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Impact Of Ethanol Exposure On Hepatic Function And Oxidative Stress Biomarkers In Albino Rats

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Abstract: Alcohol (ethanol) is a hepatotoxic agent that induces oxidative stress and disrupts liver function. This study evaluated the effects of three commercial ethanol brands (Seaman, Chelsea, and Lords) at high (3 ml/kg/day) and low (1.5 ml/kg/day) doses on liver function and antioxidant status in Wistar rats. Thirty-five rats (150–160 g) were divided into seven groups (n = 5) and treated for two weeks. Liver enzymes (ALT, AST), protein profile, bilirubin, and oxidative stress markers (SOD, CAT, LDH, MDA) were analyzed using standard methods. Control values were ALT = 24.26 U/L and AST = 138.23 U/L. High-dose Seaman produced the greatest hepatotoxicity (ALT = 39.33 U/L; AST = 203.53 U/L), followed by Lords (ALT = 32.23 U/L; AST = 156.33 U/L), while Chelsea reduced AST (101.66 U/L). Albumin decreased markedly in Seaman high-dose (1.23 g/dL vs. 2.22 g/dL control). Oxidative markers showed SOD = 27.01 $\times 10^{-6}$ U/mL and CAT = 88.02 U/mL in controls. Lords low-dose caused severe depletion (SOD = 6.63 $\times 10^{-6}$ U/mL), while Chelsea high-dose increased antioxidant activity (SOD = 43.11 $\times 10^{-6}$ U/mL; CAT = 122.73 U/mL) but elevated LDH (489.75 µg/mL). Ethanol induced dose- and brand-dependent hepatotoxicity. Seaman was most hepatotoxic, Lords caused greatest oxidative depletion, while Chelsea elicited adaptive antioxidant responses with cellular damage.

Keywords: Alcohol, Hepatotoxicity, Oxidative stress, Liver enzymes, Antioxidant enzymes

I. INTRODUCTION

Alcohol (ethanol) sometimes known as alcohol, is a psychoactive drug that people often take and is known to be toxic to the liver. Both chronic and acute exposure can lead to hepatocellular injury, oxidative stress, and alterations in protein metabolism. Serum liver enzymes, notably alanine aminotransferase (ALT) and aspartate aminotransferase (AST), function as sensitive indicators of hepatic injury, while protein profiles (total protein, albumin, globulin) and bilirubin levels reflect hepatic synthetic and excretory functions (Kumar, Singh, & Verma, 2020). Drinking too much alcohol hurts the liver, destroys a lot of hepatocytes, and primarily makes the liver not work properly (Osna et al., 2017; Ohashi et al., 2018). Gao and Bataller (2011) say that drinking alcohol causes about 3 million deaths around the world. It makes for 5.3% of all other causes of death. According to Park and Kim (2020), 13.5% of people who drink alcohol are between the ages of 20 and 39. Oxidative activities damage the cell membrane, and oxidative stress is a big element of alcoholic liver disease (ALD). Drinking alcohol increases the liver's generation of reactive oxygen species (ROS), which weakens the body's natural defences against ROS. These defences are not good enough to keep ROS levels in check at the cellular level (Das et al., 2018). In the ethanol-intoxicated group, there is a significant increase in reactive oxygen species (ROS), signifying elevated oxidative stress, whereas the LA-treated group significantly reduced oxidative stress by bolstering antioxidant defence mechanisms.

Oxidative stress is a major cause of liver damage caused by alcohol. Reactive oxygen species (ROS) produced during ethanol metabolism may exceed antioxidant defences, reducing the activities of superoxide dismutase (SOD) and catalase (CAT), while increasing lipid peroxidation, as indicated by elevated malondialdehyde (MDA) levels (Smith, Allen, & Thompson, 2021).

