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Comparative Studies of Flax and Chia Seeds Supplemented Diet on Oxidative Stress Markers and Selected Biochemical Indices of Wistar Rats Fed with Monosodium Glutamate

¹Iloh Patience N., ²Ifemeje Jonathan C,³Nwaka Andrew C., ⁴Ifemeje Mary Jane O.,⁵Ifedi,Izuchukwu C., ⁶Nwafor Paul M, ⁷Mere Chinenye A.,⁸Ilechukwu Chijioke C. & ⁹Iloh John Paul I.

Abstract: This study evaluates the impact of Linum usitatissimum (flaxseeds) and Salvia hispanica (chia seeds) supplementation on oxidative stress markers and selected biochemical indices in Wistar rats exposed to monosodium glutamate (MSG). The experimental design comprised 48 adult wistar rats distributed into seven groups, including a Normal Control (Group A), a monosodium glutamate (MSG)-treated group (Group B), 15 mg/kg MSG +20% chia seed (Group C), 15 mg/kg MSG +20% flax seed (Group D), 15 mg/kg MSG +40% chia seed (Group E), 15 mg/kg MSG +40% flax seed (Group F), and 15 mg/kg MSG +20% chia and flax seed (Group G). The rats were kept and received treatment for 28 days, after which they were sacrificed, and blood samples were collected for evaluations using the standard methods. **Parameters** assessed included lipid peroxidation (malondialdehyde, MDA), antioxidant enzyme activities (superoxide dismutase, SOD, and catalase, CAT), lipid profile (triglycerides, total cholesterol, high density lipoprotein, low density lipoprotein), and liver function tests (alanine transaminase (ALT), aspartate transaminase (AST) and alkaline phosphate (ALP)). MSG exposure significantly disrupted lipid metabolism and liver function, evidenced by elevated triglycerides, total cholesterol, LDL, and liver enzymes, alongside decreased HDL, reduced antioxidant enzyme activities, and altered MDA levels (P < 0.05). Dietary inclusion of chia and flax seeds, particularly at 40% concentrations and in combination, significantly reversed these effects by lowering lipid and liver enzyme levels, reducing MDA concentrations, and enhancing SOD and CAT activities (P < 0.05). The combined seed diet showed the greatest improvement in oxidative stress markers and antioxidant capacity, suggesting a potentiated protective effect. These findings highlight the potential of chia and flax seeds as functional food ingredients with hepatoprotective, antilipidemic, and antioxidant properties capable of mitigating MSG-induced oxidative damage and metabolic disturbances.

Keywords: Antioxidant defense, lipid peroxidation, hepatic biomarkers, dietary intervention, metabolic imbalance.

I. Introduction

Dietary interventions high in antioxidants are becoming increasingly important for preventing and managing diseases caused by oxidative stress. Oxidative stress occurs when the body's antioxidant defenses are overwhelmed by reactive oxygen and nitrogen species (ROS/RNS), resulting in damage to vital biomolecules like lipids, proteins, and nucleic acids. This imbalance is now recognized as a key factor in many conditions, including atherosclerosis, neurodegenerative diseases such as Alzheimer's and Parkinson's, cancer, diabetes mellitus, inflammatory disorders, and even psychological and age-related diseases (Egbuna and Ifemeje, 2017; Durackova, 2010).

Reactive species, especially ROS, are produced both internally, through normal cell processes and mitochondrial activity, and externally, due to environmental factors like pollutants, drugs, and dietary components. While low to moderate levels of ROS are involved in normal functions such as immune response and cell signaling, high levels cause oxidative damage, including lipid peroxidation, and interfere with metabolic and liver functions (Birbenet al., 2012; Ayala et al., 2014). The body depends on a system of enzymes (like superoxide dismutase and catalase) and non-enzymatic antioxidants to fight these effects, but this protection can be compromised under disease conditions (Egbuna and Ifemeje, 2017; Halliwell, 2007).

