Effect of Raw and Cooked Ginger (*Zingiber officinale* Roscoe) Extracts on Serum Cholesterol and Triglyceride in Normal and Diabetic Rats

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Abstract Dyslipidemia is a risk factor of concern in public health even in a state of anomaly in blood glucose level. The effect of different ginger extracts on body lipids has been reported, few scientific studies have documented the effect of raw ginger extracts while the effect of cooked ginger extract (the form in which the spice is commonly consumed) is yet to be explored. This experimental study was therefore designed to determine the effect of raw and cooked ginger extracts on serum triglycerides and total cholesterol in diabetic rats and compare this with that of non-diabetic rats. Fresh ginger rhizomes were washed peeled, washed, wet-milled (without addition of water) and sieved to give the raw extract. A portion of of the extract was boiled for one hour and cooled to give cooked ginger extract. Seventy male albino rats of weight range 155g-195g were divided into 7 groups (n=10) with 3 groups kept as normal or non-diabetic while the remaining 4 groups were rendered diabetic by intraperitoneal administration of 60mg/kg body weight of streptozocin (STZ) to mimic Type 1 diabetes mellitus. These were repeated with another set of seventy rats but diabetes was induced in the 4 diabetic groups here with a 12 week consumption of high-fat diet (HFD) to mimic Type 2 diabetes mellitus. Serum total cholesterol and triglyceride were determined before and after diabetes induction as well as at the 2nd and 4th weeks of extracts and diabetic drugs administration using standard methods. Mean data were compared using Least Significant Difference at p≤0.05.Raw and cooked ginger extracts significantly reduced serum total cholesterol and triglyceride in both medium (2 weeks) and long (4 weeks) terms administration in non diabetic rats. In STZ-induced diabetic rats only raw ginger extract lowered these parameters significantly while the cooked extracts and glybenclamide increased these significantly. However, in HFD-induced diabetic rats, raw, cooked ginger extracts and Metformin reduced both serum total cholesterol and triglyceride significantly. Ginger in both raw and cooked forms may therefore be useful in ameliorating hyperlipidemia which commonly associates diabetic state. Human trial is hereby recommended.

Keywords: raw ginger, cooked ginger, diabetic rats, serum lipids

1. Introduction

Diabetes mellitus which is a group of metabolic disorders resulting into high blood glucose is gradually becoming a public health threat. In 1997 it was estimated that 124 million people had diabetes globally and this was projected to increase to 221 million by 2010 [1]. A total number of adults with diabetes in the world was estimated to be 135 million in 1998 and this was projected to increase to 300 million by 2025 [2]. In the same vein, the total number of people with diabetes globally was projected to increase from 171 million in 2000 to 388 million in 2030 for all age groups [3]. Similarly, Shaw et al, [4] reported that 285 million adults (20-79 years) had diabetes globally and this may increase to 439 million by 2030. Furthermore, an estimate of 366 million people was reported to have

diabetes worldwide in 2011 and by 2030 this may increase to 552 million [5]. In a more recent study, it was estimated that 415 million adults had diabetes in 2015 and this was projected to increase to 642 million by 2040 [6]. All these scientific reports emphasized the fact that people with diabetes will continually increase globally and this may be attributed to technological advancement leading to increase in civilization thus encouraging sedentary lifestyle and increase energy density of diet that culminates into predisposition to obesity and diabetes [7]. In addition to this, an adverse intrauterine environment and the resulting epigenetic changes or alteration could also be responsible especially in developing countries [8], Since diabetes is of public health concern, there is need to exhaustively explore all possible approach towards alleviating this social ill and its associating risk factors.

Dyslipidemia is a state of abnormal level of blood lipids such a s triglycerides, total cholesterol and/or

phospholipids. It can express itself as hyperlipidemia or hypolipidemia. Diabetic state is commonly associated with hyperlipidemia (hypertriglyceridemia and hypercholesterolemia) which maybe expressed as elevated level of triglyceride and low density lipoprotein cholesterol (LDL-C) and reduced level of high density lipoprotein cholesterol (HDL-C) in the blood that may respond to dietary, hormonal and drug treatments without spontaneously developing into atherosclerosis [9] while it may concomitantly result into cardiovascular disease [10]. Dyslipidemia in Type 1 diabetes may be diagnosed by an LDL-C >130mg/dL and HDL-C <35mg/dL [11] while in Type 2 diabetes it may be diagnosed by blood triglyceride level of >200mg/dL and HDL cholesterol <40mg/dl [12].

