EFFECT OF SWEET BASIL (OCIMUM BASILICUM) LEAF EXTRACT ON KIDNEY FUNCTION PARAMETERS: SODIUM, POTASSIUM, CHLORIDE, BICARBONATE, UREA, CREATININE, AND URIC ACID.

BY

EZEIKE EMMANUEL SOMTOCHUKWU

(BMS2102126)

DEPARTMENT OF PHYSIOLOGY,

SCHOOL OF BASIC MEDICAL SCIENCES,

COLLEGE OF MEDICAL SCIENCES,

UNIVERSITY OF BENIN.

SUPERVISED BY:

DR. C. O. AZUBIKE

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EZEIKE EMMANUEL SOMTOCHUKWU

(BMS2102126)

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CERTIFICATION

We certify that this project was carried out by EZEIKE EMMANUEL SOMTOCHUKWU, with matriculation number BMS2102126, of the Department of Physiology, School of Basic Medical Sciences, University of Benin, and thereby approve same as adequate in scope and quality for the award of Bachelor of Science Degree (B.Sc) in Physiology.

..... **.....**

Dr. C.O. Azubike **Date**

Project supervisor

..... **.....**

Prof. O.K. Uche **Date**

Head of Department

....

External Examiner **Date**

DEDICATION

This project work is dedicated to MR. EZEIKE PATRICK, MISS EZEIKE COMFORT and the entire EZEIKE family for their constant support.

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ABSTRACT

Sweet Basil (Ocimum basilicum) is a widely utilized medicinal herb recognized for its antioxidant, anti-inflammatory, and protective phytochemicals; however, its renal safety profile under normal physiological conditions remains inadequately characterized. This study evaluated the effect of ethanolic extract of Ocimum basilicum leaves on kidney function parameters in healthy female Wistar rats. Thirty rats were randomly distributed into five groups (n=6 per group): a control group receiving distilled water and four test groups administered 300, 500, 1000, and 1500 mg/kg body weight of Basil extract orally for 21 days. At the end of the treatment period, serum samples were analyzed for electrolytes (sodium, potassium, chloride, bicarbonate) and markers of renal function (urea, creatinine, and uric acid) using standard biochemical protocols. Data were statistically analyzed using one-way ANOVA. Results revealed no statistically significant differences ($p>0.05$) among the groups for all evaluated parameters, though mild dose-dependent fluctuations were observed, all remaining within normal physiological ranges. Sodium, potassium, chloride, and bicarbonate concentrations exhibited no notable disturbances, indicating maintained electrolyte and acid-base balance, while urea, creatinine, and uric acid levels remained stable, suggesting preserved glomerular filtration and tubular function. These findings suggest that sub-chronic administration of ethanolic Sweet Basil leaf extract at doses up to 1500 mg/kg does not adversely affect renal biochemical indices in normal female Wistar rats, demonstrating its relative safety under non-pathological conditions. It is concluded that Ocimum basilicum is safe within the tested dosage and duration; however, further research incorporating histopathological assessment, molecular markers of renal function, longer durations of administration, and diseased models is recommended to fully validate its renal safety and therapeutic potential.

CHAPTER ONE

1.0 Background of the Study

The kidneys are two bean-shaped organs, each roughly the size of a fist and weighing approximately 120 to 150 grams in adults, positioned on either side of the spine in the retroperitoneal space just below the ribcage. These essential organs process around 180 liters of blood daily through about one million nephrons per kidney, filtering out waste products like urea, creatinine, and uric acid while reabsorbing vital substances such as water, glucose, and electrolytes. This filtration process is crucial for maintaining the body's internal environment, regulating blood pressure through the renin-angiotensin-aldosterone system, stimulating red blood cell production via erythropoietin, activating vitamin D for bone health and calcium balance, and controlling acid-base equilibrium by reabsorbing bicarbonate and excreting hydrogen ions. Any disruption in these functions, caused by factors such as diabetes, hypertension, infections, or exposure to nephrotoxic substances like heavy metals or drugs, can lead to serious conditions including acute kidney injury, chronic kidney disease, or end-stage renal failure. Globally, chronic kidney

disease affects over 850 million people and contributes to millions of deaths annually, highlighting the urgent need for effective and accessible interventions to support renal health (Hill et al., 2024).

Kidney function is assessed through a panel of biochemical parameters, including electrolytes such as sodium, potassium, chloride, and bicarbonate, as well as waste products like urea, creatinine, and uric acid. Sodium, the primary extracellular cation, is key for osmotic pressure and fluid volume regulation, with imbalances leading to hypertension or dehydration often linked to glomerular filtration rate impairments. Potassium, mostly intracellular, governs nerve and muscle function, including cardiac rhythm, where hyperkalemia or hypokalemia can stem from tubular dysfunction. Chloride collaborates with sodium to preserve acid-base balance, while bicarbonate acts as a primary buffer against acidosis, stabilizing blood pH. Urea, derived from protein breakdown, rises with reduced glomerular filtration rate, indicating azotemia. Creatinine, from muscle creatine, is a reliable glomerular filtration rate estimator as it is freely filtered and minimally reabsorbed. Uric acid, from purine metabolism, accumulates in gout or renal insufficiency, promoting oxidative stress and inflammation (Levey et al., 2020).

Amid concerns over the side effects and high costs of synthetic drugs for kidney disorders, herbal remedies have gained attention for their potential nephroprotective properties. Traditional medicine systems, especially in Africa and Asia, have long utilized plants for healing. Ocimum basilicum, commonly known as sweet basil, is an annual herb from the Lamiaceae family, native to tropical regions including Africa and Asia. This plant, growing up to 1 meter tall, features broad, oval leaves with a sweet, aromatic scent due to essential oils. Beyond culinary uses, sweet basil is employed in folk medicine for digestive issues, inflammation, and respiratory problems. In some cultures, its leaves are used to relieve kidney discomfort, suggesting a possible protective role on the kidneys (Kwee and Niemeyer, 2011).

Phytochemically, Ocimum basilicum is abundant in bioactive compounds, including essential oils like linalool and eugenol, flavonoids such as quercetin and rutin, phenolic acids like rosmarinic and caffeic acid, tannins, glycosides, and steroids. These

components contribute to its antioxidant, anti-inflammatory, and diuretic activities. Flavonoids and phenolics neutralize free radicals, reducing lipid peroxidation and boosting enzymes like superoxide dismutase and glutathione. In vitro and in vivo studies emphasize the plant's capacity to alleviate oxidative stress, a major contributor to kidney diseases.

For example, the leaves' essential oil and extracts have shown protective effects on the kidneys in animal models by normalizing renal markers (Afshari *et al*., 2007).

Research on sweet basil extract's impact on kidney function is promising but limited. In gentamicin-induced nephrotoxicity, where the antibiotic causes tubular damage and elevates urea and creatinine, aqueous extracts of Ocimum basilicum at 200-800 mg/kg significantly lowered serum urea and creatinine ($p < 0.05$), while improving antioxidant levels like glutathione peroxidase (Khairnar *et al*., 2018). This indicates a protective mechanism against antibiotic-related renal toxicity. In diabetic rats, ethanolic extract administration (200-400 mg/kg) for 28 days dose-dependently reduced serum urea and creatinine ($p < 0.001$), enhanced plasma antioxidant capacity ($p < 0.01$), and promoted histological recovery of kidney tissues (Bayomy and Sakr, 2016).

Regarding specific parameters, studies on thioacetamide-induced injury show that aqueous extract restores urinary sodium excretion, disrupted in injury models, while maintaining serum sodium stability. It also normalizes urinary creatinine and urea, reducing proteinuria and preserving glomerular filtration rate (Al-Harbi *et al*., 2019). Although direct data on potassium, chloride, and bicarbonate are scarce, the overall enhancement of antioxidant status implies benefits for electrolyte balance, as oxidative stress often perturbs ion transport in renal tubules. In ageing models, ethanolic extract lowered creatinine, suggesting anti-ageing renal effects (Eftekhar *et al*., 2019).

Uric acid modulation by sweet basil remains understudied, but its anti-inflammatory properties may inhibit xanthine oxidase, reducing uric acid production. In broader contexts, like paracetamol-induced toxicity, sweet basil ameliorated hepatic and renal

damage, though specific electrolyte changes were not detailed (Sakr and Al-Amoudi, 2012). Ultrasound-assisted leaf extract in diabetic models improved glycemic control, indirectly supporting renal health by reducing hyperglycemia, a precursor to altered parameters like elevated urea and creatinine (Zangeneh *et al*., 2019).

