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Hypolipidemic Effect of *Quisqualis indica (Linn)* Aerial Parts on Passive Smoking & Cholesterol Diet Fed Animals

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ABSTRACT

Objective: In the present study the effect of two different doses (100 mg/kg and 200 mg/kg) of methanolic and aqueous extracts of aerial part of *Quisqualis indica* on passive smoking and cholesterol diet (coconut, biscuit and milk powder) induced hyperlipidemia in rats has been investigated. **Methods:** In this study the plant *Quisqualis indica* aerial part was extracted in soxhlet apparatus with methanol and aqueous by following the successive extraction method and presence of various phytochemical constituent in plant extracts like flavonoids, alkaloid, amino acids, glycosides, acidic compounds etc had been estimated by using standard method. The hypolipidemic activity was analysed by reading the blood serum level in UV at 505 nm after treated with reagent present in auto span diagnostic kit. **Results:** Results showed that the simultaneously consumption of both cholesterol diet and passive smoking had increased more bad fats when compared to a particular one. Plant extracts had reduce the harmful lipid layer in blood serum at varying concentration and dose dependent manner which shows that the plant carries the hypolipidemic properties but the methanolic extracts at 200 mg/kg had been markly showed effect comparable hypolipidemic as that of standard Atorvastatin (10 mg/kg).

Key words: Hypolipidemic, *Quisqualis indica*, flavonoids, Coronary heart disease, atorvastatin.

INTRODUCTION

The products which are obtained from the natural source such as plants, microorganisms, animals or minerals is the basic needs of making drugs used for the treatment of disease which are synthesized now a days for the making of a novel drugs. During the past two decades, there has been an increasing interest in the industrialized nations to use medicinal plants. Sources of details are pharmacopoeias, indigenous knowledge, scientific literature, and other documented sources [1]. India one among 12 mega biodiverse countries of the world, which despite having only 2.5 % total land area, accounting for over 8 % of the recorded species of the world [2]. It is estimated that at least 25% of all modern medicines are derived, either directly or indirectly, from medicinal plants, primarily through the application of modern technology to traditional knowledge[2, 3].

In 21st century hyperlipidemia causing due to obesity increase their risk of developing Coronary heart disease (CHD) by 45 % to 60% [4] and the person who are exposed to second hand smoke at home or at work by 25% to 30% [5] which is a leading preventable cause of death worldwide, with increasing prevalence in adults and children, and authorities view it as one of the most serious public health problems causing heart diseases. Hyperlipidemia is an excess of fatty substances called lipids, largely cholesterol and triglycerides, in the blood and it is mainly associated with endothelial dysfunction. It is also called hyperlipoproteinemia because these fatty substances travel in the blood attached to proteins [6] and is also identified as dyslipidemia i.e. elevation of plasma cholesterol, triglycerides (TGs), or both HDL (high density lipoproteins, or "good" cholesterol) and LDL (low density lipoproteins, or "bad" cholesterol) level that contributes to the development of atherosclerosis[7]. The amount of HDL relative to LDL is considered a more important indicator of your heart disease risk.

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Nicotine present in ciggrette smoke and fats present in cholesterol diet increases the amount of bad fats (LDL, triglycerides, cholesterol) circulating in the blood vessels and decreases the amount of good fat (HDL) [4,8] and are absorbed through the lungs into the blood stream and are circulated throughout the body. When there is too much cholesterol circulating in the blood, it can create sticky deposits (called plaque) along the artery walls. Plaque can eventually narrow or block the flow of blood to the brain, heart, and other organs. And blood cells that get caught on the plaque form clots, which can break loose and completely block blood flow through an artery, causing heart attack or stroke [9].

