (SDG) respectively. Hemoglobin (Hb) values showed a steady decline with least values in group IV ($10.16 \pm 0.6\text{g/L}$) and highest value in group III ($13.33 \pm 0.15\text{g/L}$) all of which was significant ($P < 0.05$) when compared to the control ($13.65 \pm 0.08\text{g/L}$).

Table 2 shows a mean and standard deviation value of biochemical parameters of the various groups of treated male Wister Albino Rat as compared with the control. Significant ($P < 0.05$) increase in the levels of Creatinine and Gamma glutamyl transferase were recorded in groups II (AB), IV (SS), and V (SDG) ($581.18 \pm 136 \mu\text{mol/L}$; $653.86 \pm 0.59 \mu\text{mol/L}$ and $560.99 \pm 0.15 \mu\text{mol/L}$) as against the control ($531.93 \pm 0.9 \mu\text{mol/L}$) as well as Malondialdehyde (MDA) in groups IV and V ($27.71 \pm 0.97 \mu\text{mol/L}$ and $25.20 \pm 0.21 \mu\text{mol/L}$) when compared to the control ($22.02 \pm 0.03 \mu\text{mol/L}$). ALT levels were also significantly raised in groups II, III, IV and V ($22.10 \pm 0.06\text{IU/L}$; $22.09 \pm 0.09\text{IU/L}$; $29.56 \pm 0.11\text{IU/L}$ and $27.08 \pm 0.08\text{IU/L}$) respectively when compared to the control ($21.74 \pm 0.18\text{IU/L}$). AST showed level of significance only in groups III, IV and V ($84.42 \pm 0.16\text{IU/L}$; $101.24 \pm 0.09\text{IU/L}$ and $96.68 \pm 0.13\text{IU/L}$) respectively when compared to the control ($83.04 \pm 0.03\text{IU/L}$). Total Protein values recorded significant reduction ($P < 0.05$) from group II to V.

Discussion

Following the administration of GSK, our data showed that RBC count and its associated indices were not affected in group II unlike in group III, IV and V and therefore, it did not differ significantly with respect to the control group. Although only MCV was significantly higher than the control in group IV (macrocytosis). The demonstrated macrocytosis as seen in group IV can be attributed to the enlargement of the RBCs count due to presence of heavy metals accumulation (inform of nano particles in the erythrocytes), thereby causing a marked increase in their size. Ali (2003) [14] reported that the nano particle (Alumina) due to their nano size ($\leq 13\text{nm}$) are highly able to penetrate the cell membranes and accumulate inside the cells, such as RBCs leading to increase in their size and volume (macrocytosis). In addition, the phenomenon may also be considered as hemodilution mechanism to reduce and overcome

| Variables | Control Mean ± SD | Group II (GSK 'AB') Mean ± SD | Group III (GSK 'DR') Mean ± SD | Group IV (GSK 'SS') Mean ± SD | Group V (GSK 'SDG') Mean ± SD | F | DF | P |
|----------------------------|-------------------|-------------------------------|--------------------------------|-------------------------------|-------------------------------|-----------|----|-------|
| Hb (g/dL) | 13.65 ± 0.08 | 13.32 ± 0.04** | 13.33 ± 0.15** | 10.16 ± 0.06** | 12.30 ± 0.12** | 11055.166 | 4 | 0.000 |
| RBC (10 ¹² /L) | 7.38 ± 0.05 | 7.36 ± 0.04 | 7.71 ± 0.02** | 5.53 ± 0.07** | 7.64 ± 0.04** | 2180.093 | 4 | 0.000 |
| WBC (x10 ³ /μL) | 11.60 ± 0.17 | 11.72 ± 0.19 | 11.51 ± 0.08 | 16.27 ± 0.09** | 14.30 ± 0.06** | 1348.583 | 4 | 0.000 |
| PCV (%) | 44.01 ± 0.11 | 44.05 ± 0.60 | 44.09 ± 0.14 | 37.17 ± 0.44** | 43.23 ± 0.08** | 386.491 | 4 | 0.000 |
| MCV (fL) | 59.30 ± 0.35 | 55.32 ± 0.28** | 58.18 ± 0.06** | 63.70 ± 0.42** | 57.94 ± 0.41** | 424.385 | 4 | 0.000 |
| MCH (Pg) | 18.43 ± 0.12 | 18.45 ± 0.10 | 18.40 ± 0.01 | 17.78 ± 0.04** | 15.65 ± 0.09** | 998.344 | 4 | 0.000 |
| MCHC (g/dL) | 31.32 ± 0.07 | 31.33 ± 0.09 | 31.33 ± 0.09 | 26.31 ± 0.08** | 24.20 ± 0.01** | 10047.639 | 4 | 0.000 |
| Lymph (%) | 76.11 ± 0.01 | 76.12 ± 0.01 | 75.61 ± 0.01** | 75.58 ± 0.05** | 85.13 ± 0.05** | 83085.625 | 4 | 0.000 |
| Neutr (%) | 21.25 ± 0.08 | 21.25 ± 0.08 | 22.05 ± 0.05** | 13.23 ± 0.04** | 12.27 ± 0.05** | 27154.695 | 4 | 0.000 |
| Mono (%) | 1.16 ± 0.09 | 1.09 ± 0.05 | 1.03 ± 0.04** | 1.03 ± 0.04** | 1.01 ± 0.01** | 6.630 | 4 | 0.010 |
| Eosin (%) | 2.06 ± 0.05 | 2.05 ± 0.05 | 2.05 ± 0.05 | 2.05 ± 0.05 | 2.02 ± 0.05 | 0.525 | 4 | 0.718 |