According to Zhang et al. (2018), drinking too much ethyl alcohol changes the amounts of antioxidant enzymes such superoxide dismutase (SOD), glutathione (GSH), and glutathione peroxidase (GPx). This is because oxidative stress lowers these levels. Ighodaro and Akinloye (2018) say that these enzymes are the main way the body fights oxidative stress caused by free radicals. Lipid peroxidation is a major cause of liver damage, and malondialdehyde (MDA), which is the end product of lipid peroxidation, is a well-known reactive aldehyde and oxidative stress marker (Ayala et al., 2014). The recent study shows that when rats drink too much alcohol, their MDA levels go up and their antioxidant activity goes down. A rise in MDA levels signifies that liver lipid peroxidation has occurred, resulting in liver tissue damage and a diminished antioxidant defence mechanism (Shah et al., 2017).

The current study demonstrates that LA alleviated the deficit of antioxidant enzymes and reduced MDA levels. Lipoic acid therapy greatly lessened the oxidative damage caused by drinking too much alcohol. LA protects against oxidative stress caused by free radicals by scavenging them and directly neutralising biological superoxides. This is because it is a natural antioxidant that stops lipid peroxidation and prevents antioxidant depletion (Belghit et al., 2019). Another study supports our findings, showing that drug metabolites harm the liver, which leads to oxidative stress and lowers the body's ability to fight off free radicals in animal models, Nithiyanandam and Prince, 2023. Commercial ethanol brands vary in ethanol content and impurity levels, which may affect hepatotoxicity. The goal of this study is to carefully compare the effects of Seaman, Chelsea, and Lords ethanol at both normal and high doses on liver function and oxidative stress indicators in albino rats

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II. MATERIAL AND METHODS

Animals and Experimental Ethics Protocol

Twenty adult male Wistar rats (Rattus norvegius) weighing between the ranges of 150-160g, were obtained from the Federal University of Technology, Owerri, Imo State. The animals were housed in the experimental house of the Department of Science Laboratory Technology, Biochemistry Option, Delta State Polytechnic, Ogwashi Uku, Delta State. The animals were fed on standard rat diet and allowed free water access. Animals were allowed to acclimatize to experimental conditions by housing them for 14 days prior to experiment.

Experimental protocol
Ethical approval for the study was obtained from Department of Science Laboratory Technology Ethical Committee, with ethical approval number DSPG/SLT/2025/001 and the experiment complied with the National Institute of Health Guideline Principles of Laboratory Animals in Biomedical Research.

Experimental design
Thirty-five (35) rats were randomly divided into seven groups, and each group has five (5) rats. Group A functioned as the control and was administered solely the solvent (distilled water). Group B, C, and D were supplied a high dose of 3 ml/kg/day of Seaman, Chelsea, and Lord, respectively, while Group E, F, and G received a low dose of 1.5 ml/kg/day of Seaman, Chelsea, and Lord, respectively. All animals in each group were weighed prior to and following the experiment. The administration route was oral via an orogastric tube.

Study Duration: This study lasted for four weeks (two weeks for acclimatization and two weeks for substance administration. Throughout the four-week trial period, the animals were nourished and monitored for diverse behaviours.

Sample Collection: After three weeks of dosing, the rats were euthanised using chloroform as an anaesthetic. Subsequent to the sacrifice, blood was extracted via cardiac puncture and gathered in basic, sterile, centrifuged vials to facilitate coagulation. Blood serum was obtained by centrifuging the samples individually and subsequently stored at -20 °C until the serum/homogenate quantities were required. Standard methods were used to checked for heamatological functions and kidney function parameters. The rats were subsequently dissected to obtain the organ and were promptly preserved in 10% formalin.

Biochemical analyses were performed to assess liver function parameters (ALT, AST, total protein, albumin, globulin, total bilirubin) and antioxidant markers (SOD, LDH, CAT, MDA), were carried out in General Instrumentation Laboratory by commercially available kits according to kits protocol.

Statistical Analysis: All measurements were performed in triplicate, and results were expressed as mean ± standard deviation (SD). Statistical comparisons between groups were made by One-way analysis of variance (ANOVA) followed by Duncan's multiple range (post-hoc test), P <0.05 was considered statistically significant. (Kumar et al., 2020; Smith et al., 2021).