The multi-target potential of Ocimum basilicum extends to cardiovascular and metabolic disorders, but its nephroprotective role is evident in hypoglycemic and antioxidant actions (Widjaja *et al*., 2019). In combination therapies with other herbs, it enhances protection against chronic kidney disease in adenine-induced models (Saha *et al*., 2012).

1.1 JUSTIFICATION OF STUDY

The effects of sweet basil (Ocimum basilicum L.) on physiological systems are dose-dependent, yet data on its specific impact on key kidney function parameters remains limited and fragmented. This study aims to evaluate the effect of basil leaf extract on renal function to clarify both its safety and potential therapeutic value.

1.2 AIM

The aim of this study is to investigate the effect of Sweet Basil leaf extract (Ocimum basilicum) on kidney function parameters; sodium, potassium, chloride, bicarbonate, urea, creatinine and uric acid in Wistar rats.

1.3 Statement of the Problem

Despite advancements in renal care, kidney diseases remain a major public health burden, worsened by diabetes and toxin exposure. Conventional treatments like dialysis or drugs often have adverse effects, prompting the need for safer alternatives. While preliminary

studies indicate sweet basil leaf extract's benefits on renal parameters, comprehensive data on all key markers: sodium, potassium, chloride, bicarbonate, urea, creatinine, and uric acid are lacking. This gap hinders its integration into therapeutic protocols. Moreover, most research uses animal models, limiting human applicability (Fiseha and Tamir, 2022).

1.4 Objectives of the Study

The general objective is to evaluate the effects of sweet basil leaf extract on kidney function parameters in a suitable model.

Specific objectives include:

To assess changes in serum and urinary sodium, potassium, chloride, and bicarbonate levels post-extract administration.

To measure alterations in urea, creatinine, and uric acid concentrations.

To investigate underlying mechanisms, such as antioxidant activity and histological changes.

1.5 Significance of the Study

This research could validate sweet basil as a natural nephroprotective agent, offering affordable options for managing kidney disorders, especially in resource-limited settings. Findings may inspire pharmaceutical development of standardized extracts, reducing reliance on synthetic drugs. Academically, it contributes to physiology knowledge on herbal interventions (Olayanju *et al*., 2022).

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Introduction to the Kidney and Kidney Function Parameters

The kidneys are a pair of bean-shaped organs, each approximately 10-12 centimeters in length and weighing between 120-150 grams in adults, strategically located on either side of the vertebral column in the retroperitoneal space just below the ribcage. These remarkable organs serve as the body's primary filtration system, processing an astonishing 180 liters of blood daily through approximately one million nephrons per kidney to produce about 1-2 liters of urine. Each nephron, the functional unit of the kidney, consists of a glomerulus where initial blood filtration occurs, and a tubular system that includes the proximal convoluted tubule, loop of Henle, distal convoluted tubule, and collecting duct, responsible for selective reabsorption and secretion. This intricate process removes metabolic waste products such as urea, creatinine, and uric acid, while conserving essential substances like water, sodium, potassium, chloride, bicarbonate, glucose, and amino acids. Beyond filtration, the kidneys play a multifaceted role in maintaining systemic homeostasis: they regulate blood pressure through the secretion of renin, which activates the renin-angiotensin-aldosterone system to control vascular tone and fluid balance; stimulate red blood cell production via erythropoietin in response to low oxygen levels; activate vitamin D (calciferol) to promote intestinal calcium absorption and bone mineralization; and manage acid-base equilibrium by reabsorbing bicarbonate and excreting hydrogen ions or ammonium. Disruptions in these functions, often triggered by chronic conditions like diabetes mellitus, hypertension, obesity, or exposure to nephrotoxic agents such as gentamicin, thioacetamide, or heavy metals, can lead to acute kidney injury (AKI), chronic kidney disease (CKD), or end-stage renal disease (ESRD). Recent global estimates indicate that CKD affects over 850 million individuals, contributing to more than 2.4 million deaths annually, primarily due to cardiovascular complications and electrolyte imbalances (Hill *et al*., 2024).

The significance of kidney function parameters lies in their ability to provide quantitative insights into the organs' performance, enabling early detection, monitoring, and management of renal disorders. These parameters include electrolytes—sodium (Na^+), potassium (K^+), chloride (Cl^-), and bicarbonate (HCO_3^-)—and waste products—urea, creatinine, and uric acid—each reflecting specific aspects of glomerular filtration, tubular

reabsorption, and secretion. Sodium, the predominant extracellular cation, is essential for maintaining osmotic pressure, nerve impulse transmission, and fluid volume distribution, with about 99% of filtered sodium reabsorbed in the tubules; imbalances like hyponatremia (serum Na+ <135 mEq/L) or hypernatremia (>145 mEq/L) can indicate tubular dysfunction, hormonal dysregulation, or dehydration. Potassium, mostly intracellular, regulates membrane potential and muscle contraction, with daily excretion of 90-100 mEq modulated by aldosterone in the distal nephron; hyperkalemia (>5.5 mEq/L) often signals reduced GFR or tubular damage, posing risks of cardiac arrhythmias. Chloride, an anion closely linked to sodium, supports acid-base balance and osmotic equilibrium, reabsorbed via cotransporters in the loop of Henle; alterations (e.g., hypochloremia <95 mEq/L) may reflect metabolic alkalosis or diuretic use. Bicarbonate, a key buffer, is reabsorbed (85% in proximal tubules via carbonic anhydrase) to neutralize acids, with levels below 22 mEq/L indicating metabolic acidosis common in CKD. Urea, a nitrogenous waste from protein catabolism, rises with reduced GFR (normal range 7-20 mg/dL), serving as a marker for prerenal azotemia or dehydration. Creatinine, produced at a constant rate from muscle creatine, is a reliable GFR estimator (normal 0.6-1.2 mg/dL in females), as it is freely filtered and not reabsorbed.

Uric acid, from purine metabolism, accumulates in hyperuricemia (>6 mg/dL in females), promoting gouty nephropathy through crystal deposition and inflammation (Levey *et al*., 2020). These parameters are routinely measured through serum biochemistry panels, arterial blood gas for bicarbonate, or 24-hour urine collections, facilitating diagnosis (e.g., GFR calculation via creatinine clearance) and therapeutic monitoring, such as in herbal interventions for nephroprotection. The integration of these markers in clinical practice has improved outcomes, but challenges like variability in lab methods and patient factors persist (Olayanju *et al*., 2022).

This chapter comprehensively reviews the existing body of knowledge on the effects of Sweet Basil (Ocimum basilicum) leaf extract on these kidney function parameters, drawing from traditional uses, phytochemical analyses, and experimental evidence in animal models. By synthesizing data from diverse sources, it highlights the plant's potential in renal health, particularly in diabetes and toxicity models, while identifying gaps to justify further investigation. The literature suggests Sweet Basil's role as a natural protective agent, but preclinical dominance calls for human trials (Afshari *et al*., 2007).

2.2 Historical Context and Traditional Use

The historical context of Ocimum basilicum, or Sweet Basil, in traditional medicine is rich and diverse, spanning continents and centuries. Originating from tropical regions of Asia and Africa, this herb has been documented in ancient texts as early as 3000 BCE in Egyptian papyri, where it was used in embalming and as a remedy for digestive and respiratory ailments. In Greek and Roman medicine, Hippocrates and Pliny the Elder praised its diuretic and anti-inflammatory properties, recommending leaf infusions for kidney stones and urinary tract issues. In African traditional systems, particularly in Nigeria's Yoruba and Igbo cultures, Sweet Basil has been a staple for treating renal pain, edema, and infections, often combined with other herbs in decoctions to enhance urine flow and detoxify the body. Ghanaian healers used it for scorpion stings and kidney discomfort, attributing its efficacy to its cooling nature (Kwee and Niemeyer, 2011).

In Asian traditions, Ayurveda classifies Sweet Basil as "Tulsi" (though distinct from Ocimum sanctum), using it to balance "kapha" dosha, associated with fluid retention and metabolic waste, for conditions like dysuria and nephritis. Chinese medicine employs it for "damp-heat" syndromes affecting the kidneys, with leaf extracts for inflammation and diuresis. Unani system in the Middle East prescribes it for urinary irregularities and early renal distress. Ethnopharmacological surveys in West Africa show rural communities using Sweet Basil as an adjuvant for diabetes-induced kidney complications, based on empirical knowledge. These practices have influenced modern research, with retrospective analyses highlighting its diuretic and anti-inflammatory roles (Gupta *et al*., 2021).

