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Anti-hyperlipidemic Effect, Growth Performance and *in vivo* Antioxidant Enhancements *of Launaea taraxacifolia* Leaf-based Diet in High-fat Diet-induced Hyperlipidemic Rats

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ABSTRACT: The study investigated the anti-hyperlipidemic activity, *in-vivo* antioxidant and growth performance enhancements of *Launaea taraxacifolia* leaf-based diet at 6.25% , 12.5% and 25% inclusion levels in high-fat diet-induced hyperlipidemic rats. 36 female rats weighing 165.82 ± 2.10g were assigned into two groups: A (6) and B (30). Animals in group B were made hyperlipidemic by feeding on high-fat diet for six weeks and confirmed by assaying for lipid profile, body weight gain (BWG), body mass index (BMI) and hip circumference (HC). They were subsequently re-assigned into five groups: a non-treated group, an atorvastatin-treated group, and three groups treated with *Launaea taraxacifolia* leaf-based diets at inclusion levels of 6.25%, 12.5%, and 25%, respectively. All groups were maintained on their respective diets for six weeks. The feed intake (FI), BWG, BMI, HC, superoxide dismutase, catalase, glutathione reductase activity, malondialdehye (MDA), total cholesterol (TC) low density lipoprotein-Cholesterol (LDL-c), high density lipoprotein-Cholesterol (HDL-c), triglycerides (TG) and leptin concentration were determined using standard methods. The result revealed that high-fat diet significantly ($p < 0.05$) increased BWG, BMI as well as HC, MDA, TG, LDL and TC concentrations but decreased the leptin concentration and antioxidant enzymes activity however *Launaea taraxacifolia* leaf-based diet reversed the alterations caused by the high-fat diet. Overall, the results suggests that the leaf-based diet possesses anti-hyperlipidemic effect and therefore support its usage in folklore medicine.

Keywords: High-fat Diet, *Launaea taraxacifolia*, Atorvastatin, Lipid Profile, Anti-hyperlipidemic activity

Introduction

Hyperlipidemia is a condition or disorder characterized by elevated serum total cholesterol, low density lipoprotein, very low density lipoprotein and decreased high density lipoprotein levels (Victor Duniya *et al*., 2018) It is said to be a modifiable risk of cardiovascular disease, myocardial infarction, pancreatitis and heart disease (Mahima *et al.,* 2022) and it accounts for about 17 million deaths annually (Durrington, 2015). The major causes of hyperlipidemia are mainly changes in lifestyle specifically in the aspect of dieting, taking more fatty foods than the recommended amount per day (Durrington, 2015) while other factors that are culprit to high lipids level in the blood include: excessive weight, smoking, high alcohol intake, lack of exercise, diabetes, nephrological disorders, pregnancy and underactive thyroid gland (Durrington, 2015). Oxidative stress is described as an imbalance in the pro-oxidant/antioxidant balance in favour of the oxidants, leading to significant cellular damage (Federico and Bravo-San Pedro, 2021) therefore serving as one of the major risk factors for

metabolic diseases (Manz koule *et al*., 2024). In the same vein, hyperlipidemia has been are responsible for the overproduction of reactive oxygen species (ROS) due to the saturation of the electron transport chain (Hamza-reguig, 2017). Therefore, antioxidants have been reported to counteract ROS-linked oxidative damage by reacting with free radicals, and also by acting as oxygen scavengers (Shahidi and Wanasundara, 1992 ; Büyükokuroğlu *et al*., 2001). The impact of hyperlipidemia on the growth performance characteristics and various nutritional parameters cannot be underrated because it causes numerous distortations. Energy imbalance can be promoted by an excessive consumption of macronutrients, and particularly by dietary fat (Donato and Hegsted, 1985; Astrup, 1993). It has been well documented that fatty foods has a high palatability rate enhancing its consumption rate thereby increasing the risk of hyperlipidemia leading to derangement of normal serum profile. In the same vein, weight gain is a consequence of an energy imbalance (Schrode *et al*., 2003). This imbalance has been related to an increase in body mass index (BMI) and hip circumference which has also been correlated with hyperlipidemia. BMI is often directly associated with total and LDL-cholesterol plasma concentrations, whereas an inverse relationship has been reported between HDL-cholesterol and BMI (McNamara *et al*. 1992; Ernst *et al*. 1997). In the same vein, leptin, an example of adipocytokines which is a bioactive substance secreted by visceral and subcutaneous adipose tissue helps in maintaining a normal triglyceride content and protects from fat overload and lipotoxicity (Chandralekshmy, 2024). Therefore , its deficiency leads to excess fat deposition and damage to nonadipocyte. The devastating health impact caused by hyperlipidemia as led to the search for newer drugs with the capacity to lower or regulate blood cholesterol and triglyceride concentration, this to some extend has drawn attention of most medical researchers towards finding a cure or better still a relieve to the impact cause by the presence of these lipids in the blood (Abd and Jacobson, 2011). Outcomes from several of their studies has resulted to different findings stating significant properties of a variety of natural and synthetic agent, notable among which is the discovery of potent drug agent such as Pravastatin and Simvastatin which act by reducing the total circulating cholesterol in the blood by inhibiting 3-hydroxy-3-methylglutaryl coenzymes A reductase, the committed step in the biosynthetic pathway of cholesterol and thus helps in minimizing the rate of reported cardiovascular disease cases (Ito, 2012). However the side effect associated with administration of these drugs such as muscle damage skeletal disorder (Ito, 2012; Abd and Jacobson, 2011) alteration in some Liver enzymes and increased risk of diabetes mellitus is an issue to ponder upon (Naci *et al.,* 2013). This has necessitated the quest for alternative treatments that are affordable, potent and with minimal side effect. Several indigenous plants have been utilized for their beneficial effects in the treatment of diabetes and hyperlipidemia (Parikh *et al.,* 2014) among which are: *Daucus carota L, Pongamia pinnata* and *Anethum graveolens L* (Tijjani *et al.,* 2020). The plant *L. taraxacifolia* commonly known as Dandelion is a wild plant with several medicinal properties, it is of the family Asteraceae and mostly cultivated in African, West African to be specific. Leaf and seeds of the plants have been used in the treatment and ameliorating disease condition such as: Conjunctivitis, Diabetes mellitus, Hypertension, Poor bone fixation in children, yaws as well as water retention syndromes (Laleye *et al.,* 2015). The leaf of this plant is taken as sauce by south-western people of Nigeria. Hence, since adherence to drug is at an 100% rate when taken as food, nutritherapy, based on the use of food in treatment or prevention, could be one of the best strategies for combating hyperlipidemia, obesity and oxidative stress (Manz koule *et al*., 2024). This study is therefore targeted at evaluating the anti-hyperlipedemic growth performance and *in-vivo* antioxidant enhancements *of launaea taraxacifolia* leaf-based diet in high-fat diet-induced hyperlipidemic rats.