The key risk factors for hyperlipidemia are family history and lifestyle habits. Most people can lower their risk for hyperlipidemia through eating habits and exercise. Even in combination with medication, lifestyle factors are important for maintaining healthy lipid metabolism [10]. Hyperlipidemia is associated with endothelial dysfunction, an early event in atherosclerosis and predictor of risk for future coronary artery disease. Cell membranes are made of unsaturated lipids and these unsaturated lipid molecules of cell membranes are particularly susceptible to free radicals. Oxidative damage can direct to a breakdown or even hardening of lipids, which composition of all cell walls [11]. Epidemiological studies suggest that increased dietary intake of antioxidants reduces the risk of coronary artery disease [12] and there are so many natural things found in world which could have been better than the synthetic products because of their side effects or toxicity. *Quisqualis indica Linn* showing various pharmacological activities such as anti-inflammatory activity, antipyretic activity, immunomodulatory activity, anti-staphylococcal activity, anthelmintic activity, antiseptic activity, antioxidants etc due to its presence of various active constituents all over the parts of plants which had been proved [13-18]. On the contrary the presence of mainly flavonoids and other constituent together act as antihyperlipidemic as it gives an antioxidant activity [19].

MATERIALS & METHODS

Plant Material

The mature aerial parts of *Quisqualis indica* were collected in the morning

from Bhopal, Madhya Pradesh, India, in the month of January 2012. The aerial parts of *Quisqualis Indica* was collected from Bhopal, Madhya Pradesh, India & Identification and Authentication of herb by Dr. Zia ul Hassan, Professor of Botany, Safia College of Science, Bhopal, Madhya Pradesh, India (Voucher. No 323/Bot/Safia/2010).

The collected parts were washed with a normal tap water so that the sticked dirt particle had been washed and then dried in a shed area, after dried it had been crushed into small pieces for successive extraction process. About 80 gm of dry powder was taken in a soxhlet apparatus and firstly it was extracted with 400 ml methanol for about 8 days at 10-15 degree centigrade and further it was extracted with 400 ml distilled water after the collection of marc. The marc left after successive extraction was taken out and dried it separately under room temperature to get a dry mass i.e. free of solvent. The both final obtained extract was weighed, packed in a paper bags & stored in air tight container at cool place until use.

Phytochemical analysis

Preliminary Phytochemical studies of methanolic & aqueous extract of aerial parts of *Quisqualis indica* was performed for major classes of constituents like alkaloids, carbohydrates, protein and amino acid, Saponins, glycosides, steroids, tannins, flavonoid and phenolic compounds according to published standard methods [20].

Preparation of dosage forms

For in vivo studies, the standard drug Atorvastatin (10 mg/kg) was administered orally after suspending in 0.05% CMC and the concentrated methanolic and aqueous extract of *Quisqualis indica* was administered orally after suspending in Distilled water. The freshly prepared solution of both standard drug Atorvastatin & *Quisqualis indica* extract was used in each experiment.100mg/kg and 200 mg/kg per ml. Test doses were selected on the basis oral acute toxicity study in rat.

Animals

Albino wistar rats (100-200 gm) of either sex had been taken which were obtained from Sapience Bio Analytical Research laboratory, Bhopal (M.P.) animal house (Reg. No. 1413/A/11 CPCSEA) and housed 5 animals per cage made up of polypropylene, habituated at laboratory condition for 2 days prior to experiment procedure which were maintained at environment [(25^0 C ± 2) temperature, 30-50 % humidity and 12 hr light & dark condition alternately)]. The animals were fed with standard pellet diet and water ad libitum at first 12 hours and then with cholesterol diet (boiled potatoes, Biscuits, coconut, bread & milk powder at alternative days) for next 12 hours and simultaneously provided with ciggrette smoke twice a daily i.e. 1 cigarettes provided to a group of 6 animals at morning and evening by the use of smoking chambers having 1 ventilacle hole at both sides throughout the experiments except control group.

