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STANOZOLOL, AN ANABOLIC-ANDROGENIC STEROID AND HEPATIC DYSFUNCTION: A PRELIMINARY STUDY ON SERUM TRANSAMINASES PROFILE IN POSTNATAL MICE MUS MUSCULUS

Praveenkumar S. Kondaguli, Laxmi S. Inamdar (Doddamani)*

Molecular Endocrinology, Reproduction and Development Laboratory, Department of Zoology, Karnatak University, Dharwad 580 003, India.

Corresponding Author: Dr. Laxmi S. Inamdar (Doddamani)

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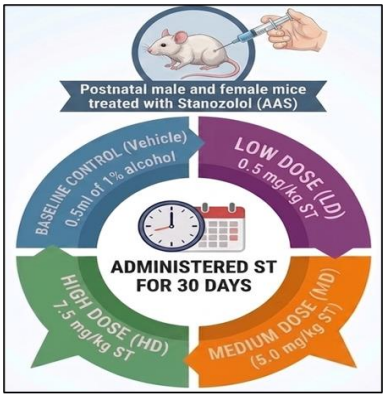
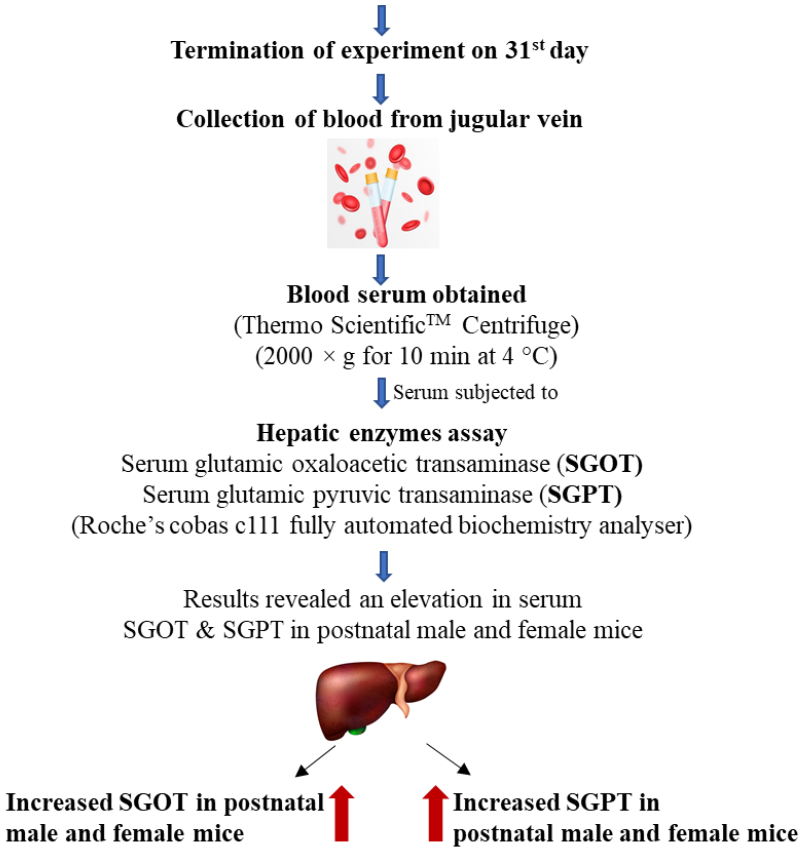
ABSTRACT

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone widely used for therapeutic purposes; however, their non-medical use has increased considerably among athletes, bodybuilders, and recreational users to enhance physical performance, endurance, and muscle mass. Nevertheless, abuse of these AAS can consequently result in various secondary adverse effects like hepatocarcinoma, coronary heart diseases, altered cholesterol and liver enzyme profiles, reproductive and endocrine disturbances. The present study was undertaken to evaluate the effects of prolonged administration of one of the AAS, ST, on serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) levels in male and female postnatal mice. A total of 40 postnatal mice (21 days old; 20 males and 20 females) were randomly divided into four groups of males and four groups of females (having 5 animals in each group). ST was dosed [0.5 mg/kg bwt (low-dose); 5.0 mg/kg bwt (medium-dose); and 7.5 mg/kg bwt (high-dose) or 1% alcohol (vehicle control)] subcutaneously for 30 consecutive days. Serum SGOT and SGPT levels were analyzed using a Roche cobas c111 fully automated biochemical analyzer. The findings revealed a significant elevation in serum SGOT and SGPT levels across ST-treated groups ($p < 0.001$), irrespective of sex, when compared to controls. These observed results indicate the AAS-induced liver diseases, such as liver cirrhosis and inflammation in the liver, that may lead to hepatic dysfunction or hepatotoxicity. These findings indicate the health risks of non-therapeutic AAS abuse and suggest the need for molecular studies to clarify the underlying mechanisms.

