ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Antihyperlipidemic Activity of *Ananas Comosus* L. Leaves Extract in Albino Rats.

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Ananas comosus Linn also named pineapple, has long been one of the most popular of tropical and subtropical fruits which is widely distributed through the world. There are some reports that *Ananas comosus* is used traditionally anti-hyperlipidemic plant, but there is no scientific anti-hyperlipidemic activity has been carried out by this plant in rats. The model used to evaluate the anti hyperlipidemic activity were high fat diet induced hyperlipidemic rats, Physical parameters like body weights, feed intake, organ weights, biochemical parameters like blood glucose, lipidprofile were monitored. In high fat diet (HFD) induced hyperlipidemia rats treated with 600mg/kg p.o HAAC showed extreme significant reduction in body weights, TC, TG, LDL, VLDL and blood glucose levels and increased HDL compared to HFD control, whereas animals treated with 400mg/kg p.o HAAC showed significant in body weights and VLDL, very significant reduction in TG, TC and significant increase in HDL compared to HFD control.

Keywords: *Ananas comosus*; anti-hyperlipidemic activity; and hyperlipidemia.

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ISSN: 0975-8585

INTRODUCTION

Health is defined as soundless of physical, mental or moral condition, especially freedom from bodily pain or disease, but true health is more than that. It includes the joy of living, the power and ability to lead a satisfying and purposeful life.

Hyperlipidaemia is a term used to describe several conditions in which high concentrations of lipids exist in the bloodstream and it results from abnormalities in lipid metabolism or plasma lipid transport or a disorder in the synthesis and degradation of plasma lipoproteins [1]. Hyperlipidaemia is a common disorder in developed countries and is the major cause of coronary heart disease that results from high levels of fats in the blood. These fats include cholesterol and triglycerides. These are important for our body to function but when they are high, they can cause heart disease and stroke. Heart disease and stroke are usually due to atherosclerosis of large and medium sized arteries. Hypercholesterolemia is the most important factor in the pathogenesis of atherosclerosis. Hyperlipidaemia is manifested as hypercholesterolemia and/or hypertriglycerolemia. However, hypercholesterolemia is the most common hyperlipidaemia. The lipids that are involved in hypercholesterolemia are cholesterol, an essential component of cell membrane and a precursor of steroid hormone synthesis and triglycerides, an important energy source. They are transported in blood as lipoproteins. The consequence of hyperlipidaemia is that with time it can cause atherosclerosis, and thus the risk of coronary heart disease and stroke is increased. Hyperlipidaemia is associated with risk factors like atherosclerosis, hypertension, Type II Diabetes mellitus, obesity, myocardial infarction, congestive cardiac failure, angina pectoris, gall bladder diseases, degenerative joint diseases, sleep apnea, and infertility. Allopathic drugs are available for counteracting liver injury and hyperlipidaemia, but the side effects and cost associated with these allopathic drugs necessitates the search for alternative which are without side effects. Management of hyperlipidemia without any side effects is still a challenge to the medical system. Although many efficacious lipid lowering drugs exist, none is effective for all lipoprotein disorders, and all such agents are associated with some adverse effects [2]. Plant products are frequently considered to be less toxic and more free from side effects than synthetic ones. The World Health Organization has estimated that perhaps 80% of earths 6 billion inhabitance rely upon traditional medicine for their primary health care needs, and a major part of this therapy involves the use of plant extracts or their active principles. *Ananas comosus* L (Family: Bromeliaceae), also named pineapple, has long been one of the most popular of tropical and subtropical fruits. Besides agricultural utilities such as being a fruit with nutritional value, some folk medicinal uses have been found. Some studies claim that, the extract of *Ananas comosus* leaves enriched with phenols has anti-hyperlipidemic, antidiabetic, and anti-oxidative effects [3]. The present study aims to evaluate the antihyperlipidemic activity of hydro-alcoholic extract of *Ananas comosus* L. Leaves (HAAC) in high fat diet induced hyperlipidemic rats.

MATERIALS AND METHODS

Collection of Plant Material

Leaves of *Ananas comosus* were collected from the pineapple fields

Ernakulam, Kerala in the month of June 2011 and its identification was confirmed by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, Sri Venkateswara University, Tirupathi, Andhra Pradesh, India.

