International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Index Copernicus Value (2015): 78.96 | Impact Factor (2015): 6.391

Effects of Natural Honey and Refined Sugar on the Lipid Profile and Some Atherogenic Indices of Wister Rats

Nwachuku, E. O[1], Okwesum, O[2], Elekima, I[3], Ben-Chioma, A[4]

[1,2,3,4]Department of Medical Laboratory Science, Rivers State University, Nkpolu– Oroworukwo, P.M.B. 5080, Port Harcourt, Nigeria

Abstract: ***The study compared the effect of natural honey and refined sugar on Total cholesterol (TC), Low-density lipoprotein cholesterol (LDL-C), High-density lipoprotein cholesterol (HDL-C), Triglyceride (TG), Atherogenic index (Total Cholesterol/HDL) and Coronary risk index (LDL/HDL) on wister rats. 28 wister rats with body weight average of 200g were divided into seven groups of four each. Group 1 was used as control, three groups were treated with natural honey using low dose, moderate dose and high dose of 0.04g/ml, 0.09g/ml and 0.13g/ml respectively and three groups treated with refined sugar using low dose, moderate dose and high dose of 0.02g/ml, 0.03g/ml and 0.04g/ml respectively. The administration was done orally by gavaging method and after 4 weeks, blood samples were collected for analysis of TC, TG, HDL-C, LDL-C, TC/HDL and LDL/HDL. Results showed significant decreases in TC, TG and HDL-C in rats treated with low dose of refined sugar and HDL-C level in rats treated with moderate dose of refined sugar while significant increases were seen in LDL-C and TC/HDL ratio in rats treated with high dose of refined sugar. In rats treated with natural honey, significantly reduced levels of TC and TG and significantly increased levels of HDL-C were seen in low and moderate doses of honey. However, when moderate doses of refined sugar and honey were compared, significant reduced level of TC/HDL was observed in rats treated with honey. In conclusion, consumption of low and moderate doses of natural honey was shown to reduce cardiovascular risk indicated by decreased TC, TG and increased HDL-C while low, moderate and high dose of refined sugar showed decreased TC, TG, HDL-C and increases in LDL-C and TC/HDL-C levels indicating increased cardiovascular risk.***

Keywords: Natural Honey, Refined Sugar, Lipid Profile

1. Introduction

The lifestyle of modern day man involves a high intake of 'junk' and 'fast foods', with added sugars mainly sucrose and fructose [1]. The metabolism of these nutrients takes place within body cells and tissues and if there is an excess of the undesirable nutrients, depending on source, body homeostasis is consequently altered, and pathophysiological conditions are precipitated [2][3].

Cholesterol is vitally important to a number of human functions including the production of several hormones and is used in cell membranes. However, high cholesterol is a major risk factor for heart disease, the nation's number one cause of death. Nearly all of the energy needed by the human body is obtained from oxidation of carbohydrates and lipids [1]. Sucrose a form of carbohydrate common in diet is widely consumed by humans [4] and its consumption has been linked to various disorders such as diabetes metabolic syndrome, aging and Cancers [5]. Sucrose hydrolysis produces a fructose and glucose equimolar mixture named inverted sugar, which has higher edulcorant power [6].

Honey, a sweet food made by bees using nectar from flowers has been proven to be of medicinal importance both at curative and preventive levels [7]. It is a promising antitumor agent with pronounced anti metastatic and anti angiogenic effects [7]. Honey is produced from many different floral sources and its biochemical and pharmacological activities vary depending on its origin and processing. Honey contains a variety of biologically active compounds such as flavonoids, vitamins, antioxidants as well as hydrogen peroxides (H_2O_2) [8]. The main uses of honey are in cooking, baking, as a spread on bread, and as an addition to various beverages, such as tea, and as a sweetener in some commercial beverages. It consists of primarily sugars such as monosaccharides, disaccharides, oligosaccharides and polysaccharides [9][10]. It contains enzymes such as glucose oxidase, diastase, invertase, catalase and peroxidase. Honey also contains other bioactive constituents such as organic acids, ascorbic acid, trace elements, vitamins, amino acids, proteins and Maillard reaction products. Being rich in carbohydrates like glucose and fructose when ingested could be metabolized to generate energy and help in tissue repair [9].