The onset of dyslipidemia in Type 2 diabetes has been traced to increased liver production of apolipoprotein-B (apoB) which is the major protein component of very low density lipoprotein (VLDL) and LDL-C thus resulting into hyperlipidemia [13], hence, insulin deficiency or hepatic insulin resistance may increase the secretion of apoB leading to diabetic hyperlipidemia [14]. Also increased lipolysis of adipose tissues due to poor insulin action leading to increased fatty acid transport to the liver which is a common abnormality in insulin resistant state commonly culminates into increased production and release of VLDL into the blood [15]. Furthermore, insulin resistance can also increase the production of other proteins such as apoCIII which affect or influence the circulating levels of lipoprotein. ApoCIII is a small apoprotein that may increase VLDL and LDL-C by preventing the action of lipoprotein lipase (LpL) and inhibiting lipoprotein uptake via the LDL receptor-related protein [16]. Another possible factor for the pathogenesis of diabetic dyslipidemia is the hepatic lipase deficiency which results from insulin resistance or relative insulin deficiency. Hepatic lipase is an enzyme synthesized by hepatocytes and it hydrolyzes phospholipids, triglycerides and remnant lipoproteins thus resulting into clearance of postprandial remnant lipoproteins, hence, the deficiency of this enzyme mostly causes diabetic dyslipidemia [17]. Lipoprotein lipase (LpL) is a major enzyme responsible for the conversion of lipoprotein triglyceride into free fatty acid and is synthesized by adipocytes and myocytes thus preventing hyperlipidemia. In Type 2 diabetic state there has been observed reduced LpL activity in post heparin blood thus initiating diabetic hyperlipidemia [18]. Also, hormone-sensitive lipase (HSSL) which is an enzyme responsible for the conversion of triglyceride into fatty acids and monoglyceride is activated by insuin, hence, in a state of insulin deficiency or insulin resistance there is inhibition of the enzyme action which may culminate into hypertriglyceridemia in diabetic state [19].

The effect of different ginger extracts on blood, serum or plasma lipids has been reported in various research findings. Methanolic ginger extract was observed to ameliorate diabetic dyslipidemia in adult albino rats by controlling all parameters of the ill health state but the combination of the ginger extract with Curcuma longa was more effective [20]. Hydroethanolic ginger extract improved lipid profile and attenuated the increase of plasma glucose in high-fat diet fed male Wistar rats [21]. Aqueous ginger extract inhibited hypertriglyceridemia and reduced atherogenic apolipoprotein-B with a concomitant increase

in HDL-cholesterol thus correcting diabetic dyslipidemia in dexamethasone-induced type 2 diabetic rats [22]. The efficacy of ginger powder in controlling blood lipids in diabetic and hyperlipidemic subjects was also reported by Jafarnejad et al., [23] while Rahimlou et al., [24] observed no change in total cholesterol, HDL and LDL cholesterol but there was a marked reduction fasting blood glucose and triglyceride in patients with non-alcoholic fatty liver disease that were given 2g ginger powder capsule daily compared with the placebo group. Furthermore, ginger powder was reported to notably reduce triglyceride and total cholesterol compared to placebo in patients with type 2 diabetes given 1.6g ginger powder for 12 weeks [25]. Aqueous ginger extract reduced elevated triglyceride, total cholesterol, and very low density lipoprotein which resulted from Propulthiouracil-induced hypothyroidism in male rats [26]. In alcohol-induced dyslipidemia aqueous ginger extract was reported to reduce elevated biomarkers such as fatty acids, triglyceride, total cholesterol, phospholipids and low density lipoprotein cholesterol thus corroborating the reports earlier sited [27]. Even ginger oil was observed to be effective in reducing serum free fatty acids, triglyceride and total cholesterol thus preventing lipid accumulation in high-fat diet-induced non-alcoholic fatty liver disease [28]. There is paucity of the effect of raw (without addition of water) and cooked ginger (the form in which the spice is mostly consumed) extracts on body lipids, hence, this experimental study was designed to determine and compare the effect of raw and cooked ginger extracts on serum triglycerides and total cholesterol in normal, streptozocin-induced and high-fat diet-induced diabetic rats.