Acute toxicity study and dose selection

The dose limits were selected on the basis of previously performed oral acute toxicity studies in albino mice, in accordance with the OECD (423) guidelines [21]. Acute toxicity studies on *Quisqualis indica aerial parts* extract were performed in mice containing 6 animals in each group, the graded doses of the methanolic and aqueous extract of *Quisqualis indica aerial parts* extract doses selected for the study were 100 mg/kg, 200 mg/kg, 400 mg/ kg, 800 mg/kg, 1600 mg/kg and 2000 mg/kg were administered orally and the animals were observed for 2 weeks following administration, change in body weight gain, food consumption, any kind of behaviorally changes and mortality were noted. It was found that the methanolic extract has produced significant toxicity at the dose of 2000 mg/kg as 2 animal of this group was died. Thus the extract was highly tolerable up to 1500 mg/kg for methanolic extract and 2000 mg/kg for aqueous extract.

Cholesterol diet & passive smoking induced hyperlipidemia

The animals were divided into following 9 groups containing 6 animals in each group they are:

Group I: Control Group
Group II: Cholesterol Diet
Group III: Passive smoking
Group IV: Cholesterol Diet + Passive smoking
Group V: Cholesterol Diet + Standard Atorvastatin (10 mg/kg p.o.)
Group VI: Cholesterol Diet + Test Methanolic extract (100 mg/kg p.o.)
Group VII: Cholesterol Diet + Test Aqueous extract (100 mg/kg p.o.)
Group VIII: Cholesterol Diet + Test Methanolic extract (200 mg/kg p.o.)
Group IX: Cholesterol Diet + Test Aqueous extract (200 mg/kg p.o.)

After 28 days rats were fasted for 10-12 hours and then they were anaesthetized with mild chloroform, blood sample was collected by retro orbital sinus puncture. Collected blood was poured slightly into tubes marked and immediately centrifuged for 2000 rpm for 15 minutes to obtain clear serum. The amount of Total-Cholesterol, HDL-Cholesterol, LDL-Cholesterol, VLDL-Cholesterol and Triglycerides were estimated by using biochemical kits simultaneously blood glucose level was also determined by Aspen Diagnostics kit Gluco-Check (Glucometer) which reads the blood glucose level manually. The amount of blood parameters was calculated in mg/dl.

Biochemical analysis

The blood serum were assayed for total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL) by reading the blood serum level in UV at 505 nm after treated with reagent of auto span diagnostic kit.

(A) Estimation of Total Cholesterol (TC)

For estimation of Total-Cholesterol 2 reagent had been used i.e. 1) Cholesterol reagent and 2) Cholesterol standard came in kit, reaction followed CHOD-PAP method.

Procedure- 3 different following solutions had been taken they are:
Blank: contained 1000 ul of reagent 1.
Standard: contained 1000 ul of reagent 1 and 10 ul of reagent 2.
Test: contained 1000 ul of reagent 1and 10 ul of test serum.

Mixed well, incubate at 37^0 C for 10 minute or at room temperature (15-30^0 C) for 30 minutes. Programme the analysers as per assay parameters given in kit.

1. Blank the analysers with reagent blank.
2. Measure absorbance of standard followed by the test at wave-length 505 nm.
3. Calculated the results as per given calculation formula.

Calculation

Cholesterol concentration (mg/dl) =Absorbance of Test/Absorbance of standard × 200.

(B) Estimation of high-density lipoprotein (HDL)

For estimation of High-density lipoprotein 3 reagent had been used i.e. 1) Cholesterol reagent 2) Precipitating reagent PEG-6000 and 3) HDL-cholesterol standard came in kit, reaction followed PEG-CHOD-PAP method, end point assayed with Lipid clearing factor(LCF).

Preparation of working reagents

Dissolve enzyme reagent with 25 ml. of diluents buffer and kept for at least 10 min. before use. The working reagent is stable for 4 weeks at 2-8^0 C.

Procedure

1. Taken 200µl of clear serum into tubes added 200 µl of reagent 2 mixed well and incubated at temperature for (15-30^0 C) for 10 min

for HDL-cholesterol separation.

2. Kept all tubes in cooling centrifugation chamber at 2000 rpm for 15 minute and separate clear supernatant which was used for HDL-Cholesterol estimation.