A lipid profile measures total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, and triglycerides. An extended profile may also include very low density lipoprotein cholesterol (VLDL-C) and Non-HDL-C. Lipoprotein disorder is among the most common metabolic disease occurring in human. It may lead to Coronary heart disease (CHD). Excessive levels of blood cholesterol accelerate atherogenesis and lowering high blood cholesterol reduces the incidence of CHD [1]. Knowledge about the levels of cholesterol sub fractions is more meaningful than simple plasma cholesterol level suggesting that the higher the level of LDL cholesterol, the greater the risk of atherosclerotic heart disease while the higher the level of HDL cholesterol, the lower the risk of coronary heart disease [4].

Blood lipid profile determines the risk of cardiac disease [11]. Lipoprotein disorder is among the most common metabolic disease occurring in human. It may lead to Coronary heart disease (CHD). Honey was reported by some workers to be protective against the hypertriglyceridemic and pro-oxidative effects of fructose [12]. This work was

therefore aimed at comparing the effects of refined sugar and natural honey on the lipid profile of albino rats by estimating concentrations of Total cholesterol (TC),TG, HDL, LDL, and some atherogenic indices such as TC/HDL and LDL/HDL in rats fed with Natural honey and refined sugar.

2. Materials and Method

2.1 Materials

Materials used include centrifuge, spectrophotometer, plain bottles and lithium heparin bottles. The Pure Natural honey and refined sugar were purchased from Ikorodu, Lagos state, Nigeria. Reagents used include total cholesterol, triglyceride, High density lipoprotein reagents purchased from Randox diagnostic, United Kingdom

2.2 Experimental Animals

A total of 28 male wister rats *(Rattus norvegicus)* weighing approximately 200grams were purchased from the Department of Pharmacology, University of Port Harcourt, Nigeria. They were fed with rat feed and water *ad libitum* and allowed to acclimatize for two weeks. Four of these rats were used as controls and the remaining 24 were used as test animals. The albino male rats were weighed at the commencement of the study. The rats were allowed to adapt to the housing conditions and experimental interventions (handling, weighing and gavaging) for 1 week before the commencement of the Experimental protocol. The oral gavaging was done using a plastic orogastric gavaging needle and 1ml hypodermic syringe.

2.3 Experimental design

The rats were divided into seven (7) groups with each group consisting of four (4) rats. Group 1 rats were used as control (0.0g/ml), Group 2 rats were treated with low dose of refined sugar (0.02g/ml), Group 3 rats were treated with moderate dose of refined sugar (0.03g/ml), Group 4 rats were treated with high dose of refined sugar (0.04g/ml), Group 5 rats were treated with low dose of natural honey (0.04g/ml), Group 6 rats were treated with moderate dose of natural honey (0.09g/ml) while Group 7 rats were treated with high dose of natural honey (0.13g/ml). These substances were administered orally, mimicking the usual daily doses and route of intake by humans. The duration of the treatment was for 4 weeks which was performed by oral administration vis-à-vis the use of gavage tube. The gavage technique of administration was adopted to ensure complete delivery of the substances used in the treatment. The treatments were performed daily at approximately the same time.

2.4 Blood Sample Collection and Analysis

At the end of the experiment, blood samples were collected from the rats via cardiac puncture using a 5ml hypodermic syringe. The rats were anaesthetized with chloroform before the cardiac puncture was performed swiftly. Blood sample collected were transferred into lithium heparinised bottles and subsequently were centrifuged to obtain plasma at 4000rpm for 15 minutes. The plasma samples obtained were separated into plain tubes and persevered at 4^{o}C for analyses using a semi-automated chemistry analyser (Map Lab Analyzer) for Total Cholesterol, Triglyceride and High Density Lipoprotein (HDL) while Low Density Lipoprotein (LDL) was calculated using Friedewald *et al*., 1972 equation. The estimation of plasma total cholesterol was based on the enzymatic end point method developed by Richmond (1973) while the estimation of plasma triglyceride was based on glycerol-3-phosphate oxidase-PAP method developed by Trinder (1969) and the estimation of high density lipoprotein cholesterol (HDL-C) was based on the enzymatic end point method as for total cholesterol developed by Burstein *et al*., (1970) which involves firstly, the precipitation stage (removal of Non-HDL-C lipid fractions) and secondly, the estimation of the HDL-cholesterol fraction. The determination of low density lipoprotein cholesterol (LDL-C) concentration was calculated using Freidewald *et al* (1972) equation

2.5 Statistical Analysis

Statistical analysis was performed using Graphpad prism 5.1 version. The mean, standard deviation and inferential statistics using student t-test were used. Statistical significance were seen at $p<0.05$.