KEYWORDS: Stanozolol, serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, hepatic dysfunction, postnatal male and female mice.

GRAPHICAL ABSTRACT





INTRODUCTION

Anabolic-androgenic steroids (AAS) are synthetic derivatives of testosterone widely used for therapeutic purposes for the treatment of diseases such as renal and hepatic failure, hypogonadism, aplastic anaemia, serious burns, delayed puberty, postsurgical recovery, muscle wasting disorders, and osteoporosis.[1-6] Nevertheless, their non-medical use/abuse has risen substantially over the past few decades, especially among athletes, bodybuilders, weightlifters, adolescent girls and boys aiming to improve muscle mass, athletic performance, endurance, and physique, which can consequently result in various secondary adverse effects like hepatocarcinoma, coronary heart diseases, altered cholesterol and liver enzyme profiles, reproductive and endocrine disturbances.[5, 7-11]

The liver is a vital organ required for survival due to its central role in regulating numerous metabolic processes, including glucose homeostasis, xenobiotic metabolism, and detoxification. It also serves as a principal site for the synthesis and degradation of steroid hormones and the production of proteins. By regulating the systemic distribution of nutrients, the liver continuously supplies energy to the body. Furthermore, it plays a crucial role in both systemic and local innate immune responses and functions as an important center for immune regulation.[12-13] Numerous experimental studies and clinical case reports have highlighted the hepatotoxic potential of AASs, including hepatotoxic effects such as cholestasis, hepatic neoplasms, hepatocellular toxicity, and peliosis hepatis.[14-15] Enzymes such as serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) are commonly measured to evaluate hepatocellular damage. Impairment in these enzymes is widely regarded as a biomarker of compromised hepatocyte integrity and impaired liver function.[16]

Among the various synthetic steroids, stanozolol (ST) is one of the most commonly abused anabolic steroids because of its potent anabolic activity and relatively lower androgenic effects.[17] However, this same property also contributes to its hepatotoxic potential.[16] Several clinical and experimental studies have reported that prolonged exposure to AAS can induce liver dysfunction, cholestasis, oxidative stress, and alterations in hepatic enzyme activity.[18-19]

A previous study from our laboratory (Inamdar and Jayamma, 2012)[20] reported that administration of ST to adult mice at doses low (0.5 mg/kg bwt), medium (5.0 mg/kg bwt) and high (7.5 mg/kg bwt) resulted in reduced total cholesterol, triglycerides and altered lipoprotein profile. A decrease in high-density lipoprotein cholesterol (HDL-c) as well as total cholesterol (TC) in all the stanozolol-treated groups and an increase in low-density lipoprotein (LDL-c) in medium and the highest dose-treated groups indicate that stanozolol alters serum lipoprotein profile.[20] Therefore, evaluating the effect of ST on hepatic enzymes (SGOT and SGPT) in both

postnatal male and female mice is essential for a better understanding of ST's systemic toxicity.

In the present study, the hepatotoxic effects of prolonged stanozolol administration were evaluated in male and female postnatal mice by assessing serum SGOT and SGPT levels following 30 days of exposure to different doses of ST. The study aimed to compare the hepatic response between sexes and determine whether prolonged ST administration induces dose-dependent alterations in liver function markers.

MATERIALS AND METHODS

Experimental animals, study design, and hormone treatment.

All protocols in this experiment adhered to the CPCSEA guidelines for the Care and Use of Laboratory Animals and were approved by the Institutional Animal Care and Use Committee (No.639/GO/02/a/CPCSEA) at the Department of Zoology, Karnatak University, Dharwad.

A total of 40 postnatal Swiss albino mice (21 days old; 20 male and 20 female) were obtained from the mouse breeding center maintained in the Department of Zoology, Karnatak University, Dharwad, and randomly divided into four groups of males and four groups of females (each group having 5 animals). All the mice were housed in individual polypropylene cages and maintained a 12 h light: dark cycle at 27±1˚C with 40-50% RH, food (pelleted diet, Goldmohur, Lipton, India), and water supplied *ad libitum*. Stanozolol (ST) was obtained from Sigma-Aldrich, USA. ST was dosed subcutaneously [0.5 mg/kg bwt (low-dose); 5.0 mg/kg bwt (medium-dose); 7.5 mg/kg bwt (high-dose) or 0.5 ml of 1% alcohol (baseline control)] for 30 consecutive days. Animals were autopsied on 31st day.