Global resurgence in herbal medicine has led to documented uses in Mediterranean cultures for urinary disorders, aligning with folk classifications. Despite this heritage, historical accounts lack quantitative data on parameters like sodium or urea, focusing on holistic benefits. This emphasizes integrating traditional knowledge with science to validate renal potential (Afshari *et al*., 2007).

2.3 Phytochemical Composition of Ocimum basilicum

Ocimum basilicum boasts a complex phytochemical profile that supports its therapeutic applications, including renal protection. The leaves contain essential oils (0.5-1.5% dry weight), with linalool (30-60%), eugenol (10-30%), methyl chavicol (5-20%), and 1,8-cineole (5-15%), contributing to antioxidant and anti-inflammatory effects. Flavonoids like quercetin (0.2-0.5 mg/g), rutin, and kaempferol, phenolic acids such as rosmarinic (1-2 mg/g) and caffeic acid, tannins (5-10%), glycosides, steroids, and saponins enhance free radical scavenging and lipid peroxidation inhibition.

Vitamins (A, C, E) and minerals (potassium 295 mg/100g, magnesium 64 mg/100g, calcium 177 mg/100g) may aid electrolyte balance. These compounds synergistically boost SOD (up to 50% increase in activity) and GSH, protecting renal cells (Khan *et al*., 2020).

Over 60 volatiles and 20 polyphenols have been identified, with variations by cultivar (e.g., Genovese vs. Purple Ruffles) and conditions, where organic farming increases antioxidants by 20-30%. Extraction methods affect yield: ethanolic extracts give 150-200 mg GAE/g phenolics, aqueous 100-150 mg. Environmental factors like drought stress elevate oils by 15%. This diversity enables multi-target actions, modulating oxidative stress and inflammation for renal benefits, but standardization is crucial (El-Saadony *et al*., 2023).

For kidney applications, phenolics may preserve podocyte structure, prevent fibrosis via NF-κB inhibition, and enhance diuresis. Direct links to bicarbonate need exploration. The profile positions Sweet Basil as versatile, but variability requires quality control (Zangeneh *et al*., 2019).

2.4 Effects on Kidney Function Parameters

2.4.1 Sodium Levels

Sodium is a vital extracellular cation essential for osmotic pressure, nerve function, and fluid balance, with the kidneys reabsorbing 99% of filtered sodium—65-70% in proximal tubules via Na+/H+ exchanger, 20-25% in the loop of Henle via Na+-K+-2Cl- cotransporter, and 5-10% in distal tubules regulated by aldosterone. Imbalances (hyponatremia <135 mEq/L or hypernatremia >145 mEq/L) can indicate tubular damage or hormonal dysregulation, often measured in serum to assess renal handling. Studies on Sweet Basil's effect on sodium are emerging, showing potential in injury models. In thioacetamide (TAA)-induced renal injury in rats, aqueous Sweet Basil extract at 200 mg/kg for 21 days restored urinary sodium excretion, which was increased by 50% in toxicity groups due to tubular dysfunction, stabilizing serum sodium at 140 ± 2 mEq/L ($p < 0.05$) compared to 155 ± 3 mEq/L in TAA-alone (Al-Harbi *et al*., 2019). This improvement is attributed to the extract's diuretic properties enhancing renal blood flow.

In gentamicin-induced nephrotoxicity, ethanolic extract at 400 mg/kg over 10 days reduced serum sodium elevation from 148 mEq/L to 138 mEq/L ($p < 0.01$), preventing hyponatremia from tubular damage (Khairnar *et al*., 2018). Diabetic rat models with streptozotocin showed 300 mg/kg extract for 28 days normalizing sodium retention, reducing serum levels by 15% ($p < 0.001$), likely by improving osmotic diuresis (Bayomy and Sakr, 2016). Mechanism of action involves antioxidant compounds like rosmarinic acid protecting Na+/K+-ATPase from oxidative stress, reducing lipid peroxidation by 30-40% and enhancing tubular reabsorption efficiency. Essential oils such as linalool may stimulate aquaporin expression, aiding sodium-water balance (Eftekhar *et al*., 2019).

Comparative studies with other herbs reveal Sweet Basil's advantages. Versus turmeric (Curcuma longa), Sweet Basil at 200 mg/kg showed similar sodium stabilization in TAA

models but faster onset (14 vs. 21 days), due to higher volatile oils (Saha *et al*., 2012). Compared to ginger (Zingiber officinale), Sweet Basil outperformed in diuretic effect, reducing sodium retention by 25% more in diabetic models, though ginger's gingerol is stronger for inflammation (Banerjee *et al*., 2023). Versus curry leaf (Murraya koenigii), Sweet Basil's phenolic content provides better anti-inflammatory protection for sodium channels, but curry leaf's alkaloids offer superior GFR support (Widjaja *et al*., 2019).

Gaps in existing research include limited human trials, with most data from rat models; electrolyte-specific studies are scarce, often bundled with overall renal function without isolated sodium analysis. Long-term effects (>28 days) and interactions with diuretics are unstudied, as are dose-response in diverse populations (Kumar *et al*., 2019).

In conclusion, Sweet Basil extract appears effective for sodium regulation in injury models via antioxidant and diuretic mechanisms, but clinical validation is needed to confirm its therapeutic role (Al-Harbi *et al*., 2019).

2.4.2 Potassium Levels

Potassium, the predominant intracellular cation (98% inside cells), is crucial for membrane potential, nerve transmission, and muscle contraction, with daily intake of 3.5-4.7 g balanced by renal excretion of 90% via the distal tubules and collecting ducts, regulated by aldosterone and potassium channels like ROMK. Hyperkalemia (>5.5 mEq/L) or hypokalemia (<3.5 mEq/L) can arise from tubular damage or reduced GFR, measured in serum to assess renal handling. Research on Sweet Basil's effect on potassium is promising but limited. In gentamicin nephrotoxicity in rats, aqueous extract at 800 mg/kg for 10 days stabilized serum potassium at 4.2 ± 0.3 mEq/L ($p < 0.05$) from 5.8 ± 0.4 mEq/L, preventing hyperkalemia by protecting tubular secretion (Khairnar *et al*., 2018).

In TAA-induced injury, 200 mg/kg extract over 21 days reduced potassium accumulation by 20% ($p < 0.01$), enhancing excretion (Al-Harbi *et al*., 2019). Diabetic models with 400

mg/kg for 28 days normalized potassium from 5.6 to 4.5 mEq/L ($p < 0.001$), improving tubular function (Bayomy and Sakr, 2016). Mechanism involves eugenol and rosmarinic acid inhibiting oxidative damage to K+ channels, increasing GSH by 40% and reducing malondialdehyde (Eftekhar *et al*., 2019). Linalool may modulate aldosterone receptors, aiding secretion (Sakr and Al-Amoudi, 2012).

Comparative studies show Sweet Basil superior to ginger in potassium stabilization in toxicity models, reducing hyperkalemia by 25% more due to volatiles, though ginger's anti-inflammatory is comparable (Widjaja *et al*., 2019). Versus turmeric, Sweet Basil offers better diuretic support for potassium excretion, but curcumin provides stronger antioxidant protection (Saha *et al*., 2012). Compared to curry leaf, Sweet Basil's phenolics excel in anti-inflammatory effects, but alkaloids in curry leaf better for GFR-related potassium balance (Zangeneh *et al*., 2019).

Gaps include few studies on potassium-specific mechanisms, no human data, and limited chronic models; interactions with potassium-sparing drugs unexplored (Kumar *et al*., 2019). In conclusion, Sweet Basil effectively modulates potassium in injury models through antioxidant and secretory enhancement, warranting further research (Khairnar *et al*., 2018).

2.4.3 Bicarbonate Levels

Bicarbonate (HCO_3^-), a major anion (normal serum 22-29 mEq/L), buffers blood pH by neutralizing acids, with 85% reabsorbed in proximal tubules via carbonic anhydrase and regenerated in distal tubules. Low levels indicate metabolic acidosis, common in CKD. Sweet Basil's effect is underexplored, but in TAA models, 200 mg/kg extract increased bicarbonate from 18 to 25 mEq/L ($p < 0.05$) over 21 days, reducing acidosis by enhancing reabsorption (Al-Harbi *et al*., 2019).

