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AMELIORATIVE EFFECT OF PSIDIUM GUAJAVA LINN AGAINST OXIDATIVE STRESS INDUCED BY HIGH DIETARY SALT

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ABSTRACT: *High table salt intake ccontribute major risk factor in multiorgan danages, impairement of endothelial tissues, obesity, stroke cardiovascular mortality. The effect of high salt diet is mediated by significant increase in reactive oxygen species and decrease in antioxidant thereby increase in oxidative cell damages and artherosclerosis. The possibility of countering and ameliorating these effect is the use of guava leaf (Psidium guajava linn). Guava leaf contain high amount of secondary metabolite such as antioxidant, polyphenol, anti-inflammatory compound, which help to mitigate the effect of reactive oxygen species.*

The aim of the study is to evaluate the effects of high salt diet on Wistar rats and to assess the possibility of ameliorating the effect with extract of guava leaf. Thirty (30) male albino Wistar rats weighing between 80-150g were randomly divided into 4 groups of 7 rats per groups. Group 1 was given commercial feed and water only, Group 2 was fed high dietary salt only, Group 3 fed high dietary salt and 100 mg/kg Psidium guajava linn and Group 4 fed high dietary salt and 200 mg/kg body to weight Psidium guajava linn.

The Wistar rats was administered high dietary salt (11 percent) through oral administration for 14 days. Rat serum, renal and hepatic post mitochondrial supernatants were analyzed for the activities of catalase, superoxide dismutase, GPx, MDA, lipid profile and liver function test. Oxidative cell damage, hyperlipidemia, oxidative stress and pathological changes were recorded in rat administered high salt diet. Administration of guava leaf at 200mg/kg attenuated the oxidative stress, and pathological damages in the liver and kidney.

Keywords: Antioxidant, Guava Leaf, High Dietary Salt, Oxidative Stress.

1.0 Introduction

High salt diet has been associated with tissues damages, responsible for cardiovascular diseases, cognitive impairment in humans (Heye *et al.,* 2016), impaired cognition, aggravation of cerebral ischemic injury, and high-stress responsivity (Ge *et al.,* 2017), reduce nitric oxide (NO) production (Kouyoumdzian *et al.,* 2016), suppress the activity of antioxidant enzymes, malfunctioned hippocampus, hypothalamus, and cerebellum of the brain leading to reduction of congnitives function, locomotion, anxiety, damage to the amygdale, Increases hyperactivity, when administered in the pre and post weaning periods (Leal *et al.,* 2022).

Rats on high salt diet has reduction of endothelium-dependent dilation in normotensive rats as a result of oxidative stress generated (Lenda *et al.,* 2000; Guzik *et al.,* 2000) shows that increased dietary salt intake results to an impaired relaxation of blood vessels to endothelium-dependent relaxations caused by vasodilator agents. Altered endothelium-dependent dilation in vessels of animals placed on high-salt diet could occur due to acetylcholine (Ach)–mediated production of nitric oxide by the endothelium is damaged leading to hypertension (Guzik *et al.,* 2000).

The oxidation reactions in the body may produce free radicals which damage the cells by various chain reactions. Free radicals (reactive oxygen species) may damage the cells causing debates, cancer, diabetes, inflammation, viral infection and neurodegenerative disorder, (Naseer *et al.,* 2018). These free radicals can be informing of hydrogen peroxide, superoxide radical, singlet oxygen and possibly hydroxyl radicals (-OH). This oxidative stress can also contribute to various pathologies, cell and tissues damages, cell fibrosis, diabetic nephropathy, trauma and stroke if not timely addressed. The removal of reactive oxygen species is fastened by superoxide dismutase (SOD), which converts O_2 into H_2O_2, which is then oxidized by catalase and glutathione peroxidase. Antioxidant enzymes such as SOD, CAT, and GPx in the human body provide the defense

mechanisms to counteract the effects of free radicals and oxidative stress. Antioxidants terminate the free radicals and stop the chain reactions.

Medicinal plants like guava leaves, rich in antioxidants, mitigate prevelence of degenerative diseases such as arteriosclerosis, inflammation, heart disease, cancer, arthritis and brain dysfunction (Feskanich *et al.*, 2000). Previous researches indicated that guava has a high content of flavanoid, polyphenol, ascorbic acid, protocatechuic acid, quercetin, ferulic acid, quercetin, gallic acid and caffeic acid which make it a good antioxidant (He and Venant, 2004; Naseer *et al.*, 2018).

The study aims to investigate the potential effects of Psidium guajava leaf in ameliorating high salt diet induced oxidative stress extract in albino rat subjected to a high salt diet.

2.1 MATERIALS AND METHODOLOGY

All reagents used are of analytical grade supplied by Sigma Aldrich Company, Louis USA and from RANDOX Laboratory Limited. A brand of table salt manufactured by Dangote company & Co, Nigeria was available commercially at a Eyiowuawi supermarket Ede.

2.2 Extraction of psidium guajava leaf

The Selected fresh whole guava leaves were weighed, air dry for 3 weeks and was grinded into fine powder. Cold extraction method was used using 98 % absolute ethanol and soaked for 72 hours at room temperature. It was filtered and concentrated to fine powdered using rotary evaporator.