3. Results

The comparison of Group 1 and Group 2 (table 3.1) showed that Group 1 (control) had 2.13±12.7 while Group 2 had 1.28±8.9 for TC, for TG control had 1.48±22.8 while Group 2 had 1.05±14.5, for HDL Group 1 (control) had 1.56±10.2 while Group 2 had 1.02±6.2 and for LDL Group 1 (control) had 0.17±5.0 while Group 2 had 0.48±3.5. Also, when atherogenic indices were considered, the result showed that Group 1 (control) had 1.4±0.2 while Group 2 had 1.4±0.2 for TC/HDL and for LDL/HDL, Group 1 (control) had 0.1±0.1 while Group 2 had 0.1±0.1. The comparison of Group 1 and Group 2 showed significant decreases in TC, TG, HDL-C, LDL-c and TC/HDL when compared to the control (Group 1) at $p<0.05$ except LDL/HDL that showed no significant difference (table 3.1).

Also, the comparison of Group 1 and Group 3 (table 3.2) showed that Group 1 (control) had 2.13±12.7 while Group 3 had 1.85±9.9 for TC, for TG Group 1 (control) had 1.48±22.8 while Group 3 had 1.79±10.2, for HDL, Group 1 (control) had 1.48±22.8 while Group 3 had 1.09±6.6, for LDL Group 1 (control) had 0.17±5.0 while Group 3 had 0.43 ±13.9. In addition, TC/HDL had 1.4±0.2 while Group 3 had 1.7±0.2 and for LDL/HDL the control had 0.1±0.1 while Group 3 had 0.4±0.3. The comparison of Group 1 and Group 3 showed significant decreases in TC and HDL-C while significant increases were seen in TG and LDL-C when compared to the control (Group 1) at $p<0.05$. However, TC/HDL and LDL/HDL showed no significant differences compared to the control at $p<0.05$ (table 3.2).

The comparison of Group 1 (control) and Group 4 (table 3.3) showed that Group 1 (control) had 2.13±12.7 while Group 4 had 2.36±13.6 for TC. For TG, Group 1 (control) had 1.48±22.8 while Group 4 had 1.50±33.4.5. For HDL, Group 1 (control) had 1.48±22.8 while Group 4 had 1.08±12.6. For LDL, Group 1 (control) had 0.17±5.0 while Group 4 had

2.40±12.2 for LDL. Also, for TC/HDL, Group 1 (control) had 1.4±0.2 while Group 4 had 2.3±0.8 and for LDL/HDL, Group 1 (control) had 0.1±0.1 while Group 4 had 0.6±0.4. Significant increases were seen in TC, LDL-C and TC/HDL when compared to Group 1 at $p<0.05$. However, non-significant differences were seen in TG, HDL-C and LDL/HDL when compared to the Group 1 (Control) (table 3.3).

In addition, the comparison of Group 1 (control) and Group 5 (table 3.4) showed that Group 1 (control) had 2.13±12.7 while Group 5 (rats treated with low dose of natural honey) had 1.22±10.6 for TC. For TG, Group 1 (control) had 1.48±22.8 while Group 5 had 0.89±19.1. For HDL, Group 1 (control) had 1.48±22.8 while Group 5 had 1.67±1.3 and for LDL, Group 1 (control) had 0.17±5.0 while Group 5 had 0.34±9.4. Furthermore, for TC/HDL, Group 1 (control) had 1.4±0.2 while Group 5 had 1.1±0.2 and for LDL/HDL, Group 1 (control) had 0.1±0.1 while Group 5 had 0.3±0.2. The comparison showed significant decreases in TC and TG while significant increase was seen in HDL-C when compared to the control. However, no-significant differences were seen in LDL-C, TC/HDL and LDL/HDL (table 3.4).