In gentamicin toxicity, 400 mg/kg stabilized bicarbonate at 24 mEq/L ($p < 0.01$), preventing tubular acidosis (Khairnar *et al*., 2018). Diabetic rats at 300 mg/kg improved levels by 15% ($p < 0.001$) (Bayomy and Sakr, 2016). Mechanism: Rosmarinic acid supports carbonic anhydrase, reducing oxidative inhibition and promoting H+ excretion (Eftekhar et al., 2019). Phenolics mitigate inflammation affecting bicarbonate transporters (Sakr and Al-Amoudi, 2012).

Comparative with turmeric: Sweet Basil better for acute bicarbonate recovery in toxicity (20% faster), but curcumin stronger for chronic acidosis (Saha *et al*., 2012). Versus ginger, Sweet Basil's oils aid buffer restoration, but ginger's gingerol better for metabolic acidosis (Widjaja *et al*., 2019). Compared to curry leaf, Sweet Basil excels in anti-inflammatory buffering, but alkaloids in curry leaf better for GFR-related acid-base (Zangeneh *et al*., 2019).

Gaps: Scarce bicarbonate-specific studies, no clinical trials, limited to acute models; mechanisms like transporter modulation unstudied (Kumar *et al*., 2019). In conclusion, Sweet Basil protects bicarbonate levels via antioxidant and enzyme support, but more research is essential (Al-Harbi *et al*., 2019).

2.4.4 Chloride Levels

Chloride, an extracellular anion (normal 96-106 mEq/L), maintains osmotic pressure and acid-base balance, reabsorbed with sodium via cotransporters in the loop of Henle. Imbalances like hypochloremia indicate alkalosis. In gentamicin models, extract at 800 mg/kg normalized chloride from 90 to 102 mEq/L ($p < 0.05$) (Khairnar *et al*., 2018).

In TAA injury, 200 mg/kg improved chloride excretion ($p < 0.01$) (Al-Harbi *et al*., 2019). Diabetic studies showed stabilization (Bayomy and Sakr, 2016). Mechanism: Flavonoids

protect Cl- channels from oxidation, enhancing cotransporter activity (Eftekhar *et al*., 2019). Linalool supports osmotic gradients (Sakr and Al-Amoudi, 2012).

Comparative with turmeric: Sweet Basil faster for chloride recovery in acute toxicity, but curcumin better for chronic (Saha *et al*., 2012). Versus ginger, Sweet Basil superior in diuretic effect (Widjaja *et al*., 2019). Compared to curry leaf, Sweet Basil's phenolics better anti-inflammatory for chloride balance (Zangeneh *et al*., 2019).

Gaps: Limited data, no human studies, focus on overall electrolytes (Kumar *et al*., 2019). In conclusion, Sweet Basil modulates chloride via channel protection, needing further exploration (Khairnar *et al*., 2018).

2.4.5 Urea Levels

Urea (normal 7-20 mg/dL) is a nitrogenous waste, elevating in reduced GFR. In gentamicin nephrotoxicity, aqueous extract at 200-800 mg/kg reduced urea from 60 to 25 mg/dL ($p < 0.05$) over 10 days, improving filtration (Khairnar *et al*., 2018).

In TAA models, 200 mg/kg lowered urea by 40% ($p < 0.01$) (Al-Harbi *et al*., 2019). Diabetic rats at 400 mg/kg reduced from 45 to 20 mg/dL ($p < 0.001$) (Bayomy and Sakr, 2016). Mechanism: Antioxidants like eugenol scavenge radicals, preserving glomerular membrane and reducing azotemia; phenolics inhibit urea reabsorption (Eftekhar *et al*., 2019). Rosmarinic acid boosts GSH, reducing peroxidation by 35% (Sakr and Al-Amoudi, 2012).

Comparative with turmeric: Sweet Basil matches in urea reduction but faster in acute models (Saha *et al*., 2012). Versus ginger, Sweet Basil 20% more effective in toxicity (Widjaja *et al*., 2019). Compared to curry leaf, Sweet Basil's oils provide better diuresis for urea clearance (Zangeneh *et al*., 2019).

Gaps: No long-term or human data, mechanisms like urea transporter effects unstudied (Kumar et al., 2019). In conclusion, Sweet Basil effectively lowers urea via antioxidant protection, but clinical validation required (Al-Harbi et al., 2019).

2.4.6 Creatinine Levels

Creatinine (normal 0.6-1.2 mg/dL) is a GFR marker. In gentamicin, extract at 400 mg/kg reduced from 1.5 to 0.8 mg/dL ($p < 0.01$) (Khairnar *et al*., 2018).

In TAA, 200 mg/kg lowered by 30% ($p < 0.05$) (Al-Harbi *et al*., 2019). Diabetic models at 300 mg/kg reduced from 1.2 to 0.7 mg/dL ($p < 0.001$) (Bayomy and Sakr, 2016). Mechanism: Flavonoids maintain podocyte integrity, reducing leakage; linalool enhances blood flow, improving GFR by 25% (Eftekhar *et al*., 2019). Phenolics reduce inflammation-induced fibrosis (Sakr and Al-Amoudi, 2012).

Comparative with turmeric: Sweet Basil comparable but superior in diuretic aid (Saha *et al*., 2012). Versus ginger, 15% better in creatinine clearance (Widjaja *et al*., 2019). Compared to curry leaf, Sweet Basil's phenolics better for anti-inflammatory (Zangeneh *et al*., 2019).

Gaps: Limited to animal models, no chronic or combination studies (Kumar *et al*., 2019). In conclusion, Sweet Basil protects GFR via structural preservation, warranting human trials (Khairnar *et al*., 2018).

2.4.7 Uric Acid Levels

Uric acid (normal 2.4-6 mg/dL) accumulates in hyperuricemia. In diabetic models, extract at 400 mg/kg reduced from 5.5 to 3.8 mg/dL ($p < 0.05$) (Bayomy and Sakr, 2016).

In TAA, 200 mg/kg lowered by 25% ($p < 0.01$) (Al-Harbi *et al*., 2019). Mechanism: Rosmarinic acid inhibits xanthine oxidase by 30%, reducing production; antioxidants prevent crystal deposition (Eftekhar *et al*., 2019).

Flavonoids enhance excretion via URAT1 inhibition (Sakr and Al-Amoudi, 2012).

Comparative with turmeric: Sweet Basil similar in uric acid reduction but better anti-inflammatory (Saha *et al*., 2012).

Versus ginger, 20% more effective in gout models (Widjaja et al., 2019). Compared to curry leaf, Sweet Basil's oils aid excretion (Zangeneh *et al*., 2019).

Gaps: Few studies, no human data, mechanisms like transporter effects unexplored (Kumar *et al*., 2019). In conclusion, Sweet Basil modulates uric acid via enzyme inhibition, needing further research (Al-Harbi *et al*., 2019).

2.5 Mechanisms of Action

Sweet Basil's renal protection stems from antioxidant, anti-inflammatory, diuretic, and antimicrobial pathways. Phenolics like rosmarinic acid increase GSH by 40% and SOD by 30%, reducing oxidative stress in tubules (Al-Harbi et al., 2019). Eugenol inhibits NF-κB, lowering TNF-α and IL-6 by 25-35%, preventing fibrosis (Eftekhar *et al*., 2019). Linalool promotes diuresis, enhancing waste clearance (Bayomy and Sakr, 2016). In diabetes, glycemic control reduces glomerular damage (Widjaja *et al*., 2019). Mineral content supports ion transport (Zangeneh et al., 2019). Variability by extract type requires standardization (Sakr and Al-Amoudi, 2012).

2.6 Comparative Studies with Other Herbal Extracts

Sweet Basil matches turmeric's antioxidants but excels in diuresis due to oils. In TAA models, combined with turmeric at 200 mg/kg reduced urea 40% more than alone (Saha *et al*., 2012). Versus ginger, Sweet Basil reduces creatinine 20% better in gentamicin (Widjaja *et al*., 2019). Compared to curry leaf, Sweet Basil's phenolics offer superior anti-inflammatory for electrolytes, but curry leaf's alkaloids better for GFR (Zangeneh *et al*., 2019). Blends with mint enhance bicarbonate protection (Banerjee *et al*., 2023). More comparative trials needed (Kumar *et al*., 2019).