For HDL-Cholesterol estimation 3 different following solutions had been taken they are:

Blank: contained 1000 ul of reagent 1.
Standard: contained 1000 ul of reagent 1and 100 ul of reagent 3.
Test: contained 1000 ul of reagent 1 and 100 ul of test serum.
Mixed well, incubate at 37^0 C for 10 minute or at room temperature (15-30^0 C) for 30 minutes. Programme the analysers as per assay parameters given in kit.

1. Blank the analysers with reagent blank.
2. Measure absorbance of standard followed by the test at wave-length 505 nm.
3. Calculated the results as per given calculation formula.

Calculation

HDL-Cholesterol concentration (mg/dl) = Abs of test/Abs of standard × 50 × 2
Abs- absorbance, *(2 dilution factor, as sample is diluted 1:1 in step 1).

(C) Estimation of Triglyceride (TG)

Triglycerides are estimated using accurate Triglycerides kit of Span Diagnostics Pvt. Ltd. Accurately triglycerides estimation kit is formulated using GPO and peroxide for quantitative estimation of serum triglycerides. The kit contain 2 reagent i.e. 1) Triglycerides mono reagent and 2) Triglycerides standard.

Table 1: Lipid serum level between control, cholesterol diet, passive smoking and cholesterol diet + passive smoking induced group.

S. No.	Treatment	TC	TG	HDL	LDL	VLDL
I	Control	52.15±0.39	41.37±1.37	21.65±0.44	24.66±1.03	8.275±0.27
II	Cholesterol diet	74.56±0.74***	64.02±0.71***	12.51±0.56***	48.19±0.85***	12.80±0.14***
III	Passive Smoking	64.05±0.89***	55.49±0.52***	12.45±0.40***	40.50±0.49***	11.10±0.10***
IV	Cholesterol diet + passive smoking	80.04±0.79***	67.94±0.95***	10.43±0.44***	56.02±0.98***	13.59±0.19***

*Values are in mean ± SE; Number of animals in each group = 6; ***p < 0.05 Vs Group I*

Preparation of working Reagent

The contents of enzyme reagent were dissolved in 10 ml of diluent buffer. The working reagent is stable for 4 - 6 week at 2 - 8^0 C.

Procedure- 3 different following solutions had been taken they are:
Blank: contained 1000 ul of reagent 1.
Standard: contained 1000 ul of reagent 1 and 10 ul of reagent 2.
Test: contained 1000 ul of reagent 1and 10 ul of test serum.
Mixed well and incubated at 37^0 C for 10 minutes. Programme the analysers as per assay parameters given in kit.

1. Blank the analyser with Reagent Blank.
2. Measure absorbance of standard followed by the test.
3. Calculate results as per given calculation formula.

Calculation- Triglycerides (mg/dl) = Absorbance of Test/Absorbance of Standard × 200.

(D) Estimation of low-density lipoprotein (LDL) and very low-density lipoprotein (VLDL)

The amount of LDL-Cholesterol and VLDL-Cholesterol were calculated using friedewald's equation.

LDL-Cholesterol =Total Cholesterol – Triglycerides/5– HDL -Cholesterol
VLDL-Cholesterol = Triglycerides/5 [22, 23].

Statistical analysis

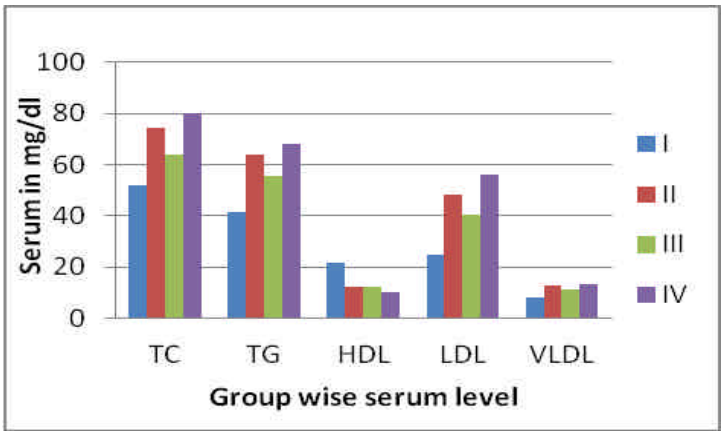
The results were expressed as mean ± S.E. Statistical analysis was carried out by using ANOVA followed by Tukey's multiple comparison tests using Graph pad PRISM software version 5.04 (2010).P values < 0.05 were considered as statistically significant.