Collection of blood and serum

Blood samples were collected from the jugular vein of control and treated mice. Serum was separated by centrifugation (Thermo Scientific^{TM} – Sorvall ST 8R Centrifuge) (2000 × g for 10 min at 4 °C) and stored at -80 °C for biochemical assay.

Analysis of Serum SGOT and SGPT

In vitro test for the quantitative determination of serum glutamic oxaloacetic transaminase (SGOT) or aspartate aminotransferase (AST) and serum glutamic pyruvic transaminase (SGPT) or alanine aminotransferase (ALT) with or without pyridoxal phosphate activation was carried out on a Roche's cobas c111 fully automated biochemistry analyser.

Statistical analysis

All statistical tests were performed using SPSS version 20. Shapiro–Wilk test was employed to evaluate the normality of all quantitative data. The serum SGOT and SGPT levels between treatment and control groups were evaluated using a one-way analysis of variance (ANOVA) to determine the significant difference between different treatment groups. To identify specific statistical significance between treatment and control cohorts, a multiple comparison analysis was performed using Tukey's HSD post-hoc test. All numerical data were expressed as mean ± standard error (SE). Both statistical tests were two-sided tests with 5%, 1% and 0.1% levels of significance ($P < 0.05$, $P < 0.01$ & $P < 0.001$).

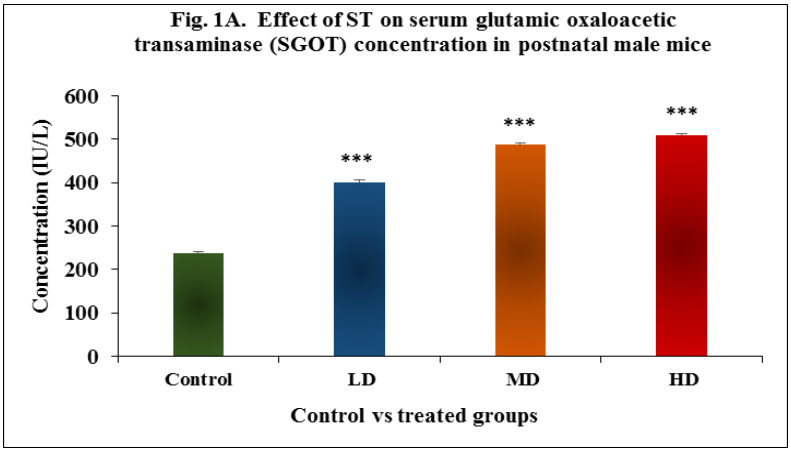
RESULTS

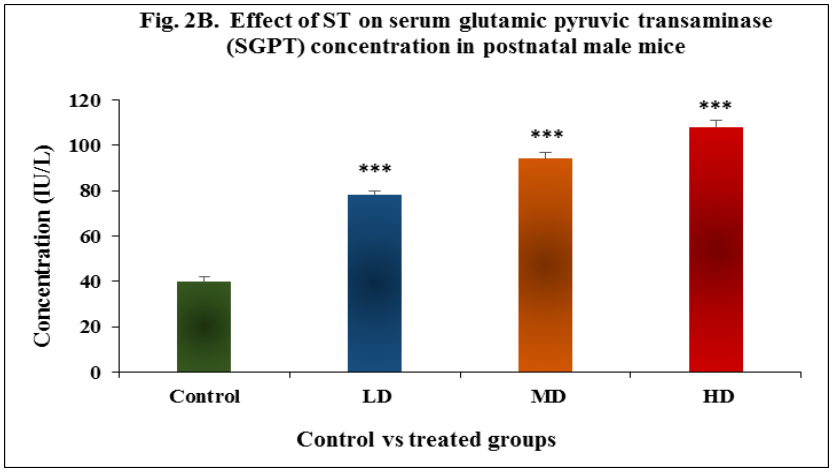
Effect of stanozolol treatment on serum SGOT and SGPT levels.

Serum glutamic oxaloacetic transaminase (SGOT/AST) and serum glutamic pyruvic transaminase (SGPT/ALT) are important hepatic enzymes involved in amino acid metabolism and are widely used as biomarkers of liver function. Elevated serum levels of these enzymes are indicative of hepatocellular damage and impaired liver integrity.