Preparation of Leaves Extract

Leaves were chopped to pieces and dried for 10 days under the shade, then dried leaves were powdered and the powder was sieved under mesh. The coarse powder was subjected to successive extraction with Hydro-alcohol (70% v/v) in Soxhlet's extractor for 18 h and the extract thus obtained was evaporated to get the semisolid mass and stored in air tight container [4].

Preliminary Phytochemical Qualitative Analysis

The extract of *Ananas comosus* leaves was prepared and subjected to qualitative test for identification of various plant constituents [5].

Animals

Adult Wistar rats of 150-200 g were used for the study. The inbred colonies of rats were obtained from the Biogen Bangalore for experimental purpose. The animals were maintained under controlled conditions of temperature (23 ± 2 C), humidity (50 ± 5%) and 12 h light-dark cycles. All the animals were acclimatized for seven days before the study. The animals were randomized into experimental and control groups and housed individually in sanitized polypropylene cages containing sterile husk as bedding. They had free assessed to standard pellets as basal diet and water *ad libitum*. Animals were habituated to laboratory conditions for 48 h prior to experimental protocol to minimize if any of non-specific stress. All the studies conducted were approved by the Institutional Animal Ethical Committee (IAEC) of Gautham College of Pharmacy, Bangalore (GCP/IAEC/003/5/2010) according to prescribed guidelines of Committee for the Purpose of Control and Supervision of Experiments on Animals (Reg No: 491/01/c/CPCSEA), Govt. of India.

Determination of Acute Toxicity (LD_{50})

Female Albino rats of weighing 160-220g were used for the study. They were nulliparous and non-pregnant. These were acclimatized to laboratory condition for one week prior to start of dosing. Hydro-alcoholic extract of *Ananas comosus* (HAAC) was dissolved in suitable solvent, to prepare a dose of 2000 mg/kg. The doses were selected according to the OECD guideline no. 425. The procedure was divided into two phases. Phase I (observation made on day one) and Phase II (observed the animals for next 14 days of drug administration). Two sets of healthy female rats (each set of 3 rats) were used for this experiment. First set of animals were divided into three groups, each of one in a group.

Animals were fasted overnight with water *ad libitum.* Animals received a single dose of 2000 mg/kg, p.o was selected for the test, as the test item was a source from herb. After administration of HAAC, food was withheld for 3-4 hrs. If the animal dies, conduct the main test to determine the LD50. If the animal survives, dose four additional animals sequentially so that a total of five animals are tested. However, if three animals die, the limit test is terminated and the main test is performed. The LD50 is greater than 2000 mg/kg, if three or more animals survive. If an animal unexpectedly dies late in the study, and there are other survivors, it is appropriate to stop dosing and observe all animals to see if other animals will also die during a similar observation period. Late deaths should be counted the same as other deaths. The same procedure was repeated with another set of animals to nullify the errors [6].

Evaluation of Antihyperlipidemic Activity

Preparation of Extract Dose

Accurately 6 gm of HAAC was weighed and suspended in 30 ml of distilled water using tween80 and thus formed suspension is sonicated 10 min at medium vibration to obtain uniform suspension. Each ml of the suspension contained 200 mg/ml.

High Fat Induced Hyperlipidemia [7]

Experimental Animals

Albino wistar rats weighing 160-220g were divided into six groups of six in each group.

High Fat Composition: Commercially available edible dalda (vanaspathy) and culinary grade coconut oil were obtained from local market. The high fat diet (HFD) was prepared by homogenously mixing dalda and coconut oil in the ratio of 3:2w/w.

Induction of Hyperlipidemia

Group I animals were administered with 10ml distilled water per kg body weight orally once daily for a period of four weeks by oral gavaging technique and served as negative control. For the Group II, III, IV, V, and VI in addition to normal diet and water prepared high fat diet was administered by gavaging to induce hyperlipidemia. HFD was gavaged at the dose rate of 10ml per kg body weight to each animal orally daily for a period of four weeks.

Treatment Protocol

Once the hyperlipidemia was induced between 0 to 4th week of the experiment, from the beginning of fifth week to the end of the eighth week, the HAAC treatment was carried out.