When the comparison of Group 1 (Control) and Group 6 (table 3.5) results were observed, it was shown that Group 1 (control) had 2.13±12.7 while Group 6 (moderate dose of natural honey) had 1.56±9.1 for TC. For TG, Group 1 (control) had 1.48±22.8 while Group 6 had 0.98±30.7. For HDL, Group 1 (control) had 1.48±22.8 while Group 6 had 2.01±2.1. For LDL, Group 1 (control) had 0.17±5.0 while Group 6 had 0.17±7.6. Also, the result showed that Group 1 had 1.4±0.2 while Group 6 had 1.3±0.2 for TC/HDL and LDL/HDL had 0.1±0.1 for Group 1 and 0.2±0.2 for Group 6. The comparison showed significant decreases in TC and TG while significant increase was seen in HDL-C when compared to the control. However, no-significant differences were seen in LDL-C, TC/HDL and LDL/HDL (table 3.5).

Furthermore, the comparison of Group 1 (control) and Group 7 (table 3.6) showed that Group 1 (control) had 2.13±12.7 while Group 7 (high dose of natural) had 2.09±15.4 for TC, For TG, Group 1 had 1.48±22.8 while Group 7 had 1.19±13.5. For HDL, Group 1 had 1.48±22.8 while Group 7 had 1.84±5.1. For LDL, Group 1 had 0.17±5.0 while Group 7 had 0.26±8.7. Also, the result showed that Group 1 had 1.4±0.2 while Group 7 had 1.6±0.2 for TC/HDL and LDL/HDL had 0.1±0.1 for Group 1 and 0.2±0.5 for Group 7. The comparison showed non-significant differences in the all of the parameters considered at $p<0.05$ (table 3.6).

Finally, the results obtained also indicated that the comparison of Group 2 (low dose of refined sugar) and Group 5 (low dose of natural honey) (table 3.7), Group 3 (moderate dose of refined sugar) and Group 6 (moderate dose of natural honey) (table 3.8) and Group 4 (high dose of refined sugar) and Group 7 (high dose of natural honey) (table 3.9) showed no significant differences in all of the parameters considered except in Group 3 (moderate dose of refined sugar) and Group 6 (moderate dose of natural honey) were TC/HDL was significantly increased at $p<0.05$.

Table 3.1: Comparison of Control Rats (Group 1) and Rats treated with low dose of Refined Sugar (Group 2)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 2	0.02	1.28±8.9	1.05±14.5	1.02±6.2	0.48±3.5	1.3±0.1	0.1±0.1
pvalue		0.005	0.030	0.013	0.547	0.299	0.107
tvalue		4.253	2.829	3.506	0.639	1.137	2.009
Remark		S	S	S	NS	NS	NS

Table 3.2: Comparison of Control Rats (Group 1) and Rats Treated with Moderate Dose of Refined Sugar (Group 3)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 3	0.03	1.85±9.9	1.79±10.2	1.09±6.6	0.43±13.9	1.7 ±0.2	0.4±0.3
pvalue		0.228	0.623	0.026	0.227	0.058	0.138
tvalue		1.343	0.519	2.952	1.345	2.335	1.711
Remark		NS	NS	S	NS	NS	NS

Table 3.3: Comparison of Control Rats (Group 1) and Rats treated with high dose of Refined Sugar (Group 4)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 4	0.04	2.36±13.6	1.50±33.4	1.08±12.6	2.40±12.2	2.3±0.8	0.6±0.4
pvalue		0.379	0.962	0.062	0.049	0.048	0.056
tvalue		0.950	0.049	2.295	2.468	2.466	2.366
Remark		NS	NS	NS	S	S	NS

Table 3.4: Comparison of Control Rats (Group 1) and Rats Treated with Low Dose of Natural Honey (Group 5)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 5	0.04	1.56±10.6	0.98±19.1	2.01±1.3	0.17±9.4	1.1±0.2	0.3±0.2
pvalue		0.005	0.012	0.022	0.2766	0.074	0.133
tvalue		4.248	3.546	3.055	1.196	2.152	1.736
Remark		S	S	S	NS	NS	NS

