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Research Article

Comparative Effects of Vitamin D3 and Sunlight on Ameliorating High-Fat Diet–Induced Obesity in Mice

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Vitamin D deficiency is linked to various chronic diseases, including obesity, insulin resistance, hyperlipidemia, liver disease, and hypertension. This study investigates the comparative effects of vitamin D3 supplementation and sunlight exposure on high-fat diet–induced obesity in albino mice. After 16 weeks on a butter-rich diet, obese mice gained 15 ± 2 g more than controls (T_0). The obese cohort was then subdivided into four groups (8 mice each): untreated obese controls (T_1), sunlight exposure (T_2), vitamin D_3 supplementation (T_3), and combined sunlight plus vitamin D_3 supplementation (T_4). After 30 days, the obese control group exhibited significant weight gain with elevated glucose, cholesterol, triglycerides, LDL, and liver enzymes, alongside reduced HDL levels ($p < 0.001$). In contrast, all treatment groups, particularly those receiving combined sunlight and vitamin D_3, showed marked improvements in these parameters, including significant reductions in metabolic markers and liver enzymes ($p < 0.001$). Serum 25 (OH) D levels remained stable in normal controls (30–32 ng/mL), declined in obese controls (15–18 ng/mL), increased moderately with sunlight (33–35 ng/mL) or synthetic vitamin D_3 (32–34 ng/mL), and reached the highest values under combined treatment (37–39 ng/mL). Histological analysis revealed fat accumulation in the liver and kidneys, as well as adipocyte infiltration in the liver and heart of obese controls, changes not observed in treated groups. Collectively, these results indicate that improving vitamin D status via sunlight exposure or supplementation may offer a simple and effective approach to alleviating obesity-related metabolic dysfunction.

Keywords: cholesterol; hyperlipidemia; hypertension; insulin resistance; triglyceride

1. Introduction

Obesity is characterized by the abnormal or excessive accumulation of adipose tissue resulting from an imbalance between energy intake and expenditure. It is strongly associated with numerous adverse health outcomes, with excess adiposity remaining a key driver of heart failure and kidney disease in Type 2 diabetes, underscoring the need for earlier weight-focused interventions [1]. Globally, the prevalence of obesity has risen at an alarming rate. Recent estimates suggest that more than 2 billion adults and 380 million children and adolescents are overweight or obese, with the fastest growth occurring in low- and middle-income countries [2]. According to the World Health Organization [3], more than 890 million adults and 160 million children and adolescents were living with obesity, highlighting its urgent status as a global public health challenge.

Vitamin D, traditionally recognized for its role in calcium-phosphate homeostasis and skeletal health, has also been implicated in several extraskeletal functions. It modulates adipocyte biology, insulin sensitivity, and glucose metabolism, processes that are often dysregulated in obesity [4]. Notably, vitamin D deficiency, commonly observed in obese individuals, has been associated with impaired adipocyte and glucose metabolism and inversely correlated with the incidence of multiple cancers, including breast, colorectal, lymphoma, lung, and prostate, underscoring its potential as a therapeutic target in obesity-related carcinogenesis [5].

Two main forms of vitamin D exist: vitamin D2 (ergocalciferol), derived from plants and fungi, and vitamin D_3 (cholecalciferol), synthesized in the skin or obtained from animal-based foods. Upon UVB exposure, 7-dehydrocholesterol in the skin is converted to previtamin D_3, which is then thermally isomerized to vitamin D_3 and transported in circulation bound to vitamin D-binding protein (VDBP) [6]. In obesity, reduced circulating vitamin D, resulting from adipose tissue sequestration and altered metabolism, impairs adipose stem cell differentiation and overall adipose tissue function by disrupting vitamin D–vitamin D receptor (VDR) signaling, thereby potentially increasing the risk of cardiometabolic disorders [7].

High-fat diets, particularly those deficient in choline and methionine, have been shown to induce obesity, hypercholesterolemia, hypertriglyceridemia, and fatty liver in rodent models [8]. Low plasma levels of vitamin D typically result in decreased calcium levels and poor intestinal absorption, which subsequently lead to increased bone turnover and reduced bone mineral density (BMD). Vitamin D deficiency is further associated with rickets, osteomalacia, osteoporosis, cardiovascular diseases, diabetes, malignancies, autoimmune and allergic diseases, chronic kidney disease, hepatic failure, malabsorption syndromes, hyperparathyroidism, granulomatous disorders, and certain lymphomas [9].

Mounting evidence highlights obesity and vitamin D deficiency as interrelated pandemics with substantial public health implications. Several studies have demonstrated an inverse relationship between body mass index and circulating 25-hydroxyvitamin D [25(OH)D] concentrations [6]. Epidemiological data further indicate that vitamin D deficiency, defined by low circulating 25(OH)D levels, is particularly prevalent in obese populations, driven by factors such as reduced sun exposure, lower dietary intake, impaired intestinal absorption, and sequestration of vitamin D in adipose tissue. Although high-dose supplementation with cholecalciferol or calcifediol can restore serum 25(OH)D levels, its effects on obesity and associated metabolic outcomes remain inconsistent [10].

Despite growing recognition of the relationship between vitamin D obesity, the precise mechanisms underlying this association remain poorly understood. Key findings indicate that vitamin D deficiency is widespread across diverse populations and may contribute to obesity through disrupted vitamin D metabolism, sequestration within adipose tissue, and regulation of adipocyte differentiation, apoptosis, and energy homeostasis, although a definitive causal link has yet to be established [11]. Current literature also lacks comprehensive meta-analyses directly comparing the metabolic effects of sunlight-mediated vitamin D synthesis and supplementation, [12].

The present study was therefore designed to evaluate and compare the effects of sunlight exposure, vitamin D3 supplementation, and their combination on obesity-related biochemical, metabolic, and histological changes in high-fat diet–induced obese mice. By addressing this knowledge gap, the study provides novel insights into the role of vitamin D pathways in obesity and its related complications.

2. Materials and Methods

2.1. Ethical Statement. All procedures were conducted in accordance with institutional guidelines at Khulna Agricultural University (KAU), Bangladesh, between January and May 2024. Experimental protocols were reviewed and approved by the Animal Experimentation Ethics Committee, Faculty of Veterinary, Animal, and Biomedical Sciences, KAU (AEEC/KAU/2024-1006).