Recent research has focused on the role of natural dietary antioxidants, such as those found in flaxseed and chia seed, in enhancing endogenous defense systems and mitigating oxidative damage. These seeds are rich in polyunsaturated fatty acids, polyphenols, and dietary fiber, which have been shown to lower lipid peroxidation, improve antioxidant enzyme activity, and support healthy lipid and liver profiles (Cahill, 2003; Newairyand Abdou, 2009). However, the comparative and combined effects of these seeds in models of chemically induced oxidative stress remain underexplored.

This study aims to fill this gap by evaluating the effects of flaxseed and chia seed supplementation, individually and combined, on lipid peroxidation, antioxidant enzyme activities, lipid profile, and liver function in Wistar rats subjected to monosodium glutamate (MSG)-induced oxidative stress.

II. Materials and Methods

A. Materials

Samples of chia seeds and flax seeds, each weighing 1.5 kg, were purchased from the Roban store in Awka. The samples were ground into powder using a corona grinder and stored in an airtight container at room temperature. A normal rat chow from Top Feed Limited, specifically Grower's Mesh, was obtained from Eke Awka. Monosodium Glutamate was purchased from Sigma. Forty-eight (48) male Wistar albino rats weighing approximately 120-150g were obtained from Chris Animal

Farm, IfiteAwka. The rats were housed in well-ventilated stainless steel cages and allowed to acclimate for 7 days before a 4-week experimental feeding period. The animals were kept under ambient conditions with free access to standard rat chow and water. The LD50 of the additives was determined by Lorke's method (1983) using thirteen (13) rats. The animals received monosodium glutamate, chia, and flax seeds and were monitored for 24 hours for signs such as excitation, paw licking, increased respiratory rate, convulsions, and death. LD50 was calculated.

B. Experimental Design

After acclimatization, the rats were randomly assigned to seven groups of five rats each (n = 5). The control group (Group A) received normal saline (3ml/kg dose). In contrast, Group B received monosodium glutamate (15mg/kg dose) dissolved in 3ml of distilled water only with normal rat chows. Group C and D received (15mg/kg MSG + 20% chia seeds and 15mg/kg MSG + 20% flax seeds), respectively, dissolved in 3ml of distilled water with normal rat chows. Group E and F received (15mg/kg MSG + 40% chia seeds and 15mg/kg MSG + 40% flax seeds), respectively, dissolved in 3ml of distilled water with normal rat chows. Group G received (feed +15mg/kg MSG + 20% chia seeds + 15mg/kg MSG + 20% flax seeds), respectively, dissolved in 3ml of distilled water. The treatment was administered orally (via intubation) for 4 weeks. At the end of the 4-week experimental feeding period, the rats were anesthetized using chloroform after an overnight fast. Blood samples were collected by cardiac puncture using a 10 mL syringe and placed in an EDTA bottle. The serum samples were obtained after centrifugation, and subsequent analyses were performed.

C. Lipid Peroxide (Estimation of Malondialdehyde Level)

MDA levels, an index of lipid peroxidation, were measured by the double-heating method of Okhawa et al. (1979). This method is based on spectrophotometric measurement of the purple color generated by the reaction of TBA with MDA. For this purpose, 2.5 mL of trichloroacetic acid solution (10% w/v) was added to 0.5 mL of homogenized tissue in each centrifuge tube; the tubes were then placed in a boiling water bath for 15 minutes. After cooling to room temperature, the tubes were centrifuged at $1000 \times \text{g}$ for 10 minutes, and 2 mL of each sample supernatant was transferred to a test tube containing 1 mL of TBA solution (0.67% w/v). Each tube was then placed in a boiling water bath for 15 minutes. After cooling at room temperature, the absorbance was read at 532 nm using a spectrophotometer.