Materials and methods

Plant collection, authentication and preparation: *Launeae taraxacifolia* (Wild lettuce), a greenish vegetable was harvested in a farm at Ojutaye Ilorin, Kwara State, Nigeria and was authenticated at the herbarium unit of the Department of Plant Biology, University of Ilorin, Kwara state where a voucher specimen (UIH1023) was deposited. The fresh leaves was thoroughly washed, dried at 60 °C and later pulverized into powder using an electric blender.

Laboratory animals: Thirty six (36) female albino rats (*Rattus novergicus*) (165.82 ± 2.10 g, 5- 7 weeks old) were obtained from the animal holding unit of the Department of Biochemistry, Faculty of Life Sciences, University of Ilorin, Ilorin, Nigeria and was kept in well ventilated house conditions with free access to rat pellets and tap water before the experiment commenced.

Feed ingredients: Yellow maize seed, soy beans and cellulose (corn corb) were obtained from Oke-Oyi market in Ilorin, Nigeria. Beef tallow was obtained from Mandate market in Ilorin. Vitamin/Mineral mix and D-Methionine were products of *Rofat Feed Nigeria Limited* in Ilorin, Nigeria while soybean, oil and sucrose were products of *Sunola Refined Soybeans, Kewalram Nigeria Limited, Nigeria* and *Saint Louis Sucre, Nigeria* respectively.

Drugs, assay kits, chemicals and reagents: Atorvastatin was a product of Juhel Nigeria Ltd while assay kits were products of Monobind Inc., Lake Forest, USA. All other chemicals and reagents used were of analytical grade which were obtained from Sigma Aldrich Limited, Buchs, Canada.

Animal grouping, composition of diet and induction of hyperlipidemia: After a week of acclimatization, the animals were weighed (initial weight) and assigned into two groups: A (6 rats {control diet}) and group (30 rats {High-fat diet induced hyperlipidemic rats}(Oladiji *et al*., 2007). They were all fasted for 12 hours to ensure complete emptying of the stomach before feeding them on their new diets (Table 1) for a period of six weeks. Hyperlipidemia was confirmed by estimation of some growth performance characteristics and determination of serum lipid profile (Determination of lipid profile was done by obtaining aliquot of the blood sample through ocular puncture and the serum was prepared according to the procedure described by Yakubu *et al*. (2008).

Table 1: Formulation of feed for control (basal diet) and high fat diet

Ingredients (g/kg)	Control diet (g/kg)	High fat diet (g/kg)
Corn Starch	516	166
Animal fat (beef tallow)	-	350
Soybean	230	230
Soybean oil	50	50
Cellulose	50	50
Sucrose	100	100
Vitamin/mineral	50	50
D-methionine	4	4
Total	**1000**	**1000**

***Vitamin/mineral mix:** Vitamin A 4,000,000 i.u; vitamin D_3 800,000 i.u.; tocopherols 400 i.u; vitamin K_3 800 mg; folacin 200 mg; thiamine 600 mg; riboflavin 1,800 mg; niacin 6000 mg; calcium pathothenate 4 mg; biotin 8 mg; manganese 30,000 mg, zinc 20,000 mg; choline chloride 80,000 mg; copper 2,000 mg; iodine 480 mg; cobalt 80 mg; selenium 40 mg; BHT 2,500 mg. Anticaking agent 6000 mg. Unit of diet composed: g/kg.