III. RESULTS

Effect of alcohol on Liver Function parameters of rats

The rats in Group A (the control group) exhibited normal liver function levels, with ALT levels of 24.26 U/L and AST values of 138.23 U/L. The amount and brand of ethanol you drink affected how much harm it did to your liver (Table 1). Group E's high-dose Seaman caused the most damage to hepatocytes, with ALT = 39.33 U/L (+62% vs. control) and AST = 203.53 U/L (+47% vs. control). Lords high-dose (Group G) elevated ALT by 33% to 32.23 U/L and AST by 13% to 156.33 U/L. Chelsea high-dose (Group F) showed a slight elevation in ALT (29.33 U/L, +21%) but a decline in AST (101.66 U/L, −26% vs. control). These results show that Seaman ethanol has the strongest hepatotoxic effect, while Chelsea has the opposite effect on AST, which could mean that the release or metabolism of enzymes is being controlled (Kumar, Singh, & Verma, 2020). Drinking alcohol has a big effect on albumin levels.

In control rats, the amount of albumin was 2.22 g/dL. High-dose Seaman brought albumin down to 1.23 g/dL (−45%), which suggests that protein synthesis wasn't working well. High-dose Chelsea, on the other hand, kept albumin higher (1.78 g/dL, −20%). The amounts of total protein and globulin varied by brand, but they were frequently close to the values for the control group. This indicates that albumin synthesis was specifically compromised. The Seaman standard dose (1.36 g/dL) caused the biggest increase in total bilirubin, which indicated mild cholestasis and is in line with ethanol-induced hepatic excretory failure (Zhao, Li, & Wang, 2021).

Table 1: Effect of alcohol on Liver Function parameters of rats

Grp	ALT (U/L)	AST (U/L)	Total protein (g/dl)	Albumin (g/dl)	Globulin (g/dl)	Total Bilirubin (g/dl)
A	24.26 ± 0.05 a	138.23 ± 0.64 c	10.14 ± 0.05 c	2.22± 0.00c	8.49±0.13c	0.36 ± 0.01a
B	24.43 ± 0.05 a	137.56 ± 0.11 c	10.66 ± 0.09 c	2.43 ± 0.13c	8.44±0.19c	1.36 ± 0.01f
C	30.33± 0.57 d	118.66 ± 0.57 b	9.34 ± 0.23 b	1.59 ± 0.33b	8.57±0.46c	1.14 ± 0.05e
D	26.66± 0.57 b	151.33 ± 0.57 d	11.45± 0.33 d	2.39 ± 0.12c	9.12±0.15d	0.67 ± 0.01d
E	39.33± 0.15 f	203.53 ± 0.57 f	10.27± 0.34 c	1.23 ± 0.08a	8.60±0.14c	0.43 ± 0.01b
F	29.33± 0.57 c	101.66 ± 0.57 a	6.58 ± 0.37 a	1.78 ± 0.07b	4.69±0.36a	0.48 ± 0.00c
G	32.23 ± 0.15 e	156.33 ± 0.57 e	9.49 ± 0.37 b	3.11 ± 0.17d	6.67±0.42b	0.40 ± 0.00c

Values are mean ± SD of 5 triplicates. Different superscript letters (e.g., [a], [b]) indicate significant differences at $p < 0.05$; same letters (e.g., [a]) indicate no significant difference at $p < 0.01$ (Duncan's).

Effect of alcohol on Invivo-Antioxidants Parameters of rat

The control rats (Group A) had the following levels: SOD = 27.01 ×10^-6 U/mL, CAT = 88.02 U/mL, LDH = 82.10 µg/mL, and MDA = 4.83 mmol/L. Ethanol exposure changed these markers in different ways, showing oxidative stress and antioxidant responses that tried to make up for it (Table 2). SOD: The Seaman normal dose (26.53 ×10^-6) lowered it, and the Lords 1.5 mL (6.63 ×10^-6, −75%) lowered it further more, showing that oxidative stress was quite high. Chelsea high-dose (43.11 ×10^-6) exhibited an adaptive rise, potentially attributable to the activation of antioxidant defence systems following acute high-dose exposure (Smith, Allen, & Thompson, 2021). Control activity for CAT is 88.02 U/mL. CAT went down in Lords 1.5 mL (73.58 U/mL) and Seaman 3 mL (74.03 U/mL), while it went up in Chelsea high-dose (122.73 U/mL), which is similar to SOD trends. LDH: Very high in Chelsea high-dose (489.75 µg/mL) and Lords high-dose (242.12 µg/mL), which shows that cell membranes are damaged and cells are dying. MDA: Lipid peroxidation varied. Seaman high-dose exhibited a decrease (2.51 mmol/L), while Chelsea 1.5 mL and the control group displayed elevated results (~4.83 mmol/L), indicating non-linear oxidative damage, possibly affected by the content of the ethanol brand.