In male mice, a significant increase in serum SGOT level was observed in all ST-treated groups when compared with the control group ($p < 0.001$), ($F_{3,16} = 789.75$; $p < 0.001$); (Fig. 1A). Likewise, serum SGPT levels were significantly elevated in all ST-treated groups than in the control ($p < 0.001$), ($F_{3,16} = 150.44$; $p < 0.001$); (Fig. 1B).

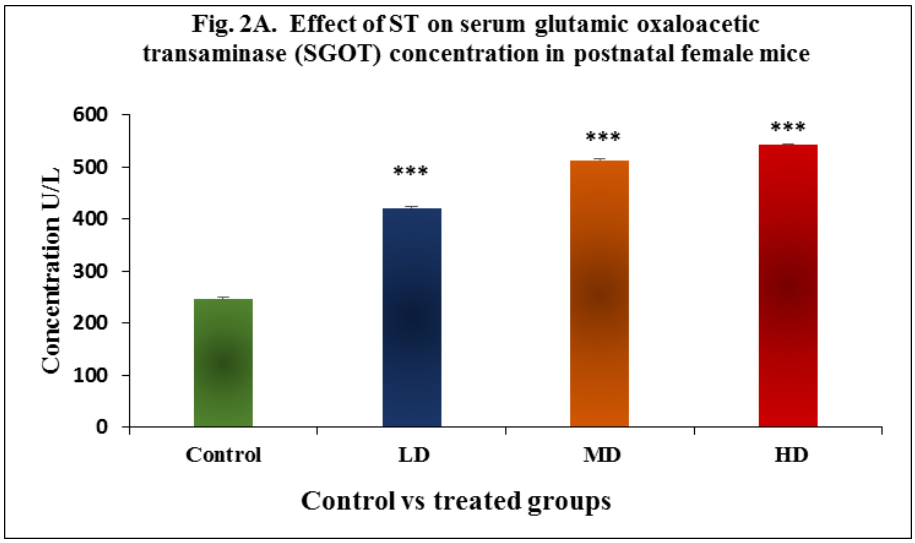
Similarly, female mice exhibited a significant elevation in serum SGOT levels in all ST-treated groups compared to the control ($p < 0.001$), ($F_{3,16} = 2944$; $p < 0.001$); (Fig. 2A). Likewise, serum SGPT levels were significantly increased in all treated groups compared to controls ($p < 0.001$), ($F_{3,16} = 306.683$; $p < 0.001$); (Fig. 2B).

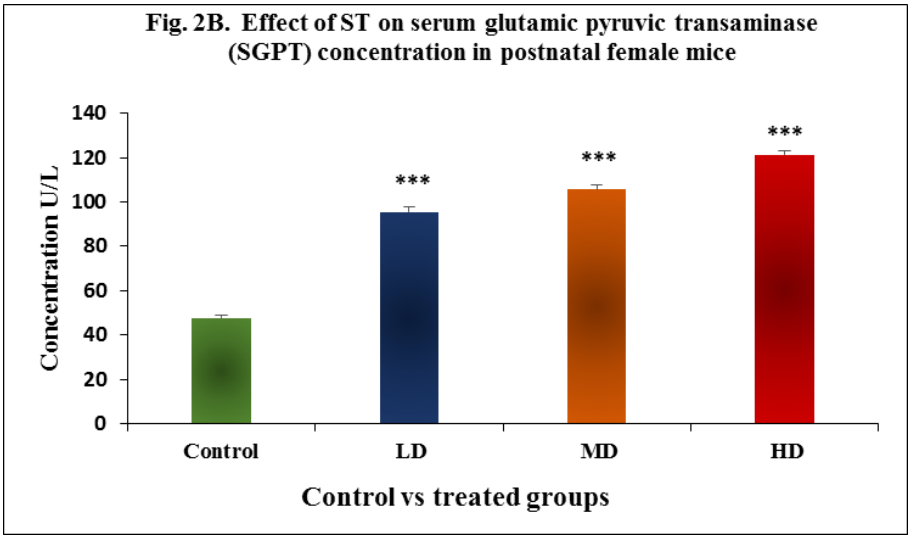




ST treatment resulted in a significant increase in serum SGOT and SGPT concentration of all the ST-treated groups in male mice. N=5 per group, *p<0.05, **p<0.01, ***p<0.001 (One-way ANOVA).
LD – low-dose, MD – medium-dose, HD – high-dose

Figure 1 A & B: The impact of ST treatment on (A) serum glutamic oxaloacetic transaminase (SGOT) and (B) serum glutamic pyruvic transaminase (SGPT) of postnatal male mice.