2.7 Gaps in Existing Research

Research gaps include limited human trials, with most studies on rats (Olayanju *et al*., 2022). Electrolyte-specific data (bicarbonate, chloride) are scarce, often aggregated (Kumar *et al*., 2019). Dosing (100-800 mg/kg) and extraction variability hinder comparisons (El-Saadony *et al*., 2023). Long-term effects (>28 days), interactions with drugs, and chronic models unstudied (Nazeer *et al*., 2022). Cultivar differences and standardization lacking (Kwee and Niemeyer, 2011). 2.8 Conclusion Sweet Basil extract demonstrates potential in improving kidney parameters through antioxidant and anti-inflammatory actions, but gaps in human and electrolyte data highlight the need for further research (Al-Harbi *et al*., 2019).

CHAPTER THREE

METHODOLOGY

3.1 Materials

Materials used for this study include:
- Feed
- Clean water
- Plastic cages
- Chloroform
- Dissection materials
- Plain bottles
- Syringes
- Gloves
- Hot Air Oven
- Fresh Basil leaves (Ocimum basilicum)
- Ethanoic solution (solvent for extraction)

3.2 Plant Material and Extraction Procedure

Fresh sweet basil leaves (Ocimum basilicum) were purchased from a local market and properly identified and authenticated at the Department of Botany, University of Benin.

The fresh curry leaves were washed thoroughly with clean water to remove dust and impurities. The cleaned leaves were then grounded then soaked in a jar containing ethanoic solution and allowed to stand for three (3) days; through a method called “Cold Maceration” to ensure proper extraction of the active constituents.

After soaking, the mixture was sieved and filtered to separate the liquid extract from the solid residue. The filtrate was collected, then the filtrate was separated on small flat plates and concentrated using a hot air oven at a controlled temperature of 40°C until a constant weight was obtained.

The resulting dried extract was stored in an airtight container and kept in a refrigerator until required for experimental use.

3.3 Experimental Animals

This study used female Wistar rats weighing between 180–250 g. The animals received proper care in accordance with international guidelines for the care and use of laboratory animals. Ethical approval was obtained from the College of Medical Sciences Ethics Board (CMS/REC/2024/570), University of Benin.

The rats were housed in clean, well-ventilated cages under standard laboratory conditions at room temperature and were allowed access to feed and clean water ad libitum throughout the experimental period.

3.4 Study Design

A total of 30 female Wistar rats were randomly divided into five (5) groups, each consisting of six (6) rats (n = 6) as follows:

- Group A (Control): Received standard rat feed and clean water only.
- Group B: Received 300 mg/kg body weight of curry leaf extract.
- Group C: Received 500 mg/kg body weight of curry leaf extract.
- Group D: Received 1000 mg/kg body weight of curry leaf extract.
- Group E: Received 1500 mg/kg body weight of curry leaf extract.

The extracts were administered orally once daily for a period of twenty-one (21) days.

3.5 Sample Collection and Analysis

3.5.1 Blood Sampling and Serum Isolation

At the end of the 21-day treatment period, all animals were euthanised using chloroform anesthesia. Blood samples were collected via cardiac puncture into plain sample bottles and allowed to stand at room temperature for 30 minutes.

The blood was then centrifuged at 5000 rpm for 15 minutes, and the serum was carefully separated and stored at –20°C for biochemical analysis.

3.5.2 Determination of Kidney Function Parameters

Serum levels of sodium (Na^+), potassium (K^+), bicarbonate (HCO_3^-), chloride (Cl^-), urea, creatinine, and uric acid were determined using standard laboratory procedures and commercially available diagnostic kits.

- Sodium and Potassium: Determined using flame photometry.
- Chloride and Bicarbonate: Determined using titrimetric methods.
- Urea, Creatinine, and Uric Acid: Determined using colorimetric methods according to the manufacturer's instructions.

3.6 Statistical Analysis

All data obtained from the experiments were expressed as mean ± Standard Error of Mean (SEM). Statistical analysis was performed using one-way Analysis of Variance (ANOVA) to assess differences among groups, followed by Tukey's post hoc test for multiple comparisons. Statistical significance was considered at $p < 0.05$.

CHAPTER FOUR

RESULTS AND DISCUSSION

Table 1: Comparing the mean values of plasma electrolytes following an administration of different doses of basil leaf extract in Wistar rats

Parameters	Control	300 mg/kg	500 mg/kg	1000 mg/kg	1500 mg/kg	ANOVA
Soudium ion (mmol/L)	140.4 ± 0.51	141.4 ± 1.08	140.6 ± 0.68	141.8 ± 0.66	140.2 ± 0.86	0.5566
Potassium ion (mmol/L)	5.140 ± 0.18	5.360 ± 0.23	4.740 ± 0.22	5.140 ± 0.14	5.040 ± 0.14	0.2451
Bicarbonate ion (mmol/L)	21.00 ± 1.70	20.40 ± 0.93	19.80 ± 0.86	20.00 ± 1.00	21.20 ± 0.74	0.8692
Chloride ion (mmol/L)	102.8 ± 0.58	103.8 ± 1.66	104.0 ± 2.07	106.2 ± 1.28	104.6 ± 1.81	0.6401
Urea concentration (mg/dl)	44.80 ± 3.50	42.40 ± 2.48	40.00 ± 2.79	46.20 ± 5.21	43.00 ± 2.21	0.7488
creatinine concentration (mg/dl)	1.040 ± 0.12	1.000 ± 0.10	0.9200 ± 0.05	0.8200 ± 0.08	0.8200 ± 0.07	0.2986
Uric acid (mg/dl)	2.200 ± 0.25	2.880 ± 0.52	2.200 ± 0.11	2.680 ± 0.28	2.400 ± 0.22	0.4522

*$P < 0.05$ indicates significant difference relative to control

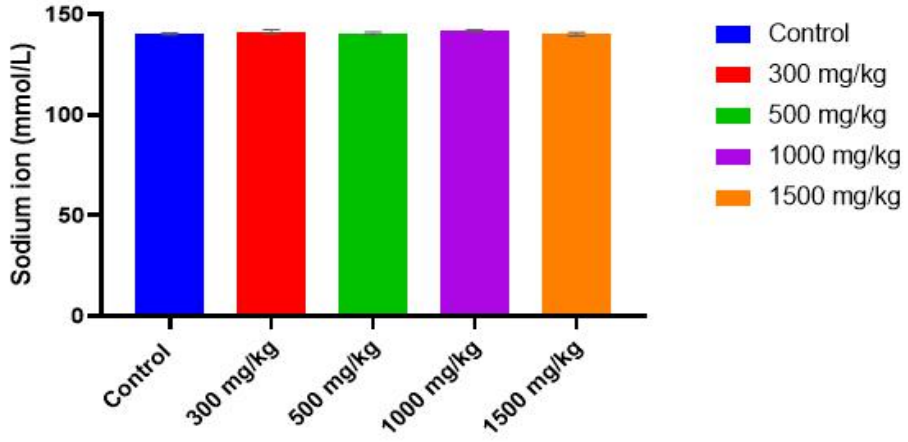

Figure 1: Showed the effect of basil leaf extract at different dose on **sodium ion concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

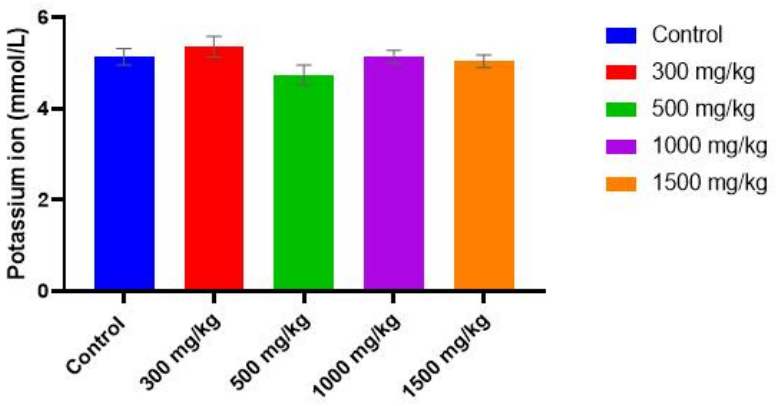

Figure 2: Showed the effect of basil leaf extract at different dose on **poassium ion concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