Table 1: Mean and standard deviation values of hematological indices of the various groups.

Key: P < 0.05 is significant, **implies significant

| Variables | Control Mean ± SD | Group II (GSK 'AB') Mean ± SD | Group III (GSK 'DR') Mean ± SD | Group IV (GSK 'SS') Mean ± SD | Group V (GSK 'SDG') Mean ± SD | F | DF | P |
|---------------------|-------------------|-------------------------------|--------------------------------|-------------------------------|-------------------------------|-----------|----|-------|
| Albumin (g/L) | 36.24 ± 0.32 | 35.59 ± 0.08** | 36.24 ± 0.15 | 55.14 ± 0.13** | 36.10 ± 0.07 | 11772.722 | 4 | 0.000 |
| Total Protein (g/L) | 70.11 ± 0.07 | 69.14 ± 0.50** | 69.60 ± 0.12** | 55.40 ± 0.40** | 56.00 ± 0.05** | 9.285 | 4 | 0.000 |
| Total Bil (μmol/L) | 1.40 ± 0.02 | 1.39 ± 0.02 | 1.39 ± 0.02 | 1.41 ± 0.01 | 1.39 ± 0.02 | 1.027 | 4 | 0.418 |
| ALP (IU/L) | 80.09 ± 0.04 | 79.58 ± 0.06** | 80.02 ± 0.25 | 60.16 ± 0.44** | 65.48 ± 0.13** | 8171.763 | 4 | 0.000 |
| AST (IU/L) | 83.04 ± 0.03 | 82.83 ± 0.30 | 84.42 ± 0.16** | 101.24 ± 0.09** | 96.68 ± 0.13** | 13622.702 | 4 | 0.000 |
| ALT (IU/L) | 21.74 ± 0.18 | 22.10 ± 0.06** | 22.09 ± 0.09** | 29.56 ± 0.11** | 27.08 ± 0.08** | 5066.963 | 4 | 0.000 |
| Glucose (mmol/L) | 6.25 ± 0.17 | 6.08 ± 0.08 | 6.14 ± 0.14 | 2.05 ± 0.07** | 4.53 ± 1.10** | 63.863 | 4 | 0.000 |
| Creat. (μmol/L) | 531.93 ± 0.90 | 581.18 ± 1.36** | 532.19 ± 0.51 | 653.86 ± 0.59** | 560.99 ± 0.15** | 18175.915 | 4 | 0.000 |
| GGT (U/L) | 2.67 ± 0.38 | 7.54 ± 0.31** | 2.91 ± 0.21 | 12.63 ± 0.11** | 9.95 ± 0.10** | 2.683 | 4 | 0.061 |
| MDA (μmol/L) | 22.02 ± 0.03 | 22.23 ± 0.42 | 21.83 ± 0.44 | 27.71 ± 0.97** | 25.20 ± 0.21** | 123.639 | 4 | 0.010 |

Table 2: Mean and standard deviation values of biochemical parameters of the various groups.