Composition of diets, animal re-grouping and sacrifices: The animals fed on high-fat diet were re-grouped into five after confirmation of hyperlipidemia with the group A still being fed on control diet. They were subsequently re-assigned into five groups: a non-treated group, an atorvastatin-treated group, and three groups treated with *Launaea taraxacifolia* leaf-based diets at inclusion levels of 6.25%, 12.5%, and 25%, respectively. All groups were maintained on their respective diets for six weeks (Table 3). During this period , the growth performance characteristiscs (feed intake (Mwale *et al*., 2008), weight gain, body mass index (Bernardis and Patterson, 1968) and hip circumference (Novelli *et al*., 2007) were re-measured. They were then fasted for 12 hours to ensure complete gastric emptying. They were then anaesthesized using diethyl ether and dissected.

Table 2: Re-grouping and treatment of animals in Group B

	Feed and Treatment
B	Rat fed on high fat diet with no treatment
C	Rats fed on high fat diet and treated with atorvastatin (0.06 mg/kg bwt)
D-F	Rats fed on high fat diet and treated with *launaea taraxacifolia* leaf-based diet at 6.25%, 12.5% and 25% inclusion levels respectively

Table 3: Feed composition with *Launaea taraxacifolia* leaf

Ingredients	Control (A)	High fat diet-induced hyperlipidemic rats				
		Untreated (B)	Atorvastatin (0.06 mg/kg bdwt) (C)	*Launaea taraxacifolia* leaf-based diet		
				6.26% (D)	12.5% (E)	25% (F)
Cotton starch	516	166	166	166	166	166
Animal fat (Beef tallow)	-	350	350	350	350	350
Soybean	230	230	230	230	230	230
Soybean oil	50	50	50	50	50	50
Cellulose	50	50	50	50	50	50
Sucrose	100	100	100	100	100	100
Vitamin/mineral	50	50	50	50	50	50
D-methionine	4	4	4	4	4	4
Total	**1000**	**1000**	**1000**	**1000**	**1000**	**1000**

***Vitamin/mineral mix:** Vitamin A 4,000,000 i.u; vitamin D_3 800,000 i.u.; tocopherols 400 i.u; vitamin K_3 800 mg, folacin 200 mg; thiamine 600 mg; riboflavin 1,800 mg; niacin 6000 mg; calcium pathothenate 4 mg; biotin 8 mg; manganese 30,000 mg, zinc 20,000 mg; choline chloride 80,000 mg; copper 2,000 mg; iodine 480 mg; cobalt 80 mg; selenium 40 mg; BHT 2,500 mg; anticaking agent 6000 mg. Unit of diet composed g/ kg.

Biochemical assays

Determination of in vitro antioxidant enzyme activities: Superoxide distumase activity was determined based on the method of Mistra and Fridovich (1972) while catalase and gluthathione reductase were determined based on the methods of Beers and Sizer (1952) and Ellman (1959) respectively.

Determination of malondialdehye and nitric oxide concentrations: The concentrations of malondialdehyde and nitric oxide were quantified according to the method of Buege and Aust (1978) and Wo *et al.* (2013) respectively.

Determination of serum lipid profile: The total cholesterol concentration was determined by colorimetric technique as described by Allain *et al.* (1974). Triglycerol concentrations in the serum were determined as described by Searcy (1969). HDL-Cholesterol was determined by a colorimeteric technique described by Burstein and Mortin (1969). The serum low-density lipoprotein cholesterol was assayed using the polyvinyl sulphate (PVS) reaction as described by Demacker *et al.* (1984).

Determination of serum leptin concentration: Serum Leptin assay was done using an ELISA kit from Labor Diagnostika Nord GmbH & Co.KG, Am Eichenhain 1,48531 Nordhorn on ELx 800MS ERBA MICROSCAN ELISA machine.

Statistical analysis: Data was expressed as the mean ± SEM of six determinations. All results were statistically analyzed using one-way ANOVA and Duncan's Multiple range Test (DMRT) (Duncan, 1957). Differences between group means were considered significant at $p < 0.05$.

Results

Feed intake (g) of rats fed on high fat diet for six weeks: The result of the feed intake of rats fed on high fat diet for six weeks is presented in Table 4. There was no significant difference ($p > 0.05$) in the feed intake of rats fed on high fat diet when compared with that of the control rats.

Table 4: Feed intake (g) of rats fed on high fat diet for six weeks

Group	Feed Intake (g)					
	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6
A (Control)	87.21± 1.82^{a}	91.32± 2.23^{a}	90.12 ± 1.23^{a}	92.45± 1.86^{a}	89.21± 1.67^{a}	91.24± 1.45^{a}
B (High Fat Diet)	88.01± 0.98^{a}	90.79 ± 1.12^{a}	92.01± 0.89^{ab}	91.84 ± 1.76^{ab}	89.10 ± 1.34^{a}	92.16± 1.67^{ab}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$).