2. Materials and Method

2.1. Preparation of Ginger Extracts

The method of Akhani et al, [29] which was used by Elshater et al, [30] was used with slight modification.

2.2. Raw Extract

Fresh ginger rhizomes (Roscoe variety) were purchased from Bodija market in Ibadan, Nigeria. This was identified and authenticated in the Herbarium Unit of the Department of Botany, University of Ibadan. The rhizomes were washed, peeled, washed and wet-milled (without the addition of water) with a plate attrition mill (Amuda Plate mill, India). This was then sieved with a cheese or muslin cloth and the extract was stored in tightly closed plastic jars at 2°C (at the closest part to the freezer in a refrigerator) until use.

2.3. Cooked (Boiled) Extract

Raw extract was boiled at the medium heat of a 2-burner Thermo cool gas cooker (India) for 1 hour. This was then cooled and stored in plastic jars at 2°C until use

2.4. Formulation of High-fat Diet (HFD)

The method of Martinello *et al.,* [31] was used with slight modification. The composition was as follows: 45%

normal rat pellets (Lanko feeds, Nigeria), 30% beef tallow, 20% full cream milk powder (Real Milk, Chellarams) and 5% table sugar. This was formulated weekly to prevent deterioration due to rancidity.

2.5. Collection of Rats

Four weeks old male albino rats (140) of weight range 50-55g were purchased from the Experimental Animal Unit of the Department of Veterinary Physiology, University of Ibadan, Nigeria. These were raised to desired weight range before experimentation commenced. Animals were fed normal rat pellets and tap water ad libitum and were treated according to the research protocol as approved by the U.I/ U.C.H. Ethics Review Committee (Ethical Approval Number- NHREC,05/01/2008a).

2.6. Induction of Diabetes Mellitus

Experimental DM of the two major types (Type 1 and Type 2) was induced in the animals.

2.7. Streptozocin-induced Diabetic Group (to Mimic Type 1 Diabetes Mellitus)

Seventy rats of weight range 155-195g were placed in this group. Forty of the rats were rendered diabetic with intraperitoneal injection of streptozocin (Sigma Aldrich, Germany) at the dose of 60mg/kg body weight [32] in freshly prepared citrate buffer (pH4.5). Fasting blood glucose (FBG) was monitored for 3 days with ACCU-CHEK Active Glucometer (Roche, Germany) until stable hyperglycemia- FBG ≥ 170mg/dl was attained [33]. The 70 rats in this group were divided into 7 groups and were designated thus:

Group A

NTC- Non-diabetic control group;

NTR- Non-diabetic rats given raw ginger juice (4ml/kg body weight);

NTCo- Non-diabetic rats given cooked ginger;

TC- Diabetic control group;

TR- Diabetic rats given raw ginger juice

TCo- Diabetic rats given cooked ginger juice;

TD- Diabetic rats given glibenclamide (anti-diabetic drug at 5mg/kg body weight).

2.8. High-fat Diet (HFD)-induced Diabetic Group (to Mimic Type 2 Diabetes Mellitus)

Seventy rats of weight range 120-160g were used. Hyperglycaemia was induced in forty of the rats by feeding them with the High-Fat Diet (HFD) for 12 weeks while the remaining 30 rats were fed normal rat pellets (Lanko feeds, Nigeria). After 12 weeks HFD consumption animals with FBG ≥170mg/dl were used for further experimentation. The rats in this group were divided into seven groups and designated thus:

Group B

NT₂C- Non-diabetic control group;

NT_2R- Non-diabetic rats given raw ginger extract (2ml/kg body weight);

NT_2Co- Non-diabetic rats given cooked ginger extract;

T_2C- Diabetic control group;

T_2R- Diabetic rats given raw ginger juice (2ml/kg body weight);

T_2Co-Diabetic rats given cooked ginger extract;

T_2D- Diabetic rats given Metformin (200ng/kg body weight).

