

Research Paper

Ameliorative Effect of Vitamin C on *Cannabis sativa*-induced Nephrotoxicity in Wistar rats

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ABSTRACT

Background:

The kidneys play a critical role in removing waste products and excess water from the blood, as well as maintaining chemical balance. Damage to the kidneys can pose serious health risks. Cannabis sativa (CS) is one of the commonly abused substances and has been associated with various adverse health effects.

Objective:

This study investigated the ameliorative effects of Vitamin C (Vit C) on CS-induced nephrotoxicity in male and female rats.

Method:

Different doses of CS (2, 4, and 6 mg/kg) and Vit C (4 mg/kg) were administered orally, both individually and in combination, for a period of three weeks. Kidney function parameters, including urea, creatinine, induced protein, albumin, sodium (Na), and potassium (K), were measured using standard methods.

Results:

CS administration significantly increased urea and creatinine levels in the high dose groups (4 and 6 mg/kg) compared with the control and low dose (2 mg/kg) groups in both sexes. Conversely, induced protein, albumin, Na, and K levels were significantly decreased in the high dose groups. However, these adverse effects were significantly ameliorated at the 4 mg/kg CS dose when co-administered with Vit C.

Conclusion:

The effects of CS on kidney function were dose dependent and sex-specific, while Vit C exhibited a protective effect, particularly at the 4 mg/kg dose. These findings suggest that further studies are needed to evaluate whether higher doses of Vit C could mitigate the nephrotoxicity observed at the 6 mg/kg CS dose, since the same dose of Vit C was administered across all treatment groups.

KEYWORDS: *Cannabis sativa*; Vitamin C; Nephrotoxicity; Renal function; Oxidative stress; Wistar rats.

INTRODUCTION

Cannabis(*C.*) is a genus of flowering plants belonging to the family *Cannabaceae*. It is generally classified into three species: *Cannabis sativa*, *Cannabis indica*, and *Cannabis ruderalis*. However, Simiyu DC, Jang JH, Lee OR. Understanding *Cannabis sativa* L.: current status of propagation, use, legalization, and haploid-inducer-mediated genetic engineering. *Plants*. 2022 May 2;11(9):1236¹. Globally, cannabis remains one of the most commonly used illicit substances², and its consumption has been linked to a range of adverse health outcomes in both humans and animals. Several studies have reported the detrimental effects of *C. sativa* use, whether through ingestion or inhalation, on multiple physiological systems²⁻¹⁵.

Vitamin C (Vit C), also referred to as ascorbic acid, is well known for its potent antioxidant activity¹⁶. It exerts its effects primarily by neutralizing free radicals and preventing oxidative damage to critical biomolecules, including DNA, proteins, and lipids¹⁷⁻¹⁸. Furthermore, Vit C contributes to the regeneration of other antioxidants, particularly Vitamin E, thereby strengthening the body's overall antioxidant defense system¹⁹. In plant systems, including cannabis, Vit C also plays a role in mitigating oxidative stress by scavenging reactive oxygen species and maintaining cellular stability²⁰. A deeper understanding of the role of Vit C in oxidative stress regulation may aid in developing strategies to enhance antioxidant capacity and improve resilience to both environmental and physiological stressors²¹.

The kidneys are paired organs situated along the posterior abdominal wall. They perform essential functions, including the excretion of metabolic waste products and toxic substances from the bloodstream, as well as the regulation of fluid and electrolyte homeostasis²². Previous studies have reported both protective and harmful effects of *Cannabis sativa* (CS) on renal function in humans and experimental animals²³⁻²⁵. However, to the best of our knowledge, no study has investigated the use of Vit C in mitigating CS-induced nephrotoxicity.

Therefore, this study was designed to fill this gap by evaluating the protective effects of Vit C against CS-induced impairment of kidney function in both male and female Wistar rats, with particular emphasis on sex-related differences and dose-dependent responses. Key renal biomarkers, including urea, creatinine, total protein, albumin, sodium, and potassium, were assessed, as they serve as important indicators of kidney function.

The objectives of this study were to: (i) evaluate the effects of *Cannabis sativa* (CS) on kidney toxicity in male and female Wistar rats; (ii) compare the extent of CS-induced kidney toxicity between sexes; (iii) examine sex-specific variations in renal toxicity in relation to dose-dependent exposure; and (iv) assess the protective effects of Vit C against CS-induced kidney toxicity in both male and female Wistar rats.

The study was guided by the following hypotheses:

Null hypothesis (H₀): Vit C has no protective effect against CS-induced kidney toxicity in male and female Wistar rats.

Alternative hypothesis (H₁): Vit C exerts a protective effect against CS-induced kidney toxicity in male and female Wistar rats.