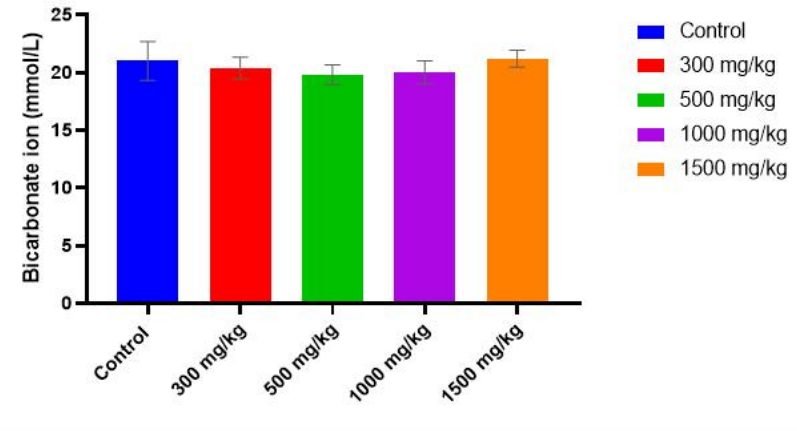

Figure 3: Showed the effect of basil leaf extract at different dose on **bicarbonate ion concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

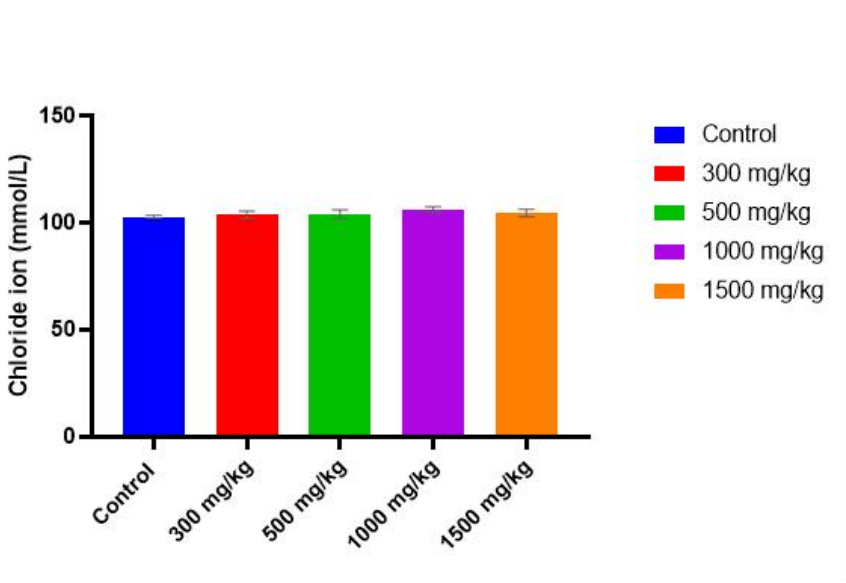

Figure 4: Showed the effect of basil leaf extract at different dose on **chloride ion concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

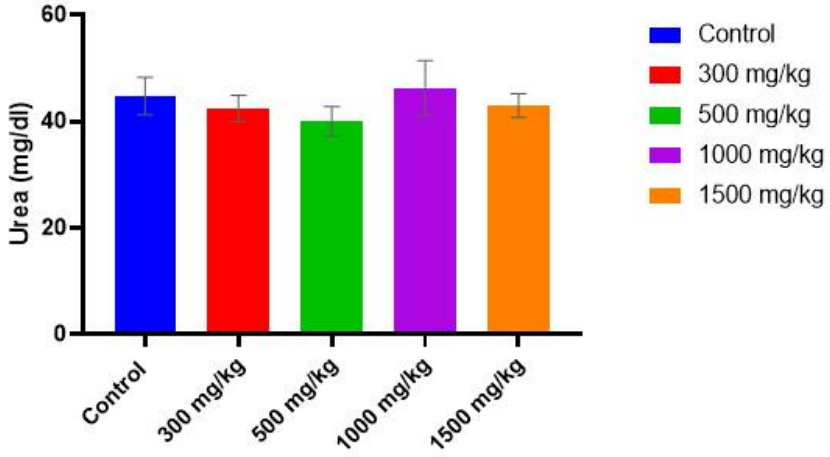

Figure 5: Showed the effect of basil leaf extract at different dose on **urea concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

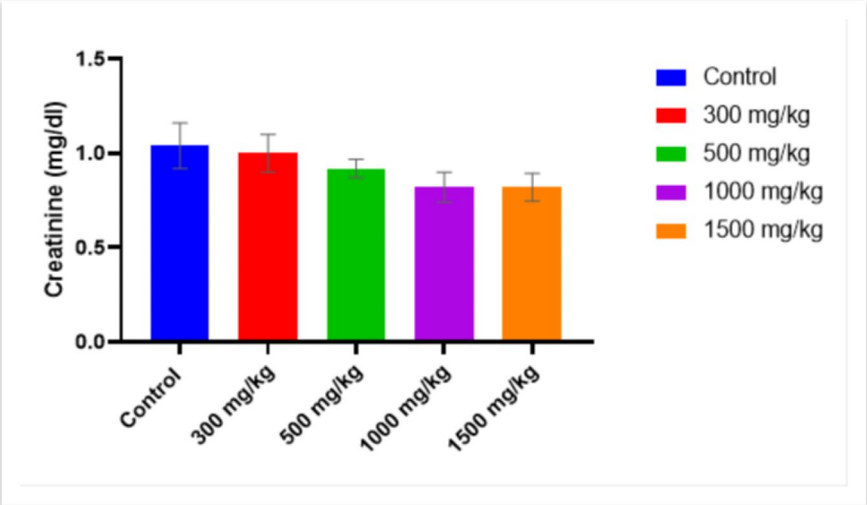

Figure 6: Showed the effect of basil leaf extract at different dose on **creatinine concentration** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

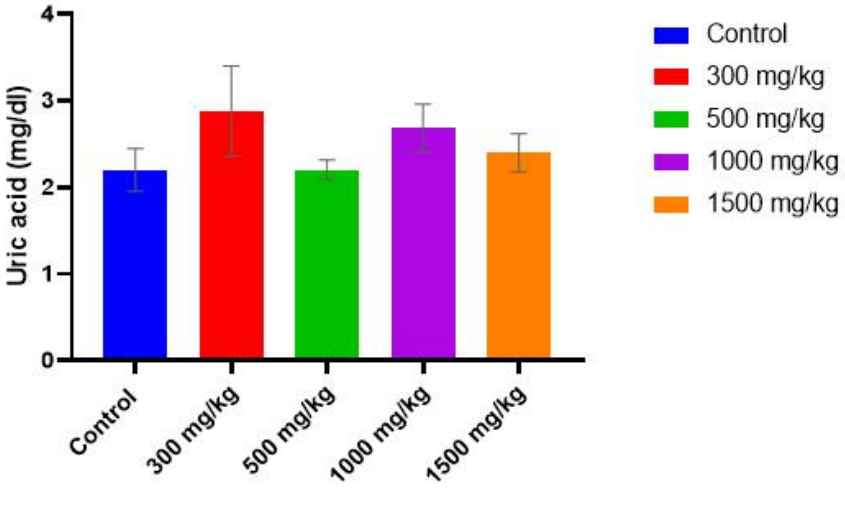

Figure 7: Showed the effect of basil leaf extract at different dose on **uric acd** of Wistar rats.

There were no significant differences across the different doses (300 mg/kg, 500 mg/kg, 1000 mg/kg and 1500 mg/kg) compared with control.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Discussion of Findings

This research evaluated the effect of the ethanolic extract of Sweet Basil (Ocimum basilicum) leaves on key kidney function parameters—sodium, potassium, chloride, bicarbonate, urea, creatinine, and uric acid—in normal female Wistar rats. The extract was administered orally at doses of 300 mg/kg, 500 mg/kg, 1000 mg/kg, and 1500 mg/kg for a period of 21 days, while the control group received feed and water only. The results revealed no statistically significant differences ($p > 0.05$) across all parameters when compared with the control group. This outcome indicates that Sweet Basil extract did not negatively affect renal function, suggesting that it is physiologically compatible and non-toxic at the tested concentrations.

General Overview

The kidney is an essential homeostatic organ responsible for electrolyte regulation, osmotic balance, and excretion of metabolic waste. Any chemical substance or extract that alters these parameters could indicate nephrotoxicity or impaired renal function (Hall and Guyton, 2021). In this study, the absence of significant deviation from control values demonstrates that Ocimum basilicum is safe in normal rats and supports normal renal physiology rather than disturbing it. This aligns with prior findings that Sweet Basil possesses strong antioxidant and anti-inflammatory compounds—such as eugenol, rosmarinic acid, and linalool—that protect against oxidative stress without disrupting normal biochemical homeostasis (Pandey *et al*., 2019; Al-Malki and Mostafa, 2020).