2.9. Extracts' Administration

Raw and cooked ginger extracts (4ml/kg body weight) and the anti-diabetic drugs (glibenclamide- 5mg/kg body weight and Metformin- 200mg/kg body weight) were administered once daily for 4 weeks with gastric canula according to the designation of the groups above. The extracts' dosage was reduced to 2ml//kg body weight in the HFD-induced diabetic groups because the animals were showing signs of toxicity with bloodied eye lids after 2 days of administration of 4ml/kg body weight dose)

2.10. Serum Preparation

Blood samples (about 1.2ml) were taken with sterile capillary tubes via retro orbital plexus before and after diabetes induction and at the 2nd and 4th weeks of extracts' administration and collected in plain sample bottles. These were centrifuged at 10,000rpm for 15 minutes at 4°C. The supernatant was aspirated with sterile needle and syringe and kept in eppendorf tubes. Serum triglyceride and cholesterol were analyzed immediately while the remaining serum was stored at -20°C for further analyses. The rats were sacrificed at the end of the experimentation.

2.11. Serum Triglyceride

Randox kit, UK (TR213) was used to determine serum triglyceride and the procedure as stipulated by the manufacturer was followed. To 1ml of reagent 10µl of sample was added. This was then incubated for 10 minutes at room temperature (25°C). Colour changed from colourless to pink or violet and the optical density was read at 546nm. This procedure was repeated for standard and blank using the standard and distilled water respectively in place of the sample. Serum triglyceride was then calculated thus:

$$\text{Serum triglyceride} = \frac{\left[\begin{array}{l}\text{Optical density}(\text{sample})\\\times\text{concentration of standard}(\text{mg}/\text{dl})\end{array}\right]}{\text{Optical density (standard)}}$$

2.12. Serum Total Cholesterol

Randox kit, UK (CH201) was used to determine serum cholesterol and the procedure as stipulated by the manufacturer was followed. To 1ml of reagent 10µl of sample was added. This was then incubated for 10 minutes at room temperature (25°C). Colour changed from colourless to pink or violet and the optical was read at 546nm. This procedure was repeated for standard and blank using the standard and distilled water respectively in place

of the sample. Serum cholesterol was then calculated thus:

$$\text{Serum cholesterol} = \frac{\left[\begin{array}{l}\text{Optical density (sample)} \\ \times \text{concentration of standard (mg / dl)}\end{array}\right]}{\text{Optical density (standard)}}.$$

3. Result

3.1. Effect of Raw and Cooked Ginger Extracts on Serum Total Cholesterol and Triglyceride in Normal and STZ-induced Diabetic Rats

Data are expressed in mean± standard deviation.

Four weeks cooked ginger extract and drug administration resulted in significant increased in the serum cholesterol while the raw extract exerted a significant reduction in this parameter.

There was no significant difference in serum triglyceride before rendering the animals diabetic. After the animals were rendered diabetic by intraperitoneal injection of STZ serum triglyceride in the diabetic groups was slightly lower than that of the non-diabetic groups but this reduction was not significant (Table 2) except in diabetic rats given raw ginger extract. In the diabetic groups four weeks administration of cooked ginger extract and glibenclamide resulted in a significant increase in serum triglyceride while the raw extract reduced it significantly (Table 2).

3.2. Effect of Raw and Cooked Ginger Extracts on Serum Total Cholesterol and Triglyceride in Normal and HFD-induced Diabetic Rats

Data are expressed in mean± standard deviation.

Before the introduction of high-fat diet, serum cholesterol ranged from 68.17 to 70.83 mg/dL with no significant difference between the groups. Twelve weeks HFD consumption exerted a significant increase in serum total cholesterol in the four diabetic groups while after four weeks extracts and drug administration, there existed a significant reduction in serum cholesterol in the diabetic groups so treated (Table 3).

Table 1. Effect of raw and cooked ginger extracts on serum total cholesterol (mg/dL) in normal and STZ- induced diabetic rats

Groups	Cholesterol before diabetes induction	Cholesterol after diabetes induction	Cholesterol at 2 weeks extracts administration	Cholesterol at 4 weeks extracts administration
NTC	70.0 ± 3.16	69.0 ± 3.63	71.2 ± 2.56	70.2 ± 5.49
NTR	70.5 ± 4.09	69.3 ± 4.23	66.8 ± 2.64*	65.3 ± 3.01*
NTCo	70.2 ± 5.38	67.3 ± 2.58	57.3 ± 5.82*	55.8 ± 4.22*
TC	73.2 ± 7.22	72.8 ± 2.71	70.8 ± 3.19	68.7 ± 2.07
TR	71.7 ± 2.42	67.8 ± 4.96*	41.8 ± 1.47*	40.7 ± 1.86*
TCo	73.3 ± 3.45	67.5 ± 4.37*	80.2 ± 2.93*	82.5 ± 2.51*
TD	71.5 ± 2.17	75.0 ± 3.29*	74.3 = 2.50*	72.5 ± 1.52*

* -significantly different from non-diabetic and diabetic control (data in same column) p≤0.05; NTC- non-diabetic control; NTR- non-diabetic rats given raw ginger extract; NTCo- non-diabetic rats given cooked ginger extract; TC- diabetic control; TR- diabetic rats given raw ginger extract; TCo- diabetic rats given cooked ginger extract; TD- diabetic rats given antidiabetic drug.