RESULTS

By the acute toxicity test it was found that the methanolic extract was non-toxic up to 1500 g/kg and aqueous extract at 200 mg/kg. So by considering the toxicity test two doses of both the extracts are taken i.e. 100 and 200 mg/kg. The preliminary phytochemicals test indicates that the extracts contain acidic compound, glycosides, alkaloid, saponin, carbohydrates, flavonoid and fixed oil. The present investigation showed that the using of highly cholesterol diet containing fats and passive smoking induced hyperlipidemia in rats particularly by the raised level of harmful lipid i.e. LDL, VLDL, TC and TG and lowering of HDL but there is highly increment of bad fats when both cholesterol diet and passive smoking had been given simultaneously shown in table 1 and graph 1. The results were discussed mainly under lipid layer.

I blue colour indicated control group
II brown colour indicated cholesterol diet induced group
III green colour indicated passive smoking induced group
IV brown colour indicated cholesterol diet + passive smoking induced group

Both the extracts had lowered the total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) and increase the high-density lipoprotein (HDL) level which was may be due to inhibition of lipid peroxidation as the plant containing flavonoids which act as antioxidants or by another constituents which was proved by the graph showing in table 2 and figure 2 lowering the harmful lipid when compare to induced group. By the results it had been concluded that the extracts is acting as hypolipidemic or as antihyperlipidemic drugs at the



I blue colour indicated control group.
II brown colour indicated cholesterol diet induced group.
III green colour indicated passive smoking induced group.
IV brown colour indicated cholesterol diet + passive smoking induced group.

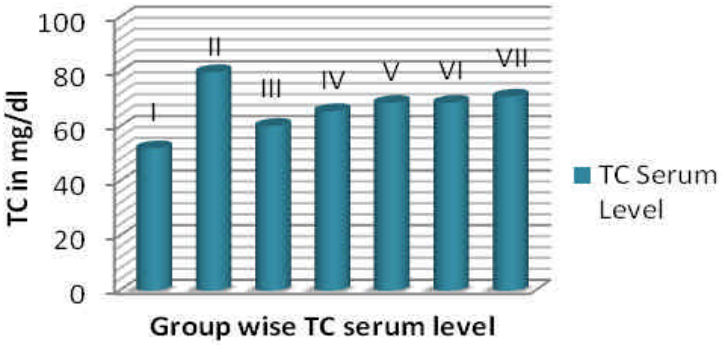
Figure 1: Graph showing variation in different lipid serum level between control, cholesterol diet, passive smoking and cholesterol diet + passive smoking induced group.

Table 2: Effect of Methanolic and aqueous extract of *Quisqualis indica* Linn on TC, TG, HDL, LDL, VLDL and BG in Serum of Control and Experimental Rats.

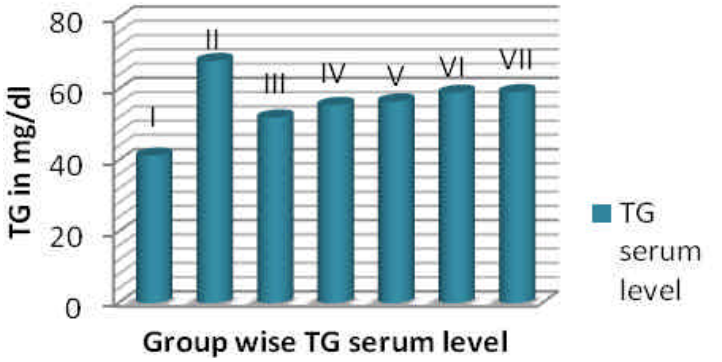
S. No.	Treatment	TC	TG	HDL	LDL	VLDL
I	Control	52.15±0.39	41.37±1.37	21.65±0.44	24.66±1.03	8.275±0.27
II	Cholesterol diet+ passive smoking	80.04±0.79***	67.94±0.95***	10.43±0.44***	56.02±0.98***	13.59±0.19***
III	Atorvastatin	60.34±0.30***	52.06±0.34***	16.46±0.44***	31.82±0.33***	10.41±0.07***
IV	Methanolic ext. (200 mg/kg)	65.78±0.63***	55.59±0.50***	15.76±0.45**	38.90±0.78***	11.12±0.10***
V	Aqueous ext. (200 mg/kg)	68.77±0.59***	56.57±0.77***	15.70±0.22***	43.11±0.54***	11.31±0.15***
VI	Methanolic ext. (100 mg/kg)	68.81±0.30***	59.02±1.41***	15.72±0.40*	43.20±0.85***	11.80±0.28***
VII	Aqueous ext. (100 mg/kg)	71.09±1.73***	59.22±1.29***	13.80±0.88***	43.28±0.50***	11.84±0.26***