Group-I: Distilled water was administered and served as negative control. Group-II: Distilled water was administered and served as positive control

Group-III: Standard drug (Fenofibrate 200mg/kg, p.o) was administered.
Group-IV: HAAC was administered at a dose rate of 200mg/kg, p.o body weight. Group-V: HAAC was administered at a dose rate of 400mg/kg, p.o body weight. Group-VI: HAAC was administered at a dose rate of 600mg/kg, p.o body weight.

After the completion of eighth week i.e., 56 days, on 57th day blood was collected for the estimation of biochemical parameters. Before collection of blood the animals were kept overnight fasting.

Parameters studied for this test were body temperature, bodyweights, average feed intake, weights of liver, kidneys, spleen, paramatrial adipose tissue, blood glucose, total cholesterol, HDL, LDL, VLDL, triglycerides, atherogenic index.

Collection of Blood and Serum Samples

At the end of the experiment, blood was collected by cardiac puncture from each rat under mild ether anaesthesia. The blood samples were used for the estimation of glucose levels and remaining was allowed to clot for 30 min at room temperature and they were centrifuged at 3000 rpm for 10 minutes. The serum was used for the study of biochemical parameters.

Physical Parameters

Determination of Body Weight

Body weight of the all animals in each group of HFD induced hyperlipidemia method was determined on the 0th, 7th, 14th, 21st, 28th, 35th, 42nd, 49th, and 56th day of the experiment period. Differences in weights were observed [8].

Determination of Average Feed Intake

During the experimental period, feed intake, of rats were measured daily during 56 days. The amount of diet ingested was calculated as the difference between the weight of feed that remained in the food bin (Da) and the amount placed one day before (Db). These data were then used to calculate a daily average feed intake (g) according to the formula [9]:

$$g= Da-Db/6$$

where 6 correspond to the animals number in each

cage.

Determination of Weights of Liver, Kidneys, Spleen, Paramatrial Adipose Tissue.

Animals were sacrificed and livers, kidneys, spleens, paramatrial adipose tissues were isolated, washed with saline and weighed by using an electronic balance [10].

Biochemical Parameters

Biochemical parameters (triglycerides (TG), total cholesterol (TC), HDLc) were estimated by using Span diognastic kits. Low density lipoprotein (LDL) and very low density lipoprotein (VLDL) values were calculated by using Friedewald's formula as given below [11].

$$VLDL = TG/5$$

$$LDL = TC-(HDL+VLDL)$$

Atherogenic index (AI) were calculated by the Friedwald formula which is given below

$$AI = TC-HDL/HDL$$

Statistical Analysis

The values are expressed as Mean ± SEM. The data was analysed by using one way ANOVA followed by Dunnett's test using Graph Pad Prism software. Statistical significance was set at $P \leq 0.05$.

RESULTS

Preparation of Extract and the Percentage Yield

Extraction of leaves of *Ananas comosus* was carried out by using the soxhlet apparatus with hydro alcoholic solvent (70%v/v alchol) and calculated percentage yield of the extract.

Table No 1. Percentage Yield of HAAC

Sl. No	Extracts	Yield (gm)	Percentage Yield
1.	Hydroalcoholic	102.35	23.9%

Preliminary Phytochemical Qualitative Analysis

There is a presence of alkaloids, carbohydrates, flavanoids, glycosides, sterols/terpenes and saponins in HAAC.

Acute Toxicity Studies (LD_{50})

In both phase I and Phase II procedures, none of the animals show any toxicity upon the single administration of HAAC (2000 mg/kg p.o). Hence, the procedure is repeated by increasing the dose of extracts (3000 mg/kg p.o). None of the animals had shown any toxicity. Thus, 200, 400, 600 mg/kg body weights were selected as doses for the present study.

ISSN: 0975-8585

Effect of HAAC on HFD Induced Hyperlipidemic Rats

A study of 56 days antihyperlipidemia was done in HFD induced hyperlipidemic rats with HAAC and the results were tabulated below.

Effect of HAAC on Physical Parameters of HFD induced hyperlipidemic rats

Body Weight

During 56 days of hyperlipidemia induction and treatment, the body weights of animals were monitored for every 7 days starting from day 0. The values were tabulated in Table No 2a and 2b.