Growth performance characteristics of rats fed on high-fat diet for a period of six weeks: The result of the growth performance evaluation of rats fed on high fat diet is presented in Table 5. It was observed that there was a significant increase ($p < 0.05$) in the weight gain (g), final body mass index and final hip circumference of rats fed on high fat diet when compared with the control.

Table 5: Growth performance characteristics of rats fed on high-fat diet for a period of six weeks

Group	Initial Weight (g)	Final Weight (g)	Weight Gain (%)	Initial BMI (g/cm^2)	Final BMI (g/cm^2)	Initial HC (cm)	Final HC (cm)
A	166.23 ± 1.28^{a}	197.21± 4.89^{a}	19.01 ± 2.01^{a}	0.42 ± 0.11^{a}	0.47 ± 0.12^{a}	1.82 ± 0.41^{a}	1.91 ± 0.22^{a}
B	165.47 ±1.42^{a}	232. 41± 6.12^{b}	41.22± 1.21^{b}	0.41± 0.09^{a}	0.71± 0.18^{b}	1.80± 0.43^{a}	2.24 ± 0.53^{b}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$). key: A – Control , B – High-fat Diet, BMI – Body Mass Index , HC – Hip Circumference

Serum lipid profile of rats fed on high fat diet for six weeks: Serum lipid profile of rats fed on high fat diet for six weeks is presented in Table 6. A significant increase ($p < 0.05$) in the total cholesterol, triglyceride and low density lipoprotein cholesterol was observed in the serum of rats fed on high fat diet when compared with the control while the reverse was seen in the values high density lipoprotein cholesterol.

Table 6: Serum lipid profile of rats fed on high fat diet for six weeks

Group	TC (mmol/L)	LDL-C (mmol/L)	TG (mmol/L)	HDL-C (mmol/L)
A	19.21± 1.10^{a}	5.393± 0.89^{a}	6.01± 0.98^{a}	12.25 ± 1.37^{a}
B	28.42± 1.32^{b}	11.23 ± 1.76^{b}	9.46 ±B0.78^{b}	7.47 ± 1.45^{b}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$). Key: A – Control , B – High-fat Diet, TC – Total Cholesterol, LDL-C – Low density lipoprotein – Cholesterol, TG – Triglycerides, HDL – C – High Density Lipoprotein –Cholesterol

Feed intake (g) of high fat diet-induced hyperlipidemic rats treated with launaea taraxacifolia leaf-based diet: The result of the feed intake of rats fed on high fat diet treated with *launaea taraxacifolia* leaf-based diet is presented in Figure 1. There was a significant increase($p < 0.05$) in the feed intake of rats fed on high-fat diet only and those fed on 6.25%, 12.5% and 25% *launaea taraxacifolia* leaf-based diet when compared with those fed on basal control diet.

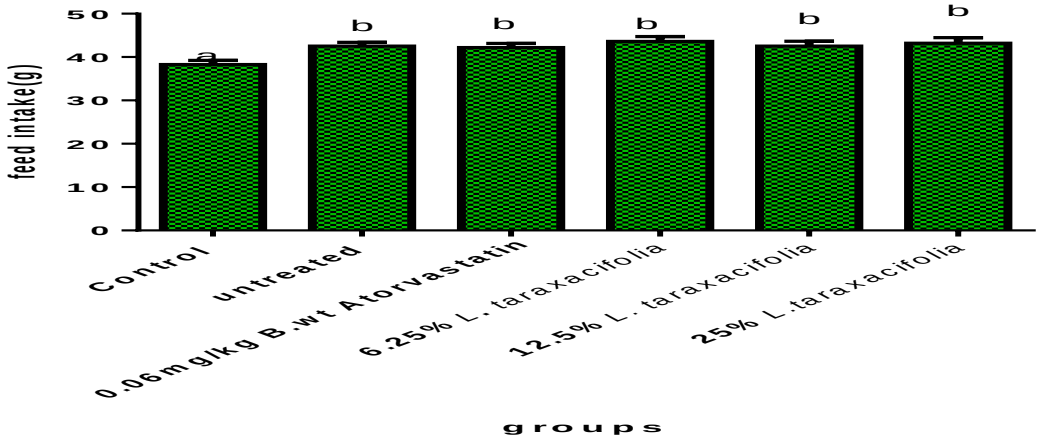

Figure 1: Feed intake (g) of high fat diet-induced hyperlipidemic rats treated with *launaea taraxacifolia* leaf-based diet

Growth performance characteristics of high fat diet –induced hyperlipidemic rats treated with Launaea taraxacifolia leaf-based diet: The above is presented in Table 7. A significant difference ($p < 0.05$) was observed when the body weight gain of rats fed on high fat diet (untreated) was compared with the control, those treated with the standard drug and those treated with the leaf-based diet at all inclusion levels. Likewise, those treated with the three inclusion levels compared favourably ($p > 0.05$) with each other. The same trend was also observed for the BMI and the HC.