Methods

Sample Collection

The leaves of CS used in this experiment were obtained from the Nigerian National Drug Law Enforcement Agency (NDLEA), solely for research study.

Extraction of *Cannabis sativa* Leaves

The extraction was done using Soxhlet apparatus after 450 grams of CS was soaked in 98% ethanol for two days. Filtration method was used to separate the filtrate, which was poured into a round bottom conical flask mounted on a rotary evaporator. It was then allowed to cool after evaporation. Approximately, 32g of the yield was obtained after drying.

Experimental Animals

The principle of ARRIVE 2.0 guideline was used throughout the experiment. Forty male (165±12 g) and forty female Wistar rats (140±9 g) were supplied by Temilade Animal Venture, Nigeria. The animals have access to food and water without restriction to day/light period. They were taken care of in line with the Principles of laboratory animal care (NIH publication No. 85-23, revised 1985). The Ethical Committee of University of Ilesa, Ilesa, Osun State, Nigeria approved the experimental protocol (UNILESA/ERC/2026/033).

Experimental Protocol

After two weeks of acclimatization, the animals (male and female) were randomly assigned into experimental groups, with five rats per group for each sex. The rats in groups IM & 1F, IIM & IIF, IIIM & IIIF, and IVM & IVF received the following treatments, respectively: 1 mL of distilled water (control), CS at 2 mg/kg body weight (bw), CS at 4 mg/kg bw, CS at 6 mg/kg bw, Vitamin C (Vit C) at 4 mg/kg bw, CS (2 mg/kg bw) + Vit C (4 mg/kg bw), CS (4 mg/kg bw) + Vit C (4 mg/kg bw), and CS (6 mg/kg bw) + Vit C (4 mg/kg bw). All treatments were administered orally once daily via gavage between 08:00 and 10:00 h for 21 consecutive days. At the end of the treatment period, the animals were sacrificed after the final dose, with free access to food and water maintained until sacrifice.

Preparation of Serum

ketamine (80mg/kg bw) was used to induce sleep in the animal through intraperitoneal injection, after which, sample plain bottles were used for blood collection. After 30 min, the blood

clotted and centrifuged at $625\times g$ for 10 min with the help of a Uniscope Laboratory Centrifuge (Model SM800B, Surgifield Medicals, Essex, England). With the aid of a Pasteur pipette, the serum was collected into plain bottles. Sera were used within 12 hours of preparation after being stored in a freezer maintained at -5°C .

Determination of Albumin and IP

Albumin, induced protein (IP), and total protein levels were determined spectrophotometrically using a SpectraMax Plus spectrophotometer (Molecular Devices, Sunnyvale, CA, USA), according to the manufacturer's instructions (Product Code: BXC0243; Fortress Diagnostics, UK).

Determination of Urea

Urea was determined by Urease method²⁶ shown below:

Urea is hydrolyzed by the enzyme urease to produce ammonia and carbon(iv)oxide. The ammonia released reacted with a phenol-hypochlorite reagent in an alkaline medium to form blue-coloured indophenols dye. The intensity of the blue colour was measured spectrophotometrically and this was directly proportional to the urea concentration in the sample.

Determination of Creatinine

In an alkaline medium, creatinine reacts with **Picric acid** to form an orange-red colored complex (creatinine-picric acid complex). The intensity of the color produced is directly proportional to the creatinine concentration in the sample²⁷.

Determination of Sodium

Sodium reacts with sodium tetraphenylborate in a specially prepared buffer to form a colloidal suspension. The degree of turbidity produced is directly proportional to the sodium concentration in the sample.

Determination of Potassium

Potassium reacts with potassium tetraphenylborate in a specially prepared buffer to form a colloidal suspension. The resulting turbidity is directly proportional to the potassium concentration in the sample.

Statistical Analysis

Mean $\pm$ standard error of mean was used to express the data obtained from the results. Students Package for Social Science, version 20.0 (SPSS Inc., Chicago, USA) and Graph Pad Prism (version 9.0) were used to interpret the data. Two-way analysis of variance was used to analyze the data with the significant differences determined by the least significance difference (LSD) post-hoc test for all the parameters. Shapiro-Wilk and Bartlett's Tests were used to test for normality and homogeneity among groups. The statistically significant was $P < 0.05$ for all values.