D. Estimation of Superoxide Dismutase Activity (SOD)

Superoxide dismutase (SOD) activity was assayed by the nitro blue tetrazolium (NBT) method as described by Beauchamp *et al.* (1971). In this method, the reaction mixture consists of 0.5 mL of supernatant, 1 mL of 50 mM sodium carbonate, 0.4 mL of 25 μ M NBT, and 0.2 mL of 0.1 mM EDTA. The action is then initiated by the addition of 0.4ml of 1mMhydroxylamine hydrochloride. The change in absorbance is recorded at 560 nm using a UV spectrophotometer. The control is simultaneously run without homogenization. Units of SOD activity are expressed as the amount of enzyme required to inhibit the reduction of NBT by 50%. Specific activity of total SOD is expressed as units per milligram of protein.

$$SOD = \frac{ODsample \times 100 \times 10^6}{4020} \div protein content$$

E. Estimation of Catalase Activity (CAT)

Catalase (CAT) activity was determined by catalytic reduction of hydrogen peroxide using a standard method described by Aebi (1984). The mixture consists of 1.95 ml of phosphate buffer (0.05 M, pH 7), 1 ml of H2O2 (0.019 M), and 0.05 ml of sample (10% w/v) in a final volume of 3 ml. The control cuvette contains all the components except the substrate. Change in absorbance is then recorded at 240 nm, and the results are expressed as micromoles of product formed per minute per milligram of protein of the tissue.

$$CAT = \frac{ODsample \times 15 \times 10^3}{40} \div protein content$$

F. Serum Lipid Profile Assay

Total serum cholesterol, HDL fraction, LDL fraction, and Triglyceride fraction were quantified using specific methods. Total serum cholesterol was determined enzymatically following the protocol outlined by Allain et al, (1974) by applying the Randox Cholesterol Kit (Randox, England). The serum (10µL) was mixed with 1000µL of cholesterol reagent and allowed to stand for 10 minutes at room temperature, after which the total cholesterol content was measured using a spectrophotometer. The HDL fraction was isolated using the HDL-Cholesterol precipitant method described by Friedwald WT et al., (1972). LDL was calculated using a formula derived from total cholesterol, triglycerides, and HDL cholesterol levels (LDL-Cholesterol = Total Cholesterol - (Triglycerides/5 + HDL cholesterol)). Triglyceride followed the method of Fossati and Prencipe (1982). The triglyceride content of the serum was measured at 546nm using a UV-visible spectrophotometer. The serum (10µL) was mixed with 1000µL of Triglyceride reagent and allowed to stand for 10 minutes at room temperature, after which the Triglyceride content was measured using a UV-visible spectrophotometer.

G. Liver Function Test

Randox kit was used for AST and ALT activity determination based on the modified method of Reitman and Frankel (1957). The blood samples were centrifuged, and the sera were used for the analysis. A sample of 0.1 ml was pipetted into test tubes. 0.5ml of phosphate buffer was added to the sample and blank, mixed, and incubated at 370 °C for 30 minutes. 2, 4-dinitrophenylhydrazine was added to the sample test tubes, mixed, and allowed to stand for exactly 20 minutes. Sodium hydroxide was added to the sample and blank, mixed, and the absorbance of the sample was read against the blank at 546nm after 5min. The AST activity was determined by the calibration curve provided in the kit.

H. Alkaline Phosphatase Activity Determination (ALP)

A Randox ALP Kit was used. The method of DGKC, 1972, was adopted. The working reagent was prepared by pipetting 10ml of the buffer (mixture of Diethanolamine buffer (1mol/l, pH 9.8 and MgCl₂, 0.5mol/l) into one vial of the substrate (p-nitrophenylphosphate (p-NPP)).1 mL of this mixture was then added to 20 μ L of serum. The absorbance was read after 30 seconds and again after 1, 2, and 3 minutes.

III. Statistical Analysis

The data obtained were expressed as the mean ± SD of three replicates. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 23. One-way analyses of Variance were adopted for comparison, and the results were subjected to a post hoc test using the least significant difference (LSD) method. p<0.05 was considered significant for all the results.