High salt diet preparation: 11 percent of high salt diet was produced using commercially sold animal feed (grower) i.e. 11g of salt and 89g of rats feed was mixed together, distilled water was sparkle on it for through mixing. The feed was given to each rats based on each body weight (oral administration)

Experimental design

Thirty (30) healthy male Wistar rats weighing approximately 80-150g were gotten from animal house of Obafemi Awolowo University, Ife, Osun state, Nigeria. They were quarantined for 7 days and divided into 4 groups each containing 7 rats. They were kept in a wire mesh cage. They were fed with rat pellet and supplied with water. All rats received human care according to the criteria outlined in the care for the care and use of laboratory animal prepared by the National Academic of Science and published by the National Institute of Health (Garner *et al.*, 2011). The test animals were fed daily to 11 percent high salt diet for 14 days. Raw guava extract (crude extract; CE) of guava leaf was prepared in two different doses of 100 mg/kg and 200 mg/kg body weight, orally using a metallic feeding canola once daily for 14 days and different concentrations of the extract were administered to the albino rats by using an oral metallic feeding canola (Mahfoud *et al*., 2022).

Dosage of Administration

Group 1: Normal control

Group 2: High salt diet only (positive control)

Group 3: High salt diet and 100 mg/kg crude extract once daily.

Group 4: High salt diet and 200 mg/kg crude extract once daily.

After 14 days of administration the rats were sacrificed, and the kidneys and liver tissues was harvested quickly and washed with ice cold 1.15 % KCl solution. The kidney was homogenized in ice-cold 0.25 M sucrose solution buffered with 40 mMTris.HCl at pH 7.4, using Potter Elvherjem Teflon-lined homogenizer. The homogenate obtained was centrifuged at 8000 g for 10 minutes. The pellet so obtained was taken up in 10ml. of the medium and re-centrifuged. The supernatants were recentrifuged at 12000 g for 10 minutes in order to remove light mitochondria. The final supernatants were combined and used as post-mitochondrial fraction for the biochemical analyses. The blood was obtained by heart punched and collected inside sample, it was centrifuged to obtained the serum. Portion of kidney and liver was reserved for Histopathology examination according to a modified method of Avwioro (2010).

1.2 Biochemical assay

Assay of Aspartate Aminotransferase (AST) activity, Alanine Aminotransferase (ALT) Activity, Total protein (TP), Creatinine kinase (CK), Alkaline phosphatase (ALP), low density lipoprotein cholesterol (LDL-C), high-density lipoprotein-cholesterol (HDL-C), and Very low density lipoprotein (VLDL) was determined using Randox laboratory assay kits UK. Total cholesterol level was determined based on the method of Trinder (1969) using commercially available Randox laboratories, kits UK. Triglycerides level was determined based on the method of Tietz (1990) using commercially available kits (Randox laboratories, UK). Albumin activity was carried out using colorimetry method described by (Reinhold, 1953). Bilirubin activity was determined spectrophotometry using procedure designed by Jendrassik and Grof 1938. Serum urea concentration was determined by colorimetric determination of ammonia using commercially available Urease Berthelot's reagent kit MAK 120 (Sigma-Aldrich). In the post-mitochondrial supernatants, superoxide dismutase (SOD) activity was determined spectrophotometrically (Marklund and Marklund, 1974). Reduced glutathione (GSH) concentration in the sample was determined by the method of Owens and Belcher (1965). Lipid peroxidation was determined by monitoring the degrees of lipid peroxides in the supernatant fraction of plasma using the procedure described by Niehaus and Samuelsson, (1968). The activity of catalase in the liver and kidney were determined by method of Claiborne *et al.* (1984).

Statistical Analysis

The results of the experiments were expressed as Means ± SEM (n = 7). Data analysis was done using ANOVA on Graph pad prism software. Statistical significance was taken at $p<0.05$.

2.0 Results

The result of the effects of the leaf extract of Psidium guajava on Lipid profile of rats fed high dietary salt

2.1 There was significant increase of cholesterol, triglycerides, low density lipoprotein, very low density lipoprotein and castelli risk index in rat given HSD only when compared with HSD + 200 mg/kg CE and control. Significant reduction was observed in HSD + 200 mg/kg CE when compared to control

Table 1: Results of Lipid Profile of Rats Exposed to High Salt Diets

Parameters	CHOL(mg/dl)	TRIG(mg/dl)	HDL(mg/dl)	LDL(mg/dl)	VLDL(mg/dl)	CRI
Control	68.39±2.03^{a}	39.26±1.09^{b}	21.82±0.93^{c}	38.72±1.06^{a}	7.85±0.45^{a}	3.13±0.12^{a}
HSD only	129.70±3.11^{d}	63.37±1.30^{d}	15.65±0.45^{a}	101.38±3.40^{d}	12.67±0.33^{c}	8.29±0.17^{c}
HSD+100mg/kg	93.18+1.27^{c}	55.49±1.17^{c}	19.17±0.88^{b}	62.92+1.42^{c}	11.09+0.64^{b}	4.86+0.14^{b}
HSD+200mg/kg	74.53±2.18^{b}	35.61±1.06^{a}	24.31±0.79^{d}	43.10 ±2.05^{b}	7.12±0.70^{a}	3.07±0.07^{a}

Values are expressed as mean ± standard deviation (n=5). Values with the different superscript(s) in a column are significantly different ($P<0.05$).

Key: CHOL = Cholesterol, TRIG = Triacylglyceride, HDL = High density lipoprotein, LDL = Low density lipoprotein VLDL = Very low density lipoprotein, CRI= Castelli risk index

2.2 The **result of the effects of the leaf extract of Psidium guajava on Lipid profile of serum of rats fed high dietary salt**

Increase in ALP, ALT, AST CK and Bilirubin was observed in HSD group only when compared to HSD + 200 mg/kg, there was reduction in albumin in rats administer HSD only when compared with control. Whereas rats adminsterd both HSD and 200 mg/kg showed increase in bilirubin when compared to HSD +100 mg/kg.