2.2. Experimental Animals. Forty male Swiss Albino Mice (*Mus musculus*), aged 30–35 days and weighing of 27 ± 1 g, were obtained from the International Center for Diarrheal Disease Research, Bangladesh (icddr'b). Mice were acclimatized for 14 days under controlled conditions (12 h light/dark cycle, 31 ± 2°C, 70%–80% relative humidity) with free access to commercial pellets and drinking water. Animals were housed in polypropylene cages maintained under standard hygienic conditions.

2.3. Experimental Designs. Mice were initially divided into two groups (Figure 1):

- Control (T0): Eight mice fed standard diet and water *ad libitum*.

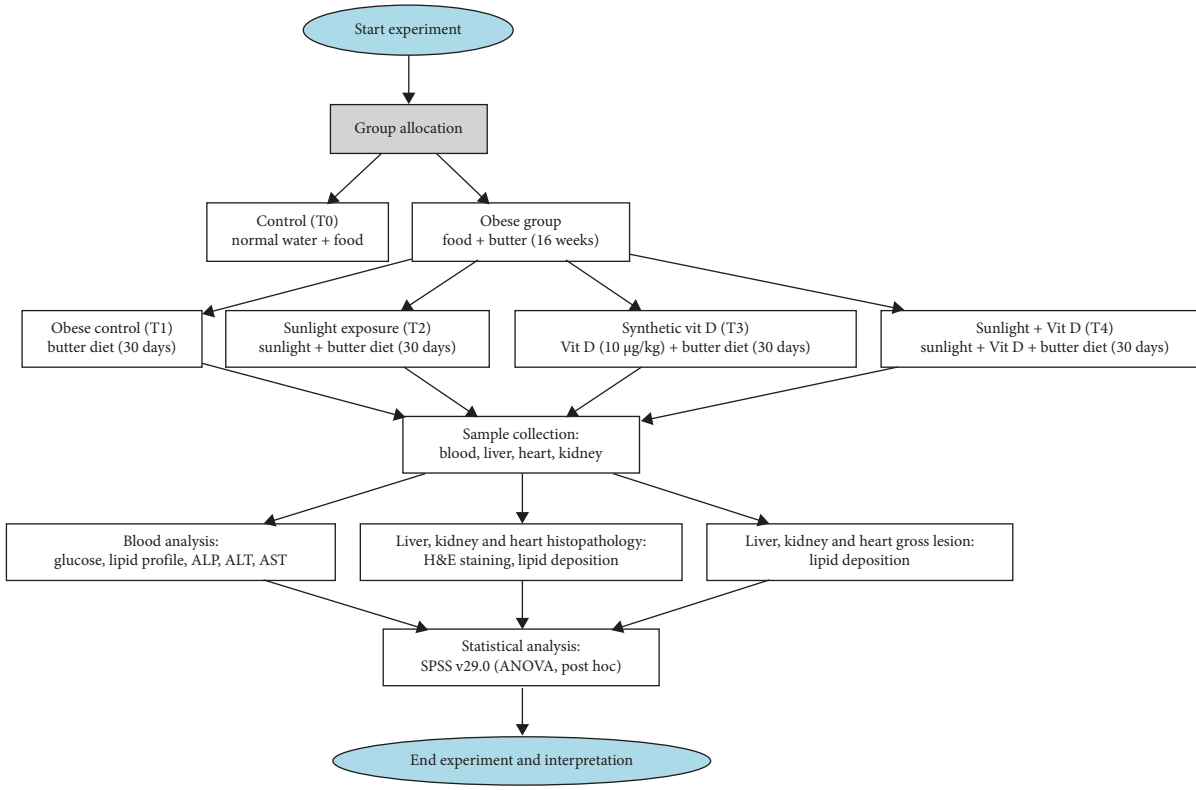


Figure 1: Methodology of the experiment.

- Obese group: Thirty-two mice fed a high-fat diet formulated by mixing 7 g butter (≈50% calories from fat; PRAN Company Ltd., Dhaka, Bangladesh) with 15 g pellets per 100 g feed. Butter provided 720 kcal/100 g, primarily from milk cream and salt. This diet was prepared based on previous reports indicating that > 30% energy from fat induces obesity in mice [13]. Composition of diets provided to experimental groups is shown in Table 1.

After 16 weeks of feeding, the obese group exhibited a body weight increase of 15 ± 2 g compared with controls. These mice were then subdivided ($n = 8$/group): control obese (T1), sunlight exposure (T2), synthetic vitamin D3 exposure (T3), and both sunlight and synthetic vitamin D3 (T4) exposure.

- Control obese (T1): Continued high-fat diet and water *ad libitum*.
- Sunlight exposure (T2): Daily 20 min of sunlight exposure after 12 p.m. plus high-fat diet and water ad libitum. Exposure time was based on recommendations that 10–30 min of unprotected sun exposure can stimulate dermal vitamin D synthesis [14, 15].
- Synthetic vitamin D3 (T3): Synthetic vitamin D3 (cholecalciferol BP 400 IU, D-RISE 2000 IU Solution, ACI Limited, Bangladesh) was given orally (10 μg/kg) with normal water and butter mixed feed *ad libitum* for 30 days. The dosage of vitamin D3 was determined based on previous research indicating its safety and efficacy in a high-fat diet–induced obese mice model, where similar doses improve vitamin D3 status, lipid metabolism, and anti-inflammatory effects without exerting toxicity [16, 17].
- Combined sunlight and synthetic vitamin D3 (T4): Both sunlight exposure (as in T2) and vitamin D3 supplementation (as in T3).

Treatments continued for 30 days.

2.4. Collection of Blood and Tissue. At the end of the experiment, mice were anesthetized with diethyl ether, and blood was collected via cardiac puncture (1.5–2 mL per animal) into plane tubes. Samples were allowed to clot at room temperature for 6 h, refrigerated overnight (4°C), and centrifuged to separate serum (0.4–0.5 mL/mouse), which was stored at −20°C.

2.5. Biochemical Assays. Serum glucose, lipid profile (total serum cholesterol, triglycerides, HDL, and LDL), liver enzymes (ALT, AST, and ALP), were analyzed in Microlab diagnostic and consultation center, Khulna, using diagnostic kits from Roche Diagnostics (Germany) on a Roche Cobas c311 fully automated clinical chemistry analyzer in accordance with the manufacturer's instructions. Serum 25(OH) D concentrations were measured using a commercial ELISA kit (ImmunoDiagnostic Systems, Scottsdale, AZ, USA) following the manufacturer's instructions.