IV. Results and Discussion

A. Results

(a) Lipid peroxidation (Malondialdehyde)

The data in Table 1 showed the levels of MDA in different groups exposed to various treatments involving MSG, flax, and chia seeds. The analysis results showed a significant decrease in MDA levels in the monosodium glutamate-treated group (1.92 \pm 1.02 μ mol/ml \times 10^ (-8)) compared to the control group (2.35 \pm 1.06 μ mol/ml \times 10^ (-8)). On the contrary, Groups C and D, supplemented with 20% chia and flax seeds, respectively, demonstrated an increase in MDA levels compared to the control. Increasing the concentration of the seeds, Group E (15mg/kg MSG + 40% Chai seed) and Group F (15mg/kg MSG + 40% Flax seed),

both exhibited lower MDA levels of 2.06 ± 0.90 and 1.85 ± 0.51 µmol/ml x 10^{-8} , respectively, compared to the monosodium glutamate-treated group. Furthermore, the combination of the seeds in Group G (15mg/kg MSG + 20% Chai seed + 20% Flax seed) demonstrated the lowest MDA level among all groups, indicating a potential enhanced effect of combining chai and flax seeds in reducing oxidative stress, with a value of 1.24 ± 0.79 µmol/ml x 10^{-8} .

Table 1: Malondialdehyde test of flax and chai seeds

Groups	MDA (µmol/ml x 10 ⁻⁸)
Group A: Normal Control	2.35±1.06
Group B: 15mg/kg MSG	1.92±1.02
Group C: 15mg/kg MSG + 20% Chaiseed	2.40±0.18
Group D: 15mg/kg MSG + 20% Flax seed	2.25±0.70
Group E: 15mg/kg MSG + 40% Chai seed	2.06±0.90
Group F: 15mg/kg MSG + 40% Flax seed	1.85±0.51
Group G: 15mg/kg MSG + 20% Chai seed + 20% Flax seed	1.24±0.79

Values are presented as mean \pm SD (n=5) p < 0.05

(b) Antioxidant Capacity

The superoxide dismutase (SOD) and catalase (CAT) activities of the supplemented seeds are shown in Figures 2 and 3, respectively. The results demonstrated that both SOD and CAT showed reduced activities inthe treatment of MSG. The treatment with monosodium glutamate (MSG) resulted in a significant decrease in SOD activity, from $0.00014 \pm 0.00002 \, \mu \text{mol/min}$ in the normal control group to $0.00037 \pm 0.00037 \, \mu \text{mol/min}$. The concentration of flax at 40% resulted in a significant increase in SOD, $0.0010\pm0.00060 \, \mu \text{mol/min}$, compared to chia seed supplementation alone. Administration of MSG resulted in a reduction in CAT activity, with a mean value of $0.72 \pm 0.12 \, \mu \text{mol/min}$, compared to the control group of $0.82 \pm 0.35 \, \mu \text{mol/min}$, indicating a decrease in antioxidant capacity. However, feeding with flax seeds resulted in a significant (P< 0.05) increase in CAT activity ($1.33 \pm 0.12 \, \mu \text{mol/min}$), particularly at higher concentrations. Furthermore, the combination of chia and flax seeds exhibited an enhanced effect, leading to a higher increase in CAT activity of $2.59\pm1.83 \, \mu \text{mol/min}$ compared to either seed alone.