Table: 2Results of Lipid profile in the serum of rats fed high dietary salts.

Conc./ Parameters	ALP(mg/d)	ALT(U/L)	AST(U/L)	CK(U/I)	ALB(mg/d)	BIL(mg/dl)
Control	29.63±1.02^{a}	45.71±1.10^{a}	25.30±1.19 ^{a}	21.50±1.55^{a}	37.18±1.11^{c}	56.60 ±1.39^{a}
HSD only	55.75±1.24^{d}	78.24±2.72^{d}	47.56±1.27^{d}	35.09±0.84^{d}	21.49±0.72^{a}	97.01±3.12^{d}
HSD+100mg/kg	41.16±1.09^{c}	63.84±1.61^{c}	36.93±0.89^{c}	30.73+0.70^{c}	29.30+0.88^{b}	78.46±2.10^{b}
HSD+200mg/kg	34.50±1.30^{b}	49.11±1.55^{b}	28.61±1.33^{b}	24.62±2.01^{b}	40.51±1.22^{d}	90.33±2.53^{c}

Values are expressed as mean ± standard deviation (n=5). Values with the different superscript(s) in a column are significantly different ($P<0.05$). **Key:** ALP = Alkaline phosphatase, ALT = Alanine amino transferase, AST= Aspartate amino transferase, CK= Creatinine kinase, ALB = Albumin,BIL = Bilirubin

2.3 The result of the effects of the leaf extract of *Psidium guajava* on Lipid profile in the liver tissues of rats fed high dietary salt

from the liver biomarkers in liver sample ALT, ALP, AST, GGT and Bilirubin activity in HSD treatment group when compared to control and HSD+200mg/kg CE. Whereas the specific activity was reduced was observed in HSD+100mg/kg CE and HSD treatment group.

Table 3: Results of lipid profile in the liver tissues of rats fed high dietary salt

Conc.	ALP (mg/dl)	ALT (U/L)	AST (U/L)	GGT (mg/dl)	ALB (mg/dl	BIL (mg/dl)
Control	48.15 ±1.21^{a}	77.18 ± 2.81^{a}	53.22±1.43^{a}	10.19±0.51^{a}	47.06±2.13^{c}	32.83 ± 1.17^{a}
HSD only	84.57±2.57^{d}	110.38±3.09^{d}	95.55±2.00^{d}	18.32±0.69^{c}	34.28±1.03^{a}	61.40± 1.58^{d}
HSD+100mg/kg	66.29±2.13^{c}	92.40±3.15^{c}	73.32±1.71^{c}	15.66±0.52^{b}	39.16±1.21^{b}	47.62±1.21^{c}
HSD+200mg/kg	52.03±1.20^{b}	80.63±2.65^{b}	58.40±2.02^{b}	10.71±0.61^{a}	46.51±1.27^{c}	35.81±2.08^{b}

Values are expressed as mean ± standard deviation (n=5). Values with the different superscript(s) in a column are significantly different (P<0.05). **Key:** ALP = Alkaline phosphatase**,** ALT = Alanine amino transferase AST= Aspartate amino transferase, GGT = Gamma glutamyltransferase, ALB = Albumin, BIL = Bilirubin

2.4 The result of the effects of the leaf extract of *Psidium guajava* on kidney function test in the serum of rats fed high dietary salt

From the kidney homogenate, creatine kinase specific activity increase was obtained in HSD group when compared to HSD + 100 mg/kg CE, and its reduction in HSD + 200 mg/kg CE treatment group when compared to HSD only, high increase in urea and uric acid concentration was observed in HSD group, when HSD + 200 mg/kg CE, there was reduction in urea and uric acid concentration when compared to HSD group.

Table 4: Result of kidney function test in the serum of rat fed high dietary salt

Conc./ Parameters	ALP(mg/d)	ALT(U/L)	AST(U/L)	CK(U/I)	Urea(mg/d)	Uric(mg/dl)
Control	23.41±1.03^{a}	31.05±2.10^{a}	11.01±1.01 ^{a}	15.30±1.20^{a}	52.60±1.17^{a}	26.07±1.59^{a}
HSD only	38.07±1.32^{d}	55.68±2.15^{d}	27.52±1.42^{d}	28.15±0.74^{d}	93.81±2.05^{d}	50.38±2.14^{d}
HSD+100mg/kg	30.50±1.04^{c}	41.27±1.27^{c}	17.30±0.93^{c}	24.53+1.53^{c}	71.44+1.40^{c}	33.65±1.90^{c}
HSD+200mg/kg	25.33±2.33^{b}	34.80±1.31^{b}	12.97±1.26^{b}	17.19±1.60^{b}	57.30±1.64^{b}	27.25±1.25^{b}

Values are expressed as mean ± standard deviation (n=5). Values with the different superscript(s) in a column are significantly different (P<0.05). **Key:** ALP = Alkaline phosphatase, ALT = Alanine amino transferase, AST= Aspartate amino transferase CK= Creatinine kinase

2.5 The result of the effects of the leaf extract of *Psidium guajava* on the antioxidant enzyme of rats fed high dietary salt

From table 5, table 6 and table 7 there was Significant reduction of antioxidant in serum, kidney tissue sample and liver sample such as SOD, catalase, GPx, GSH, and GSH in HSD group when compared to control group, and these enzymatic specific action increment was recorded in HSD + 200 mg/kg group when compared to HSD + 100 mg/kg treatment group.