Table 7: Growth performance characteristics of high-fat diet–induced hyperlipidemic rats treated with *Launaea taraxacifolia* leaf-based diet

Group	Initial Weight (g)	Final Weight (g)	Weight Gain (%)	Initial BMI (g/cm^2)	Final BMI (g/cm^2)	Initial HC (cm)	Final HC (cm)
A	197.21 ± 4.89^{a}	229.12 ± 3.11^{a}	15.95 ± 2.01^{a}	0.47 ± 0.12^{a}	0.58 ± 0.11^{a}	1.91 ± 0.22^{a}	1.99 ± 0.10^{a}
B	232.41 ± 6.12^{b}	291.22 ± 3.01^{b}	25.30 ± 1.10^{b}	0.71 ± 0.18^{b}	0.93 ± 0.09^{b}	2.24 ± 0.53^{b}	2.67 ± 0.77^{b}
C	232.41 ± 6.12^{b}	262.11 ± 2.12^{c}	13.21 ± 2.01^{a}	0.71 ± 0.18^{b}	0.82 ± 0.07^{c}	2.24 ± 0.53^{b}	2.44 ± 0.56^{c}
D	232.41 ± 6.12^{b}	275.11 ± 2.82^{d}	18.38 ± 1.67^{c}	0.71 ± 0.18^{b}	0.89 ± 0.08^{bd}	2.24 ± 0.53^{b}	2.54 ± 0.46^{d}
E	232.41 ± 6.12^{b}	273.01 ± 1.99^{d}	17.77 ± 1.98^{c}	0.71 ± 0.18^{b}	0.85 ± 0.09^{de}	2.24 ± 0.53^{b}	2.49 ± 0.57^{c}
F	232.41 ± 6.12^{b}	271.00 ± 2.45^{d}	17.01 ± 2.11^{cd}	0.71 ± 0.18^{b}	0.86 ± 0.10^{de}	2.24 ± 0.53^{b}	2.51 ± 0.43^{cd}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$). key : A- Control B – Untreated C – Atorvastatin (0.06 mg/kg bdwt) treated D F – 6.25%, 12.5% and 25% inclusion of *Launaea taraxacifolia* leaf-based Diet

Serum lipid profile of rats of high fat diet-induced hyperlipidemic rats treated with Launaea taraxacifolia leaf-based diet: Serum lipid profile of rats of high fat diet-induced hyperlipidemic rats treated with *launaea taraxacifolia* leaf-based diet is presented in Table 8. A significant increase ($p < 0.05$) in the total cholesterol, triglyceride and low density lipoprotein cholesterol was observed in the serum of rats fed on high fat diet when compared with the control and those fed *launaea taraxacifolia* leaf-based diet at all inclusion levels while the reverse was seen in the values of high density lipoprotein cholesterol. However, for the total cholesterol and low density lipoprotein cholesterol, the groups fed on 12.5 and 25% of the leaf-based diet compared favourably ($p > 0.05$) with each other and those fed on the standard drug while for triglycerides, groups fed on 6.25 and 12.5% of the diet compared favourably ($p > 0.05$) with each other and the standard drug. Interestingly, for the high-fat diet, the group fed on 25% of the diet compared favourably ($p > 0.05$) with the control and group treated with the standard drug.

Table 8: Serum lipid profile of rats of high fat diet-induced hyperlipidemic rats treated with *Launaea taraxacifolia* leaf-based diet

Group	TC (mmol/L)	LDL-C (mmol/L)	TG (mmol/L)	HDL-C (mmol/L)
A	18.23± 1.10^{a}	4.39± 0.89^{a}	6.92 ± 0.98^{a}	13.25 ± 1.37^{a}
B	34.13 ± 1.23^{b}	12.37 ± 1.12^{b}	11.48 ± 1.10^{b}	5.68 ± 0.86^{b}
C	22.45 ± 1.23^{c}	6.23 ± 0.95^{c}	8.22 ± 1.11^{c}	12.23 ± 1.67^{c}
D	26.32 ± 2.01^{d}	8.89 ± 1.10^{d}	10.22 ± 1.10^{d}	8.03 ± 0.98^{d}
E	23.12 ± 1.97^{c}	6.47 ± 0.87^{c}	10.01 ± 0.99^{d}	10.22 ± 2.18^{e}
F	22.30 ± 1.89^{c}	5.68 ± 0.96^{ce}	8.98 ± 1.87^{c}	12.45 ± 1.64^{ac}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$). key: A- Control B – Untreated C –Atorvastatin (0.06 mg/kg bdwt) treated D F – 6.25%, 12.5% and 25% inclusion of *Launaea taraxacifolia* leaf-based diet

In vivo antioxidant status of selected tissues of rats of high fat diet-induced hyperlipidemic rats treated with Launaea taraxacifolia leaf-based diet: The above is presented in Figures 2-4. A significant increase ($p < 0.05$) was observed in the activities of the three antioxidant enzymes in all the tissues assayed in the groups fed with the leaf-based diet at all inclusions when compared with the group fed on high fat diet and not treated.