Key P < 0.05 is significant, **implies significant

the irritability of erythrocytes through indirect dilution of heavy metals invading these cells [15]. These metals are capable of also causing the splitting of the DNA strands, resulting into nuclear DNA damage, disruption in the amino acids sequence, and production off abnormally large erythroblasts with nuclear cytoplasmic asynchrony in the bone marrow [16].

The microcytic anaemia (microcytosis) as demonstrated by significant decrease in MCV in groups II, III and V post 30 days administration of GSK, may be suggestive of loss of the content of Hb inside the RBCs, disturbances in the erythropoietin the medullary and extra medullary sites like the spleen, sequel to the hypoxia induced by metal toxicity and the deficiency of folic acid and cobalamin (Vitamin B₁₂) that might have been caused by the effect of the

alcohol as well as following the accumulation of the metals in the gut, it competes with the cobalamin to use the H₂-histamine blockers leading to the atrophy or loss of the gastric mucosa that will not be able to synthesize the intrinsic factor which inhibits folic acid absorption and vitamin B₁₂ needed for erythropoiesis thus leading to megaloblastic anaemia [17]. However, the significant reduction in Hb concentration of the rats across the groups may be due to the oxidation of Hb at the molecular level and also haem biosynthesis failure due to deficit in the components needed for its synthesis (protein, enzymes, iron) etc. Earlier on our data revealed bioaccumulation of heavy metal resulting to marked liver damage by reactive oxygen species [14], leading to significant depletion of the total protein contents generated from our data which might be due to the excessive utilisation as well as consumption of albumin as an antioxidant and as a scavenger of the generated free radicals in order to safeguard blood cells against the oxidative damage [18] and as a result, shortage of the globin proteins required for Hb synthesis ensued. The heavy presence of these metals might have well affected the functions of transferrin expelling it from tissues and molecules [19] or inhibited ceruloplasmin, which is known to promote the incorporation of ferric ions into the transferrin [20] thus resulting to a decrease uptake Fe³⁺ needed for Hb synthesis and consequently causing anaemia [21] supporting the fact that, GSK is detrimental to health [22]. In addition, the presence of lead (Pb) as detected in GSK, could as well have affected the hemopoietic tissues, inhibited the activity of the enzyme δ -aminolevulinic acid dehydratase (δ -ALAD) [23] and haem synthase [20], leading to total reduction in Hb synthesis and hypoxia as a result of reduced O₂ carrying ability by Hb.

On the other hand, the Leucocytosis as reported from our data especially among those albino rats that were administered GSK brands 'SS', and 'SDG' (Groups IV and V), which was significantly higher than the corresponding controls, may be attributed to the production of leucocytes in hemopoietic tissues following an immune response to the inflammation due to oxidative stress of the reactive oxygen species elicited by the presence of heavy metals in these brands of the GSK.

The lymphocyte inflammatory cells detected in this study also indicate the production of chronic inflammatory disease under the effect of GSK [24]. Ashry and his cohorts had earlier shown that chronic active cells were accompanied by inflammatory cells in the hepatocytes after administration of Codeine.

Measurements of transaminases (AST and ALT) and alkaline phosphatase (ALP) enzymes activity in serum are most frequently measured for diagnosis of liver diseases particularly infective hepatitis, alcoholic cirrhosis, biliary obstruction, toxic hepatitis and liver cancer [25-29]. And research has shown that both the Liver and Kidney are known to play a cardinal role in drug metabolism and this predisposes them to toxic injury. Biochemical analysis results in this research showed that there was an increase in the levels of (AST, ALT, γ -glutamyl transferase and total Bilirubin indicating tissue damage and inefficiency of the Liver due to use of GSK over time. Exposure to heavy metals detected in GSK, particularly (Mn, Pb, Cr, Cd, Ni, and Co) could have induced liver damage as they are known to cause infiltration and fibrosis of hepatocytes thus increasing the effectiveness of enzyme amino transporter AST, and ALT. The presence of these metals is capable of affecting the anti-oxidants defence system as well thus leading to tissue damage [30]. Inside the liver cells, heavy metals might have also interfered with phosphate and ATP metabolism [31], resulting to the depletion of cellular energy and ultimately affected also the membrane potentials thus causing necrosis as confirmed by our histological findings. This could be responsible for the leakage of transaminases into the circulation [32]. However the elevation of ALT has also been reported in non-Liver conditions e.g. muscle injury [33]. Furthermore, a significant elevated level of ALT has been reported in rats receiving hepatotoxic compounds e.g., carbon tetrachloride (CCL₄) for a long time compared to control group [34]. This finding is also supported by the direct relationship with the levels of MDA established.