Table 5: Results of Serum Antioxidant Enzymes of rats fed high dietary salt

Parameter	SOD Activity (U/mg protein)	CAT Activity (µmol/min/mg-protein)	GPx mg/100 mg tissue	GSH Concentration (mmole/min/mg-protein)	TP (mg protein/ml serum)
Control	5.36±0.41^{d}	4.18±0.20^{c}	14.66±0.15^{d}	6.07±0.10^{d}	4.51±0.13^{c}
HSD only	2.55±0.25^{a}	2.30±0.12^{a}	9.81±0.21^{a}	3.72±0.05^{a}	3.02±0.07^{a}
HSD+100mg/kg CE	3.61±0.14^{b}	3.16±0.11^{b}	12.29+0.26^{b}	4.88±0.16^{b}	3.80±0.17^{a}
HSD+200mg/kg CE	4.94±0.17^{c}	3.89±0.06^{bc}	13.54±0.10^{c}	5.79±0.12^{c}	4.39±0.11^{b}

Table 6: Results of Antioxidant Enzymes in the liver tissues of rats fed high dietary salt

Parameters	**SOD** Activity (U/mg protein)	**CAT** Activity (µmol/min/mg-protein)	**GPx** mg/deciliter	**GSH** Concentration (mmole/min/mg-protein)	**TP** (mg protein/ml serum)
Control	7.83±0.20[c]	4.11±0.21[c]	21. 67±1.01[c]	8.45±0.15[d]	5.06 ± 0.24[c]
HSD only	3.09±0.22[a]	2.30±0.15[a]	16.22±1.03[a]	5.61±1.00[a]	3.12 ±0.11[a]
HSD+100mg/kg	6.15±0.31[b]	3.52±0.14[b]	20.36+0.42[b]	6.08±0.62[b]	3.98± 0.19[a]
HSD+200mg/kg	8.01±0.24[d]	4.03±0.51[c]	22.92±1.26[d]	7.53±0.59[c]	4.79 ± 0.08[b]

Table 7: Results of Kidney antioxidant enzymes

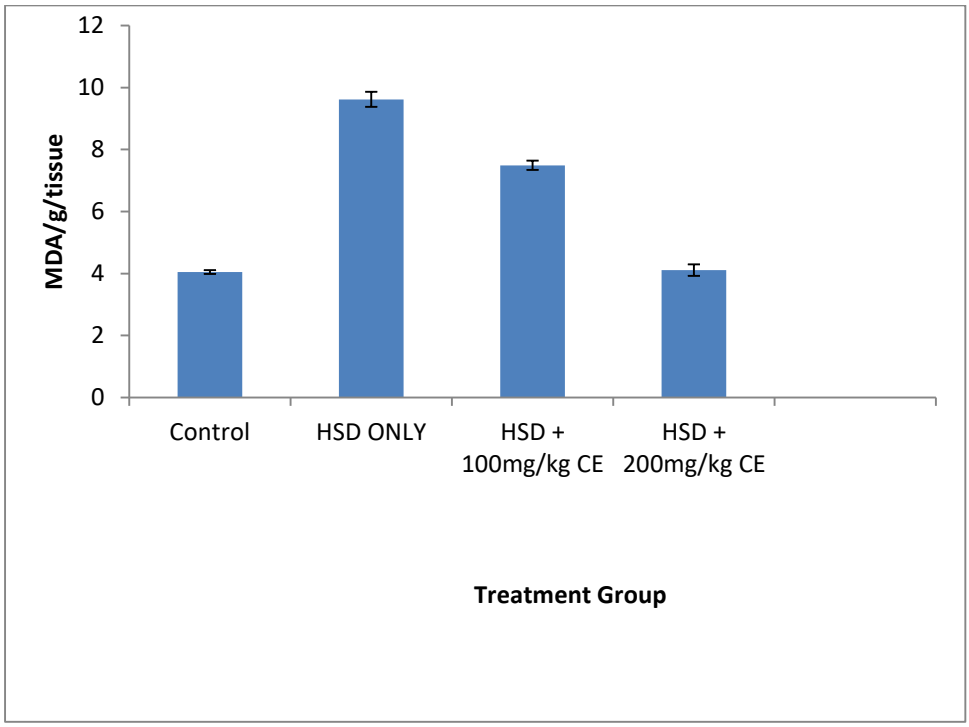
Parameters	**SOD** Activity (U/mg protein)	**CAT** Activity (µmol/min/mg-protein)	**GPx** mg/deciliter	**GSH** Concentration (mmole/min/mg-protein)	**TP** (mg protein/ml serum)
Control	3.09±0.32[c]	2.44±0.17[b]	9. 10±1.00[d]	7.47±0.35[c]	3.25 ± 0.40[c]
HSD only	1.24±0.27[a]	1.46±0.10[a]	5.33±0.40[a]	4.14±0.49[a]	1.63 ±0.15[a]
HSD+100mg/kg	2.65±0.06[b]	1.85±0.21[a]	6.17+0.61[b]	6.81±0.08[b]	2.40± 0.31[b]
HSD+200mg/kg	3.18±0.13[c]	2.31±1.10[b]	8.72±0.22[c]	7.39±0.16[c]	3.12 ± 0.44[c]

Values are expressed as mean ± standard deviation (n=5). Values with the different superscript(s) in a column are significantly different (P<0.05). **Key:** SOD = Superoxide dismutase, CAT = Catalase, GPx = Glutathione peroxidase, GSH = Glutathione, TP = Total protein.