In first 28 days i.e., from day 0 to day 28, animals in the Groups II to VI increased their weights with extreme significance (336.0±3.19, 326.0±3.19, 330.3±3.08, 317.2±3.26, 320.8±1.85 respectively) when compared to normal control group (217.7±3.33gm).

From day 29 to day 56 animals in all the groups decreased their weights. But animals treated with 200mg/kg p.o HAAC did not show significance in decrease in weights up to day 56. Animals treated with 400mg/kg p.o showed significant decrease in weight from day 42 and on day49, 56 it showed extreme significance in body weight reduction 260.8±1.35gm, 249.8±1.10gm respectively. P<0.001. Animals treated with 600mg/kg p.o HAAC showed very significant weight decrease on day 35 301.0±3.56gm P<0.01, and showed extreme significant reduction in body weights from day 42 to day 56, 269.8±1.19gm, 226.5±0.92gm, 207.7±1.45 respectively P<0.001.

Fenofibrate showed its antihyperlipidemic activity effectively on body weights and exhibited extreme significant reduction in weights from day 35 to day 56 .

Table No 2a: Effect of HAAC on Body Weights in HFD Induced Hyperlipidemic Rats

Groups	Treatment	Body weights (gm)				
		Day 0	Day 7	Day 14	Day 21	Day 28
Group-I	Distilled water	188.2±3.877	197.3±3.92	204.3±3.94	210.5±3.51	217.7±3.33
Group-II	HFD	189.2±5.83	230.0±10.04**	278.0±9.48***	303.5±7.52***	336.0±3.19***
Group-III	HFD+fenofibrate	189.3±3.20	223.0±3.93*	256.0±3.64***	283.2±2.71***	326.0±3.19***
Group-IV	HFD+HAAC200mg/kg	206±18.4	211.5±3.11	255.2±4.39***	289.3±2.51***	330.3±3.08***
Group-V	HFD+HAAC400mg/kg	188.2±3.57	220.2±2.77*	255.5±3.56***	287.7±2.36***	317.2±3.26***
Group VI	HFD+HAAC600mg/kg	188.5±2.79	216.5±2.06*	259.0±2.69***	288.8±2.57***	320.8±1.85***

Values are Mean ± SEM (n=6) one way ANOVA followed by Dunnett's test. Where, *** P<0.001, ** P<0.01 and * P<0.05. All values are compared with normal control.

ISSN: 0975-8585

Table No 2b: Effect of HAAC on Body Weights in HFD Induced Hyperlipidemic Rats

Groups	Treatment	Body weights (gm)			
		Day 35	Day 42	Day 49	Day 56
Group-I	Saline	222.0±3.32***	226.5±2.95***	230.2±3.06***	235.3±2.65***
Group-II	HFD	321.0±6.97	307.7±4.97	298.2±5.08	287.2±4.88
Group-III	HFD+ fenofibrate	287.5±4.28	260.2±3.47***	220.3±4.14***	189.5±3.31***
Group-IV	HFD+HAAC200mg/kg	313.5±1.60	297.2±2.27	288.2±1.74	274.3±3.66*
Group-V	HFD+HAAC400mg/kg	309.52.81	290.8±2.46**	260.8±1.35***	249.8±1.10***
Group VI	HFD+HAAC600mg/kg	301.0±3.56**	269.8±1.19***	226.5±0.92***	207.7±1.45***

Values are Mean ± SEM (n=6) one way ANOVA followed by Dunnett's test. Where, *** P<0.001, ** P<0.01 and * P<0.05.
All values are compared with normal control.

Average Feed Intake

Quantities of feed consumed by rats in group I were more when compared to remaining groups, this was because rats were fed with extra HFD for 1 to 28 days during hyerlipidemia induction. The quantities of feed intake were the same in all groups, although body weight gain differed significantly between control groups, standard and HAAC treated groups. This difference was probably due to the different doses of HAAC extracts seems to exert a protective effect against overweight in HAAC treated group as compared to HFD control group. The values of average feed intake were tabulated in Table No 3.

Table No 3: Effect of HAAC on Feed Intake in HFD Induced Hyperlipidemic Rats.