*Values are in mean ± SE; Number of animals in each group = 6; ***p < 0.05 Group I Vs Group II; *** ** * p <0.05 Vs Group II.*

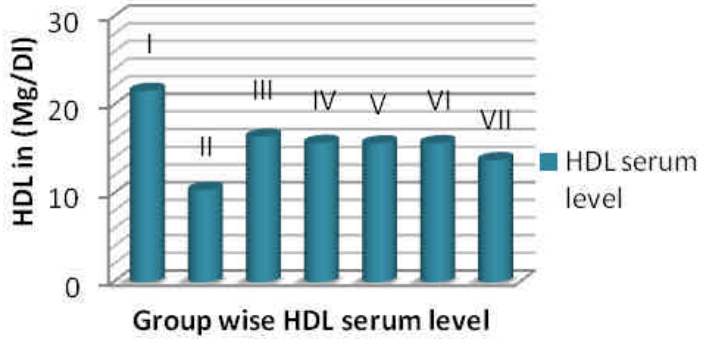
dose level of 100 and 200 mg/kg by lowering the harmful effects of lipid level in a dose dependent manner but the methanolic extracts at dose of 200 mg/kg was found to be more effective than others extracts comparable to standard drug Atorvastatin.



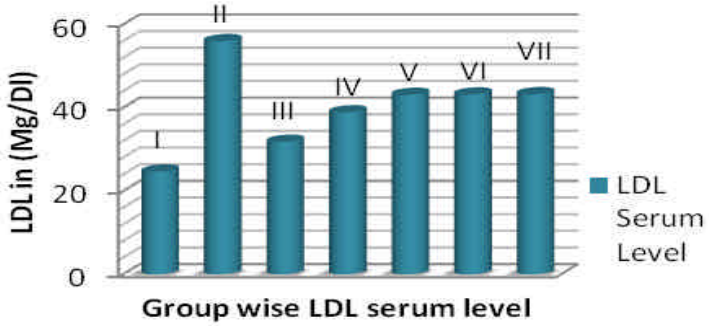
Graph 1: Group wise TC serum level.



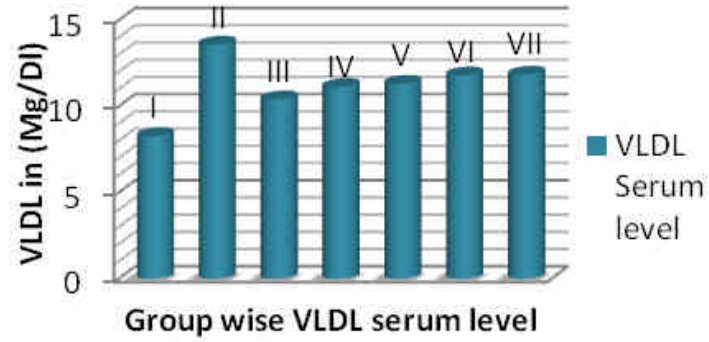
Graph 2: Group wise TG serum level.



Graph 3: Group wise HDL serum level.