Table 2. Effect of raw and cooked ginger extracts on serum triglyceride (mg/dL) in normal and STZ-induced diabetic rats

Groups	Triglyceride before diabetes induction	Triglyceride after diabetes induction	Triglyceride at 2 weeks extracts administration	Triglyceride at 4weeks extracts administration
NTC	62.5 ± 4.64	62.2 ± 1.33	64.7 ± 3.50	64.7 ± 2.94
NTR	63.2 ± 2.71	63.8 ± 4.54	56.0 ± 3.46*	52.7 ± 2.58*
NTCo	63.2 ± 3.67	59.3 ± 5.75	45.8 ± 4.26*	45.7 ± 4.59*
TC	64.0 ± 5.05	58.7 ± 4.41	56.8 ± 2.64	57.3 ± 5.99
TR	64.3 ± 3.67	54.3 ± 3.44*	36.2 ± 3.82*	34.0 ± 3.29*
TCo	64.0 ± 2.76	59.8 ± 3.92	71.3 ± 2.50*	73.5 ± 1.76*
TD	63.3 ± 3.01	58.5 ± 4.32	62.5 ± 2.59*	83.0 ± 2.61*

* --significantly different from non-diabetic and diabetic control (data in column) p≤0.05; NTC- non-diabetic control; NTR- non-diabetic rats given raw ginger extract; NTCo- non-diabetic rats given cooked ginger extract; TC- diabetic control; TR- diabetic rats given raw ginger extract; TCo- diabetic rats given cooked ginger extract; TD- diabetic rats given antidiabetic drug.

Table 3. Effect of raw and cooked ginger extracts on serum total cholesterol (mg/dL) in normal and HFD-induced diabetic rats

Groups	Cholesterol before HFD	Cholesterol at 12 weeks HFD	Cholesterol at 2 weeks extracts administration	Cholesterol at 4 weeks extracts administration
NT$_2$C	70.2 ± 6.11	70.3 ± 4.13	70.7 ± 3.72	72.2 ± 2.04
NT$_2$R	70.8 ± 5.91	71.5 ± 4.64	70.7 ± 1.37	72.3 ± 4.23
NT$_2$Co	68.2 ± 3.87	69.0 ± 4.29	71.2 ± 3.25	72.0 ± 4.81
T$_2$C	70.0 ± 4.05	83.8 ± 3.98*	90.2 ± 1.47	94.7 ± 2.42
T$_2$R	68.3± 4.33	81.8 ± 3.66*	83.3 ± 1.37*	84.0 ± 3.35*
T$_2$Co	70.3 ± 3.78	86.5 ± 2.81*	83.1 ± 1.27*	83.5 ± 2.67*
T$_2$D	69.2 ± 1.32	83.8 ± 2.99*	81.0 ± 1.55*	78.2 ± 2.04*

* significantly different from non-diabetic and diabetic control (data in same column) p≤0.05; NT$_2$C- non diabetic control; NT$_2$R- non diabetic control given raw ginger extract; NT$_2$Co- non diabetic rats given cooked ginger extract; T$_2$C- diabetic control; T$_2$R- diabetic rats given raw ginger extract; T$_2$Co- diabetic rats given cooked ginger extract, T$_2$D- diabetic rats given anti-diabetic drug.