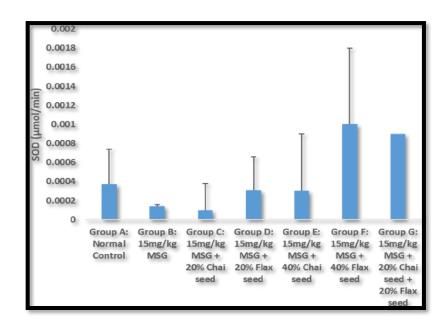


Figure 1: Superoxide activities

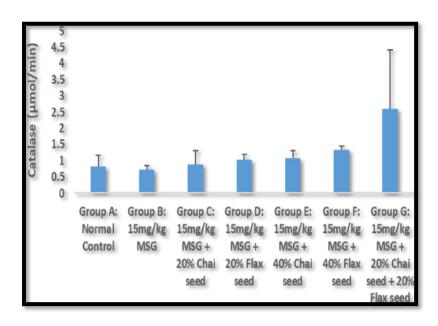


Figure 2: Catalase activities

(c) Lipid Profile

The results of the lipid profile test, including Triglycerides (TRIG), Total Cholesterol (TCHOL), High-Density Lipoprotein (HDL), and Low-Density Lipoprotein (LDL), are shown in Figures 3, 4, 5, and 6. The result of the analysis demonstrated that both seeds have antilipidemic effects. From the result, it is evident that rats treated with MSG only (+ve control) had higher levels of TRIG and TCHOL compared to the normal control group (-ve control). The mean values of TRIG and TCHOL in the MSG group were significantly increased (P < 0.05) compared to the normal control group. Rats fed diets containing 40% flax seed showed the most significant decrease in TRIG and TCHOL levels compared with the MSG group. Diets with 40% chia seed also reduced these parameters significantly, while the combination of 20% chia and 20% flax seeds resulted in a moderate but significant reduction compared to the MSG group. The mean value of HDL in the MSG group was significantly lower (P < 0.05) than in the normal control group. Rats fed with 20% chia seed and 20% flax seed showed significant increases in HDL levels compared to the MSG group, with chia seed having a slightly greater effect. The combination group also showed a significant increase in HDL compared to the MSG-only group. The mean LDL levels were significantly elevated in the MSG group compared to the normal control. The lowest LDL levels were observed in rats fed with 40% flax seed and the combination of 20% chia and 20% flax seeds, both showing significant reductions (P < 0.05) compared to the MSG group. The 40% chia seed group also demonstrated a significant decrease in LDL levels. Overall, rats fed diets containing chia and flax seeds, especially at higher concentrations or in combination, showed significant improvements in serum lipid parameters when compared with the MSG-only group.

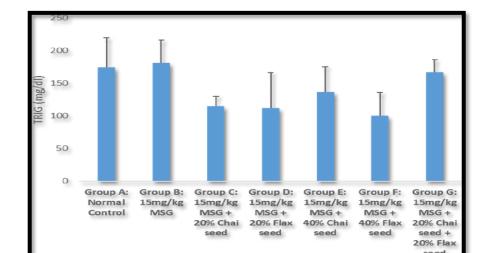
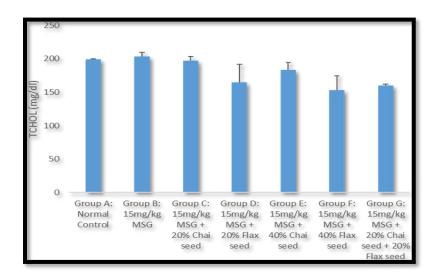


