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# Impacts of *Moringa oleifera* Seed Nanoparticles on Growth, Serum Biochemistry, and Hepatic Enzymes in Diet-Induced Hyperlipidemic Male Wistar Rats

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**Abstract**

This study was conducted on male Wistar rats to examine the protective effects of *Moringa oleifera* seed nanoparticles (MOS-NPs). 120 rats were distributed into five groups: normal control (NC) fed a standard diet, high-fat diet control (HC), and HC groups receiving MOS-NPs at 25 (MOS-NP-L), 100 (MOS-NP-M), and 200 (MOS-NP-H) mg $kg^{-1}$ BW/day for 60 days. The HC group showed significantly elevated body weight gain (92.3 $\pm$ 3.3g), epididymal fat percentage (8.12 $\pm$ 0.28%), liver weight (4.77 $\pm$ 0.17%), and spleen index (0.45 $\pm$ 0.04%) compared to the NC group (56.1 $\pm$ 2.4 g, 4.12 $\pm$ 0.15%, 3.32 $\pm$ 0.12%, 0.24 $\pm$ 0.02%). The Serum analysis described increases significantly in glucose, creatinine, urea, ALT, AST, and ALP, along with reduced total protein and albumin in the HC group versus the NC. MOS-NP dose-dependently reversed these variations. The MOS-NP-H group demonstrated the most potent effect, restoring all parameters to levels not significantly different from the NC group, with body weight gain at 60.4 $\pm$ 2.4 g ($p = 0.008$), epididymal fat at 3.36 $\pm$ 0.12% ($p = 0.008$), ALT at 42.1 $\pm$ 2.4 U $L^{-1}$ (p = 0.007), AST at 86.4 $\pm$ 3.4 U $L^{-1}$ ($p = 0.007$), and creatinine at 0.79 $\pm$ 0.03 mg $dL^{-1}$ ($p = 0.007$). In conclusion, MOS-NPs at 200 mg $kg^{-1}$, as a natural therapeutic agent, significantly enhanced organ dysfunction.

## 1. Introduction:

Hyperlipidemia is categorized by raised levels of cholesterol, low-density lipoprotein, abnormal serum lipids, including total and triglycerides that also recognized as the main risk factor of metabolic and vascular diseases [1]. It increased worldwide mainly due to the high intake of saturated fats, refined carbohydrates, which are significantly involved in the prevalence of diseases [2]. It also tempts systemic oxidative stress, atherosclerosis, chronic inflammation, and coronary artery disease. Furthermore, it is described as a metabolic condition that damages in the internal body organs such as the liver and kidneys [3]. In the rodent models of hyperlipidemia widely investigate the metabolic disturbances, organ indices, and metabolic disorders furthermore it also assesses

the nutritional balance and metabolic health with the dietary interventions [4, 5].

The physiological response to dietary treatments in hyperlipidemic states with extreme caloric intake distresses fat deposition and abnormal weight gain predominantly in visceral adipose tissue, such as epididymal fat, which is powerfully associated with cardiovascular risks and metabolic syndrome [6]. Similarly, organ catalogues, with liver and spleen weight relative to body mass, serve as markers of lipid assembly, immune function, and systemic inflammation [7]. Metabolic excess imitates steatosis and designates chronic inflammation and immune dysregulation [8]. The biomarkers, such as albumin and total protein, are commonly used biomarkers that play a critical role in transporting metabolites and keeping oncotic pressure, and are used to demonstrate the nutritional adequacy, liver synthetic function, and metabolic status [9]. The low level of serum albumin and total protein caused malnutrition, hepatic dysfunction, and chronic inflammation. In contrast, the regularization of these biomarkers is a gauge of enhanced metabolic parameters [10].