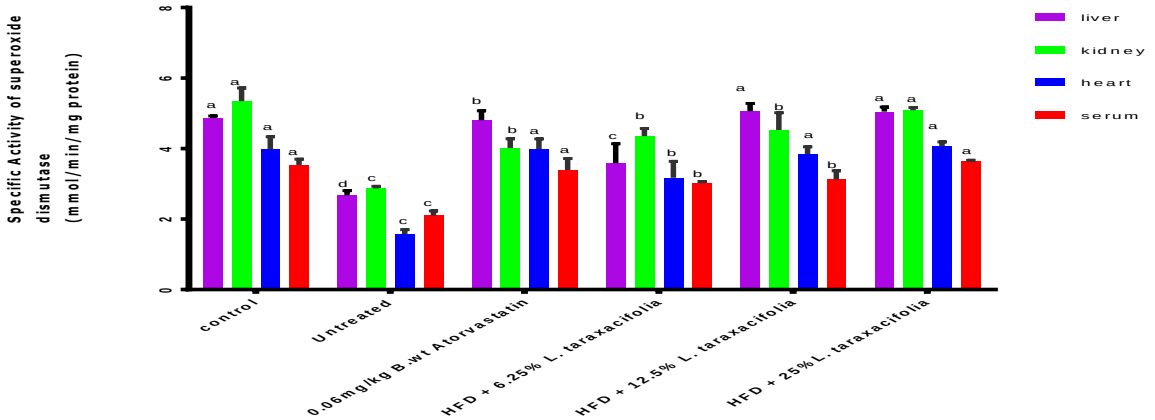

Figure 2: Superoxide dismutase activity of some selected tissues of rats of high fat diet-induced hyperlipidemic rats treated with *Launaea taraxacifolia* leaf-based diet

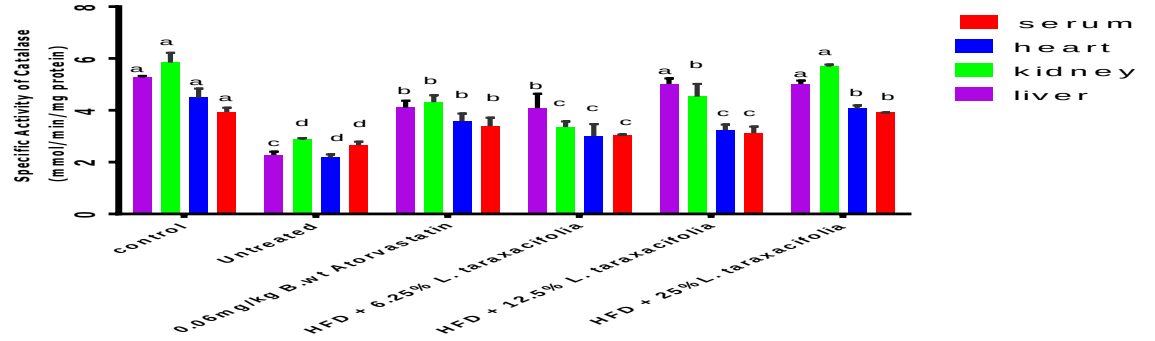

Figure 3: Catalase activity of some selected tissues of rats of high fat diet-induced hyperlipidemic rats treated with *Launaea taraxacifolia* leaf-based diet

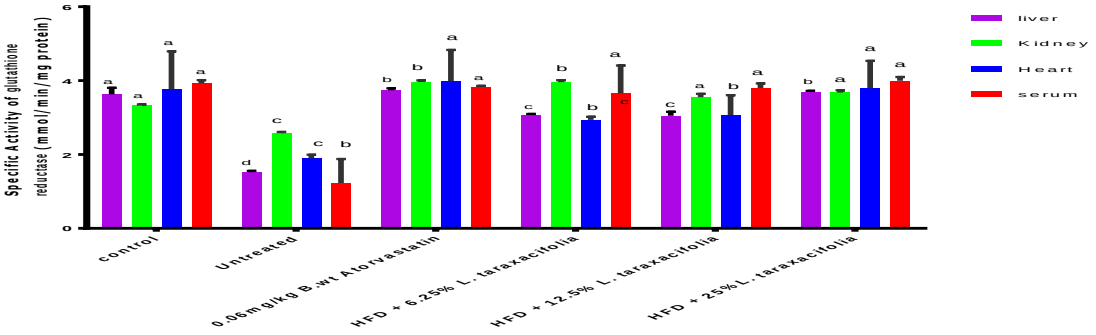

Figure 4: Glutathione reductase activity of some selected tissues of rats of high fat diet-induced hyperlipidemic rats treated with *Launaea taraxacifolia* leaf-based diet

Serum malondialdehyde and nitric oxide concentration of rats of high fat diet-induced hyperlipidemic treated with Launaea taraxacifolia leaf-based diet: Serum malondialdehyde and nitric oxide concentration of rats of high fat diet-induced hyperlipidemic treated with *launaea taraxacifolia* leaf-based diet is presented in Table 9. There was a significant increase ($p < 0.05$) in the malondialdehyde concentration of rats fed on high fat diet and not treated when compared with the control, those treated with the standard drug and *launaea taraxacifolia* leaf-based diet at all inclusion level while the reverse is the case of nitric oxide.