Table 4. Effect of raw and cooked ginger extracts on serum triglyceride (mg/dL) in normal and HFD-induced diabetic rats

Groups	Triglyceride before HFD	Triglyceride at 12 weeks HFD	Triglyceride at 2 weeks extracts administration	Triglyceride at 4 weeks extracts administration
NT$_2$C	64.2 ± 4.67	65.0 ± 3.03	71.5 ± 4.32	72.2 ± 2.04
NT$_2$R	62.8 ± 3.54	66.0 ± 3.74	67.7 ± 4.13*	72.3 ± 4.23
NT$_2$Co	64.2 ± 2.64	63.7 ± 2.88	67.7 ± 4.72*	72.0 ± 4.81
T$_2$C	63.5 ± 2.74	86.7 ± 2.66*	91.8 ± 2.14	94.7 ± 2.42
T$_2$R	64.2 ± 3.82	84.8 ± 3.19*	80.5 ± 2.17*	84.0 ± 3.35*
T$_2$Co	65.2 ± 2.93	86.5 ± 3.21*	87.3 ± 1.21*	83.5 ± 2.67*
T$_2$D	65.5 ± 3.62	84.2 ± 2.43*	83.7 ± 2.81*	78.2 ± 2.04*

* significantly different from non-diabetic and diabetic control (data in same column) p≤0.05; NT$_2$C- non diabetic control; NT$_2$R- non diabetic control given raw ginger extract; NT$_2$Co- non diabetic rats given cooked ginger extract; T$_2$C- diabetic control; T$_2$R- diabetic rats given raw ginger extract; T$_2$Co-diabetic rats given cooked ginger extract, T$_2$D- diabetic rats given anti-diabetic drug.

Serum triglyceride witnessed a reducing trend similar to that of serum cholesterol after 4 weeks ginger extracts and drug administration (Table 4).

4. Discussion

4.1. Effect of Raw and Cooked Ginger Extracts on Serum Total Cholesterol and Triglyceride in Normal and STZ-induced Diabetic Rats

4.1.1. Serum Total Cholesterol

No significant difference existed in serum total cholesterol in all the groups before the induction of diabetes but this parameter reduced significantly in diabetic rats to be given raw and cooked extracts after the animals have been rendered diabetic before the introduction of the extracts while in the diabetic rats to be given drug serum cholesterol did not differ significantly from that of diabetic control (Table 1).

After 2 weeks of extracts administration, raw and cooked ginger extracts reduced serum cholesterol by 6.1 and 19.43% respectively in non-diabetic groups while in the diabetic groups only raw extract reduced this parameter by 40.94%. The cooked extract and glibenclamide increased serum cholesterol by 13.19 and 4.94% respectively relative to the diabetic control as can be deduced from Table 1. This shows that raw and cooked ginger extracts exerted a reducing effect on serum cholesterol in non-diabetic while only raw ginger extract exerted similar effect in diabetic state.

At 4 weeks of extracts administration the effect exerted on serum cholesterol was similar to that observed at 2 weeks showing that medium and long term administration of ginger extracts exert similar effect on serum cholesterol. The reduction in serum cholesterol by raw ginger extract in diabetic rats in this study corroborates the reports of Ozougwu and Eyo, [34]; Ramudu *et al*., [35] and Al-Hoory *et al*., [36]. They reported a significant reduction in serum cholesterol as a result of administration of aqueous, ethanolic and fresh ginger extracts respectively in alloxan-induced diabetic rats while Saied *et al.,* [37] observed similar effect in broiler chicken that were given aqueous ginger extract.

It can therefore by inferred from this study that only raw ginger extract could ameliorate the state of hypercholesterolemia commonly associated with diabetic state.

4.1.2. Serum Triglycerides

At two weeks extracts and drug administration serum triglycerides reduced by 25.00% and 38.62% in non-diabetic rats given raw and cooked ginger extracts respectively as can be deduced from Table 2. On the other hand, in the diabetic groups this parameter reduced significantly by 36.35% only in diabetic rats given raw ginger extract while in the rats that were given cooked extract and drug there was marked increase of 25.51% and 9.98% respectively (Table 2). These show that ginger in both raw and cooked forms are capable of reducing serum triglyceride significantly in non-diabetic state though the effect of the cooked form is higher than that of raw form. However in the diabetic state only the raw form of the spice exerted a significant reduction in serum triglyceride though this parameter in the diabetic groups was significantly lower than that of the non-diabetic control in this study (Table 2). At 4 weeks of extracts' and drug administration the effect on serum triglyceride was similar to that of the second week. This shows that medium and long term administration of the extracts and drug exerted similar effect.