2.6 **The result of the effect of the leaf extract of Psidium *guajava* on lipid peroxidation (MDA) in rats fed with high dietary salt**

Significant increase in MDA concentration was observed in serum of HSD group, when compare to HSD+100mg/kg (Fig 1) and reduction of MDA concentration was recorded in HSD+200mg/kg CE subject group when compare to control.

Fig 1: Result of lipid peroxidation (MDA) in the serum of rats fed high dietary salt



The results of histopathology of the renal tissues of the male rats are presented as Figures 2 to 5. The observed features and the pathological lesions are indicated with arrows in the micrographs

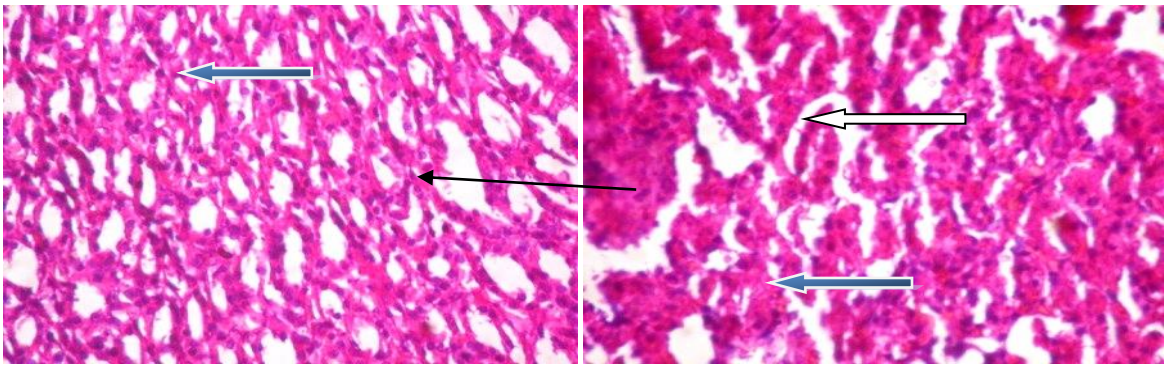


Fig 2: Photomicrographs (H& E) of kidney sections stained by Haematoxylin and Eosin of rats given normal feed only for 14 day depict normal architecture, the renal cortex show normal glomeruli with normal mesengial cells and capsular spaces (white arrow), the renal tubules appear normal (blue arrow), the interstitial spaces appear normal (slender arrow). Mag X 400

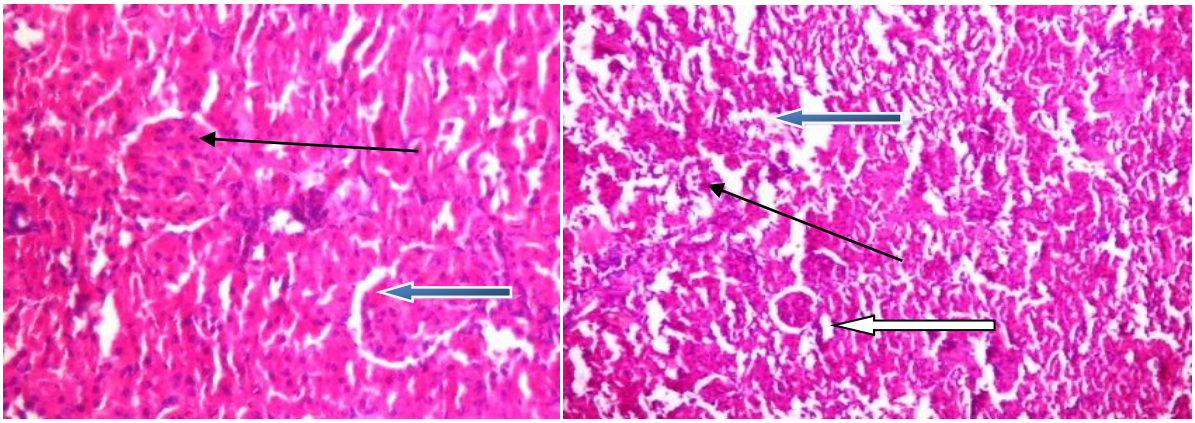


Fig 3: Photomicrographs (H& E) of kidney sections stained by Haematoxylin and Eosin of rats exposed to high salt diet for 14 days indicate poor architecture, renal tubules appeared mildly degeneration (white arrow), while other tubules appear normal (blue arrow), the interstitial spaces show mild vascular congestion (slender arrow). Mag X 400