Table 1: Composition of diets provided to experimental groups.

Component	Control diet	High-fat diet (obese group)
Pellet feed	100% (15 g)	68.2% (15 g)
Butter (PRAN, Bangladesh)	—	31.8% (7 g)
Total feed	15 g	22 g
Energy from fat (%)	~10–12%	~50%
Source of fat	—	From butter
Calories per 100 g feed	~350 kcal (estimated)	~580—600 kcal (based on butter energy density: 720 kcal/100 g)

2.6. Histological Analysis. After perfusion with phosphate-buffered saline, liver, kidney, and heart were collected, fixed in 10% neutral buffered formalin, dehydrated in graded ethanol, cleared with xylene, and embedded in paraffin. Sections (4–5 μm) were sliced using a microtome and placed on clean glass slides. The slides were then stained using hematoxylin and eosin (H&E) following a standard histological staining method [18] within the Department of Physiology and the Department of Anatomy and Histology in Khulna Agricultural University.

2.7. Statistical Analysis. Data were recorded in MS Excel and analyzed in SPSS version 29.0 (IBM SPSS Statistics, Chicago, IL, USA). Statistical significance was set up at $p < 0.05$. Normality was assessed using the Shapiro–Wilk test and histogram. The univariate generalized model was built using replication as a random effect and treatment as a fixed effect. Results were expressed as least squares mean ± SEM. Multiple comparison was adjusted using Bonferroni correction. In addition, data visualization was conducted in R (version 4.4.3).

3. Result

3.1. Body Weight Gain. According to the results, mice in treatment group T_1 (obese group) gained their greatest body weight (Table 2), averaging 48 g at the end of the experiment, more than the others in treatment Group T_2 (43 g), Group T_3 (44 g), and Group T_4 (42 g). The study findings represent that after the treatment period, the average body weight gain was significantly higher ($p < 0.001$) in T_1 group (obese control), followed by T_3 (synthetic vitamin D3), T_2 (sunlight), T_4 (sunlight + synthetic vitamin D3), and T_0 (control) groups. Among the treatment groups, combined sunlight and vitamin D3 (T4) produced the highest reduction in weight gain. Based on multiple comparisons with the Bonferroni correction, values with different superscripts in a similar row are significantly similar ($p < 0.001$) in Groups T_2, T_3, and T_4 but significantly different ($p < 0.001$) in Groups T_0 and T_1. The box plot with jittered points shows the weight of different groups of mice (Figure 2).

3.2. Glucose. From the serum analysis, after the treatment period, glucose level was significantly higher in Group T_1 (11 mg/dL) (Table 2). However, in other treatment groups, the glucose levels were almost similar to the control group. From the table, the values of glucose levels were Group T_0 (8 mg/dL), Group T_2 (8 mg/dL), Group T_3 (8 mg/dL), and Group T_4 (8 mg/dL). Again, based on multiple comparisons with the Bonferroni correction, values with different superscripts in a similar row are significantly similar ($p < 0.001$) in Groups T_0, T_2, and T_3 and but significantly different ($p < 0.001$) in Group T_1. Overall, T_1 had the highest glucose levels; T_2–T_4 restored it to near control levels.

3.3. Lipid Profile. The average mean value of different blood parameters such as cholesterol, triglyceride, and LDL value was higher in T_1 group (obese control); 228 mg/dL, 209 mg/dL, and 100 mg/dL, respectively, but HDL levels are significantly lower (29 mg/dL) (Table 2). Again, cholesterol, triglyceride, and LDL values were lower in T_2 group (sunlight exposure group): 118 mg/dL, 102 mg/dL, and 69 mg/dL, respectively. Moreover, HDL level is significantly higher in Group T_2 (59 mg/dL) than in others such as Group T_0 (48 mg/dL), Group T_3 (51 mg/dL), and Group T_4 (56 mg/dL). Overall, T_2 showed the best lipid profile, especially HDL.

3.4. Liver Enzyme. The study findings showed that after the treatment period, liver enzymes (ALT, AST, and ALP) were significantly higher in T_1 group (obese control) followed by T_2 (sunlight), T_3 (synthetic vitamin D3), T_4 (sunlight + synthetic vitamin D3), and T_0 group (control). From Table 2, ALT, AST, and ALP values of group T_1 were 82 mg/dL, 197 mg/dL, and 122 mg/dL, respectively. Conversely, the lowest value of liver enzymes (ALT, AST, and ALP) was in Group T_4 (sunlight + synthetic vitamin D3): 50 mg/dL, 148 mg/dL, and 106 mg/dL, respectively. Overall, T_4 had the lowest enzyme levels, suggesting liver protection.

3.5. 25(OH)D Concentrations. The serum 25(OH)D concentrations varied markedly among experimental groups. The control group maintained stable values with means around 30–32 ng/mL, whereas the obese control group exhibited a pronounced reduction, averaging approximately 15–18 ng/mL. Supplementation with sunlight exposure elevated 25(OH)D levels to 33–35 ng/mL, while the combined treatment of sunlight and synthetic vitamin D_3 produced the highest concentrations, averaging 37–39 ng/mL. Synthetic vitamin D3 alone also increased 25(OH)D relative to controls, with mean values of 32–34 ng/mL. The violin and boxplot distributions (Figure 3) illustrate these trends,

Table 2: Univariable association of weight and different blood parameters with five separate replications ($n = 25$) under five dietary treatments.