Laboratory evaluation of serum blood Creatinine and Blood Urea Nitrogen (BUN) levels are considered as a good marker for the determination of renal function [35]. The impairment of renal biomarkers in this study is reflected by a significant increase in Creatinine. Effect of GSK brands on Kidney function parameters shows the mean Kidney function values of the rats treated with GSK and as could be seen in Creatinine levels were significantly raised when compared with the control which was above the normal reference values (Group II, IV and V).

Remarkable elevated levels of Creatinine were seen in the rats that were administered ("AB", "SS" and "SDG") GSK brands orally at dose level 0.31 mls/gm of body weight over a period of 30 days

when compared to control. Serum Creatinine and Urea increases as the ability of the kidney to produce urine within the body declines. Hence in general terms, a rising level of Creatinine signals an increasing problem with poor performing Kidneys [33]. All of these findings reinforced those of Koechel, *et al.* (1984) and Damjananov (1996) [35]. They both demonstrated that many chemicals had a direct nephrotoxic action and exerted their effects majorly on the PCT [35].

Conclusion

The study showed changes that were statistically significant in some haematological parameters and biochemical parameters studied as a result of administration of the local indigenous alcohol beverage in the male albino rats.

Bibliography

- World Health Organization. Global status report on alcohol and health; World Health Organization: Geneva (2014): 1-43.
- Odejide OA. "Alcohol policies in Africa". *African Journal of Drug and Alcohol Studies* 5 (2006): 27-39.
- Jernigan DH. "Global status report: Alcohol and young people". Geneva: World Health Organization (2001).
- Alcohol and public policy group. "Alcohol: no ordinary commodity- a summary of the second edition". *Addiction* 105 (2010): 769-779.
- Das SK., *et al.* "Non-alcoholic fatty liver diseases: an under recognizes cause with emerging importance". *Current Science* 90 (2006): 659-665.
- Ebirim IC and Morakinyo OM. "Prevalence and perceived health effect of alcohol use away make undergraduate students in Owerri, South - East Nigeria: a descriptive cross-sectional study". *BMC Public Health* (2011).
- National Highway Traffic Safety Administration. Traffic safety facts 2000. Washington, DC (2000).
- O' Neill., *et al.* "Clinical Relevance of heavy drinking during the college year: cross sectional and prospective perspectives". *Psychology of Addictive Behavior* 15 (2005): 350-359.
- Bonomo Y., *et al.* "Teenage drinking and the onset of alcohol dependence: a cohort study over seven years". *Addiction* 99 (2004): 1520-1528.
- Arata CM., *et al.* "High school drinking and its consequences". *Adolescence* 38 (2003): 567-579.
- Hewitt BG. "Alcoholism: Developmental patterns of drinking and prevention of alcohol use disorders".in Tsuang, Mt., Stone, WS., and Lyons, MJ. (Eds.), Recognition and prevention of major mental and substance use disorders". *American Psychiatric Publishing Inc* (1998): 297-316.
- Lami Sadiq. "How Gaskolo is turning Plateau Youth into slaves". *Online Daily Trust Newspaper* (2017): 1.
- Jos Metropolitan Development Board. Jos greater master plan (2009): 11-15.
- Ali AA. "Studies on fate and toxicity of nano Alumina in male albino rats". PhD Thesis, Cairo University, Faculty of Science, Zoology Department, Cairo, Egypt (2013).
- Bhagwant S and Bhikajee M. "Induction of hypochromic macrocytic anaemia in Oreochromis hybrid (cichlidae) exposed to 100 mg/l (sublethal dose) of aluminium". *Science and Technology Research Journal* 5 (2000): 9-20.
- Aslinia F., *et al.* "Megaloblastic anemia and other causes of macrocytosis". *Clinical Medicine Research* 4 (2006): 236-241.
- Ahmed HH., *et al.* "Potential role of some nutraceuticals in the regression of Alzheimer's disease in an experimental animal model". *Turkish Journal of Medical Sciences* 41.3 (2011): 455-466.
- Tolia C., *et al.* "Copper (II) interacting with the non-steroidal antiinflammatory drug flufenamic acid: Structure, antioxidant activity and binding to DNA and albumins". *Journal of Inorganic Biochemistry* 123C (2013): 53-65.
- Mahieu S., *et al.* "Aluminium toxicity: haematological effects". *Toxicology Letters* 111 (2000): 235-242.
- Chmielnicka J., *et al.* "Disturbances of morphological parameters in blood of rats orally exposed to aluminium chloride". *Bio-Logical Trace Element Research* 42.3 (1994): 191-199.