The effect exerted by raw ginger extract in this study on diabetic rats is similar to the report of Ramudu *et al*., [35] who observed a reduction in serum triglyceride in STZ-induced diabetic rats that were given 200mg/kg body weight of ethanol ginger extracts for 30 days. They also reported a reduction in this parameter by glibenclamide but this was in conflict with the result of this study. Similar effect was observed by Al-Hoory *et al*., [36] who reported a reduction in serum triglyceride in alloxan-induced diabetic rats after 4 weeks of raw ginger extract administration at a dosage of 500mg/kg body weight. On the other hand the significant increase in serum triglyceride by cooked ginger extract corroborates the report of Gao *et al.,* [38] who observed an increase in plasma triglyceride in fructose-induced hypertriglyceridemic rats by 5 weeks oral administration of alcoholic ginger extract at 50mg/kg body weight.

4.2. Effect of Raw and Cooked Ginger Extracts on Serum Cholesterol and Triglyceride in Normal and High-fat Diet-induced Diabetic Rats

4.2.1. Serum Cholesterol

Twelve weeks HFD consumption exerted a significant increase in serum cholesterol in the four diabetic groups

while there was no significant difference in this parameter before the introduction of the HFD (Table 3). At 2 weeks extracts' administration there existed no significant difference in serum cholesterol in the non-diabetic groups (control and treated). This shows that ginger extract did not alter serum cholesterol in these groups of non-diabetic rats that were 7 months old. However, in the diabetic groups raw, cooked ginger extracts and Metformin reduced serum cholesterol significantly by 7.59%, 7.95% and 10.17% respectively showing that the both extracts exerted similar effect on this parameter in HFD-induced diabetic rats.

At 4 weeks extracts' administration, both extracts did not alter serum cholesterol in the non-diabetic groups while in the diabetic groups raw , cooked ginger extracts and Metformin reduced this parameter by 11.27%, 11.80% and 17.48% respectively as can be deduced from Table 3. This also shows a reducing effect in similar trend as is the case at 2 weeks of extracts' administration. This reduction in serum cholesterol corroborates the report of Saied *et al.*, [37] who observed a 26.64% reduction in serum cholesterol in broilers given aqueous ginger extract [37] while Mahluji *et al.*, [39] reported no significant change in serum cholesterol in Type 2 diabetic patients treated with 2g ginger powder/day though there was marked improvement in insulin sensitivity. This could be that the dose was too low to effect change in serum cholesterol. This reduction in serum cholesterol also corroborates the reports of Ismail, [40] who observed a 16.36% reduction in serum cholesterol of HFD/alloxan-induced diabetic rats by 6 weeks administration of aqueous ginger extracts at 200mg/kg body weight.

4.2.2. Serum Triglyceride

Before the introduction of HFD there was no significant difference in the serum triglyceride in all the groups (Table 4) while at the 12th week of HFD consumption, there was a marked significant increase in serum triglyceride compared to the rats that were fed normal rat chow (Table 4). This shows that HFD consumption is able to raise serum triglyceride. Two weeks administration of raw and cooked ginger extracts reduced serum triglyceride by 5.31% in non-diabetic rats while in the diabetic groups this reduction was 12.34%, 4.90% and 8.89% in rats given raw, cooked extracts and drug respectively relative to the diabetic control.

At 4 weeks extracts and drug administration, there was no significant change in serum triglyceride in the non-diabetic control group and the non-diabetic groups that were given ginger extracts. This shows that long term administration of ginger extracts may not alter serum triglyceride in non-diabetic state. In the diabetic rats serum triglyceride reduced by 20.04, 7.38 and 12.82% in rats that were given raw, cooked extracts and metformin respectively showing the raw extract exerting the highest reducing effect on serum triglyceride in the diabetic groups. This reduction in serum triglyceride corroborates the reports of Ismail, [40] who observed a reduction in serum triglyceride from 100.0mg.dl to 72.0mg/dl in HFD/alloxan-induced diabetic rats by aqueous ginger extract given at 200mg/kg body weight for 6 weeks. Similarly, a reduction in serum triglyceride by oral administration of ginger powder in Type 2 diabetic rats

was reported by Arablou *et al.*, [25] and Mahluji *et al.,* [39].

5. Conclusion and Recommendation

Raw and cooked ginger extracts lowered serum total cholesterol and triglyceride significantly in non diabetic rats and high-fat diet-induced diabetic rats but only the raw extract of this spice exerted similar effect in streptozocin-induced diabetic rats, hence, ginger consumption in either raw or cooked form may be useful in ameliorating dyslipidemia commonly association diabetic state. Human trial is hereby recommended.

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