Table 9: Serum malondialdehyde and nitric oxide concentration of high fat diet-induced hyperlipidemic treated with *Launaea taraxacifolia* leaf-based diet

Group	Malondialdehyde (mmol/mg protein)	Nitric oxide (mg/dl)
A	2.64 ± 0.49^{a}	29.83 ± 1.34^{a}
B	5.43 ± 1.02^{b}	46.04 ± 2.28^{b}
C	2.98 ± 0.56^{a}	31.23 ± 1.98^{a}
D	3.58 ± 0.78^{b}	33.21 ± 1.87^{ac}
E	3.17 ± 0.56^{b}	30.45 ± 1.56^{a}
F	2.88 ± 0.87^{a}	32.34 ± 2.11^{a}

Values are expressed as means of five replicates ± S.E.M and those with different superscripts across the column are significantly different ($p > 0.05$). Key: A- Control B - Untreated C - Atorvastatin (0.06 mg/kg bdwt) treated D F – 6.25%, 12.5% and 25% inclusion of *Launaea taraxacifolia* leaf-based diet

Serum leptin concentration of rats of high fat diet-induced hyperlipidemic treated with Launaea taraxacifolia leaf-based diet: Serum leptin concentration of rats of high fat diet –induced hyperlipidemic treated with *launaea taraxacifolia* leaf-based diet is presented in Figure 5. There was a significant decrease ($p < 0.05$) in the serum leptin concentration of rats fed on high fat diet when compared with those control, those treated with the standard drug and the *launaea taraxacifolia* leaf-based diet at all inclusion levels. The groups fed on 6.25% and 12.5% inclusion levels compared favourably ($p > 0.05$) with each other and that of the standard drug group. Likewise, the group fed on 25% of the diet also compared favourably ($p > 0.05$) with the control group.

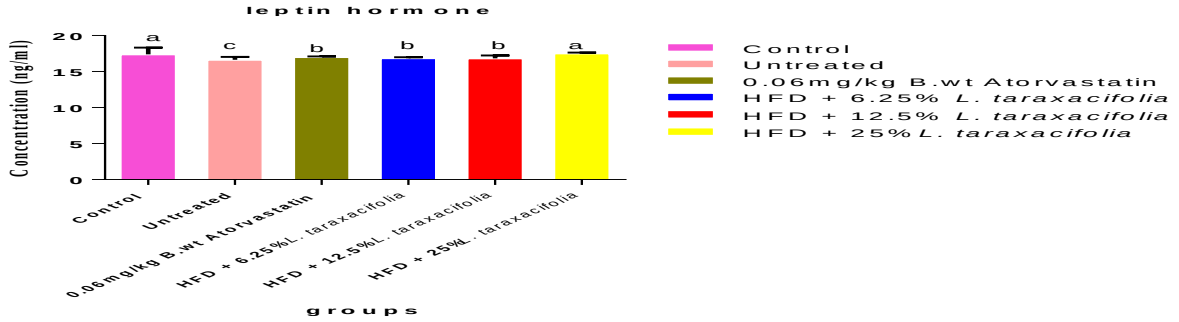

Figure 5: Serum leptin concentration of rats of high fat diet-induced hyperlipidemic treated with *Launaea taraxacifolia* leaf-based diet

Discussion

This study presents the outcomes of treatment of high fat diet-induced hyperlipidemia with *launaea taraxacifolia* leaf-based diet at 6.25%, 12.5% and 25% inclusion levels. Several studies have suggested that long-term feeding with a high-fat diet results in the successful induction of a reliable model of hyperlipidemia in rodents (Marques *et al.*, 2015; Poznyak *et al.*, 2020). This is the method we have adopted for this study and has been confirmed by the derangement of serum lipid profile and the significant increase in the growth performance characteristics (body weight gain, body mass index and the hip circumference) after feeding on high fat diet for six weeks. It is well documented that elevated levels of lipid parameters, particularly values of total cholesterol and triglycerides, are major hyperlipidemic markers (Pehlivanovi´c Kelle *et al.*, 2024) and increase in these markers have further proven that hyperlipidemia has been established. Growth performance / anthropometric indices are simpler and non-invasive tests which can be applied to predict lipid profile abnormality and at-risk population for future cardiovascular and other endocrine events. Hence the study correlate simple anthropometric measurements with lipid profile parameters which further buttress its use to confirm the state of hyperlipidemia (Reddy and Nambiar, 2017).