Results

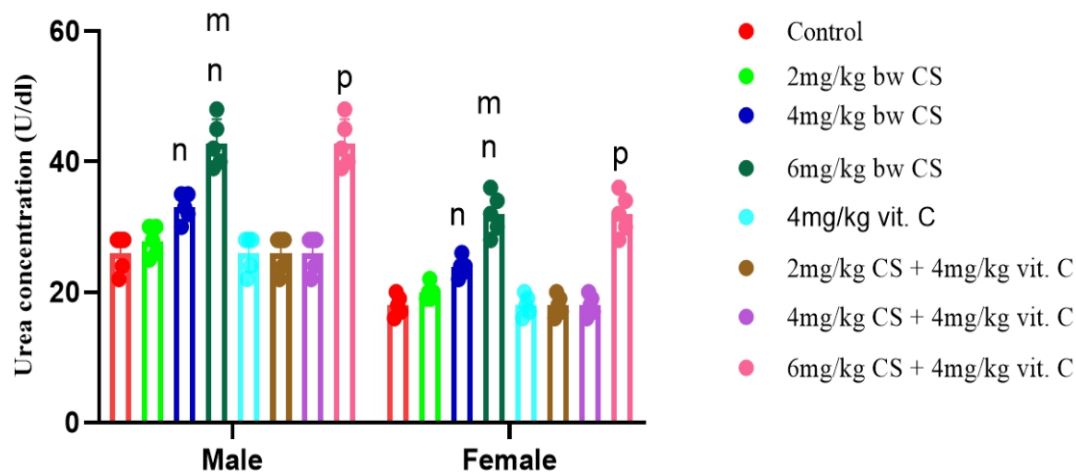


Figure 1: Urea Concentration for Control, 2 mg/kgbw CS, 4 mg/kgbw CS, 6 mg/kgbw CS, 4 mg/kgbw Vit. C, 2 mg/kgbw CS + 4 mg/kgbw Vit. C, 4 mg/kgbw CS + 4 mg/kgbw Vit. C, and 6 mg/kgbw CS + 4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: $P < 0.05$ vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: $P < 0.05$ vs CS (4 mg/kgbw) (male and female)

p: $P < 0.05$ vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

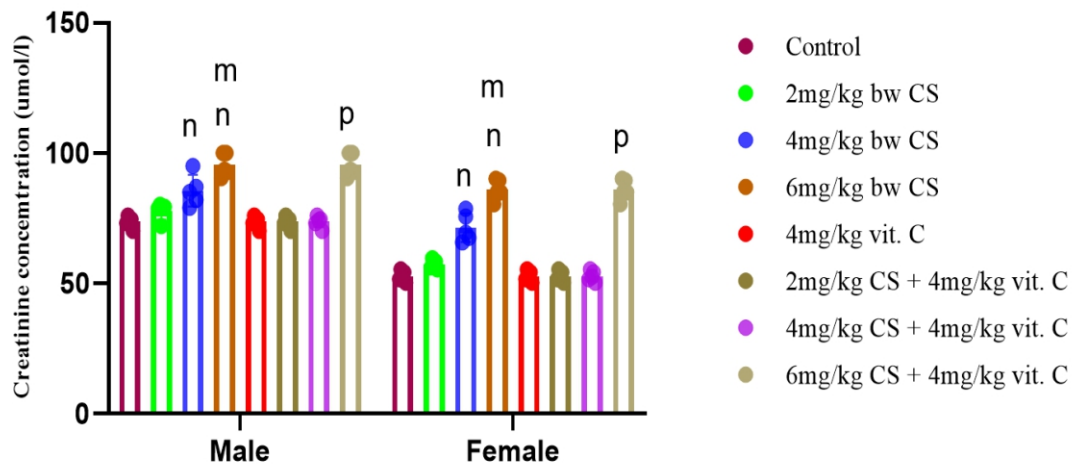


Figure 2: Creatinine Concentration for Control, 2mg/kgbw CS, 4mg/kgbw CS, 6mg/kgbw CS, 4mg/kgbw Vit. C, 2mg/kgbw CS+4mg/kgbw Vit. C, 4mg/kgbw CS+4mg/kgbw Vit. C, and 6mg/kgbw CS+4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: $P < 0.05$ vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: $P < 0.05$ vs CS (4 mg/kgbw) (male and female)