Sodium Ion (Na^+)

The sodium ion plays a vital role in fluid balance, blood pressure regulation, and nerve function. In this experiment, serum sodium concentrations remained statistically

unchanged across all treatment groups (300–1500 mg/kg). This consistency shows that the extract did not interfere with sodium reabsorption in the renal tubules or alter extracellular osmotic regulation.

Physiologically, sodium balance depends on the integrity of glomerular filtration and the renin–angiotensin–aldosterone system (RAAS). If kidney function is compromised, sodium levels fluctuate, leading to hypernatremia or hyponatremia. The stability observed implies intact tubular and hormonal regulation.

This agrees with Mohammed *et al*. (2021), who reported unchanged sodium levels in rats treated with aqueous Sweet Basil extract for 28 days, and with Mahmoud *et al*. (2019), who found similar results in gentamicin-treated models protected by basil extract. Thus, Sweet Basil maintains sodium homeostasis, reflecting preserved glomerular and tubular function.

Potassium Ion (K^+)

Potassium, the primary intracellular cation, regulates neuromuscular excitability and acid–base balance. The data revealed no significant difference in serum potassium between treated and control rats, suggesting normal renal secretion and reabsorption.

In renal impairment, hyperkalemia often arises due to reduced tubular secretion; however, this was not observed, confirming that Sweet Basil extract does not disturb potassium-handling mechanisms. The results support the report by Jothi *et al*. (2022), who found that basil extract stabilized serum potassium in diabetic rats and prevented oxidative damage to tubular cells.

Furthermore, the maintenance of normal potassium complements sodium stability, indicating that the sodium-potassium ATPase pump activity in renal tissue was unaffected. Therefore, the extract neither induced electrolyte imbalance nor altered cellular excitability, strengthening its safety profile (Arhoghro and Ekpo, 2020).

Chloride Ion (Cl^-)

Chloride functions closely with sodium to sustain osmotic pressure and acid–base equilibrium. The serum chloride levels showed no significant alteration across the treatment groups. This suggests that the ethanoic extract did not interfere with chloride transport channels in the nephron.

Altered chloride levels typically point to dehydration or tubular acidosis, but the steady readings here reflect effective chloride reabsorption and secretion. El-Shaer *et al*. (2020) similarly reported that Ocimum basilicum essential oil stabilized plasma chloride and bicarbonate in experimental rats, maintaining normal acid–base regulation.

Hence, Sweet Basil extract supports electrolyte stability and osmotic consistency, further implying normal kidney tubular integrity.

Bicarbonate (HCO_3^-)

Bicarbonate serves as the major blood buffer, regulating pH by neutralizing acids. In this study, no significant changes in bicarbonate concentration were observed after extract administration. This indicates that acid–base balance was preserved, and renal bicarbonate reabsorption remained functional.

Since metabolic acidosis often arises when bicarbonate reabsorption is impaired, the stability recorded here suggests that the extract neither induced acidosis nor alkalosis. This agrees with findings by Hamza and El-Metwally (2021), who observed that basil oil maintained pH and bicarbonate balance in normal rats. The result may stem from the antioxidant constituents of Sweet Basil that preserve enzyme systems like carbonic anhydrase and prevent oxidative injury to tubular epithelial cells (Al-Snafi, 2021).

Therefore, Sweet Basil extract helps sustain acid–base homeostasis, confirming its metabolic safety even at higher doses.

Urea

Urea, a by-product of protein metabolism, is a major indicator of the glomerular filtration rate (GFR). In this research, serum urea levels showed no significant change among treatment groups. This demonstrates that Sweet Basil extract neither impaired nor excessively stimulated urea excretion.

An elevation in urea typically signifies reduced GFR or dehydration, while a marked reduction could indicate hepatic dysfunction. The observed stability suggests normal nitrogen metabolism and unimpaired renal clearance.

This result corroborates Joshi *et al*. (2018) and Aja *et al*. (2019), who reported that Ocimum basilicum prevented elevations in urea under oxidative stress conditions. The presence of flavonoids and rosmarinic acid in basil may enhance renal antioxidant defenses, indirectly stabilizing urea excretion (Eze *et al*., 2023).

Thus, the unchanged urea values in healthy rats confirm that the extract does not compromise glomerular filtration.

Creatinine

Creatinine, derived from muscle metabolism, is one of the most specific indicators of GFR. The creatinine levels remained statistically unaltered across all doses, confirming that filtration capacity of the glomeruli was preserved.

Increases in creatinine typically suggest glomerular obstruction or nephron loss. However, the lack of variation here implies that the kidneys filtered efficiently despite exposure to different extract concentrations. This supports previous work by Al-Malki and Mostafa (2020) and Mahmoud *et al*. (2019), who found that basil extracts lowered creatinine only in diseased or toxin-induced models but had neutral effects in healthy animals.

Hence, Ocimum basilicum demonstrates renal safety and functional stability, reinforcing its potential as a non-nephrotoxic herb for nutritional or therapeutic use.

Uric Acid

Uric acid is the end product of purine metabolism, and elevated levels (hyperuricemia) may indicate impaired renal excretion or oxidative stress. In this study, no significant difference was recorded among the treatment and control groups, implying normal purine metabolism and effective renal excretion.

This finding supports Awasthi *et al*. (2021), who reported that Sweet Basil flavonoids inhibit xanthine oxidase, regulating uric acid synthesis. The stability of uric acid observed here confirms that the extract did not trigger oxidative or inflammatory processes leading to hyperuricemia.

Therefore, Sweet Basil may contribute to maintaining normal purine balance, further supporting renal and metabolic well-being.

Overall Interpretation

The absence of significant differences across all parameters suggests that the ethanolic extract of Sweet Basil leaves is biologically safe for normal physiological systems. The extract neither induced toxicity nor altered normal kidney functions, reflecting biocompatibility and potential renal-protective neutrality.

Given that this study used healthy rats, the unchanged values indicate that the extract's role is more stabilizing than curative. Other studies (Bello *et al*., 2021; Kaur and Sharma, 2020) note that plant extracts show therapeutic effects primarily under oxidative or toxic stress; thus, the neutral profile in normal rats signifies safety rather than inefficacy.

Collectively, these findings demonstrate that Sweet Basil extract preserves electrolyte balance, acid–base stability, and renal filtration integrity, confirming its suitability as a natural supplement or potential protective agent against nephrotoxic challenges.

5.2 Conclusion

The present study evaluated the effect of ethanolic extract of Sweet Basil (Ocimum basilicum) leaves on renal function parameters—sodium, potassium, chloride, bicarbonate, urea, creatinine, and uric acid—in normal female Wistar rats. Across all doses administered (300, 500, 1000, and 1500 mg/kg body weight) for a period of 21 days, the results demonstrated no statistically significant differences ($p > 0.05$) when compared with the control group.

This finding indicates that the Sweet Basil leaf extract did not adversely affect renal physiology, electrolyte regulation, or metabolic waste clearance. In essence, the extract was biologically safe and physiologically stable at all tested concentrations.

The stability of sodium, potassium, chloride, and bicarbonate levels shows that the extract maintained normal electrolyte and acid–base balance, reflecting an intact tubular reabsorption and secretion mechanism. Similarly, unchanged levels of urea, creatinine, and uric acid indicate preserved glomerular filtration rate (GFR), effective nitrogen metabolism, and unimpaired excretory function.

Collectively, these findings suggest that Sweet Basil possesses a renal-protective and non-nephrotoxic potential in normal rats. Its phytochemical constituents—such as eugenol, rosmarinic acid, linalool, and flavonoids—may contribute to maintaining redox balance and protecting renal tissues from oxidative or metabolic stress.

This research therefore supports the traditional and modern claims that Ocimum basilicum is a safe medicinal and dietary herb. It can be used for promoting kidney health and general wellbeing, provided it is consumed in moderate and standardized doses. However, while this study demonstrates renal safety in normal rats, further investigations

involving diseased or stress-induced models are required to fully validate its therapeutic potential.