Parameters	Mean					SE	p
	T0	T1	T2	T3	T4	M	
Weight (g)	38 ± 2.6^{a}	48 ± 2.6^{b}	43 ± 2.6^{c}	44 ± 2.6^{c}	42 ± 2.6^{c}	0.92	< 0.001
Glucose (mg/dL)	8 ± 0.5^{a}	11 ± 0.5^{b}	8 ± 0.5^{a}	8 ± 0.5^{a}	8 ± 0.5^{a}	0.19	< 0.001
Cholesterol (mg/dL)	134 ± 8.1^{a}	228 ± 8.1^{b}	118 ± 8.1^{c}	$125 \pm 8.1^{a,c}$	117 ± 8.1^{c}	2.85	< 0.001
Triglyceride (mg/dL)	111 ± 7.8^{a}	209 ± 7.8^{b}	102 ± 7.8^{c}	105 ± 7.8^{c}	100 ± 7.8^{c}	2.75	< 0.001
HDL (mg/dL)	48 ± 5.5^{a}	29 ± 5.5^{b}	59 ± 5.5^{c}	$51 \pm 5.5^{a,c}$	56 ± 5.5^{c}	1.94	< 0.001
LDL (mg/dL)	72 ± 8.5^{a}	100 ± 8.5^{b}	69 ± 8.5^{a}	75 ± 8.5^{a}	65 ± 8.5^{a}	2.99	< 0.001
ALT (mg/dL)	52 ± 7.5^{a}	82 ± 7.5^{b}	51 ± 7.5^{a}	53 ± 7.5^{a}	50 ± 7.5^{a}	2.65	< 0.001
AST (mg/dL)	159 ± 8.7^{a}	197 ± 8.7^{b}	153 ± 8.7^{a}	159 ± 8.7^{a}	148 ± 8.7^{c}	3.06	< 0.001
ALP (mg/dL)	91 ± 16.4^{a}	122 ± 16.4^{b}	$108 \pm 16.4^{a,b}$	114 ± 16.4^{b}	$106 \pm 16.4^{a,b}$	5.80	0.001

Note: Different superscripts in a row differ from one another significantly ($p < 0.05$) in multiple comparison by Bonforoni correction. Here, T0 = control; T1 = control obese, T2 = sunlight, T3 = synthetic vitamin D, T4 = sunlight + synthetic vitamin D.
Abbreviation: SEM = Standard Error of Mean.

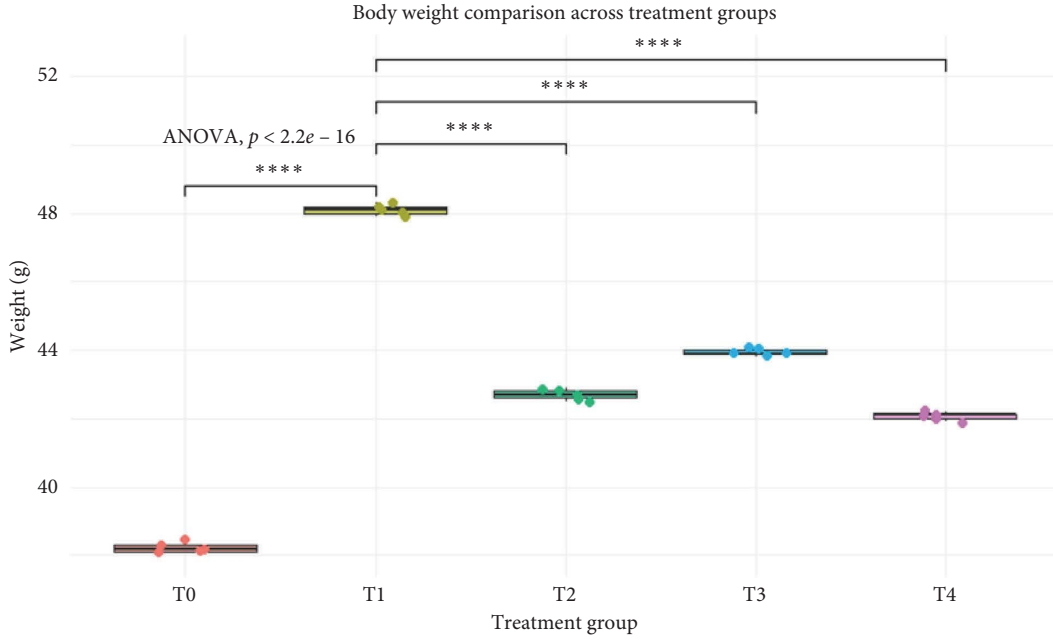


Figure 2: Box plot with jittered points shows the weight of different groups of mice.

showing a clear downward shift in the obese control group compared with all other treatments, and a marked upward shift in the combined sunlight plus vitamin D_3 group.

3.6. Gross Study. Gross examination revealed normal liver and kidney morphology in all groups except the obese control group. Mice in this group exhibited enlarged, pale-yellow livers and pale tan to yellow kidneys, consistent with fatty liver–kidney syndrome (Figure 4). However, no gross abnormalities were observed in heart across any groups.

3.7. Histoarchitectural Study. Liver section from the control group showed normal histoarchitecture without pathological alterations (Figure 5). In contrast, liver sections from the obese control group (T_1) exhibited hepatocyte steatosis, indicating fatty deposition and the presence of adipocytes in the liver hepatocytes. These alterations might be due to increased body weight and fat deposition in hepatocytes. However, no significant architectural changes were found in other treatment groups (T_2, T_3, and T_4). Kidney sections from the control group displayed normal cortical and medullary structures, with no evidence of glomerular or tubular lesions, cellular infiltration, or parenchymal damage (Figure 6). Histomorphological studies of heart tissue showed adipocyte infiltration in the obese control group (T_1), indicating fatty changes (Figure 7). Other groups displayed only mild, nonspecific alterations, with nuclei and myocardial architecture remaining normal.

Overall, the obese control group (T_1) consistently exhibited the poorest results across all treatments, including weight, glucose, lipid profile, and organ pathology. The most significant