Serum glucose helps reduce the risk of metabolic syndrome, restore carbohydrate metabolism, and assess the effectiveness of dietary interventions or bioactive compounds. hyperglycemia is a metabolic disorder that occurs due to reduced insulin sensitivity and extreme hepatic glucose output [11]. The liver plays a vital role in lipid metabolism with the storage, synthesis, and catabolism of lipids [12]. Under hyperlipidemic situations, extreme lipid growth causes nonalcoholic fatty liver disease that can lead to steatohepatitis, cirrhosis, and fibrosis [13]. The Serum transaminases, such as ALT and AST, together with alkaline phosphatase (ALP), are biochemical markers of biliary function and hepatocellular integrity. Still, elevated levels of these enzymes can cause metabolic imbalance, hepatic dysfunction, and hepatocyte injury [14]. Serum creatinine is a marker of glomerular filtration efficiency, urea imitates renal excretory capacity, and protein catabolism. Moreover, elevated serum creatinine and urea levels indicate renal damage, nephrotoxicity, and systemic metabolic excess [15].

*Moringa oleifera*, as a medicinal plant, gained increasing attention with broad-spectrum pharmacological activities [16]. It has great anti-inflammatory and lipid-lowering properties due to its rich in polyphenols, isothiocyanates, essential amino acids, and flavonoids [17]. Moringa seed extracts are reported to improve serum lipid profiles and restore antioxidant defense [18]. Nanotechnology enhanced the potential of natural bioactive compounds by increasing solubility, consistency, and bioavailability, thereby improving cellular uptake and biological efficacy [19].

*M. oleifera* seed nanoparticles (MOS-NPs) are considered a safe, natural, and effective substitute to conservative drugs, which are frequently related to adverse side effects and described as a new approach to fight hyperlipidemia-induced immunological and biochemical disorders [20]. Therefore, the present study was undertaken to assess the protective properties of *M. oleifera* seed nanoparticles in diet-induced hyperlipidemic Wistar rats.

## 2. Materials and Methods:

### 2.1 Study Site and Ethical Approval:

The present study was conducted at the Department of Zoology, Government College University Lahore, under precise environmental conditions. All measures involving animals complied with the rules and were accepted by the Institutional Animal Ethics Committee of the University Lahore (ZDLU Code, 2943).

### 2.2 Characterization of MOS-NPs:

In this study, *Moringa oleifera* seed nanoparticles were plant-derived organic nanoparticles and synthesized via an aqueous green method. Briefly, finely crushed seeds were exposed to aqueous extraction and controlled processing to produce nanoscale phytochemical assemblies. The subsequent nanoparticles were within the nanometer range (¡200 nm) and experienced physicochemical depiction. Morphology was assessed by using scanning electron microscopy (SEM), crystalline features were examined by X-ray diffraction (XRD), functional groups were recognized by Fourier transform infrared spectroscopy (FTIR), elemental composition was examined by using energy-dispersive X-ray spectroscopy (EDS), and nanoparticle formation and optical behavior were evaluated by UV–visible spectroscopy [19].

### 2.3 Experimental Animals and Housing:

Wistar rats (Rattus norvegicus) were obtained from the Department of Zoology, Government College University Lahore. 120 male R. norvegicus weighing 150–200 g and aged 8–10 weeks were taken, acclimatized, and maintained for one week before the experiment under standard laboratory conditions such as $22 \pm 2$ °C, 50–60% relative humidity, 12 h light/dark cycle. Clean drinking water and feed were given throughout the experiment, except for the fasting period before sampling. Polypropylene cages with sterile paddy husk bedding were provided for housing, which was altered twice weekly to keep hygienic conditions [21].

### 2.4 Experimental Design and Diet Formulation:

Five experimental groups were designed, and a total of 120 rats were randomly distributed into three replicates of each group (n = 8 rats per replicate) for a 60-day study. The first group received a standard laboratory chow with 0.5% carboxymethyl cellulose (CMC) as a vehicle and was recognized as the normal control (NC) group. The second group was recognized as a high-fat control (HC) group that received a high-fat diet (HFD) to induce hyperlipidemia, along with 0.5% CMC (Carboxymethyl Cellulose). The third group with HFD