21. Flora SJ, *et al.* "Aluminium-induced oxidative stress in rat brain: response to combined administration of citric acid and HEDTA". *Comparative Biochemistry and Physiology Part C* 134 (2003): 319-328.
22. Andrew OE. "Histological studies of the effects of monosodium glutamate on the kidney of adult Wistar rats: Department of Anatomy, School of Basic Medical Science, College of Medical Sciences, University of Benin, Edo State Nigeria". *The Internet Journal of Health* (2000): 1528-8315.
23. Pimentel-Vieira V, *et al.* "Effect of aluminium on δ -aminolevulinic acid dehydratase from mouse blood". *Toxicology Letters* 117 (2000): 45-52.
24. Ashry M, *et al.* "Histopathology and histochemical changes in response to the administration and withdrawal of Codeine on Liver of rat". *Egyptian Journal of Histology* 13 (1990): 3-12.
25. Varshneya C, *et al.* "Toxicological effects of dietary malathion in cockerels". *The Indian Journal of Animal Sciences* 58.4 (1988): 411-414.
26. Kaneko J, *et al.* "Clinical Biochemistry of Domestic Animals". 5th edition., Academic press, Inc. San Diego, London, New York (1997).
27. Davidson V and D Sittman. "Biochemistry". 4th edition". Lip-pinicott Williams and Wilkins Publishers (2010).
28. Zaahkoug SAM, *et al.* "Carbamate toxicity and protective effect of vit. A and vit. E on some biochemical aspects of male albino rats". *The Egyptian Journal of Hospital Medicine* 1 (2000): 60-77.
29. Abdel-Wahhab MA, *et al.* "Zizyphus spina-christi extract protects against aflatoxin B1-intitiated hepatic carcinogenicity". *African Journal of Traditional, Complementary and Alternative Medicines* 4.3 (2007): 248-256.
30. El-Sokkary G, *et al.* "Melatonin protects against lead-induced hepatic and renal toxicity in male rats". *Toxicology* 213 (2005): 25-33.
31. Silva VS, *et al.* "Effect of chronic exposure to aluminium on iso-form expression and activity of rat (Na⁺/K⁺) ATPase". *Toxicological Sciences* 88 (2005): 485-494.
32. Nehru B and Anand P. "Oxidative damage following chronic aluminium exposure in adult and pup rat brains". *Journal of Trace Elements in Medicine and Biology* 19 (2005): 203-208.
33. Edwards MB and Bouchier AD. "Principle and Practice of Medicine.16th edition", ELBS Churchill Living Stone, Man Group Ltd., Hong Kong (1994): 606-745.
34. Hai ZH, *et al.* "Hepato-protective and antioxidant effects of Licorice extract against CCL4 induced oxidative damage in Rats". *International Journal of Molecular Sciences* 12 (2011): 6529-6543.
35. Koechel DA, *et al.* "The pentobarbital anesthetized dog: An animal model for assessing chemically induced changes in renal function and ultra-structure". *American Journal of Veterinary Research* 45.12 (1984): 2565-2573.
36. Damjanov I. "Histopathology: A Color Atlas and Textbook". Williams and Wikins. A Waverly Company. Baltimore. Philadelphia and London (1996): 257-287.