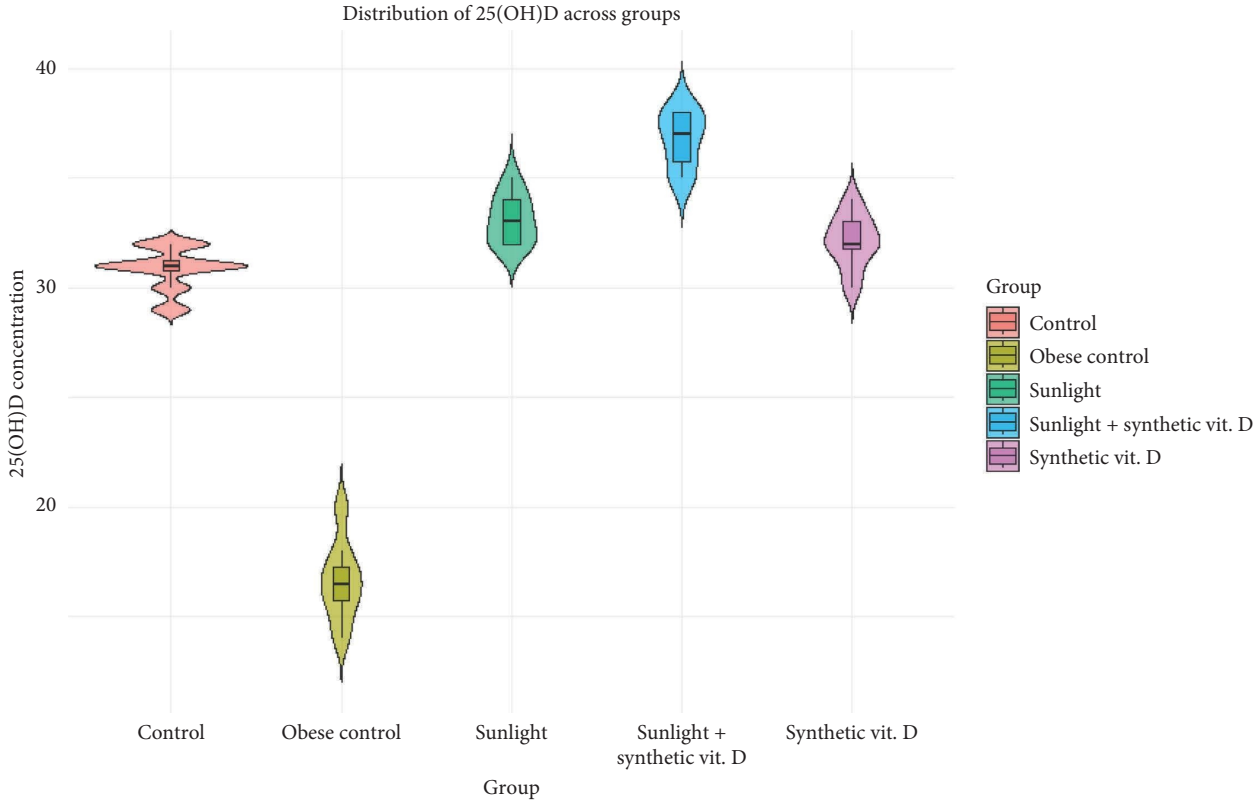


Figure 3: Distribution of serum 25(OH)D concentrations across experimental groups.

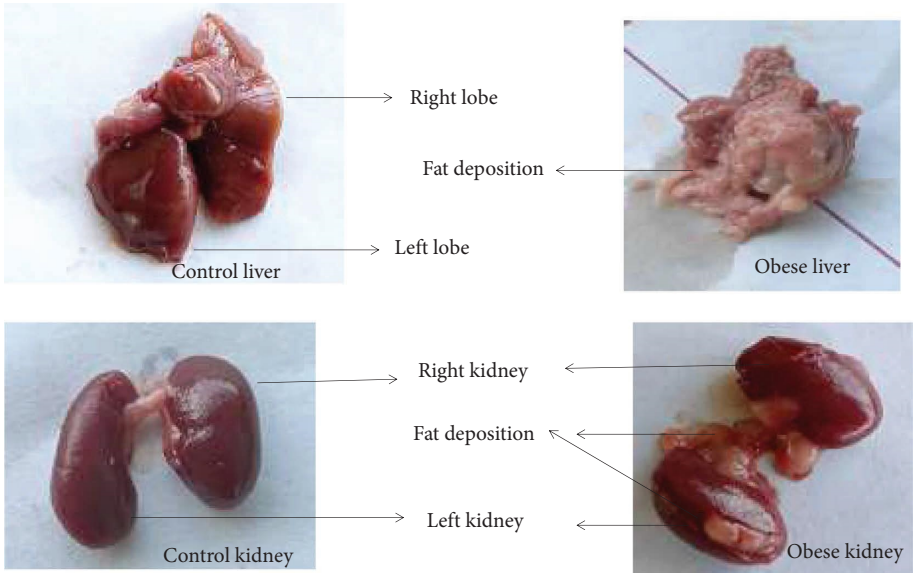
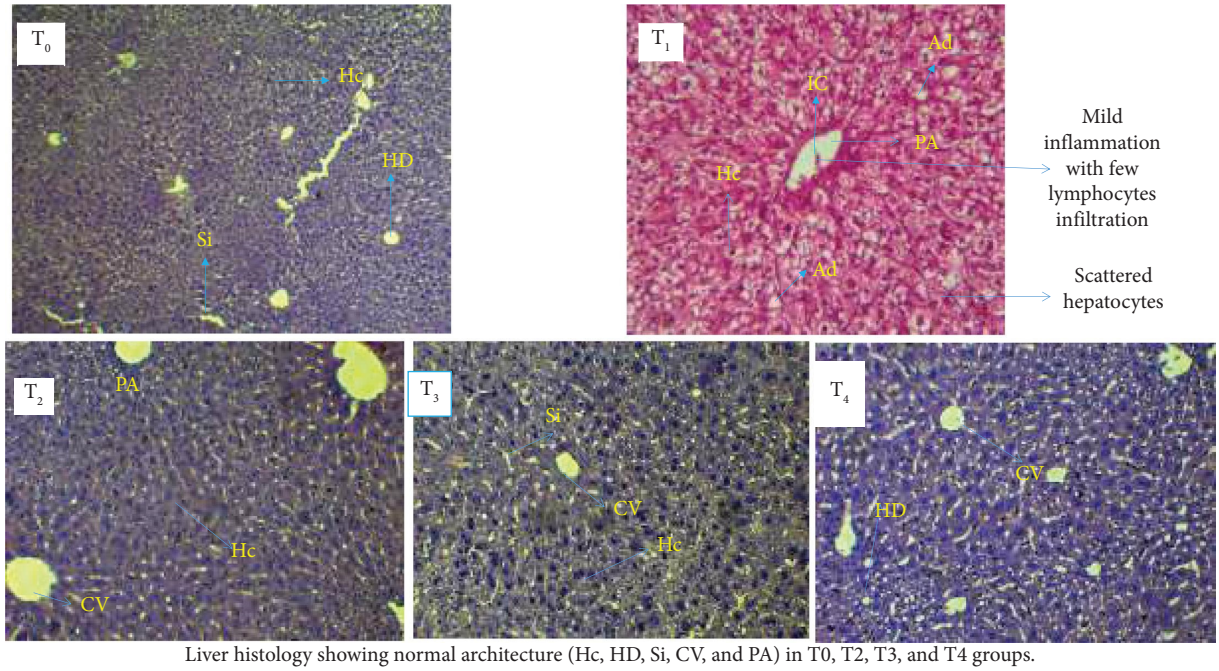


Figure 4: Gross lesion of liver (control and obese group) and kidney (control and obese group).

reliable improvements in weight gain, liver enzymes, and tissue structure were observed in T_4 (sunlight + vitamin D_3), followed by T_2 and T_3. These findings suggest a synergistic effect of combined sunlight and vitamin D_3 supplementation.