supplemented with *M. oleifera* seed nanoparticles was a low dose MOS-NP-L (25 mg $kg^{-1}$ body weight/day). The fourth group, HFD supplemented with *M. oleifera* seed nanoparticles, was designated the medium-dose MOS-NP-M (100 mg $kg^{-1}$ body weight/day). The fifth group HFD supplemented with *M. oleifera* seed nanoparticles was a high dose MOS-NP-H (200 mg $kg^{-1}$ body weight/day). Freshly prepared MOS-NPs were suspended in 0.5% CMC each day and administered orally by gavage at 10 mL/kg body weight to ensure precise dosing. The HFD was prepared following the composition described by [22], containing 20% protein, 35 carbohydrates, and 45% fat. The Diets were stored in airtight containers at 4 °C. The configuration of the studied diet presented in Table 2.4.

### 2.5 Growth Parameters Evaluation and Sample Collection:

All the animals were subjected to a fast of about 12 h at the end of the sixty-day study period. The body weight was measured by an electronic balance. Daily Food intake was recorded by subtracting the residual chow from the given food. The rats were anesthetized by intraperitoneal injection (ketamine, 80 mg $kg^{-1}$ body weight, and xylazine, 10 mg $kg^{-1}$ body weight) followed by sacrifice via cardiac puncture. Blood samples were centrifuged at 3000 rpm for 15 min at 4 °C. The separated serum was aliquoted and kept at 80 °C till biochemical studies [23]. The following formulas were used to evaluate growth parameters.

Body weight gain (g) =Final body weight (g)Initial body weight (g)

Food intake (g/day) = Total food consumed (g)/Number of days

Epididymal fat index (% bw) = Epididymal fat weight (g)/Final body weight (g)×100

Liver index (% bw) =Liver weight (g)/Final body weight (g)×100

Spleen index (% bw) = Spleen weight (g)/Final body weight (g)×100

### 2.6 Biochemical Assays:

Serum biochemical parameters were evaluated using standard diagnostic kits (Randox Laboratories Ltd., UK). Albumin and Total protein were measured by the bromocresol green and biuret methods, respectively, both widely used to assess metabolic and nutritional status in rodent studies [24, 25]. The reliable assay for carbohydrate metabolism, the glucose oxidase–peroxidase (GOD–POD) method described by [26] was used, and Serum glucose was quantified. Liver function enzymes, including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), were estimated by the Reitman and Frankel (1957) [27] method, while alkaline phosphatase (ALP) activity was determined following [28]. The Renal function was assessed by recording the creatinine using the Jaffé reaction [29] and urea using the diacetyl monoxime method [30].

### 2.7 Statistical Analysis:

One-way analysis of variance (ANOVA) was executed by using SPSS software (version 26.0) followed by Tukey's HSD post hoc test, to identify significant differences between the groups.

## 3. Results:

Table 2 reveals the effects of MOS-NPs on the growth and relative organ. The NC group showed the lowest values for all parameters. Body weight gain was 56.1 ± 2.4 g, food intake 19.4 ± 0.7 g/day, epididymal fat 4.12 ± 0.15%, liver weight 3.32 ± 0.12%, and spleen index 0.24 ± 0.02% (p = 0.007). The HC group displayed significantly higher levels than the NC group. Administration of low-dose MOS-NPs-L (25 mg $kg^{-1}$) partially reduced these elevations, with body weight gain 84.4 ± 2.3 g, food intake 22.0 ± 0.6 g/day, epididymal fat 6.02 ± 0.21%, liver weight 4.38 ± 0.11%, and spleen index 0.38 ± 0.07% (p = 0.008).

Although improved relative to HC, these values remained higher than NC. The MOS-NPs- M (100 mg $kg^{-1}$) indicated the enhancements. Body weight gain decreased to 74.3 ± 2.3 g, food intake to 19.6 ± 0.7 g/day, epididymal fat to 4.42 ± 0.23%, liver weight to 3.74 ± 0.11%, and spleen index to 0.31 ± 0.04% (p = 0.007). The MOS-NPs-H (200 mg $kg^{-1}$) demonstrated the strongest protective effect. Body weight gain was 60.4 ± 2.4 g, food intake 19.2 ± 0.9 g/day, epididymal fat 3.36 ± 0.12%, liver weight 3.32 ± 0.14%, and spleen index 0.28 ± 0.07% (p = 0.008). These values were not significantly different from NC but were markedly lower than HC.