Groups	Treatment	Avg feed intake 1-28 days	Avg feed intake 29-56 days
Group I	Normal saline	17.28±0.303	15.83±0.215
Group II	HFD	10.42±0.375	16.64±0.648
Group III	HFD+ fenofibrate 200mg/kg	10.25±0.250	17.29±0.500
Group IV	HFD + HAAC 200mg/kg	10.04±0.426	16.62±0.693
Group V	HFD+HAAC 400mg/kg	9.99±0.474	16.95±427
Group VI	HFD+HAAC 600mg/kg	10.75±0.320	17.25±0.309

Different Organ Weights

Weights of different organs like liver, kidney, spleen, paramatrial adipose tissue were observed in HFD induced hyperlipidemic rats. The weights of these organs were increased slightly in HFD control group compared to normal control group, and group of 200mg/kg HAAC did not show any effect whereas groups of 400mg/kg and 600mg/kg HAAC showed slight decrease in organ weights but not significant compared to HFD control group. The values of these weights were tabulated in Table No 4.

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Table No 4: Effect of HAAC on Organ Weights in HFD Induced Hyperlipidemic Rats.

Groups	Treatment	Liver weight (gm)	Right kidney weight(gm)	Left kidney weight (gm)	Spleen weight (gm)	Adipose tissue weight (gm)
Group I	Distilled water	7.9±0.09	0.61±0.01	0.61±0.007	0.805±0.007	6.950±0.095
Group II	HFD	8.96±0.06	0.73±0.007	073±0.007	0.900±0.005	12.25±0.519
Group III	HFD+ fenofibrate 200mg/kg	7.93±0.08	0.61±0.006	0.60±0.007	0.795±0.007	7.417±0.319
Group IV	HFD + HAAC200mg/kg	8.71±0.08	0.70±0.007	0.71±0.003	0.863±0.07	12.43±0.194
Group V	HFD+HAAC 400mg/kg	7.95±0.04	0.66±0.007	0.64±0.010	0.830±0.005	9.167±0.266
Group VI	HFD+HAAC 600mg/kg	7.81±0.11	0.60±0.007	0.62±0.009	0.793±0.008	7.317±0.267

Blood Glucose Levels

After 56 days of hyperlipidemia induction and treatment with HAAC in HFD induced hyperlipidemic rats blood glucose levels were monitored and the values were tabulated in Table No 5

Animals in HFD group exhibited very significant increase in blood glucose levels (94.50±3.43 mg/dl) compared to normal control group (72.67±1.82 mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC did not show significant reduction in blood glucose levels, animals treated with 400mg/kg HAAC showed significant decrease in blood glucose level 84.00±2.38 $P<0.05$., and animals treated with 600mg/kg showed very significant decrease in blood glucose levels 80.33±3.58 $P<0.01$. Fenofibrate decreased blood glucose levels very significantly 80.17±1.70mg/dl $P<0.01$.

Table no 5: Effect of HAAC on Blood Glucose Levels in HFD Induced Hyperlipidemic rats

Groups	Treatment	Blood glucose levels
Group I	Distilled water	72.67±1.82***
Group II	HFD	94.50±3.43
Group III	HFD+ fenofibrate200mg/kg	80.17±1.70**
Group IV	HFD + HAAC 200mg/kg	85.53±2.33
Group V	HFD+HAAC 400mg/kg	84.00±2.38*
Group VI	HFD+HAAC 600mg/kg	80.33±3.58**

Values are Mean ± SEM (n=6) one way ANOVA followed by Dunnett's test. Where, *** $P<0.001$, ** $P<0.01$ and * $P<0.05$. All values are compared with HFD control.

Serum Lipid Profile

The lipid profile was evaluated by estimating triglycerides (TG), total cholesterol (TC), HDL-Cholesterol (HDL-C), LDL-Cholesterol (LDL-C) and VLDL-Cholesterol (VLDL-C) in normal and hyperlipidemic animals. The values were tabulated in Table No 6

Triglycerides (TG)

Animals in HFD group exhibited very significant increase in triglycerides levels (339.1±3.44mg/dl) compared to normal control group (141.9±3.72mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC showed significant reduction in triglycerides levels 322.8±1.86mg/dl P<0.05, animals treated with 400mg/kg HAAC showed very significant decrease in triglycerides levels 235.5±4.92mg/dl P<0.01. And animals treated with 600mg/kg showed extremely significant decrease in triglycerides levels 173.1±4.53mg/dl
P<0.001.