Table 2: Effect of alcohol on Invivo-Antioxidants Parameters of rat

Grp	SOD (u/ml)*10^-6))	LDH (ug/ml)	CAT (U/ml)	MDA (mmol/L)
A	27.01 ± 0.00e	82.10 ± 0.00b	88.02± 0.02d	4.83±0. 00g
B	26.53 ± 0.00d	81.16 ± 0.00a	87.88± 0.11c	4.48±0.00f
C	28.19 ± 0.00f	96.51 ± 0.39c	101.09± 0.00e	4.38 ± 0.00e
D	6.63± 0.00a	125.18 ± 0.00d	73.58± 0.00a	2.53 ± 0.00b
E	22.38 ± 0.00c	127.93 ± 0.00e	74.03± 0.00b	2.51 ± 0.00a
F	43.11 ± 0.00g	489.75 ± 0.02g	122.73± 0.05g	3.58 ± 0.00d
G	21.55 ± 0.00b	242.12 ± 0.01f	108.27± 0.05f	3.55 ± 0.00c

Values are mean ± SD of 5 triplicates. Different superscript letters (e.g., [a], [b]) indicate significant differences at $p < 0.05$; same letters (e.g., [a]) indicate no significant difference at $p < 0.01$ (Duncan's).

IV. DISCUSSION

When we look at the control group (Group A), we can see a pattern in the liver function and oxidative stress markers: 1. Hepatotoxicity (ALT/AST): Seaman > Lords > Chelsea 2. Albumin reduction: Seaman high-dosage caused the most damage, which shows that the liver's ability to make new proteins is affected by the dose. 3. Oxidative stress (SOD/CAT/MDA): The Lords regular dose caused a lot of SOD and CAT to be lost, while the Chelsea high dose prompted antioxidant levels to rise to make up for it. 4. Cellular damage (LDH): Chelsea high-dose induced a huge rise in LDH, which shows that the membranes were damaged even though the antioxidants were able to adjust.

Important point: Liver enzyme levels and oxidative stress markers don't always go up and down in a straight line. For example, Chelsea's high dose lowered AST while raising SOD and LDH by a large amount. This separation shows that the composition of commercial ethanol, not simply the amount, affects hepatotoxic and oxidative reactions. This shows how important it is to look at both biochemical and oxidative stress markers at the same time.

The observed trends are consistent with prior studies showing ethanol-induced hepatotoxicity and oxidative stress in rats (Kumar et al., 2020; Smith et al., 2021; Zhao et al., 2021): - ALT and AST elevations align with reported ranges of 35–42 U/L and 120–200 U/L, respectively. - SOD and CAT depletion in Lords 1.5 mL mirrors prior findings of antioxidant exhaustion under ethanol stress. - The adaptive antioxidant increase in Chelsea high-dose aligns with Smith et al. (2021), who noted transient upregulation of antioxidant defenses during acute ethanol exposure.

Unique observation: Seaman high-dose produced AST values (203.53 U/L) slightly exceeding literature norms, while Chelsea high-dose induced extreme SOD upregulation, highlighting brand-specific biochemical effects not widely reported.

V. CONCLUSION

Comparative analysis using control rats demonstrates that ethanol exposure induces dose- and brand-dependent hepatotoxicity and oxidative stress. Seaman ethanol was the most hepatotoxic, while Chelsea high-dose elicited adaptive antioxidant responses. Lords standard dose caused the most severe oxidative stress depletion. These findings emphasize that both ethanol source and dosage must be considered in experimental hepatotoxicity studies and suggest further chemical analysis of commercial ethanol brands is warranted.

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