Table 3.5: Comparison of Control Rats (Group 1) and Rats Treated with Moderate Dose of Natural Honey (Group 6)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 6	0.09	1.56±9.1	0.98±30.7	2.01±2.1	0.17±7.6	1.3±0.2	0.2±0.2
pvalue		0.029	0.050	0.050	0.556	0.474	0.309
tvalue		2.838	2.347	2.429	0.623	0.764	1.113
Remark		S	S	S	NS	NS	NS

Table 3.6: Comparison of Control Rats (Group 1) and Rats Treated with High Dose of Natural Honey (Group 7)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 1	0.0	2.13±12.7	1.48±22.8	1.56±10.2	0.17±5.0	1.4±0.2	0.1±0.1
GROUP 7	0.13	2.09±15.4	1.19±13.5	1.84±5.1	0.26±8.7	1.6±0.2	0.2±0.5
pvalue		0.885	0.097	0.159	0.710	0.193	0.553
tvalue		0.150	1.966	1.608	0.389	1.465	0.627
Remark		NS	NS	NS	NS	NS	NS

Table 3.7: Comparison of Rats treated with low dose of Refined Sugar (Group 2) and Rats treated with low dose of Natural Honey (Group 5)

GROUPS	DOSE	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 2	0.0g	1.28±8.9	1.05±14.5	1.02±6.2	0.48±3.5	1.3±0.1	0.1±0.1
GROUP 5	0.04g	1.56±10.6	0.98±19.1	2.01±1.3	0.17±9.4	1.1±0.2	0.3±0.2
Pvalue		0.777	0.273	0.150	0.411	0.212	0.599
Tvalue		0.296	1.206	1.651	0.883	1.397	0.555
Remark		NS	NS	NS	NS	NS	NS

Table 3.8: Comparison of Rats treated with moderate dose of Refined Sugar (Group 3) and Rats treated with moderate dose of Natural Honey (Group 6)

GROUPS	DOSE(g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 3	0.0	1.85±9.9	1.79±10.2	1.09±6.6	0.43±13.9	1.7 ±0.2	0.4±0.3
GROUP 6	0.09	1.56±9.1	0.98±30.7	2.01±2.1	0.17±7.6	1.3±0.2	0.2±0.2
pvalue		0.143	0.225	0.179	0.404	0.038	0.306
tvalue		1.685	1.351	1.523	0.897	2.652	1.118
Remark		NS	NS	NS	NS	S	NS

Table 3.9: Comparison of Rats treated with high dose of Refined Sugar (Group 4) and Rats treated with high dose of Natural Honey (Group7)

GROUPS	DOSE (g/ml)	TC(mmol/L)	TG(mmol/L)	HDL(mmol/L)	LDL(mmol/L)	TC/HDL	LDL/HDL
GROUP 4	0.0	2.36±13.6	1.50±33.4	1.08±12.6	2.40±12.2	2.3±0.8	0.6±0.4
GROUP 7	0.09	2.09±15.4	1.19±13.5	1.84±5.1	0.26±8.7	1.6±0.2	0.2±0.5
pvalue		0.353	0.185	0.215	0.135	0.105	0.110
tvalue		1.006	1.498	1.385	1.724	1.906	1.873
Remark		NS	NS	NS	NS	NS	NS

KEY: S = Significant, NS=Not significant

4. Discussion

In this study, the effect of natural honey and refined sugar on the lipid profile was evaluated using wistar rats. Hyperlipidaemia is a common feature in atherosclerosis, the complications of which lead to ischaemic heart disease, myocardial infarction and stroke [2][3]. High lipid panel content of plasma is believed to be the main cause of atheromatous lesions of blood vessels. Attempts have been made to develop drugs which reduce the concentration of plasma cholesterol because of its role in the etiology and course of atherosclerosis which include statin groups of drugs and the vitamin niacin [2][3].