The NC group recorded baseline values, including total protein (6.6 ± 0.4 g $dL^{-1}$ ), albumin (4.4 ± 0.3 g $dL^{-1}$), glucose (92.4 ± 3.5 mg $dL^{-1}$), creatinine (0.68 ± 0.03 mg $dL^{-1}$), and urea (32.3 ± 1.4 mg $dL^{-1}$) (p = 0.006). In contrast, the HC group exhibited significant alterations, with decreased total protein (5.3 ± 0.6 g $dL^{-1}$) and albumin (3.2 ± 0.4 g $dL^{-1}$), while glucose (152.4 ± 5.6 mg $dL^{-1}$), creatinine (1.32 ± 0.06 mg $dL^{-1}$), and urea (58.1 ± 2.4 mg $dL^{-1}$) were markedly elevated compared with NC (p = 0.007). Administration of low-dose MOS-NPs-L partially improved these parameters. The glucose, creatinine, and urea levels were decreased (p = 0.006) and The Total protein was increased to 5.8 ± 0.4 g $dL^{-1}$ and albumin was recorded 3.6 ± 0.3 g $dL^{-1}$, respectively.

The MOS-NPs-M recorded improvement, with total protein and albumin values at NC levels, while glucose, creati-

**Table 1.** Composition of Experimental Diets.

| Diet Group | Fat(%) | Protein(%) | Carbohydrate(%) | Additives |
|---|---|---|---|---|
| Normal Control (NC) | 10 | 20 | 70 | 0.5% CMC |
| High-Fat Control (HC) | 45 | 20 | 35 | 0.5% CMC |
| MOS-NP-L | 45 | 20 | 35 | MOS-NPs 25 mg $kg^{-1}$ BW/day + 0.5% CMC |
| MOS-NP-M | 45 | 20 | 35 | MOS-NPs 100 mg $kg^{-1}$ BW/day + 0.5% CMC |
| MOS-NP-H | 45 | 20 | 35 | MOS-NPs 200 mg $kg^{-1}$BW/day + 0.5% CMC |

**Table 2.** Effects of *M. oleifera* Seed Nanoparticles on Growth Performance, Organ Indices, and Food Intake in Diet-Induced Hyperlipidemic Wistar Rats

| Group | Body weight gain (g) | Food intake (g/day) | Epididymal fat (% BW) | Liver (% BW) | Spleen index (% BW) | p-value |
|---|---|---|---|---|---|---|
| NC | 56.1±2.4$^{a}$ | 19.4±0.7$^{a}$ | 4.12±0.15$^{a}$ | 3.32±0.12$^{a}$ | 0.24±0.02$^{a}$ | 0.007 |
| HC | 92.3± 3.3$^{c}$ | 22.4± 1.4$^{c}$ | 8.12± 0.28$^{c}$ | 4.77±0.17 | 0.45±0.04$^{c}$ | 0.006 |
| MOS-NP-L (25) | 84.4± 2.3$^{b}$ | 22.0±0.6$^{b}$ | 6.02±0.21$^{b}$ | 4.38±0.11$^{b}$ | 0.38±0.07$^{b}$ | 0.008 |
| MOS-NP-M (100) | 74.3±2.3$^{b}$ | 19.6± 0.7$^{b}$ | 4.42± 0.23$^{b}$ | 3.74± 0.11$^{b}$ | 0.31± 0.04$^{b}$ | 0.007 |
| MOS-NP-H (200) | 60.4±2.4$^{a\,b}$ | 19.2±0.9$^{a\,b}$ | 3.36±0.12$^{a\,b}$ | 3.32±0.14$^{a\,b}$ | 0.28±0.07$^{a\,b}$ | 0.008 |

nine, and urea were significantly lower as compared to HC (p = 0.008). The MOS-NPs- H displayed total protein (6.9 ± 0.4 g $dL^{-1}$) and albumin (4.2 ± 0.4 g $dL^{-1}$) close to NC levels with the protective effect. Glucose (99.4 ± 3.8 mg $dL^{-1}$), creatinine (0.79 ± 0.03 mg $dL^{-1}$), and urea (34.7 ± 1.5 mg $dL^{-1}$) were recorded and found not significant compared to NC, but reduced greatly compared to HC (p = 0.007) as shown in Table 3.