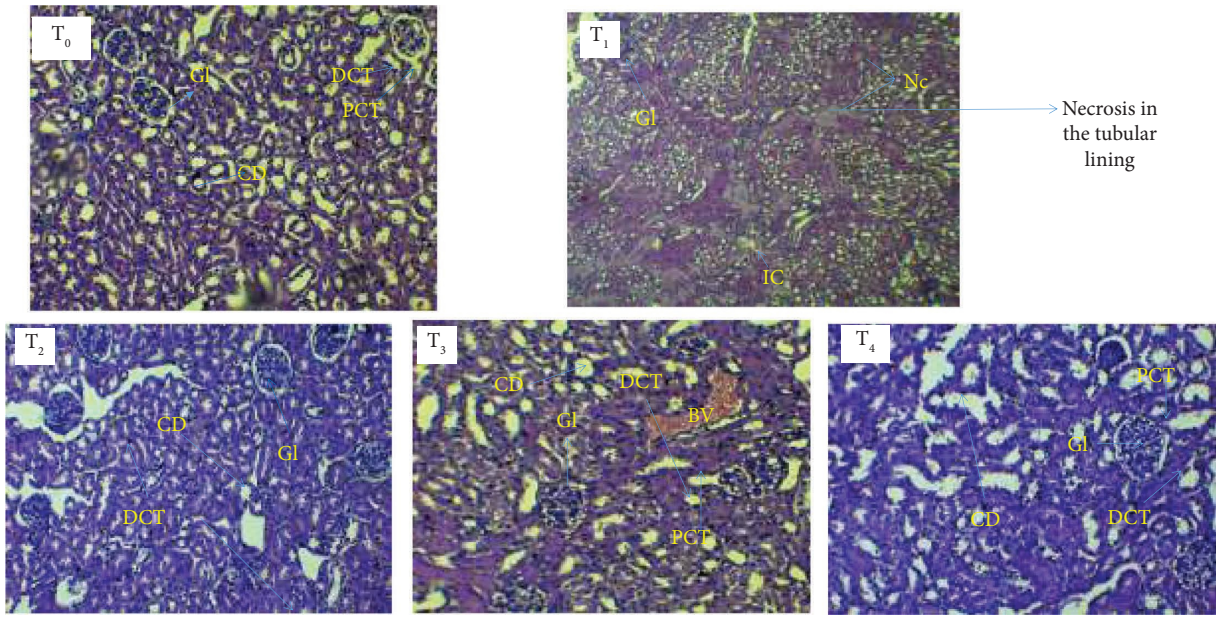
4. Discussion

The potential role of vitamin D in mitigating obesity has attracted considerable public health interest, given its



Liver histology showing normal architecture (Hc, HD, Si, CV, and PA) in T0, T2, T3, and T4 groups.
T1 group shows adipocytes (Ad), mild inflammatory cell infiltration (IC), and disorganized hepatocytes (H&E; 40x).

FIGURE 5: Photomicrograph of liver tissue of T0, T2, T3, and T4 groups showing normal hepatocytes (Hc), hepatic ducts (HD), sinusoids (Si), central vein (CV), and portal area (PA) where T1 group showing adipocytes (Ad) with mild inflammation of inflammatory cells (IC) and scattered hepatocytes (Hc) (H&E; 40x).

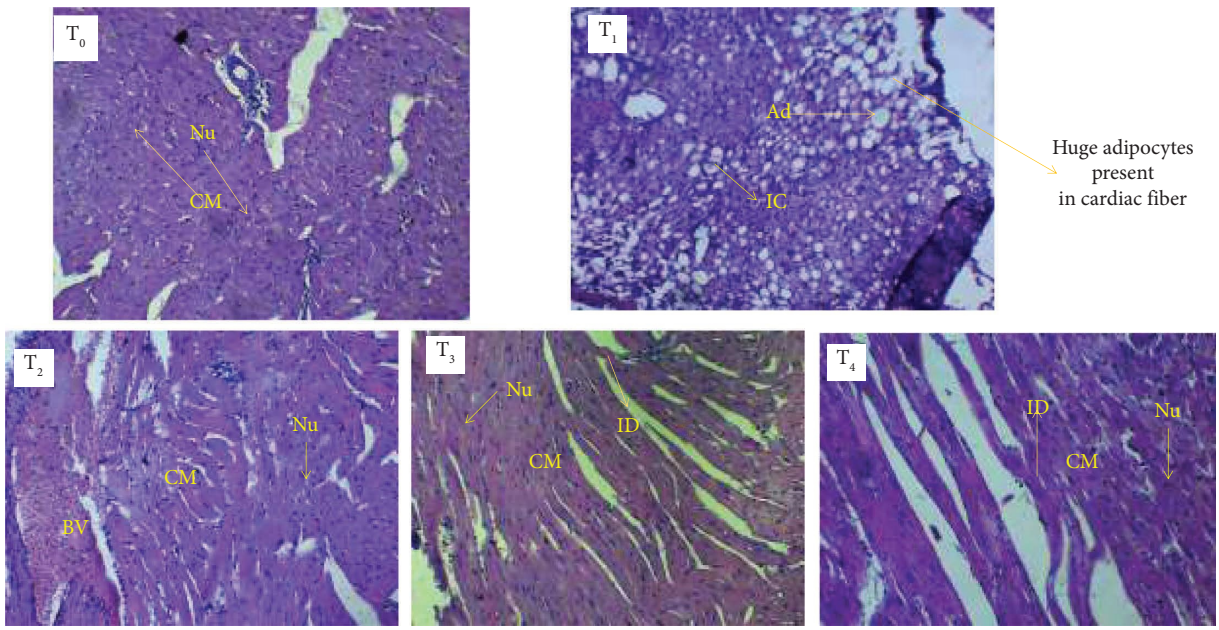


Kidney histology showing normal structures (Gl, PCT, DCT, CD) in T0, T2, T3, and T4 groups. T1 group exhibits mild necrosis (Nc) and inflammatory infiltration (IC)(H&E; 40x).