Fig 3: Triglycerides level



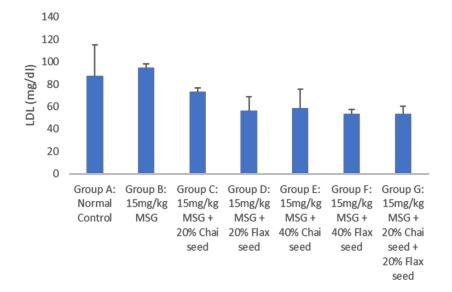


Figure 6: Low density Lipoprotein

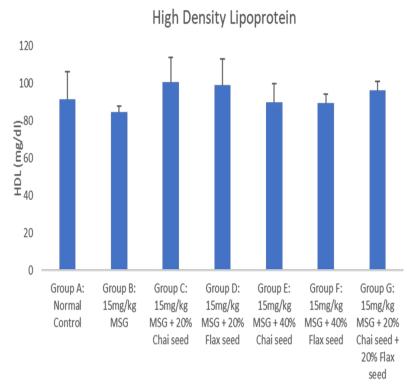


Figure 5: High Density

(d) Liver Function Test

The result of the liver function test measuring alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) levels across different experimental groups are shown in Figures 7, 8, and 9. The analysis showed that both chia and flax seeds have hepatoprotective effects. The mean values for ALT, AST, and ALP in the MSG-only group (Group B: $36.00\pm15.13\,\text{U/L}$, $91.63\pm24.13\,\text{U/L}$, and $98.21\pm26.30\,\text{U/L}$, respectively) were significantly higher (P<0.05) compared to those recorded for the normal control group (Group A: $21.28\pm11.46\,\text{U/L}$, $85.83\pm55.48\,\text{U/L}$, and $88.52\pm7.69\,\text{U/L}$, respectively). Among the intervention groups, the lowest ALT and AST levels were observed in Group F (40% flax seed: ALT $22.98\pm2.99\,\text{U/L}$, AST $74.33\pm9.83\,\text{U/L}$). For ALP, the most significant reduction was found in Group G (combined 20% chia and flax seeds: $35.42\pm47.63\,\text{U/L}$). All groups receiving chia and flax seeds, especially at higher inclusion rates (Groups E, F) or in combination (Group G), exhibited significantly

reduced (P<0.05) ALT, AST, and ALP levels compared to the MSG-only group, indicating improved liver enzyme profiles following seed supplementation.

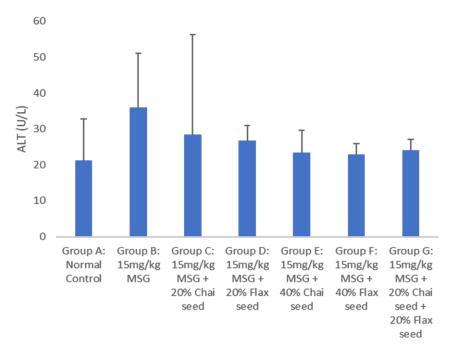


Figure 7: Alanine transaminase

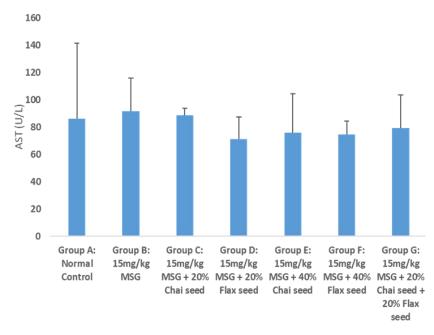
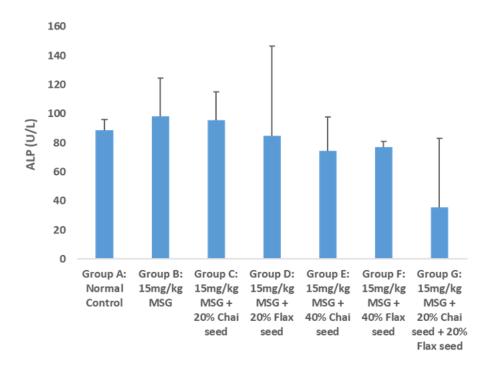


Figure 8: Aspartate aminotransferase (AST)



B. Discussion

Flax and chia seeds have been shown to play a protective role against oxidative stress, which is evident by the significant decreases in MDA levels and increased SOD and CAT activities, indicating enhanced antioxidant defense. According to Wadley et al. (2013), oxidative stress can cause damage by generating reactive oxygen species, which in turn lead to chronic inflammation. Oxidative stress also increases lipid peroxidation and the depletion of antioxidant enzymes, promoting further tissue injury and ischemia (Mittal et al., 2014).

The Malondialdehyde (MDA) test is a standard method used to measure oxidative stress levels in the body. (Ghosh et al., 2019). Treatementwith MSG significantly increased serum MDA levels in rats, indicating enhanced lipid peroxidation and oxidative stress. This is consistent with the findings of Egbuna and Ifemeje (2017), who reported that excessive ROS generation leads to peroxidative damage of membrane lipids, with MDA serving as a sensitive biomarker. Similar observations were made by Ayala et al. (2014), who highlighted the role of MDA in reflecting the extent of oxidative injury in various disease models. However, dietary supplementation of chia and flax seeds significantly reduced MDA levels, thus suggesting their potential in mitigating MSG induced oxidative stress and biochemical alterations. Flaxseed oil contains alpha-linolenic acid, an omega-3 fatty acid, and various phytoestrogens, which may contribute to its antioxidative properties (Yasmeen et al., 2019). The mechanisms underlying its effects on MDA

levels involve upregulation of the LDL receptor and peroxisome proliferator-activated receptor gamma (PPAR- γ), potentially shielding MDA from free radicals (Elimam and Ramadan, 2018).