Table 4 describes the MOS-NPs on hepatic enzyme activities in hyperlipidemic Wistar rats. In the NC group, baseline values were observed with ALT (37.4 ± 1.8 U $L^{-1}$), AST (74.5 ± 4.4 U $L^{-1}$), and ALP (132.2 ± 4.3 U $L^{-1}$) (p = 0.005). However, the HC group displayed higher ALT, AST, and ALP with hepatic dysfunction as compared with NC group significantly (p = 0.007). The low-dose MOS-NPs-L decreased these elevations and recorded significant (p = 0.005) improvements to HC but persisted higher than NC. The MOS-NPs-M reveals more pronounced protective effects, with ALT (55.6 ± 2.4 U $L^{-1}$), AST (112.4 ± 4.6 U $L^{-1}$), and ALP (168.6 ± 6.5 U $L^{-1}$), all significantly reduced compared with HC (p = 0.006). The MOS-NPs-H (200 mg $kg^{-1}$) described a considerable hepatoprotective outcome, with enzyme activities to normal values. These parameters are different from NC and recorded not significant (p = 0.007) but lower than HC.

## 4. Discussion:

The present results provide a comprehensive insight into the role of MOS-NPs in modifying diet-induced metabolic stress. High-fat diet feeding distinctly improved epididymal fat growth, food intake, body weight gain, and relative liver and spleen indices compared with the normal control group.

**Table 3.** *M. oleifera* Seed Nanoparticles Impact on Serum Biochemical Parameters in Hyperlipidemic Male Wistar Rats.

| Group | Total protein (g $dL^{-1}$) | Albumin (g $dL^{-1}$) | Glucose (mg $dL^{-1}$) | Creatinine (mg $dL^{-1}$) | Urea (mg $dL^{-1}$) | p-value |
|---|---|---|---|---|---|---|
| NC | 6.6± 0.4[a] | 4.4± 0.3[a] | 92.4± 3.5[a] | 0.68± 0.03[a] | 32.3 ± 1.4[a] | 0.006 |
| HC | 5.3± 0.6[c] | 3.2± 0.4[c] | 152.4± 5.6[c] | 1.32± 0.06[c] | 58.1± 2.4[c] | 0.007 |
| MOS-NP-L | 5.8± 0.4[b] | 3.6± 0.3[b] | 136.5± 5.2[b] | 1.14± 0.04[b] | 47.4± 2.7[b] | 0.006 |
| MOS-NP-M | 6.5± 0.5[b] | 3.8± 0.4[b] | 116.5± 4.1[b] | 0.94 ± 0.07[b] | 42.4± 1.6[b] | 0.008 |
| MOS-NP-H | 6.9 ± 0.4 [a b] | 4.2± 0.4 [a b] | 99n.4 ± 3.8 [a b] | 0.79± 0.03 34.7± 1.5 [a b] | 0.007 | |

**Table 4.** The relation between thyroid hormones with sex for obese participants.

| Group | ALT (U/L) | AST (U/L) | ALP (U/L) | p-value |
|---|---|---|---|---|
| NC | 37.4± 1.8[a] | 74.5± 4.4[a] | 132.2 ± 4.3[a] | 0.005 |
| HC | 87.3± 4.3[c] | 157.4± 3.1[c] | 234.2± 9.4[c] | 0.007 |
| MOS-NP-L | 74.1± 3.4[b] | 132.2± 4.2[b] | 196.4± 7.4[b] | 0.005 |
| MOS-NP-M | 55.6± 2.4[b] | 112.4± 4.6[b] | 168.6± 6.5[b] | 0.006 |
| MOS-NP-H | 42.1± 2.4 [a b] | 86.4± 3.4 [a b] | 135.3± 5.6 [a b] | 0.007 |

This outcome is consistent with previous studies showing that chronic exposure to fat-enriched diets brings ectopic fat deposition and obesity in rodent models [31, 32].