FIGURE 6: Photomicrograph of kidney tissue of T0, T2, T3, and T4 groups showing normal glomerulus (GL), proximal convoluted tubules (PCT), distal convoluted tubules (DCT), and collecting ducts (CD) where T1 group showing mild necrosis (Nc) with mild inflammation of inflammatory cells (IC) (H&E; 40x).

implications for clinical recommendations. In the present study, vitamin D supplementation in fat-induced obese mice was associated with attenuated weight gain and improved glucose homeostasis. Consistent with previous reports, mice fed a high-fat diet (butter-based) gained significantly more weight than healthy controls.

Vitamin D appears to modulate adipose tissue dynamics by inhibiting adipogenesis, reducing lipid accumulation, and



Heart tissue showing normal cardiac muscle (CM), nuclei (Nu), intercalated discs (ID), and blood vessels (BV) in T0, T2, T3, and T4. T1 group reveals large adipocytes (Ad) and mild inflammatory cell infiltration (IC)(H&E; 40x).

FIGURE 7: Photomicrograph of heart tissue of T0, T2, T3, and T4 groups showing normal nuclei (Nu) in cardiac muscle fiber (CM), intercalated disc (ID), and blood vessel (BV) where T1 group showing huge adipocytes (Ad) in the lining of cardiac fiber with mild inflammation of inflammatory cells (IC) (H&E; 40x).

promoting lipolysis [19]. Furthermore, vitamin D is vital for muscle growth and repair, which enhances metabolism and leads to increased calorie burning during rest. Several clinical studies support these mechanisms, reporting reductions in fat mass among overweight and obese women following vitamin D supplementation [20]. Additionally, vitamin D also influences the activity of two key proteins that help in the development of fat cells, PPAR-γ and C/EBPα [21]. In contrast, several recent randomized clinical trials indicate that vitamin D treatment does not appear to lead to weight loss in those who are overweight or obese [22].

Obesity-related adipose dysfunction alters vitamin D metabolism, rendering 25-hydroxyvitamin D_3 more effective than vitamin D_3 in achieving sufficiency, while deficiency (< 25–30 nmol/L) and suboptimal status (< 50–100 nmol/L) remain closely associated with metabolic disorders, raising questions about body size–based dietary requirements [23, 24]. Consistent with these observations, previous studies demonstrate that combined sunlight exposure and cholecalciferol supplementation ameliorate obesity-related vitamin D disturbances [6, 25].

VDR expression in insulin-sensitive tissues and pancreatic β-cells mediates vitamin D's role in glucose metabolism, enhancing peripheral insulin sensitivity and insulin production [26]. Our findings indicate that while the vitamin D and sunlight-treated groups maintained normoglycemia, the obese control group (T_1) exhibited considerably higher glucose levels. Although some meta-analyses show that vitamin D has little effect on glucose regulation in obese people [27, 28], our animal model supports the hypothesis that vitamin D can reverse insulin resistance brought on by high-fat diets, perhaps by inhibiting inflammatory cytokines like TNF-α and IL-6, which are known to interfere with insulin signaling [29].

The present results demonstrated that high-fat diet–induced obesity resulted in significant dyslipidemia, characterized by elevated serum total cholesterol (TC), triglycerides (TGs), and low-density lipoprotein cholesterol (LDL-C) levels accompanied by a reduction in high-density lipoprotein cholesterol (HDL-C). These alterations are consistent with earlier reports linking obesity to adverse lipid metabolism [30, 31]. Intervention with vitamin D and sunlight exposure effectively improved the lipid profile. Both treatments significantly reduced serum TC, TGs, and LDL-C concentrations, while HDL-C levels increased, particularly in the sunlight-exposed group. The findings of this study are consistent with previous meta-analysis showing that vitamin D supplementation benefits the lipid profile and serves as a useful dietary intervention for managing dyslipidemia [32, 33]. Remarkably, the group that received only sunlight (T_2) outperformed the group that received both sunlight and vitamin D3 (T_4) in several metabolic markers, including HDL levels. Interestingly, the sunlight-only group (T_2) showed the most favorable lipid profile, particularly higher HDL levels, despite similar serum 25(OH)D increases compared to the vitamin D-supplemented groups. This effect may be attributed to ultraviolet (UV)-induced nitric oxide (NO) synthesis, which exerts vasodilatory, insulin-sensitizing, and anti-inflammatory effects, thereby complementing vitamin D's metabolic actions [34, 35]. These additional photoproducts likely contributed to the observed improvements in glucose regulation, lipid metabolism, and liver enzyme profiles, highlighting that sunlight provides

benefits beyond vitamin D synthesis alone. Although Hu et al. [36] reported no significant changes in lipid profiles following vitamin D supplementation in overweight or obese individuals, evidence from broader studies suggests potential benefits. A systematic review and meta-analysis by Dibaba [37] indicated that vitamin D supplementation can reduce triglycerides (TG) and total cholesterol (TC) while increasing HDL-C. Similarly, Ge et al. [32] found improvements in HDL-C and TG in individuals with obesity-related metabolic syndrome, though LDL-C and TC were unchanged. Surdu et al. [38] reported that low baseline vitamin D levels are linked to atherogenic lipid profiles, and supplementation can modulate LDL-C, TC, TG, and HDL-C. Discrepancies may result from variations in study design, population, supplementation duration, baseline vitamin D status, and interactions with nitric oxide (NO), which influences lipid metabolism. Overall, vitamin D may beneficially affect lipid profiles, but further research is needed to clarify the mechanisms and conditions for efficacy.

Additionally, liver enzyme levels of ALT, AST, and ALP are elevated in the obese group, whereas lower levels were observed in the control and vitamin D or sunlight-treated groups (T_0, T_2, T_3, T_4). These findings are consistent with previous reports demonstrating that vitamin D supplementation and lifestyle interventions ameliorate hepatic dysfunction associated with obesity [20, 39, 40]. According to the results of current research, although post-intervention levels of both AST and ALT decreased, this reduction was not significant, a finding consistent with previous research [41]. Furthermore, an epidemiological study indicated that the risk of elevated ALT and AST is higher in patients with vitamin D deficiency [40]. Supplementing vitamin D helps enhance liver enzymes by inhibiting lipogenesis, the process by which fat is synthesized, and promoting fat oxidation, which prevents fat accumulation in the liver.