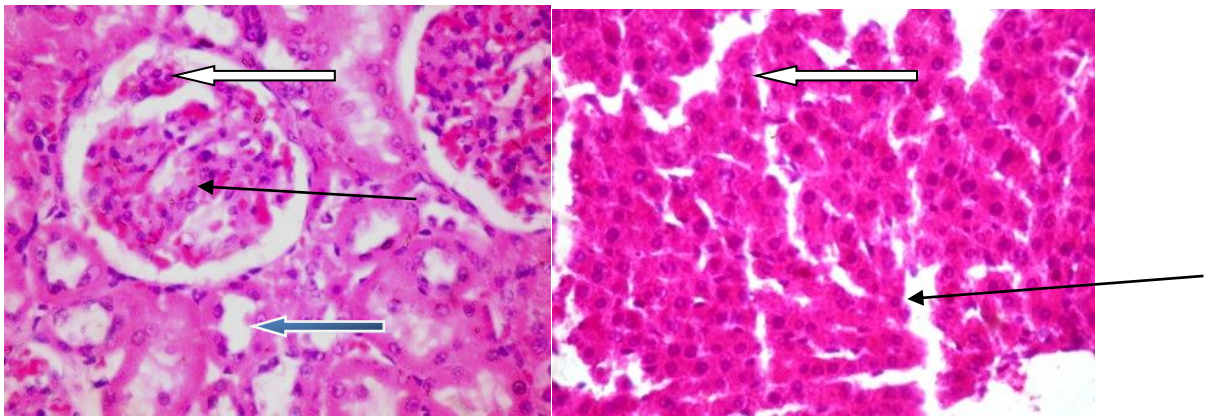


Fig 4: Photomicrographs (H&E) of kidney sections stained by Haematoxylin and Eosin of rats given high salt and administered 100mg/kg crude extract depicts moderate architecture, the renal cortex shows normal glomeruli with normal mesengial cells and capsular spaces (white arrow), most of the renal tubules appear normal (blue arrow) while few are collapsed with lack of luminar spaces, the interstitial spaces appear normal (slender arrow). Mag X 400

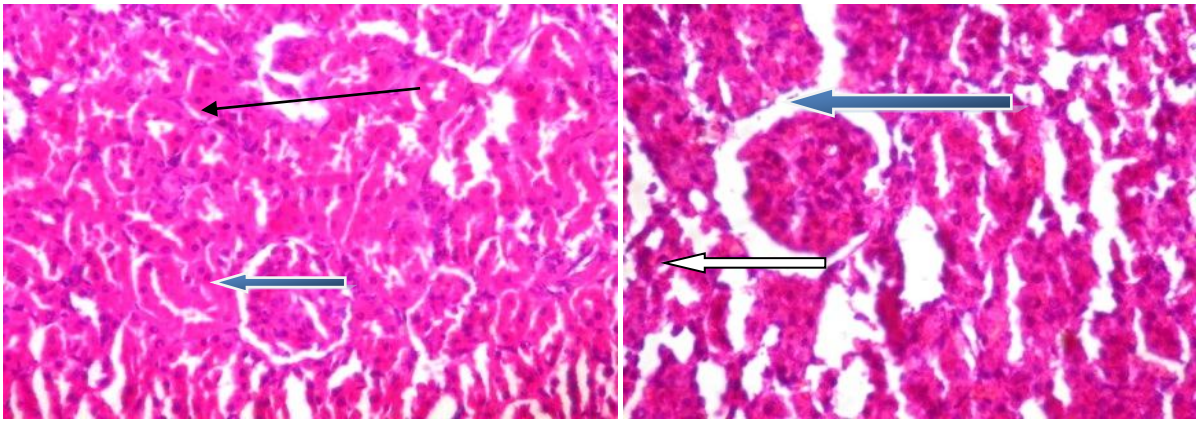


Fig 5: Photomicrographs (H&E) of kidney sections stained by Haematoxylin and Eosin of rats administed high salt and 200 mg/kg bd crude extracts depects normal architecture, the renal cortex show normal glomeruli with normal mesengial cells and capsular spaces (white arrow), the renal tubules appear normal (blue arrow), the interstitial spaces appear normal (slender arrow).

Histopathology of liver tissues

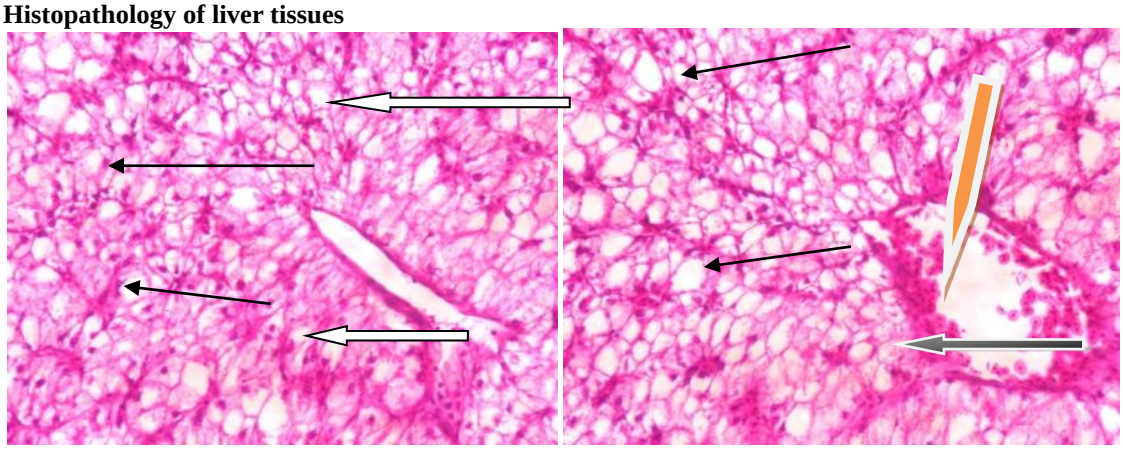


Fig 6: Photomicrograph (H&E) of a liver section stained by Haematoxylin and Eosin of rats administered normal feed (control) shows normal venule without congestion (white arrow), the morphology of the hepatocytes appear normal (black arrow), the sinusoids appear normal and not infiltrated (slender arrow). Mag X 400.