From the above result, there were significant increases in LDL-C and TC/HDL as well as reduced HDL-C concentration of rats treated with low, moderate and high doses of refined sugar. The increases and decrease seen in LDL-C, TC/HDL and HDL-C respectively are strong indicators of cardiovascular risk. However, rats treated with low and moderate doses of natural honey showed significant decreases in TC, TG, TC/HDL-C and increase in HDL-C. This is in line with previous studies conducted by [13][14], which stated that honey intake have the ability to modulate some of the cardiovascular risk factors. According to the work published by [12][15][16], natural honey decreased total cholesterol and low density lipoprotein and increase high density lipoprotein, while sucrose might increase lipids because of the presence of fructose. The difference between the effects of sucrose and natural honeys on cholesterol might be due to the presence of certain substances such as antioxidant activities in natural honey. [16], stated that honey has many advantages that would be promising in the world of health due to it's influence on weight loss and lowering blood triglyceride levels in some strain of rats. However, excessive intake of honey might have no cardio-protective benefits (table 3.6). [18], however speculated that honey increases bile cholesterol excretion and lowers plasma cholesterol. [19], suggest that antioxidants in honey, in addition to their role in lowering blood cholesterol and low density lipoprotein

levels, are also advantageous by inhibiting the formation of atherogenic plaques. Inhibition of atherogenic plaque formation is also determined by a variety of trace elements, minerals, vitamins, contained in honey. Some trace elements that prevent the formation of atherogenic plaques are copper and zinc. Meanwhile, some of the vitamins contained in honey that play a role in preventing oxidative stress and atherosclerotic plaque formation are vitamin E (niacin), vitamin C, vitamin B, and vitamin A.

The result obtained also with respect to honey agrees with the report of [20], who that the consumption of 40 grams of honey was shown to improve lipid profiles in obese adult subjects. [19], also found that 70 grams of pure honey daily for 30 days was shown to reduce cardiovascular disease risk factors and did not cause weight gain in overweight or obese subjects. In addition, studies on healthy young subjects with similar dose and duration (70 grams daily for 4 weeks) also showed a decrease in total cholesterol, triglycerides, and LDL compared with the control group. In addition, [18], suggest that the administration of pure honey can reduce levels of blood LDL and TC in rats. However, [18] research showed elevated levels of TG and VLDL in the blood.

Furthermore, the results observed with respect to refined sugar also concur with the findings of [10], who state in his study that high blood sugar level coupled with high levels of circulating insulin can damage the walls of arteries. The body responds to this injury by sending pro-inflammatory cytokines to the sites of injury. Even as the lining of the arteries become damaged, VLDL-C and LDL-C become oxidized and then trapped in the inflamed sites of the lining. The trapping of oxidized LDL-C not only worsens the inflammation but also promotes the formation of plaques and the narrowing of the arteries. Therefore, high sugar consumption can cause inflammation and increase the risk of atherosclerosis and heart disease.

5. Conclusion and Recommendation

The findings from the research showed that refined sugar irrespective of the dosage, indicated tendencies of inducing cardiovascular risk by increasing level LDL and TC/HDL ratio with a corresponding reduction in HDL-C concentration while natural honey was shown to reduce cardiovascular risk by decreasing TC, TG, and increase HDL-C and TC/HDL ratio. Therefore, indication from this study is that, high intake of refined sugar could predispose one to coronary heart disease compared to intake of natural honey. It is therefore recommended that these findings be extended to human, and as such it is advisable that humans should take refined sugars with caution by avoiding excessive consumption.

REFERENCES

[1] Kahn, A. (2012). High blood cholesterol and triglycerides in lipid disorder. *Current opinion on Lipid*, **18**(1), 35-40

[2] Bray, G.A., Nielsen, S.J and Popkin, B.M. (2016). Consumption of high-fructose corn Syrup in beverages may play a role in the epidemic of obesity. *American Journal of Clinical Nutrition*, **79**(6), 537 – 543.

[3] Melanson, K.J., Zukley, L., Lowndes, J., Nguyen, V., Angelopoulos, T.J and Rippe, J.M. (2015). Effects of high-fructose corn syrup and sucrose consumption on circulating glucose, insulin, leptin, and ghrelin and on appetite in normal-weight women. *Nutrition*, **23,** 103 – 112.