Another study [33] found that increased adiposity is the hallmark of hyperlipidemia, which causes systemic inflammation, leading to metabolic dysregulation. MOS-NPs at medium and high doses reduced epididymal fat index and organ weights, indicating diminished fat accumulation and improved lipid metabolism, consistent with previous findings that *M. oleifera* extracts suppress adipogenesis and increase lipid clearance [34, 35]. studied that the spleen indices normalization specifies immunomodulatory effects, heightened systemic inflammation, and is described as splenic hypertrophy. In this study, albumin and serum protein levels in hyperlipidemic control rats were significantly reduced, accompanied by systemic swelling and reduced hepatic protein synthesis. Similar reductions were documented in non-alcoholic fatty liver disease and obesity-linked hepatopathy [24]. It was reported by [26] that the elevated serum glucose levels in high-fat diet-fed rats are an indicator of diet-induced insulin resistance and altered glucose homeostasis.

MOS-NPs supplementation reduces the glucose levels, with the maximum dose reestablishing near-normal values. This is consistent with previous studies showing that *M. oleifera* improves insulin secretion and insulin sensitivity and decreases fasting glucose. [36, 37]. [38] Recorded the hypoglycemic activity of *M. oleifera*, which controls carbohydrate metabolism by increasing glucose uptake and stopping gluconeogenesis. Creatinine and urea levels were significantly raised in hyperlipidemic controls, demonstrating nephrotoxic change and impaired renal clearance. Similar changes were reported in rodents fed high-fat diets, in which lipid accumulation in the kidney leads to decreased glomerular filtration rate, glomerulosclerosis, and oxidative stress [39, 40].

These results also agree with previous studies reporting that *M. oleifera* seed and leaf extracts restore normal purification markers, lessening inflammatory cytokines in renal tissue, and improve renal oxidative injury[41, 42]. The present findings confirm hepatic dysfunction and biliary stress due to significantly raised ALT, AST, and ALP in hyperlipidemic controls, consistent with earlier findings [43, 44]. described that ALT and AST are delicate markers of hepatocyte membrane injury, while higher ALP designates altered biliary flow and cholestasis. MOS-NPs-H restored values close to those of the normal control, but significantly decreased these enzymes in a dose-dependent manner. These results align with earlier studies showing that *M. oleifera* extracts prevent the decrease in oxidative stress, hepatic lipid accumulation, and protect hepatocyte membranes from peroxidative damage [18, 45, 46].

Described the greater effectiveness of MOS-NPs over traditional extracts in restoring liver function and studied nanotechnology-based delivery systems to advance the cellular uptake of plant-derived compounds, solubility, and bioavailability. Furthermore, the novel nanoparticle formulation overcomes the limitations of poor bioavailability associated with conventional plant extracts. This study provides a strong indication that MOS-NPs represent a safe, natural, and effective approach for stopping hyperlipidemia-associated metabolic imbalances.

## 5. Conclusion:

The findings of this study demonstrate that *M. oleifera* seed nanoparticles (MOS-NPs) significantly impair high-fat diet-induced hyperlipidemia in Wistar rats. Administration of MOS-NPs, particularly at 200 mg/kg, effectively normalized body weight, reduced epididymal fat accumulation, restored serum biochemical parameters, and improved liver and kidney function markers.

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**Declarations:**

**Conflict of interest:** The author confirms that all illustrations, and tables, in this document are our own original work.

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**Author contributions:** All authors contributed significantly to this work. M.N., performed the experimental work and material collection, while H.A.M., conducted the data analysis. The manuscript was written by M.N., H.A.M., and A.J.A., and B.S.A. revised and finalized the manuscript. All authors reviewed and approved the final version.