p: $P < 0.05$ vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

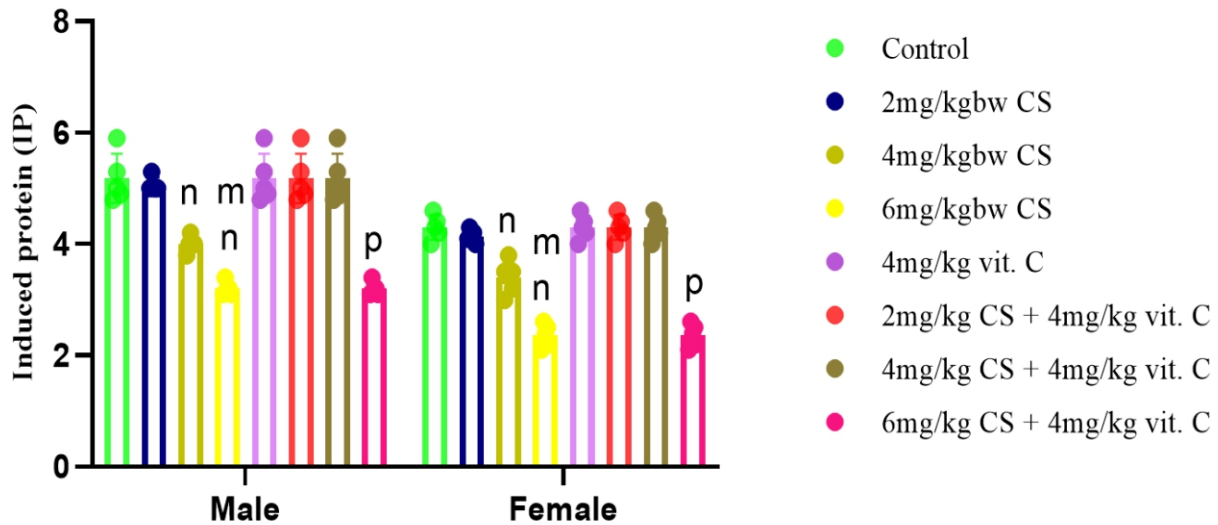


Figure 3: IP for Control, 2 mg/kgbw CS, 4 mg/kgbw CS, 6 mg/kgbw CS, 4 mg/kgbw Vit. C, 2 mg/kgbw CS + 4 mg/kgbw Vit. C, 4 mg/kgbw CS + 4 mg/kgbw Vit. C, and 6 mg/kgbw CS + 4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: $P < 0.05$ vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: $P < 0.05$ vs CS (4 mg/kgbw) (male and female)

p: $P < 0.05$ vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

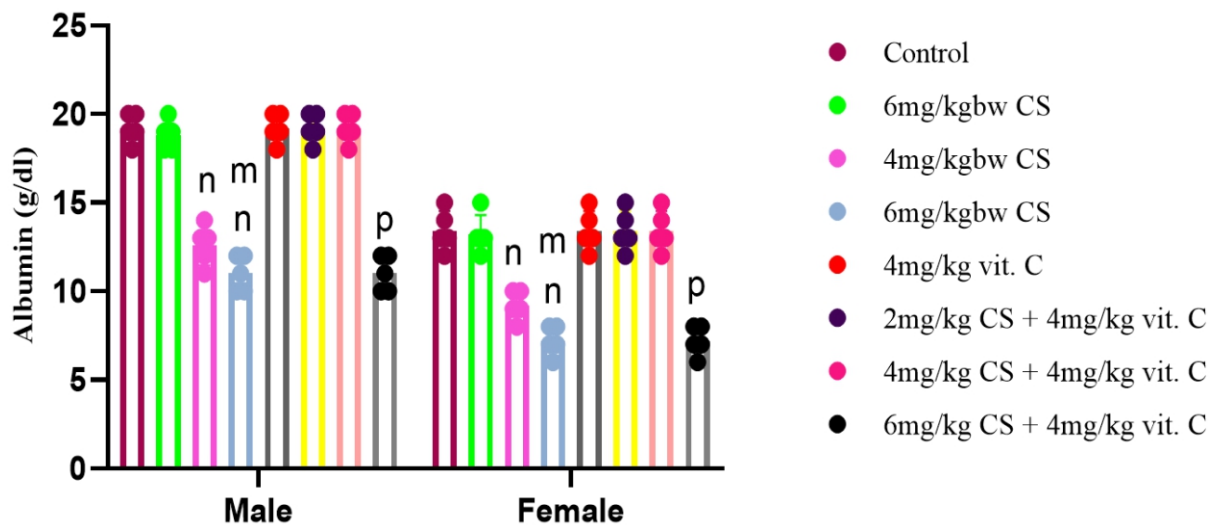


Figure 4: Albumin for Control, 2 mg/kgbw CS, 4 mg/kgbw CS, 6 mg/kgbw CS, 4 mg/kgbw Vit. C, 2 mg/kgbw CS + 4 mg/kgbw Vit. C, 4 mg/kgbw CS + 4 mg/kgbw Vit. C, and 6 mg/kgbw CS + 4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: $P < 0.05$ vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: $P < 0.05$ vs CS (4 mg/kgbw) (male and female)