Graph 4: Group wise LDL serum level.



Graph 5: Group wise VLDL serum level.

I: Control Group.
II: Cholesterol Diet + passive smoking induced hyperlipidemia group.
III:Cholesterol Diet + passive smoking + Standard Atorvastatin (10 mg/kg p.o.) group.
IV: Cholesterol Diet + passive smoking + Test Methanolic extract (100 mg/kg p.o.) group.
V: Cholesterol Diet + passive smoking + Test Aqueous extract (100 mg/kg p.o.) group.
VI:Cholesterol Diet + passive smoking + Test Methanolic extract (200 mg/kg p.o.) group.
VII:Cholesterol Diet + passive smoking + Test Aqueous extract (200 mg/kg p.o.) group.

Figure 2: Graph showing variation in different lipid serum level between control, induced and treated group.

DISCUSSION

Oxidative damage can direct to a breakdown or even hardening of lipids, which is a composition of all cell walls and cell membranes are made of unsaturated lipids and these unsaturated lipid molecules of cell membranes are particularly susceptible to free radicals. Breakdown or hardening is due to lipid peroxidation leads to death of cell or it becomes unfeasible for the cell to properly get its nutrients or get signals to achieve another and increases excess of fatty substances called lipids, largely cholesterol and triglycerides, in the blood. These free radicals form sticky substance plaque

so that the blood cannot flow properly and can speed up a process called atherosclerosis or hardening of the arteries and block the arteries which cause heart disease, heart attack and stroke [24] and today heart disease can be linked to high fatty substances [10] which largely comes through highly cholesterol diet [4] and passive smoking [5] which was also proved by the present investigation.

The present results shows that the extracts of aerial parts of *Quisqualis indica* linn produce a significant reduction in harmful lipids and raised the HDL level which is good cholesterol and thus it act as antihyperlipidemic drug. Antioxidants cause protective effect by neutralizing free radicals i.e. inhibition of lipid peroxidation or lipolysis, which is toxic byproduct of natural cell metabolism [12]. It is well known that HDL-Cholesterol levels have a protective role in Coronary artery disease [25]. Similarly increased level of serum LDL-cholesterol results in increased risk for the development of atherosclerosis [24]. The increased level of HDL- cholesterol and decreased cholesterol level along with its LDL and VLDL fraction which is evident from the results could be due to a decreased oxidation of Lipid so that it cannot breakdown to form plaque and block the artery so the atherosclerosis doesn't exists which is due to the presence of flavonoids which act as antioxidant [12] which scavenge the formation of free radical between endothelial cells. Results also shows that the plant extracts also decrease blood glucose level. Thus the decreasing harmful lipid levels in the body under the influence of plant *Quisqualis indica* linn could have stop the formation of more oxygen between endothelial spaces.

The result positively suggests that the antihyperlipidemic activity of these herbal plants could be attributed to the presence of the valuable flavonoids in the extracts simultaneously it also reduce cholesterol, triglycerides and blood glucose which strongly strengthen the hypolipidemic activity of the plant. The antihyperlipidemic activity of *Quisqualis indica* (100 and 200 mg/kg) against cholesterol diet and passive smoking showed significant activity when compared to Atorvastatin treated groups in a dose dependant manner.

Thus, our present study showed that administration of Methanolic and aqueous extract of 100 and 200 mg/kg of *Quisqualis indica* was effective to manage hyperlipidemia. The active ingredients present here may recover the disorders in lipid metabolism noted in hyperlipidemic state.