In hyperlipidemic condition, enzymatic (superoxide dismutase, catalase, glutathione reductase) as well as non-enzymatic (reduced glutathione) antioxidative defense system is altered leading to reactive oxygen species mediated damage (Devi and Sharma, 2004). In same vein, oxidative stress causes the lipids in the membrane to mutate thereby leading to abnormal lipid metabolism (Lai *et al*., 2023). This therefore necessitate the need to assay for the *in-vivo* antioxidation status of animals fed on high fat diet. Likewise , lipid peroxidation marker, malondialdehyde, is commonly used as a measure of the oxidative stress in cells has been proven to be elevated in hyperlipidemic condition while nitric oxide acts as a potent inhibitor of the lipid peroxidation reaction by scavenging propagatory lipid peroxyl radicals (Hogg and Kalyanaraman, 1999).

Leptin, an hormone, inhibits fatty acid synthase in the adipocytes, contributing to an overall fat mass reduction has been linked in many literature to hyperlipidemia (Chandralekshmy, 2024). It does this by acting as an inducer of lipoprotein lipase production which significantly decreses plasma triglycerides (Stary, 1994) and also blocks the overaccumulation of triglycerides in nonadipocytes confining its storage to fat cells (Chandralekshmy, 2024). Therefore, its deficiency results in excess fat results in excess fat deposition and damage to nonadipocytes.

The leaf of *Launaea taraxacifolia* has been scientifically proven to contain various nutritional/ chemical constituents (Adinortey *et al.*, 2013, Arawande *et al*., 2013 and Oyegoke e*t al*., 2021). The results reveal the presence of crude fibre, phenolics, tannins, triterpenoid, flavonoids, coumarins and many vitamins in appreciable amounts. The results of Oyegoke *et al*., 2021 also revealed that the leaves possess high antioxidant activity after assaying for the *in-vitro* antioxidant potential which could be helpful in preventing or slowing the progression of various oxidative stress related disorders. The nutritional/chemical constituents of the leaf might therefore be responsible for its ant-hyperlipidemic activity which might work through the antioxidant mechanism or others. The high quantity of polyphenolic compounds and vitamin Cand E reported by Oyegoke *et al*., 2021 might be used to support the antioxidant mechanism of action of its hyperlipidemic activity . Polyphenols exhibit its antioxidant property by its hydrophobicity which is intermediate between that of vitamic C (highly hydrophilic) and that of vitamin E (highly hydrophobic). Polyphenols are thus expected to be involved in oxidation regeneration pathway with vitamin C and E (Manach, 2004).

The significant increase in the feed intake of rats fed on high fat diet only which were not treated and those treated with *Launaea taraxacifolia* leaf- based at all inclusions when compared with the control might be due to the fact that the leaf of the plant contains substaintial amount of vitamins, minerals and crude fibre which plays a crucial role in stimulating the appetite of the rats (Dutta *et al*., 2004) while that of those fed on high fat diet might be due to high fat contant as fats has been proved to increase the palatability of food and also has a weak-satiety signaling property (Erlanson-Albertsson, 2010). The increase in weight gain after feeding with high fat diet resulted from the fact that dietary fat is the most energy-dense macronutrients therefore consumption of it resulted in high weight gain which resulted into obesity and hyperlipoproteinemia but this was averted by the leaf-based diet which might be due to presence of some lipid lowering compounds in it such as alkaloids polyphenols, flavonoids and saponins (Lansky, 1993, Haliwell, 1994, Lipkin, 1995, Jeffery and Harborne, 2000). Body mass index and hip circumferences are two anthropometric data that has been linked to obesity and hyperlipidemia conditions as they are used to distinguish between those at increased risk as a result of "abdominal fat distribution" , android obesity or gynoid fat distribution (Reddy and Nambiar, 2017) thereby also predicting lipid profile abnormality. The reduction in their levels in rats fed the leaf based-diet of *Launaea taraxacifolia* acts as pointer that hyperlipidemia has been alleviated. The leaf based-diet was able to decrease the concentration of total cholesterol, low density lipoprotein cholesterol and triglyceride while increasing that of high-density lipoprotein cholesterol. and this implies that there is a continous transport of excess cholesterol to the liver for excretion into the bile, thereby reducing the risk of hypertension and atherosclerosis (Patil *et al*., 2004).

The enhancement of the *in-vivo* antioxidant status of the animals fed on the leaf-based diet can be extrapolated to the results of the research carried out by Oyegoke *et al*. (2021) in which the leaf of *launaea taraxacifola* was able to scavenge free radicals such as 2,2 –Diphenyl -1- picrylhydrazyl, Nitric oxide, hydrogen peroxide, ferric reducing antioxidant power e.t.c. proving its antioxidant capacity. Likewise , its ability to increase the concentration of leptin hormone at the three inclusion levels signifies that induction of lipoprotein lipase is activated which decreases triglycerides and in turn prevents hyperlipidemia. Overall , the results from this research work confirmed the anti-hyperlipidemic effect of *Launaea taraxacifolia* leaf-based diet through its import on the growth performance characteristics and antioxidant potentials.

Conclusion

The present study concludes that the leaf of *Launaea taraxacifolia* exhibits anti-hyperlipidemic effect, enhanced growth performances and *in-vivo* antioxidants upmost at 12.5% and 25% inclusion in the diet.

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