p: $P < 0.05$ vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

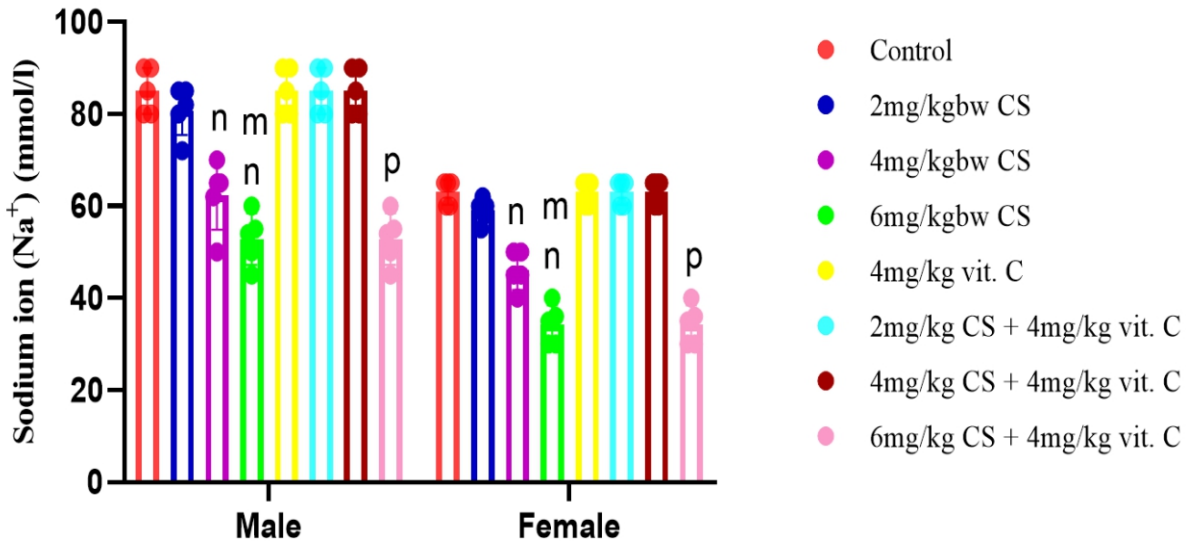


Figure 5: Na⁺ for Control, 2 mg/kgbw CS, 4 mg/kgbw CS, 6 mg/kgbw CS, 4 mg/kgbw Vit. C, 2 mg/kgbw CS + 4 mg/kgbw Vit. C, 4 mg/kgbw CS + 4 mg/kgbw Vit. C, and 6 mg/kgbw CS + 4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ standard error of mean. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: P<0.05 vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: P<0.05 vs CS (4 mg/kgbw) (male and female)

p: P<0.05 vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

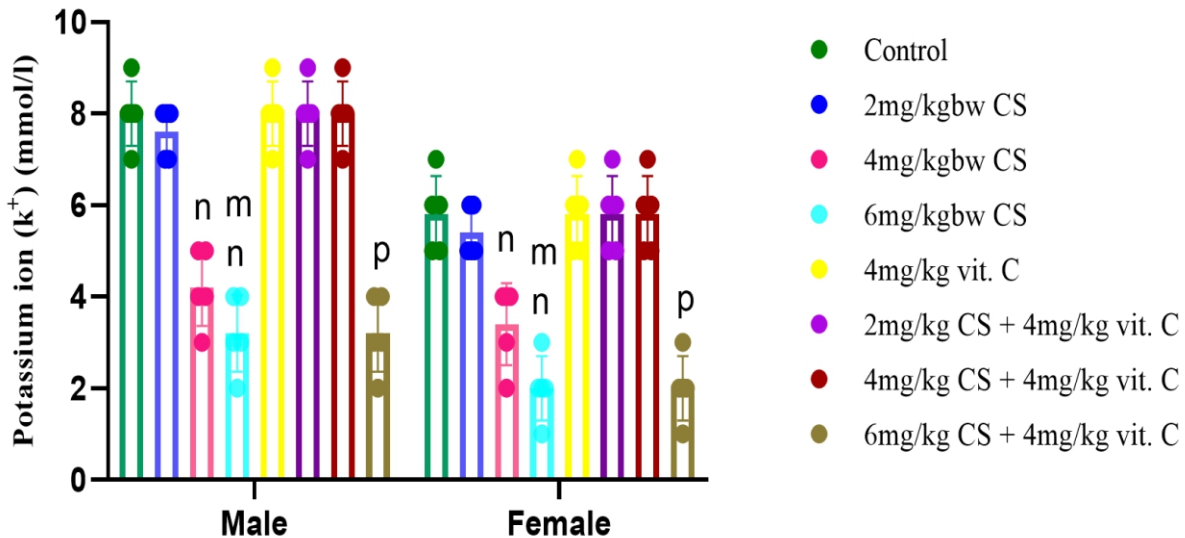


Figure 6: K⁺ for Control, 2 mg/kgbw CS, 4 mg/kgbw CS, 6 mg/kgbw CS, 4 mg/kgbw Vit. C, 2 mg/kgbw CS + 4 mg/kgbw Vit. C, 4 mg/kgbw CS + 4 mg/kgbw Vit. C, and 6 mg/kgbw CS + 4mg/kgbw Vit. C respectively. Values are expressed as mean $\pm$ standard error of mean. Values are expressed as mean $\pm$ SEM; Number of experiments (n=80, 40 males and 40 females)