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REFERENCES

1. Kessler RC, Eisenberg DM, et al, Annals of Internal Medicines, 135(4), 2001, 262-8.
2. Sucher NJ, Carles MC et al., Genome-based approaches to the authentication of medicinal plants. Planta Medica, 74(6), 2008, 603–623.
3. Calixto JB, Efficacy, safety, quality control, marketing and regulatory guidelines for herbal medicines (phytotherapeutic agents), Brazilian Journal of Medicine and Biological Research, 33, 2000, 179-189.
4. Barness LA, Opitz JM et al., Obesity: genetic, molecular, and environmental aspects, Am. J. Med. Genet. A, 143A (24), 2007, 3016–34.
5. Centers for Disease Control and Prevention (CDC). Vital signs: nonsmokers' exposure to secondhand smoke: United States, 1999–2008. MMWR Morb Mortal Wkly Rep, 59, 2010, 1141–1146.
6. Hubert Chen, review updated on the health central network, 2005.
7. Harlanogdanchon, what is the best way to lower cholesterol and blood pressure naturally, 2012.
8. Mitchell, B, et al., Tobacco Use and Cessation: The Adverse Health Effects of Tobacco and Tobacco-Related Products, Primary Care: Clinics in Office Practice, 26(3), 1999, 463-98.
9. Society for Vascular Surgery, North Point Domain, 2010.
10. Griffin R M, WebMD LLC, 2005-2012.
11. Vadnere G P et al., Investigation on in-vitro Antioxidant activity of whole plant of *Cassia occidentalis* Linn. (Caesalpiniaceae)", IJPRIF, Vol 3, 2011, 1985-1991.
12. Engler M. M, Engler M. B., Antioxidant Vitamins C and E Improve Endothelial Function in Children with Hyperlipidemia: Endothelial Assessment of Risk from Lipids in Youth (EARLY), American Heart Association, 2003, 0009-7322,
13. Yadav Yashraj, Mohanty PK, et al, Anti-inflammatory activity of hydroalcoholic extract of *Quisqualis indica* Linn. flower in rats, International Journal of Pharmacy & Life Sciences, 2, 2011, 977-981.
14. Yadav Yashraj, Mohanty PK, et al, Evaluation of immunomodulatory activity of hydroalcoholic extract of *Quisqualis indica* Linn. flower in wistar rats, IJPLS, 2, 2011, 689-686.
15. Nitu Singh et al., Antipyretic activity of methanolic extract of leaves of *Quisqualis indica* linn, IJPRD, 2, 2010, 122-126.
16. Jahan Fatima N, Rahman Mohammad S. et al, Diphenylpropanoids from *Quisqualis indica* Linn. and their Anti-staphylococcal Activity, Latin American Journal of Pharmacy, 28 (2), 2009, 279-83.
17. Kaisar Md. Abul, Islam Mohammad Rashedul, et al, Total Phenolic Content, Free Radical Scavenging Activity and Reducing Power of *Quisqualis indica* Linn, Dhaka Univ. J. Pharm. Sci. 8(2), 2009, 173-175.
18. Wetwitayaklung Penpan, Limmatvapirat Chutima, et al, "Kinetics of Acetylcholinesterase Inhibition of *Quisqualis indica* Linn. Flower Extract" Silpakorn U Science & Tech J, 1(2), 2007, 20-28.
19. Bagchi Manashi, Mark Milnes et al, Acute and chronic stress-induced oxidative gastrointestinal injury in rats, and the protective ability of a novel grape seed proanthocyanidin extract, Nutrition Research 19 (8), 1999, 1189–1199.
20. Paech D., Tracey M. V., Modern Methods of Plant Analysis, Vol. IV. Berlin, Springer- Verlag, 1995, 373-374.
21. Ecobichon DJ., The basis of toxicity testing. CRS Press, New York. 1997, 43-49.
22. Kaplal A, lavernel L.S., Lipid metabolism, In clinical Chemistry : interpretation and techniques,2nd ed., Lea & febiger, Philadelphia, 1983, 333-336.
23. Executive summary on the 3rd report of the NCEP expert Panel on detection, evaluation and treatment of high blood cholesterol in adults (adult treatment Panel-III).
24. Warnholtz.A, Mollnau.M et al, Antioxidants and endothelial dysfunction in hyperlipidemia. Curr. Hytertens. Rep, 3, 2001, 53-60.
25. Wilson, P.W., Abbott R.D et al., High density lipoprotein cholesterol and mortality, The Framingham heart study. Arteriosclerosis, 8, 1988, 737-740.

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