ST treatment resulted in a significant increase in serum SGOT and SGPT concentration of all the ST-treated groups in male mice. N=5 per group, *p<0.05, **p<0.01, ***p<0.001 (One-way ANOVA).
LD – low-dose, MD – medium-dose, HD – high-dose

Figure 2A & B: The impact of ST treatment on (A) serum glutamic oxaloacetic transaminase (SGOT) and (B) serum glutamic pyruvic transaminase (SGPT) of postnatal female mice.

DISCUSSION

The present study demonstrates a significant elevation in serum transaminases – SGOT and SGPT following prolonged administration of ST in postnatal male and female mice, indicating the AAS-induced liver diseases, such as liver cirrhosis, inflammation in liver cells, which may lead to hepatic dysfunction or hepatotoxicity.

Stanozolol, a 17α-alkylated derivative of dihydrotestosterone (DHT), contains a methyl substitution at the C17-α position that protects the steroid from extensive first-pass hepatic metabolism, thereby enhancing its bioavailability and prolonging systemic activity.[5,21] However, this structural modification also imposes a substantial metabolic stress on hepatocytes and has been associated with hepatotoxic outcomes, including transaminase elevation, cholestasis, and hepatic dysfunction.[19,22] The significant increase in SGOT and SGPT levels observed in the present study aligns with these mechanisms, as leakage of these intracellular enzymes into circulation is a hallmark of hepatotoxicity.

Recent clinical and experimental evidence further supports these findings. A prospective evaluation of ST-associated liver injury demonstrated that AAS misuse is increasingly recognized as a major cause of drug-induced liver injury, often presenting with biochemical alterations and hepatic dysfunction.[23] Additional clinical reports have documented that even therapeutic or low-dose exposure to ST can lead to transient but significant increases in liver enzymes, further emphasizing its hepatotoxic potential.[24] Similarly, Abeles et al. (2020) reported that anabolic steroids induced liver injury frequently manifests with elevated liver enzymes and may progress to severe hepatotoxicity depending on dose and duration.[16] These reports corroborate the significant elevations in SGOT and SGPT observed in the current study. Also, the consistent increase in SGOT and SGPT across all treated groups in both postnatal male and female mice suggests that ST exerts a systemic effect even at low doses, reinforcing concerns.

Furthermore, experimental studies have also reinforced the hepatotoxic potential of AAS. Chronic exposure to anabolic steroids induces hepatic steatosis and metabolic disturbances in rodents, suggesting that prolonged steroid administration disrupts lipid metabolism and increases hepatic distress.[18] Increased SGOT and SGPT levels have been documented in both clinical cases of anabolic steroid abuse and experimental animal studies exposed to synthetic androgens.[25,19] According to Petrovic et al. (2022), the underlying mechanisms of AAS-induced liver injury involve oxidative stress, mitochondrial dysfunction, disturbance of antioxidant defense systems, increased bile acid synthesis, and hepatocyte hyperplasia. These alterations compromise hepatocellular integrity, leading to leakage of intracellular enzymes into the circulation and subsequent impairment in liver function.[26] Such mechanisms may underlie the elevations of serum SGOT and SGPT transaminases observed in the present investigation. Previous case reports have documented severe outcomes, including cholestatic jaundice and liver injury following stanozolol misuse, further supporting the pathological relevance of elevated transaminases.[27] Although histopathological analysis of liver tissue was not performed in the present study, the biochemical alterations in transaminases -

SGOT and SGPT indicate underlying structural damage such as hepatocellular degeneration and fatty liver disease (steatosis).

Based on the above results, the present findings indicate that irrespective of sex, prolonged administration of ST may induce liver disease, such as liver cirrhosis, steatosis, and inflammation in liver cells, which may lead to hepatic dysfunction and hepatotoxicity in postnatal male and female mice. Further investigations integrating histological, molecular and recovery-based analyses are warranted to elucidate the full spectrum and reversibility of ST-induced hepatic damage.

CONCLUSION

It is concluded that long-term administration of ST to postnatal male and female mice leads to an elevation in both SGOT and SGPT liver enzymes, irrespective of sex, which indicates ST-induced hepatotoxicity leading to hepatic dysfunction.

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