Where:

n: P<0.05 vs control, 2 mg/kgbw CS, and Vit C groups (male and female)

m: P<0.05 vs CS (4 mg/kgbw) (male and female)

p: P<0.05 vs control, Vit C, and 4 mg/kgbw CS + 4mg/kgbw Vit C (male and female) groups

Vit C: Vitamin C

CS administration significantly increased urea and creatinine levels in the high-dose groups (4 and 6 mg/kg) compared with the control and low-dose (2 mg/kg) groups in both sexes [Figures 1 and 2]. In contrast, IP, albumin, Na⁺, and K⁺ levels were significantly reduced in the high-dose groups [Figures 3–6]. However, these alterations were significantly attenuated in animals co-administered Vitamin C at the 4 mg/kg CS dose [Figures 1–6]. Furthermore, significant sex-related differences ($p < 0.05$) were observed across all measured parameters, with males exhibiting more pronounced changes than females in the high-dose groups.

DISCUSSION

This study demonstrated that low-dose CS exposure does not pose a significant risk to renal physiological function over a short duration, whereas high-dose exposure exerts detrimental effects on kidney function irrespective of exposure period.

The use of cannabis may have important implications for renal health. Evidence suggests that heavy cannabis consumption is associated with an increased risk of chronic kidney disease (CKD)²⁸, a condition characterized by a progressive decline in kidney function over time^{28–30}. Cannabis may also influence renal hemodynamics, potentially altering kidney function³⁰. $\Delta 9$ -tetrahydrocannabinol (THC), the primary psychoactive constituent of cannabis, affects vascular tone by modulating vasodilation and vasoconstriction, thereby influencing renal blood flow and glomerular filtration³⁰. In addition, cannabis use has been associated with alterations in urinary parameters, including increased urine output and frequency, which may reflect its effects on hydration status and the regulation of renal function³¹.

In the kidney, cannabinoid receptors 1 (CB₁) and 2 (CB₂) play important roles in regulating renal blood flow, glomerular filtration rate (GFR), glomerular permeability, interstitial collagen deposition, and tubular function³². The observed increase in urea and creatinine levels at high doses in this study is consistent with the findings of Rein³³ and Nedumaran et al.³⁴, who reported elevated urea and creatinine levels following CS administration in rats. This effect may be attributed to alterations in glomerular filtration rate and renal blood flow³³, which impair the kidney's ability to effectively eliminate creatinine and urea, both end products of protein and muscle metabolism³⁵.

However, these findings contradict those of Mobisson et al.³⁶, who reported decreased creatinine and urea levels following CS administration in rats. These discrepancies may be due to differences in sample types used for analysis; whereas their study utilized bile samples, the present study analyzed blood serum.

Moreover, the observed decrease in induced protein (IP), albumin, sodium, potassium, and total protein in the high-dose

groups suggests abnormal loss of these substances through urine rather than their retention in the bloodstream. This may be due to activation of cannabinoid receptors in the renal vasculature and kidney, leading to altered renal handling and increased excretion of these biomolecules³⁷. However, Vitamin C ameliorated CS-induced renal toxicity at the 4 mg/kg dose, likely due to its potent antioxidant properties and ability to reduce oxidative stress^{4,18–21}.

Furthermore, this study reveals sex-specific differences in renal responses to CS, with males exhibiting more pronounced toxicity than females. One of the contributing factors may involve differences in the expression of cannabinoid receptor associated with signaling pathways. It has been reported that males possess more cannabinoid receptors than females³³. Also, CS may induce epigenetic alterations by modifying global DNA methylation patterns, which could be due to cannabis-associated immunosuppression³⁹.

This study has some limitations. First, it remains unclear whether prolonged administration of low-dose CS (2 mg/kg body weight) beyond the three-week period used in this study would produce effects similar to those observed at higher doses (4 and 6 mg/kg body weight). Second, it is not known whether administration at different times of the day (e.g., afternoon or night) would yield outcomes comparable to those observed with morning administration in this study. Third, it is uncertain whether increasing the dose of Vitamin C would provide protection against CS-induced renal toxicity at the highest dose (6 mg/kg), as observed at 4 mg/kg.

A major strength of this study is that Vitamin C demonstrated potential as a mitigating agent against CS-induced kidney toxicity at moderate doses specifically when CS is being used for recreational and medicinal purposes.

This suggests that further studies are needed to determine whether higher doses of Vitamin C could also attenuate the renal toxicity observed in the 6 mg/kg group, given that the same dose of Vitamin C was administered in both the 4 mg/kg and 6 mg/kg treatment groups.