Administration of monosodium glutamate demonstrated an antioxidant defense mechanism, which aligns with previous research indicating that MSG may have potential adverse effects on antioxidant status. However, supplementation with chia and flax seeds, especially flax seeds, showed a significant increase in SOD activity. Additionally, the combination of chia and flax seeds demonstrated a significant increase in SOD activity compared to either seed alone. This suggests that combining these seeds could offer enhanced antioxidant protection against MSGinduced oxidative stress. This result aligns with a study performed by Jangaleet al., (2013), where administration of flaxseed oil significantly increased antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD), and GSH. Similarly, the analysis of Catalase (CAT) activity corroborated the findings observed with SOD activity. Treatment with MSG resulted in a reduction in CAT activity, indicating a decrease in antioxidant capacity. However, supplementation with flax seeds resulted in a significant (P< 0.05) increase in CAT activity (1.33 \pm 0.12 μ mol/min), particularly at higher concentrations. Furthermore, the combination of chia and flax seeds exhibited a synergistic effect, resulting in a 2.59 ± 1.83 µmol/min increase in CAT activity compared to either seed alone. This suggests that combining chia and flax seeds could offer superior antioxidant protection against MSG-induced oxidative stress compared to individual supplementation. In a study conducted by Naqshbandi et al. (2013), which explored the potential of flaxseed oil to counteract oxidative damage to the kidneys induced by anticancer therapy, the researchers observed a significant increase in the activity of key antioxidant enzymes, namely SOD, CAT, and GPx. Similarly, Czech et al. (2019) conducted research investigating the effects of replacing soybean oil with flaxseed oil in turkey diets. Their findings revealed elevated levels of SOD and CAT activity in the bloodstream, alongside an increase in the FRAP value attributed to heightened vitamin E concentrations.

The lipid profile test examined various lipid parameters, including triglycerides (TRIG), total cholesterol (TCHOL), high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels in response to different supplementations such as MSG, chia seed, and flax seed supplementation. The results showed that both flax and chia seed supplements have significant (P<0.05) triglyceride, total cholesterol, and low-density lipoprotein lowering effects against monosodium glutamate exposure. Triglyceride levels are an important marker of lipid metabolism and cardiovascular health. Elevated TRIG levels are associated with an increased risk of cardiovascular diseases. The study observed that administration of MSG alone

led to an increase in TRIG levels compared to the control group. At the same time, supplementation with chia seed and flax seed resulted in significant reductions in TRIG levels compared to the MSG-alone group. This result is in agreement with previous research supporting the hypolipidemic properties of chia and flax seeds. Ayerza and Coates (2007) reported that chia seeds reduced TRIG levels in animal studies, while Guevara-Cruz et al., (2012) demonstrated the triglyceride-lowering effects of chia seed consumption in patients with metabolic syndrome. This result is also similar to the result of Zhao et al., (2004), who demonstrated an 18% decrease in blood TG levels when patients ingested 17.5 g of ALA/day over a six-week intervention. Total cholesterol levels consist of both HDL and LDL cholesterol, providing an overall assessment of cholesterol metabolism and cardiovascular The present study demonstrated that MSG administration resulted in an increase in TCHOL levels compared to the control group. However, supplementation with flaxseed resulted in significant (P < 0.05) reductions in TCHOL levels, with higher concentrations leading to further decreases. The cholesterol-lowering effects of flaxseed consumption have been documented in previous research. Rodriguez-Leyva et al. (2013) reported that flaxseed and its omega-3 fatty acid content, alpha-linolenic acid, have cardiovascular benefits, including reductions in total cholesterol levels. Patadeet al. (2008) also reported that mild to moderate hypercholesterolemic Native American postmenopausal women who consumed flaxseed (30 g/day) for three months exhibited a 7% reduction in TCHOL and a 10% reduction in LDL cholesterol, with minimal changes in HDL cholesterol and TG concentrations. HDL cholesterol is often referred to as "good" cholesterol because it helps remove LDL cholesterol from the arteries, reducing the risk of cardiovascular disease. This study found that MSG administration resulted in a reduction in HDL levels compared to the control group. However, supplementation with chia seed showed a significant (P<0.05) increase in HDL levels, with a further increase noted in the combination group receiving both chia and flax seeds. Previous research has highlighted the cardioprotective properties of chia seed consumption. Oliveiraet al., (2018) demonstrated that chia seed supplementation improved HDL cholesterol levels in patients with type 2 diabetes. LDL cholesterol is often referred to as "bad" cholesterol because high levels are associated with an increased risk of cardiovascular disease. An elevated concentration of plasma lipoprotein is a risk factor for CAD, cerebrovascular disease, atherosclerosis, thrombosis, and stroke (Wilde, 2003). This study revealed that flaxseed exhibited a more pronounced cholesterol-lowering effect than chia seed, as indicated by a significant reduction in LDL levels. The combination of chia seed and flax seed showed a further reduction in LDL levels, suggesting potential synergistic effects. The result agrees with previous studies, which reported the