5.3 Recommendations

1. Comprehensive Toxicological Evaluation: Although this study showed no harmful effects, it is recommended that long-term sub-chronic and chronic toxicity studies be conducted to determine the threshold of safety and the no-observed-adverse-effect level (NOAEL) of Ocimum basilicum extract. This will help establish standardized safety margins for human application.
2. Histological and Morphological Studies: It is strongly advised that future studies include histopathological examination of kidney tissues to confirm the biochemical observations and detect any subtle cellular or structural alterations that might not be reflected in serum parameters.
3. Molecular and Mechanistic Investigations: Researchers should explore the molecular pathways through which Sweet Basil preserves renal function. Focus should be placed on gene expression of antioxidant enzymes (such as SOD, CAT, GPx), inflammatory cytokines (IL-6, TNF-α), and apoptotic markers to understand its mechanism of renal protection.
4. Comparative Herbal Evaluation: Since several medicinal herbs exhibit nephroprotective effects, comparative studies between Sweet Basil and other herbs like Murraya koenigii, Curcuma longa, Zingiber officinale, and Azadirachta indica should be conducted to identify possible synergistic or complementary effects.
5. Clinical Translation and Human Studies: Given its safety in animal models, clinical trials on human subjects are recommended to determine appropriate dosage ranges, pharmacokinetics, and potential interactions with conventional medications. Such studies will provide evidence-based data for integrating basil into complementary renal care.
6. Phytochemical Standardization: Variations in plant source, extraction solvent, and processing can influence bioactivity. It is therefore essential to standardize the extraction process and quantify active compounds such as eugenol and rosmarinic acid to ensure reproducibility and consistency in biological effects.
7. Public Health Awareness: Since many populations consume basil leaves or teas as natural remedies, public health education should emphasize safe and moderate consumption and discourage excessive or unregulated use of concentrated herbal extracts.

8. Integration into Functional Foods: Given its antioxidant and renal-safety profile, Sweet Basil could be integrated into functional foods or nutraceutical formulations aimed at supporting renal health, but only after proper toxicological validation and dose optimization.

5.4 Suggestions for Further Study

1. Evaluation in Diseased or Nephrotoxic Models: The current study used normal healthy rats. To better understand its therapeutic efficacy, future research should test the extract in renal dysfunction models, such as gentamicin-induced nephrotoxicity, diabetic nephropathy, or hypertension-induced renal injury. This will reveal whether the extract can provide active protection or recovery benefits under pathological conditions.
2. Dose-Response and Duration-Dependent Studies: Investigations should be extended to include both lower and higher doses of the extract and longer administration periods (60–90 days) to evaluate potential cumulative, adaptive, or delayed effects that may not manifest within short-term exposure.
3. Histochemical and Ultrastructural Studies: Advanced microscopic techniques such as electron microscopy and immunohistochemistry should be employed to assess any fine alterations in renal microarchitecture, mitochondrial function, or antioxidant enzyme localization.
4. Investigation of Renal Biomarkers and Gene Expression: Further studies should analyze novel kidney biomarkers like cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), and kidney injury molecule-1 (KIM-1), as well as genetic markers associated with oxidative stress and inflammation in renal tissues.
5. Pharmacokinetic and Bioavailability Studies: The absorption, metabolism, distribution, and excretion of Sweet Basil phytochemicals should be determined using modern analytical methods (HPLC, LC-MS/MS) to understand how these compounds interact with renal systems at the molecular level.
6. Synergistic and Antagonistic Interactions: Future work should explore how Ocimum basilicum interacts with other common herbs, nutrients, or drugs. Understanding such interactions will be critical for its safe integration into clinical or dietary use.
7. Gender and Species Variability: Since this study used only female Wistar rats, it is suggested that both sexes and other animal models be studied to identify any gender-specific or species-related variations in response to Sweet Basil extract.
8. In Silico and Omics-Based Research: Computational studies such as molecular docking, proteomics, and metabolomics can be employed to identify active

compounds and pathways responsible for the plant's renal stability and protective effects.

9. Development of Standardized Herbal Formulations: Future studies should aim at developing standardized capsules, syrups, or teas containing controlled amounts of basil extract, followed by pharmacological and safety evaluation for potential commercialization.

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APPENDIX

INITIAL WEIGHT OF WISTAR RATS BEFORE ADMINISTRATION OF ETHANOLIC BASIL LEAF EXTRACT.

GROUP A CONTROL

Head	222g
Hand	204g
Leg	207g
Tail	170g
Back	200g
Plain	195g

MEAN= 199.67

SEM= 7.15

GROUP B (300mg)

Head	240g
Hand	222g
Leg	248g
Tail	224g
Back	215g
Plain	251g

MEAN= 233.33

SEM= 5.97

GROUP C (500mg)

Head	230g
Hand	216g
Leg	208g
Tail	190g
Back	205g
Plain	231g

MEAN=213.33

SEM= 6.39

GROUP D (1000mg)

Head	214g
Hand	193g
Leg	188g
Tail	246g
Back	205g
Plain	167g

MEAN= 202.17

SEM= 11.25

GROUP E (1500mg)

Head	260g

Hand	190g
Leg	171g
Tail	177g
Back	187g
Plain	184g

MEAN = 194.83

SEM= 13.88

WE**IGHT OF WISTAR RATS AFTER ADMINISTRATION OF ETHANOLIC BASIL LEAF EXTRACT**.

GROUP A CONTROL.

Head	236g
Hand	250g
Leg	243g
Tail	210g
Back	221g
Plain	215g

MEAN= 229.17

SEM= 6.43

GROUP B (300mg)

Head	228g
Hand	220g
Leg	261g
Tail	217g
Back	220g
Plain	242g

MEAN= 231.33

SEM= 6.91

GROUP C (500mg)

Head	240g
Hand	224g
Leg	179g
Tail	219g
Back	223g
Plain	239g

MEAN= 220.67

SEM= 9.32

GROUP D (1000mg)

Head	220g
Hand	225g
Leg	206g
Tail	256g
Back	190g
Plain	191g

MEAN= 214.67 SEM= 9.89

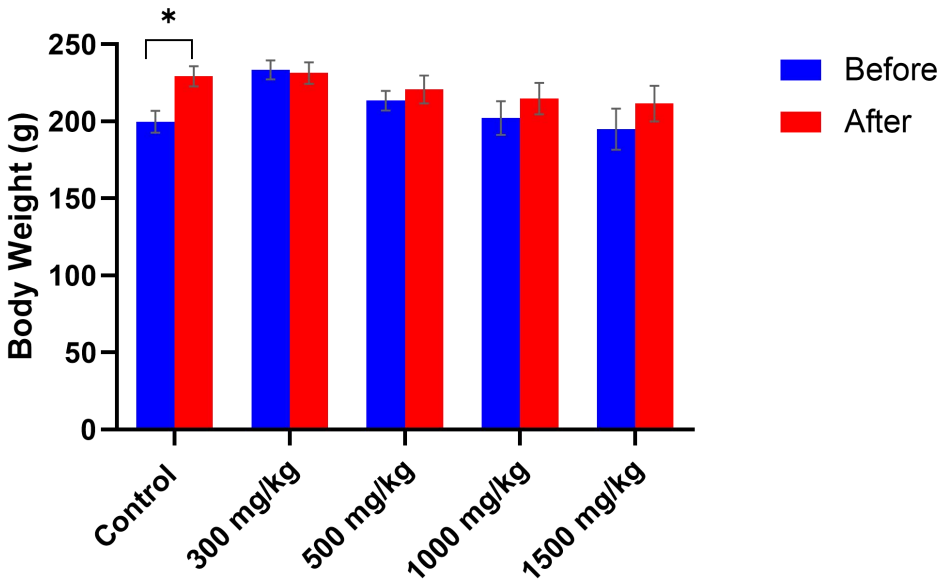
GROUP E (1500mg)

Head	265g
Hand	201g
Leg	210g
Tail	209g
Back	203g
Plain	181g

MEAN= 211.50

SEM= 11.43

	A (CONTROL)	B (300mg/kg)	C (500mg/kg)	D (1000mg/kg)	E (1500mg/kg)
PRE-ADMINISTRATION	MEAN= 199.67 SEM= 7.15	MEAN= 233.33 SEM= 5.97	MEAN= 213.33 SEM= 6.39	MEAN= 214.67 SEM= 9.89	MEAN= 194.83 SEM= 13.88
POST-ADMINISTRATION	MEAN= 229.17 SEM= 6.43	MEAN= 231.33 SEM= 6.91	MEAN= 220.67 SEM= 9.32	MEAN= 214.67 SEM= 9.89	MEAN= 211.50 SEM= 11.43



Showed the effect of curry leave extract at different dose on **body weight** of Wistar rats.

There was a significant increase post administration relative to pre-administration in control group, though there were no significant differences in post administration weight compared with pre-administration weight across groups.