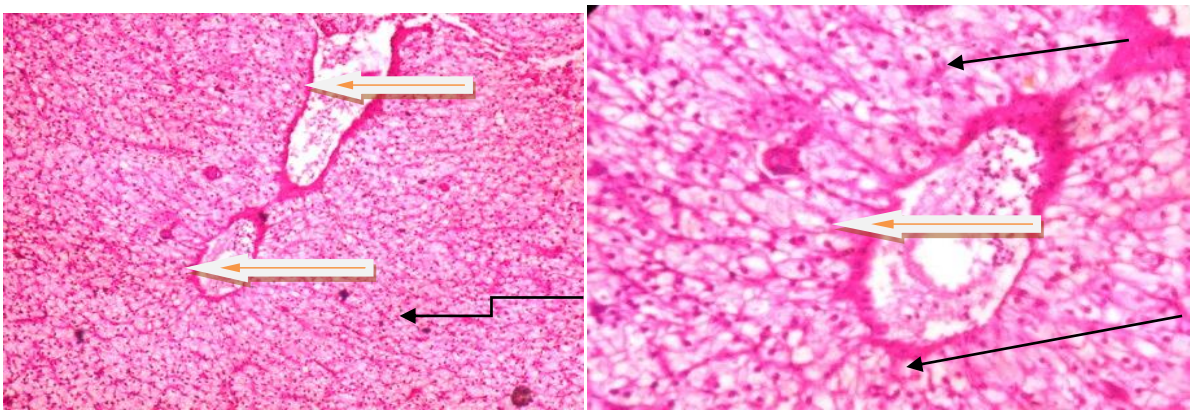


Fig 7: Photomicrograph (H&E) of a liver section stained by Haematoxylin and Eosin of rats exposed to high salt diet for 14 days showed mild congestion of portal vein (white arrow), the liver parenchyma also show area with destroyed liver plates with severe hemorrhage and necrosis (black arrow), the sinusoid appears normal and not infiltrated (slender arrow). Mag X 400

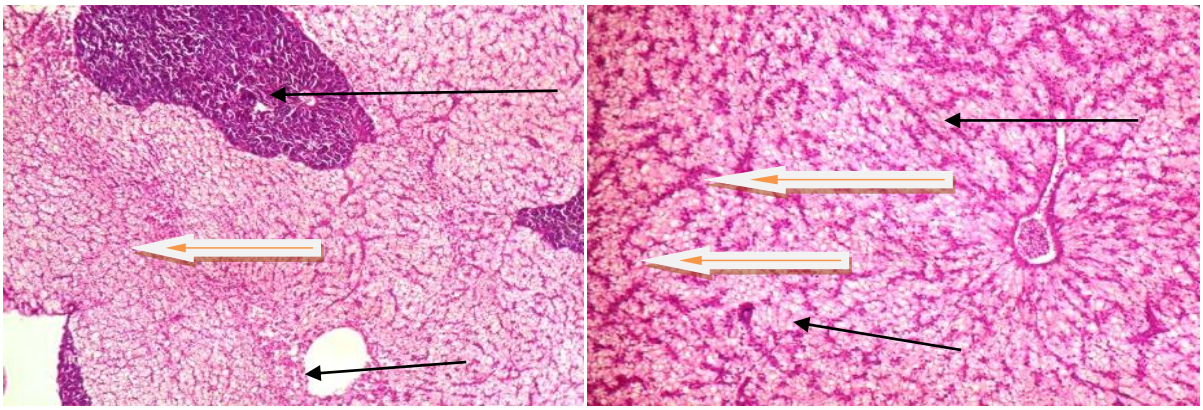


Fig 8: Photomicrograph (H&E) of a liver section stained by Haematoxylin and Eosin of rats exposed to high salt diet and administered 100mg/kg bd crude extract showed hepatocytes show normal glycogen infiltration (slender arrow), normal venules without congestion (white arrow), the sinusoid appear normal and not infiltrated (black arrow). Mag X 400

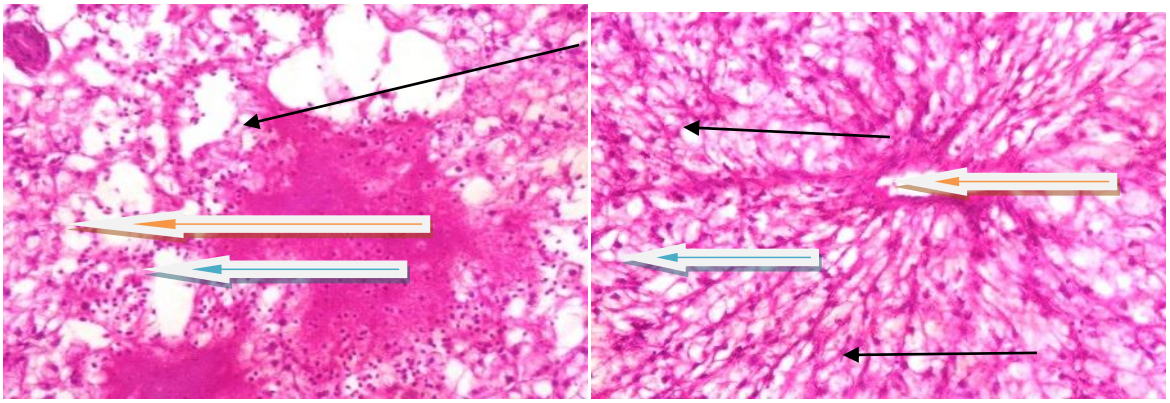


Fig 9: Photomicrograph (H&E) of a liver section stained by Haematoxylin and Eosin of rats exposed to high salt diet and chewed 200 mg/kg crude extracts indicated normal central venules and portal tract without congestion (white arrow) , the morphology of the hepatocytes appear normal (black arrow), the sinusoids appear normal and not infiltrated (slender arrow), no pathological lesion seen. Mag X 400