Total cholesterol (TC)

Animals in HFD group exhibited very significant increase in total cholesterol levels (193.4±2.61mg/dl) compared to normal control group (122.8±2.18mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC did not show significant reduction in total cholesterol levels, animals treated with 400mg/kg HAAC showed very significant decrease in total cholesterol levels 180.3±3.21mg/dl P<0.01. and animals treated with 600mg/kg showed extremely significant decrease in total cholesterol levels 132.0±2.61mg/dl
P<0.001.

High Density Lipoprotein (HDL)

Animals in HFD group exhibited very significant decrease in HDL levels 23.40±1.43mg/dl) compared to normal control group (40.07±1.43mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC did not show significant increase in HDL levels, animals treated with 400mg/kg HAAC showed significant increase in HDL levels 29.36±1.32mg/dl P<0.05 and animals treated with 600mg/kg showed extremely significant increase in HDL levels 36.90±1.01mg/dl P<0.001.

Low Density Lipoprotein (LDL)

Animals in HFD group exhibited very significant increase in LDL levels (101.0±3.33mg/dl) compared to normal control group (54.38±2.94mg/dl).

Hyperlipidemic animals treated with 200mg/kg and 400mg/kg HAAC did not show significant reduction in LDL levels, and animals treated with 600mg/kg showed extremely significant decrease in LDL levels 60.51±2.67mg/dl P<0.001.

ISSN: 0975-8585

Very Low Density Lipoprotein (VLDL)

Animals in HFD group exhibited very significant increase in VLDL levels (68.81±0.92mg/dl) compared to normal control group (28.38±0.74mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC showed very significant reduction in VLDL levels 64.20±0.59mg/dl P<0.01, animals treated with 400mg/kg and 600mg/kg HAAC showed extremely significant decrease in VLDL levels 47.10±0.98mg/dl, 34.62±0.90 respectively P<0.001.

Fenofibrate treated group showed potent antihyperlipidemic activity and showed decrease in triglyceride, total cholesterol, LDLc, VLDLc levels, increase in HDL c levels with extreme significance compared to HFD control group.

Table no 6: Effect of HAAC on Lipid Profile in HFD Induced Hyperlipidemic Rats

Groups	Treatment	Serum Lipid Profile mg/dl				
		TG	TC	HDL-C	LDL-C	VLDL-C
Group-I	Distilled water	141.9±3.72***	122.8±2.18***	40.07±1.43***	54.38±2.94***	28.38±0.74***
Group-II	HFD	339.1±3.44	193.4±2.61	23.40±1.43	101.0±3.33	68.81±0.92
Group-III	HFD+ fenofibrate 200mg/kg	164.5±2.76***	129.4±2.26***	38.49±2.07***	58.01±1.38***	32.90±0.55***
Group-IV	HFD + HAAC 200mg/kg	322.8±1.86*	185.6±1.70	24.20±1.13	98.85±3.95	64.20±0.59**
Group-V	HFD+HAAC 400mg/kg	235.5±4.92**	180.3±3.21**	29.36±1.32*	100.5±1.69	47.10±0.98***
Group- VI	HFD+HAAC 600mg/kg	173.1±4.53***	132.0±2.61***	36.90±1.01***	60.51±2.67***	34.62±0.90***

Values are Mean ± SEM (n=6) one way ANOVA followed by Dunnett's test. Where, *** P<0.001, ** P<0.01 and * P<0.05. All values are compared with HFD control

Atherogenic Index

Atherogenic index values were calculated in HFD induced hyperlipidemic rats and the values were tabulated in Table no 7.

Animals in HFD group exhibited very significant increase in Atherogenic index value (7.42±0.56mg/dl) compared to normal control group (2.08±0.12mg/dl).

Hyperlipidemic animals treated with 200mg/kg HAAC did not show decrease in Atherogenic index, animals treated with 400mg/kg and 600mg/kg HAAC showed extremely significant decrease in Atherogenic index 4.80±0.16mg/dl, 2.59±0.15 respectively P<0.001.