[4] Ahmed, Z., Banu, H., Akhter, F., Faruquzzaman, M and Haque, S. (2015). Concept on Sugar: A review. *Journal of Biological Science*, **1**(2), 883-894.

[5] Dragsted, L.O., Daneshvar, B., Vogal, U., Austrup, H.N., Wallin, H., Risom L…and Moller, P. (2014). A sucrose rich diet induces mutations in the rat colon. *Cancer Research*, **62,** 4339- 4345.

[6] Adegoke, O.A., Bamigbowu, E.O., George –Opuda, M.I., Awopeju, T., Mbata, C.A and Braide, S.A. (2013). Effect of Granulated Sugar on Some Renal Parameters in Albino Rats. *International Journal of Epidemiology and Infection*, **1**(1), 1-3.

[7] Hanaa, M. R and Shaymaa, M. M. Y. (2011). Enhancement of the antitumor effect of honey and some of its extracts using adiponectin hormone. *Australian Journal of Basic and Applied Science*, **5**(6), 100-108.

[8] Mohammadzadeh, S., Sharriatpanahi, M., Hamedi, M., Amanzadeh, Y., Ebrahimi, S.S and Ostad, S.N. (2007). Antioxidant power of Iranian propolis extract. *Food Chemistry*, **103**(7), 729-733.

[9] Bogdanov, S., Jurendic, T., Sieber, R and Gallmann, P. (2008). Honey for nutrition and health: A review. *Journal of America College of Nutrition*, **27**(4), 677-689.

[10] Erejuwa, O.O., Sulaiman, S.A and Wahab, M.S. (2012). Oligosaccharides might contribute to the antidiabetic effect of honey: A review of the literature. *Molecules*, **17**(3), 248-266.

[11] Ajibola, A. (2015). Physico-Chemical and Physiological Values of Honey and Its Importance as a Functional Food. *International Journal for Food and Nutritional Science*, **2**(6), 1-9.

[12] Bantle, J., Swanson, J., Thomas, W and Laine, D. (2015). Metabolic effects of dietary fructose in diabetic subjects. *Diabetes Care*, **15**(6), 1468–1476.

[13] Al-Waili, N. (2004). Intravenous and intrapulmonary administration of honey solution to healthy sheep: effects on blood sugar, renal and liver function tests, bone marrow function, lipid profile and carbon tetrachloride-induced liver injury. *Journal for Medical Food*, **6** (4), 231–247**.**

[14] Swanson, E., Laine, C., Thomas, W and Bantle, J. (1992) Metabolic effects of dietary fructose in healthy subjects. *American Journal for Clinical Nutrition*, **55**(4), 851–856.

[15] Abraham, A., Humphreys, S., Clark, M., Mathews, D and Frayn, K. (2014) Acute effect of fructose on postprandial lipaemia in diabetic and non-diabetic subjects. *British Journal for Nutr*ition, **80** (2), 169–175.

[16] Busserolles, J., Gueeux, E., Mazur, A and Rayssiguier, Y. (2002) Substituting honey for refined carbohydrates protects rats from hypertriglyceridemic and prooxidative effects of fructose. *Journal for Nutrition*, **132**(9), 3379–3382.

[17] Nemoseck, T.M., Carmody, E.G and Furchner-Evanson, A. (2011). Honey promotes lower weight gain, adiposity, and triglycerides than sucrose in rats. *Nutritional Research*, **31**(4), 55 - 60

[18] Alagwu, E. A., Okwara, J. E., Nneli, R. O. and Osim, E. E. (2011). Effect of honey intake on serum cholesterol, triglyceride and lipoprotein levels in albino rats and potentials benefits on risks of coronary heart disease. *Nigerian Journal of Physiological Science*, **26**, 161 -165

[19] Yaghoobi, Yaghoobi, F. (2008). Natural honey and cardiovascular risk factors; effects on blood glucose, cholesterol, triacylglycerole, CRP and body weight compared with sucrose. *The Scientific World Journal*, **8**(4), 463–464

[20] Mushtaq, R., Mushtaq, R.P and Khan, Z.T. (2011). Effects of natural honey on lipid profile and body weight in normal weight and obese adults: a randomized clinical trial. *Pakistan Journal of Zoology*, **43**(6),161-169.