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# تأثيرات جسيمات النانوية لبذور مورينغا أوليفيرا على النمو، معايير الكيمياء الحيوية ، وإنزيمات الكبد لمصل الدم في ذكور جرذان المصابة بفرط شحميات الدم الناتج عن النظام الغذائي

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الخلاصة

أُجريت هذه الدراسة على ذكور الجرذان لتقييم التأثيرات الوقائية للجزيئات النانوية لبذور مورينغا أوليفيرا ($MOS-NPs$) . تم تقسيم 120 جرذاً على خمس مجموعات: مجموعة الضابطة الطبيعية ($NC$) مع نظام غذائي قياسي، ومجموعة ضابطة تتغذى على نظام غذائي عالي الدهون ($HC$) ، ومجموعات $HC$ التي تلقت جسيمات النانوية من نوع $MOS-NPs$ بجرعات 25 ($MOS-NP-L$) ، و 100 ($MOS-NP-M$) ، و 200 ($MOS-NP-H$) ملغم من وزن الجسم لمدة 60 يوماً. أظهرت مجموعة $HC$ زيادة ملحوظة في وزن الجسم ( $92.3\pm3.3$ غرام)، وكانت نسبة الدهون في البربخ ( $8.12\pm0.28\%$ )، ووزن الكبد ( $4.77\pm0.17\%$ )، ومؤشر الطحال ( $0.45\pm0.04\%$ ) مقارنةً بمجموعة $NC$ ( $56.1\pm2.4$ غرام، $4.12\pm0.15\%$ ، $3.32\pm0.12\%$ ، $0.24\pm0.02\%$ ). أظهر تحليل مصل الدم ارتفاعاً ملحوظاً في مستويات الجلوكوز، والكرياتينين، واليوريا، وإنزيم ناقلة أمين الألانين ($ALT$) ، وإنزيم ناقلة أمين الأسبارتات ($AST$) ، وإنزيم الفوسفاتاز القلوي ($ALP$) ، بالإضافة إلى انخفاض في مستويات البروتين الكلي والألبومين في مجموعة $HC$ مقارنةً بمجموعة $NC$ . وقد عكست جزيئات $MOS-NP$ هذه التغيرات بشكلٍ متناسب مع الجرعة. وأظهرت مجموعة $MOS-NP-H$ التأثير الأقوى، حيث رجعت جميع المؤشرات إلى مستويات لا تختلف بشكلٍ ملحوظ عن مجموعة $NC$ ، مع زيادة في وزن الجسم بلغت $60.4\pm2.4$ غرام ( $p=0.008$ )، ونسبة دهون البربخ $3.36\pm0.12\%$ ( $p=0.008$ )، وإنزيم ($ALT$) $42.1\pm2.4$ وحدة لتر $^{-1}$ ( $p=0.007$ )، وإنزيم ($AST$) $86.4\pm3.4$ وحدة لتر $^{-1}$ ( $p=0.007$ )، والكرياتينين $0.79\pm0.03$ ملغ ( $p=0.007$ ) .

**الكلمات الدالة:** المورينغا أوليفيرا، أداء النمو، الكيمياء الحيوية في مصل الدم، إنزيمات الكبد، جرذان ويستر.

**التمويل:** لم يتلق المؤلفون أي تمويل محدد لهذا المشروع من أي جهة عامة أو تجارية.

**بيان توفر البيانات:** البيانات التي تدعم نتائج هذه الدراسة متاحة من المؤلف المسؤول.

**اقرارات: تضارب المصالح:** يؤكد المؤلف أن جميع الرسوم التوضيحية والجداول الواردة في هذه الوثيقة هي من عملنا الأصلي.

**الموافقة الأخلاقية:** تمت الموافقة علتم دعم البيان الأخلاقي لهذا البحث من قبل قسم علم الحيوان، جامعة كلية الحكومة لاهور، باكستان (الرمز: 2024 : 00963 : *DZGCUL* ).

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