Table no 7: Effect of HAAC on Atherogenic Index in HFD Induced Hyperlipidemic Rats

ISSN: 0975-8585

Groups	Treatment	Atherogenic Index
Group I	Distilled water	2.08±0.12***
Group II	HFD	7.42±0.56
Group III	HFD+ fenofibrate 200mg/kg	2.39±0.14***
Group IV	HFD + HAAC 200mg/kg	6.76±0.45
Group V	HFD+HAAC 400mg/kg	4.80±0.16***
Group VI	HFD+HAAC 600mg/kg	2.59±0.15***

Values are Mean ± SEM (n=6) one way ANOVA followed by Dunnett's test. Where, *** P<0.001. All values are compared with HFD control.

DISCUSSION

The study was carried out to evaluate the antihyperlidemic activity of *Ananas comosus* leaves extract in different models of rats. It was demonstrated by body weights, feed intake, organ weights, and lipid profile. The body weights during induction period of hyperlipidemia were increased with extreme significance, and up on treatment with HAAC, body weights were decreased. During the total experiment period feed intake by animals was quite similar. So feed intake did not affect the body weight reduction in animals and it might be due to the HAAC administration. Different organ weights were also observed which showed slight decrease in weights compared to HFD control but not significant. Plasma lipid levels were determined by exogenous lipid absorption and endogenous lipid synthesis and metabolism in body, which usually involves targets for lipid regulating drugs. The possible mechanism for the antihyperlipideimic activity in the present study shown by HAAC may be attributed to its estrogenic constituents that indirectly increase thyroid hormone levels that will shift lipid metabolism towards its catabolic side [12]. The reduction of serum total cholesterol was associated with a decrease of its LDL fraction which is a major, potentially modifiable risk factor of cardio vascular diseases and the target of many suggest that cholesterol lowering activity of this product appears to be due to the enhancement of LDL – C catabolism through hepatic receptors [13,14]. Flavonoids present in HAAC may augment the activity of lecithin acyl tranferase (LCAT), which regulates blood lipids. In the trial of hyperlipidemia, serum total cholesterol level was determined mainly by endogenous cholesterol metabolism when high-fat diets were replaced with normal chow. HAAC accelerated the fall in serum total cholesterol level, which suggested that HAAC inhibited the synthesis of endogenous cholesterol. The hypolipidemic effect of HAAC may also be attributed to increase excretion of fecal bile components that results in increased total bile output and subsequent increase of conversion of cholesterol to bile acids and salts, prevention of accumulation of lipids in liver [15] some components of HAAC may compete with cholesterol binding sites or interfere with cholesterol bio synthesis in liver, thereby reducing blood cholesterol level [16]. The probable hypolipidemic mechanism of HAAC may be due an increasing in HDL indicating that components in HAAC may involved in mobilizing cholesterol from extra hepatic tissue to liver where it is catabolised. These results established the idea that the hypolipidemic effect of HAAC *In-vivo* was might be associated

with flavonoids enriched in HAAC since flavonoids could inhibit HMGCoA reductase activity [17]. Administration HAAC provided a beneficial action on rat lipid metabolism in regard to the reduction of AI. In fact, the AI was decreased in all treated groups.

CONCLUSION

The present study indicated that administration of HAAC at a dose of 600mg/kg produced significant antihyperlipidemic activity in HFD induced hyperlipidemic rats. HAAC at a dose of 400mgkg shows less effect than the 600mg/kg in reducing the lipid levels and body weights. The acute toxicity study indicated that the extracts are devoid of major toxic effects. The effect of HAAC in normal rats, HFD rats indicated that it have better hypolipidemic control compared with the normal control animals. Besides this, the drug administered to treat HFD induced hyperlipidemic rats showed a significant reduction in blood glucose levels and the other serum lipids such as TC, TG, LDL, VLDL levels, AI and also increases the HDL levels. These observations concluded that the hydro alcoholic extracts of the plant *Ananas comosus* possess antihyperlipidemic effects. The mechanism has point towards inhibiting cholesterol and triglyceride synthesis.

ACKNOWLEDGEMENTS

I would like to sincerely thank **Mrs. Kavitha Sarvesh,** Chairperson and **Mrs. Anitha Prasad** Management membe rof Gautham College of Pharmacy, for providing facilities and opportunity to accomplish this endeavour successfully.

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