DISCUSSION

Lipid peroxidation can be described as a process in which free radicals (oxidant) attack lipid contain carbon-carbon double bond example polyunsaturated fatty acids leading to oxidative damages (oxidative stress) in tissues, organ and cells (Ayala *et al.,* 2014). High level of oxidant such as reactive oxygen species or free radicals causes this oxidative damages as a result of imbalance the proxoidants and antioxidant levels, whereby prooxidant is more favourable. Lipid peroxidation undergoes series of enzymatic chain reaction resulting in MDA, propanal, hexanal etc. with MDA being the most mutagenic products.

Oxidative stress marker such as MDA has been known to cause various pathology disorder such as Alzheimer diseases, cardiovascular diseases, liver diseases, Parkinson diseases, neurological disorder etc (Ayala *et al.,* 2014).

From the study of research conducted it shows high salt diet induces oxidative stress in the serum, kidney and liver sample in the rats given high salt only, thereby causing oxdative damages in serum, liver and kidney tissues. This was more buttresses from Haematoxylin and Eosin staining (fig 6) where oxidative damage causes poor architecture, destroyed liver plate, severe hemorrhage, liver necrosis, mild congestion of portal vein in the liver cell, and mild congestion of interstitial spaces.

The oxidative stress was attenuated and ameliorated with the use of guava leaf which was assumed to improve the antioxidant activities of the metabolic system. Superoxide dismutase synthetase activities, catalase and glutathione (GSH) has been known to be an antioxidant whose role is to neutralize and dismutate reactive oxygen species (ROS). Magnificent Increase in SOD, GSH and Catalase in rats given high salt diet and administered 200mg/kg extracts indicated that guava leaf may serve as a good antioxidant mechanism to counter oxidative damage.

Serum assessment of lipid profiles lipid entails knowing the concentration and amount of high density lipoprotein (HDL) cholesterol, triglycerides, total cholesterol and its fractions; very low density lipoprotein (VLDL) and low density lipoprotein (LDL) cholesterol which is being carried out to assess the cardiovascular, coronary artery and peripheral vascular layers. Sharp increase of cholesterol (cholestoriemia) was recorded in rats administered high salt diet group, several studies have reported high cholesterol leads to the development atherosclerosis, heart attacks and cardiovascular accidents (Adeyemi and Orekoya, 2014) Significant increase in Low density lipoprotein also known as bad cholesterol, (LDL) castelli risk index (CRI) also known as cardiac risk ratio and triglycerides was detected in rats administered high salt diet which is a risk factor for development of coronary heart diseases, myocardial infarction, future cardiovascular diseases, deposition of lipid on the arterial wall called plaque thereby causing atherosclerosis, stroke, peripheral vascular diseases and heart failure (Adeyemi and Orekoya, 2014). Rats given high salt diet and 200mg of the extract ameliorate the effect of the dyslipidemia and hyperlipidemia indicating that guava leaf has good potential of reducing the prone to cardiovascular risk factors. Disruption of liver plasma membrane and liver cells damages was noticed in rats administered high salt diets, thereby causing increase in ALP, ALT, AST, GGT and ALB (Olorunnisola *et al.*, 2021). Previous work showed that high amount of these liver enzymes implicates cirrhosis of liver, liver damage, chronic renal failure making the enzyme to leak out to the outside cells and accumulates in the body.

When liver damaged it lead to loss of metabolic function and hypoglycemia. High amount of Bilirubin and GGT was also recorded; An increase in total bilirubin may indicate that the excretory function of the liver has been affected by damage caused to the liver cells thereby causing hyperammonemia. This was also complemented by histopathology staining (fig7) of destruction of liver plate and necrosis. The result is in line gotten from Olorunnisola *et al*., (2021).

High amount of urea, creatinine and uric acid was found in serum and kidney tissues indicating high salt causes damages to the kidney tissues, nephron, tubules and as a result of decrease in glomerular filtration rate (GFR). The function of the kidney is to remove toxic and waste metabolic product from the body. Since the kidney is affected (renal failure) they are not removed from the body, thereby making them to over accumulate in the metabolic body. The damages to the liver and kidney might be to high salt diets induces oxidative stress (damages). The guava leaf serves as a good antioxidant which help to reduces and ameliorate the damages caused by oxidative stress, this was justified when the rats was administered 100mg/kg and 200mg/kg crude extracts of the guava leaf (Paul *et al.,* 2012).

CONCLUSION

Indiscriminate and abuse of salt for taste, aroma seasoning, flavour enhancer for food without minding the possible effects on the body system over time. should be utterly discouraged as it can cause various degree of pathological effects in the body

High amount of salt in food can induces oxidative damages in liver, kidney, lungs, heart and serum (plasma) cell. However, (*Psidium guajava* Linn), guava leaf (200mg/kg) exhibits a good antioxidant mechanism by reducing and ameliorating damages caused by the oxidative stress.

RECOMMENDATION

Based on our findings 200mg/kg guava leaf *Psidium guajava* Linn showed a good significant antioxidant against oxidative stress caused by high salt in the kidney and liver. It is therefore necessary to educate and regulate the use of table salt in our diets in Nigerian. By emphasizing the dangers and metabolic disorder that results in abuse of salt in our diet.

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