Acknowledgements

I acknowledge Dr. Emeka for rendering the laboratory assistance for the analysis. I also extend my warm appreciation to the Nigerian National Drug Law Enforcement Agency (NDLEA), for willingly donating CS leaves used for this research study.

DECLARATIONS

Funding: None Declared.

Conflict of Interests Disclosure: The author has declared that no competing interests exist.

Declaration of Generative AI and AI-Assisted Technologies: The author declares that no generative AI or AI-assisted technologies were used in the preparation of this manuscript.

ARTICLE INFORMATION

Accepted for Publication: June 9, 2026

Published: June 27, 2026

Peer Review: Externally Peer Reviewed.

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REFERENCES

1. Simiyu DC, Jang JH, Lee OR. Understanding Cannabis sativa L.: current status of propagation, use, legalization, and haploid-inducer-mediated genetic engineering. *Plants*. 2022;11(9):1236.
2. Chouvy PA. Cannabis cultivation in the world: heritages, trends and challenges. *EchoGéo* 2019;13(48).
3. Oluwasola A. Melatonin ameliorates the effects of *Cannabis sativa*-induced oxidative stress in Wistar rats: A dose-dependent and gender-specific study. *J. Cannabis Res*. 2026a;8(27):2-7.
4. Oluwasola A. A comparative study on the protective effect of ascorbic acid (Vitamin c) on *cannabis sativa*-induced oxidative stress in male and female Wistar rats. *Pac J Med Sci*. 2026b;8(27):2-7.
5. Ayoola OE, Oluwasola A, Garba S. *Cannabis sativa* exacerbates inflammatory responses in male and female Wistar rats. *Pac J Med Sci*. 2025;25(2):50-55.
6. Oluwasola A, Lukman J, Comfort AM. Effect of *Cannabis sativa* on Haematological Parameters in Male and Female Wistar Rats. *Asian Pac J Med Toxicolo*. 2024;13(3):90-94.
7. Oluwasola A, Ayoola OE, Garba S, Adepoju MA, Biliaminu SA, Olayaki LA. Melatonin Mitigates Hormonal Toxicity in Cannabis-Treated Female Wistar Rats. *Trop J Health Sci*. 2023a;30(2):14-19.
8. Oluwasola A, Ayoola OE, Garba S, Adepoju MA, Balogun ME, Biliaminu SA et al. The Ameliorative effects of Melatonin on Cannabis- induced Liver toxicity in male and female Wistar rats. *Trop J Health Sci*. 2023b;30(3):20-26.
9. Oluwasola A, Ayoola OE, Odetayo AF, Garba S, Olayaki LA. Ameliorative Effect of Melatonin on Reproductive Hormones in Ethanol Extracts of *Cannabis sativa*-Treated Female Wistar Rats. *NISEB*. 2022a;22(2):27-32.
10. Oluwasola A, Balogun ME, Muzzammill SS, Zainab SO, Adekunle OJ, Mariam EB et al. Effect of Acute Administration of Ethanol Extract of *Cannabis sativa* Leaf on Liver Function Biomarkers in Male Wistar Rats. *NISEB*. 2022b;22(1):20-26.
11. Abdulrahim HA, Alagbonsi IA, Oluwasola A, Noah AO, Abdul-Rahuf AF, Luqman AO. *Cannabis sativa* and/or melatonin do not alter brain lipid but alter oxidative mechanisms in female rats. *J Cannabis Res*. 2021; 3:38-48.
12. Oluwasola A, Olayaki LA, Ayinde TO. Effects of Melatonin and Ethanolic Extract of *Cannabis sativa* Leaves on Sexual Behaviour Parameters of Female Wistar Rats. *BiosResJ*. 2020a;32(1):29-36.
13. Olayaki, Luqman & Oluwasola. PhD. Fipmld, Amuda. (2020). Melatonin Mitigates Ovarian Toxicity In Cannabis-treated Female Wistar Rats. *The FASEB Journal*. 34. 1-1. 10.1096/fasebj.2020.34.s1.03900.
14. Oluwasola A, Olayaki LA, Ayinde T. Effects of Melatonin on Estrous Cycle Changes Induced by Ethanolic Extract of *Cannabis sativa* in Female Wistar Rats. *NISEB*. 2019a;19(12):55-60.
15. Oluwasola A, Ayinde TO. Comparative Study on the Antioxidant Potentials of Honey and Some Selected Beverages in Male Wistar Rats. *AJE*. 2019b;6(2):1-7.
16. Agwu, Eunice & Ezihe, Christian & Kaigama, Gyelkur. (2023). Antioxidant Roles/Functions of Ascorbic Acid (Vitamin C). 10.5772/intechopen.110589.