cholesterol-lowering effects of flax seed consumption, supporting the findings of this study (Rodriguez-Leyva et al., 2013).

Elevation of serum liver enzymes (ALT, AST, ALP) in MSG-treated rats indicates hepatic injury and compromised liver function (Egbuna and Ifemeje, 2017). The significant (P< 0.05) decrease in ALT, AST, and ALP levels with chia and flaxseed supplementation is in agreement with previous research indicating the hepatoprotective effects of these seeds. Previous studies by Asmaa (2021) and Mahfouz (2020) demonstrated that chia seed extract reduced ALT, AST, and ALP levels in rats with hepatic steatosis. These findings align with prior research. MarineliRdaet al. (2015) demonstrated that consumption of white chia seeds reduced liver damage by lowering AST and ALT levels. The study also noted improvements in lipid profiles and liver function following ingestion of both black and white chia seeds, attributed to their high omega-3 fatty acid content. Similarly, Fernandez-Martinez et al. (2019) linked the hypolipidemic and hepatoprotective effects of chia to its abundance of α -linolenic acid (omega-3), fiber, protein, and phenolic compounds. Additionally, Kumagai et al. (2013) demonstrated that bilirubin, a breakdown product of hemoglobin, is crucial for liver function. They found that hepatic dysfunction led to increased levels of both direct and total bilirubin, indicating impaired bilirubin clearance from the bloodstream. Furthermore, these observations align with the findings of Alamri (2019), who noted that the levels of ALT and AST were markedly elevated in both the negative and positive control groups compared to the rats that received chia seeds in their diet. These studies collectively support the notion that chia seeds possess hepatoprotective properties due to their nutritional composition, particularly their omega-3 fatty acid content. The flaxseed supplementation demonstrated a higher decrease in AST levels, which is consistent with previous studies highlighting the hepatoprotective properties of flax seeds

V. Conclusion

This study demonstrates the potential protective effects of flaxseed and chia seed supplementation against MSG-induced oxidative stress and associated biochemical alterations in Wistar rats. The observed reduction in oxidative stress markers, improvement in lipid profiles, and normalization of liver function indices suggested that flax and chia seeds, particularly when combined, can significantly mitigate the adverse effects of MSG. These findings, therefore, highlighted the importance of dietary interventions in managing oxidative stress and related metabolic disturbances.

Author Address:

^{1,2,3,7,8}Department of Biochemistry, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra State, Nigeria.

⁴Department of Medical Biochemistry, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra State, Nigeria.

⁵Department of Physiology, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra State, Nigeria.

⁶Department of Food Science and Technology, Chukwuemeka Odumegwu Ojukwu University, Igbariam Campus, Anambra State, Nigeria.

⁹Department of Electrical Electronics, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra State, Nigeria.

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