In addition to its hepatic benefits, vitamin D may also impact obesity-related hormonal pathways that regulate appetite and fat storage. Adiponectin, leptin, and neuropeptide Y (NPY) play essential roles in controlling appetite, maintaining energy balance, and regulating fat metabolism. Leptin, a hormone produced by fat cells, typically acts to suppress hunger and boost energy expenditure. However, in obesity, the body often develops leptin resistance, which limits this regulatory effect. Adiponectin, which supports insulin sensitivity and the breakdown of fatty acids, is usually found at lower levels in obese individuals. Meanwhile, NPY, a neuropeptide secreted mainly by the hypothalamus, promotes food intake and tends to be elevated in obesity, thereby facilitating fat accumulation [42–44]. Although these specific markers were not analyzed in the current study, existing research suggests that vitamin D and/or sunlight exposure might influence these pathways, potentially contributing to the observed improvements in body weight and metabolic health.

The present study demonstrates that obesity is associated with markedly reduced serum 25(OH)D concentrations, whereas interventions with sunlight exposure, synthetic vitamin D supplementation, or their combination significantly improved vitamin D status. In particular, the obese control group exhibited a pronounced decline in circulating 25(OH)D levels, supporting earlier findings that obesity is linked to impaired vitamin D bioavailability. This reduction has been attributed primarily to the sequestration of vitamin D in adipose tissue and volumetric dilution effects, as previously reported by other scientists [24, 45, 46].

Gross examination revealed notable fat accumulation in both the liver and kidneys of the obese control group (T_1), whereas no such deposition was observed in the other treatment groups. This finding is consistent with evidence that lipid accumulation in these organs is a hallmark of metabolic disorders, including fatty liver disease (FLD) and chronic kidney disease (CKD). High-fat diet feeding in aged murine models exacerbates ectopic fat deposition, promoting pronounced hepatic steatosis, inflammation, and fibrosis, evidenced by elevated serum ALT, macrophage infiltration, pro-inflammatory cytokines, fibrogenic markers, oxidative stress, and TLR4 expression, while also inducing glomerular alterations and renal inflammation, though age-related differences in HFD-induced renal injury are less marked [47, 48].

Recent studies further suggest that perirenal, renal sinus, and intrarenal fat contribute to the pathogenesis of obesity-related complications, including insulin resistance, atherosclerosis, hypertension, and chronic kidney disease [49]. Mechanistically, dysregulation of the energy-sensing enzyme AMP-activated protein kinase (AMPK) may drive tissue inflammation and fibrosis, while circulating mediators such as Fetuin-A and adiponectin facilitate lipid-induced metabolic disturbances and interorgan communication. Although cardiac lipid deposition was considered, this study did not provide definitive evidence of myocardial fat accumulation in obese mice, highlighting the need for further targeted investigations.

Again, histomorphological studies of liver showed that adipocytes were found in hepatocytes in obese group T_1 but alteration of hepatocytes was absent in other groups. The study showed that obese mice had hepatocyte steatosis, which is a sign of fatty material buildup in the liver [50–52]. The results demonstrate the effects of a high-fat diet on the liver health of obese mice by showing notable alterations in the fatty acid levels in liver. Moreover, inflammatory cell infiltration near the glomerulus in the kidneys suggested early signs of obesity-induced kidney damage in the obese group T_1 [49, 52]. Similarly, a high-fat diet caused renal dysfunction in Sprague-Dawley rats by impairing glomerular filtration, damaging mitochondrial integrity, and increasing oxidative stress [53]. In contrast to normal healthy control mice heart tissue, which had normal architecture with regular myocardial cell morphology, the histology of heart tissue of fat-induced obese mice revealed the deposition of fat globules in myocardial cells with noticeable fatty alterations in the portal area [54]. Further research on gerbils revealed that a high-calorie diet resulted in interstitial edema and structural disarray linked to the buildup of lipids and infiltrating cells, as well as interstitial myocardial and perivascular fibrosis in the heart muscle [55]. Histological signs of hypertrophy, inflammation, possible intra- and extracellular fat accumulation and fibrosis were found in interstitial and perivascular areas of heart [56, 57].

Although both sunlight and oral supplementation ultimately produce the same form of vitamin D_3, sunlight exposure may confer additional benefits through non–vitamin D pathways. Ultraviolet B radiation induces the release of nitric oxide and other photoproducts, which have been associated with vasodilation, enhanced insulin sensitivity, and anti-inflammatory effects [34, 58, 59]. These mechanisms may help explain why sunlight exposure alone produced a more favorable lipid profile compared with vitamin D_3 supplementation in our study. Although the findings are promising, the research is limited by the short duration of treatment and the absence of gene/protein expression data to elucidate molecular pathways. As new research indicates that the vitamin D-gut axis plays a critical role in obesity and metabolic disorders, future research should explore VDR expression in target tissues, cytokine profile, and gut microbiota interactions [60].

This study benefits from a controlled experimental design, the inclusion of both natural and supplemental vitamin D sources, and comprehensive biochemical and histological assessments. However, its short intervention period and the absence of detailed mechanistic analyses, such as gene expression, cytokine profiling, or nitric oxide signaling, limit the ability to fully distinguish vitamin D–dependent from non-vitamin D effects. Future research with longer durations and molecular investigations is warranted to clarify these mechanisms.

5. Conclusion

This research demonstrates that both vitamin D_3 supplementation and sunlight exposure mitigate high-fat diet–induced obesity and associated metabolic disturbances in mice. Notably, the combined intervention (T_4) offered the greatest protection against hepatic injury, whereas sunlight exposure alone (T_2) produced the most significant improvements in lipid profiles. These findings highlight the promising role of vitamin D_3, particularly when acquired through natural sunlight, as a straightforward and accessible approach to managing obesity-associated metabolic disorders. Further investigations are needed to elucidate the underlying mechanisms involved and to translate these findings into clinical practice.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

Tithe Saha: conceptualization, methodology, funding acquisition, and writing–original draft; Sabuj Kanti Nath: writing–review and editing, data visualization, supervision, and validation; Papia Khatun: resources and project administration; Swarup Kumar Kundu: resources and conceptualization; Anil Yadav: investigation and data curation; Zahid Hasan Rocky: investigation and data curation; and Tishita Sen Ape: formal analysis and software; Purbita Devi: writing–review and editing. Tithe Saha and Sabuj Kanti Nath contributed equally to this work.

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