17. McClain R. Cheating Death: The New Science of Living Longer and Better. Ben Bella Books; 2023.
18. Sundaram SS, Shukla AK. Free radicals versus antioxidants. In Potential Therapeutic Applications of Nano-antioxidants. Springer Singapore; 2021: p.1-17.
19. Khadim RM, Al-Fartusie FS. Antioxidant Vitamins and their effect on immune system. In J Phys. 2021;1853(1):012065.
20. Mladenov M, Lubomirov L, Grisk O, Avtanski D, Mitrokhin V, Sazdova I. et al. Oxidative stress, reductive stress and antioxidants in vascular pathogenesis and aging. Antioxidants.2023;19;12(5):1126.
21. Foyer CH, Kyndt T, Hancock RD. Vitamin C in plants: novel concepts, new perspectives, and outstanding issues. Antioxid Redox Signal. 2020;1;32(7):463-485.
22. Unny N, Madhavan & Aziz, Zarina & Vakayil, Beena. (2023). Fluid and Electrolyte Balance. 10.1007/978-981-19-9410-4_8.
23. Rein JL, Zeng H. A retrospective cohort study that examined the impact of cannabis consumption on long-term kidney outcomes. Cannabis and Cannabinoid Res.2024;9(2):635-45.
24. Collister D, Pyne L Bhasin AA. Sex and gender in randomized controlled trials of adults receiving maintenance dialysis: A meta-epidemiologic study. AJKD.2023;81(5):575-82.
25. Młynarska E, Kustosik N, Mejza M, Łysoń Z, Delebis D, Orliński J, Rysz J, Franczyk B. Renal Outcomes and Other Adverse Effects of Cannabinoid Supplementation. Nutrients. 2024 Dec 27;17(1):59. doi: 10.3390/nu17010059. PMID: 39796493; PMCID: PMC11722839.
26. Ali SM, Ibupoto ZH, Salman S, Nur O, Willander M, Danielsson B. Selective determination of urea using urease immobilized on ZnO nanowires. Sens Actuators B Chem.2011;160(1):637-43.
27. Miller Z, Miller BF. Specific determination of serum creatinine. Proc Soc Exp Biol Med. 1951;78(2):471–473.
28. Potukuchi PK, Moradi H, Park F. Cannabis use and risk of acute kidney injury in patients with advanced chronic kidney disease transitioning to dialysis. Cannabis and cannabinoid Res. 2023;8(1):138-47.
29. Tonial N. Non-Renal Alterations of Drug Disposition in Chronic Kidney Disease (Doctoral dissertation. The University of Western Ontario (Canada)); 2023.
30. Gitau K, Howe HS. Therapeutic cannabis use in kidney disease: A survey of Canadian nephrologists. Kidney Med. 2022;4(5):100453.
31. Golosova D, Levchenko V. Acute and long-term effects of cannabinoids on hypertension and kidney injury. SciRep. 2022;12(1):6080.
32. Pressly JD, Soni H. Activation of the cannabinoid receptor 2 increases renal perfusion. Physiol Genomics. 2019;51(3):90-96.
33. Rein JL. The nephrologist's guide to cannabis and cannabinoids. Curr Opin Nephrol Hypertens.2020;29(2):248-57.
34. Nedumaran B, Rudra P, Gaydos J, Kumar S, Meacham RB, Burnham EL, Malykhina AP. Impact of Regular Cannabis Use on Biomarkers of Lower Urinary Tract Function. Urology. 2017 Nov;109:223.e9-223.e16. doi: 10.1016/j.urology.2017.08.011. Epub 2017 Aug 18. PMID: 28827195; PMCID: PMC6541002.
35. Onyema JO, Onyieke EN, Amadi BA. Mitigating Marijuana-Induced Nephrotoxicity: The Role of Selected Local Spice Diets in Wistar Rats. Int J Innov Biochem Microbiol Res. 2024;12(4):1-15.
36. Mobisson SK, Onyebuagu PC, Wopara I. Cannabidiol Oil and Prednisolone Treatment Altered Hematologic Indices, Serum Urea, Creatinine and Cellular Architecture of Kidney on Cadmium Induced Toxicity in Male Wistar Rats. J Pharm Res. Int. 2023;35(21):14-27.
37. Okon VE, Obembe AO, Osim EE. Decreased biliary secretion, biliary composition and serum electrolytes with chronic administration of *Cannabis sativa* in albino Wistar rats. NJMRD.2023;8(2):21-25.
38. Liu X, Li X, Zhao G. Sexual dimorphic distribution of cannabinoid receptor mRNA in adult C57BL/6J mice. J Comp Neuro. 2020;528(12):1986-99.
39. Smith RC, Sershen H, Janowsky DS. Changes in Expression of DNAMethyltransferase and Cannabinoid Receptor mRNAs in Blood Lymphocytes After Acute Cannabis Smoking. Front Psychiatry. 2022;13:887700.