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RESEARCH ARTICLE **OPEN ACCESS**

IN VITRO PANCREATIC LIPASE INHIBITION POTENTIAL OF *PANCHAVALKALA* AQUEOUS AND ETHANOLIC BARK EXTRACTS.

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ABSTRACT:

Background: Hyperlipidemia and obesity are excessive accumulation of fat in *Asthayi* (unstable form) and *Sthayi roopa* (stable form) within the body that may have deleterious health effects. One of the approached methods of treating obesity and impaired fat metabolism is inhibition of pancreatic lipase activity. Pancreatic lipase breaks down triglycerides into fatty acids which then get absorbed through the duodenal mucosa. A pancreatic lipase inhibitor prevents the formation of fatty acids and hence prevents accumulation of fats in the body. **Objective:** To evaluate *Panchavalkala dravyas* individually and together both in aqueous and ethanol extracts for inhibitory activity against pancreatic lipase. **Materials and methods:** In the present study, aqueous and ethanolic extracts of *Nyagrodha (Ficus benghalensis L.), Udumbara (Ficus racemosa L.), Ashwatha (Ficus religiosa L.), Plaksha (Ficus virens Aiton) and Parisha (Thespesia populnea (L.)Sol.ex Correa)* their mixture (1:1:1:1:1 combination) were evaluated for their inhibitory effect on Tri Acyl Glycerol Lipase enzyme using in vitro assay. In vitro pancreatic lipase inhibition activity study is carried out on both aqueous and ethanoic extracts of PVK *dravyas* together and on individual drugs. Orlistat is used as standard synthetic drug and caffeine as natural positive control. **Results:** PVK *dravyas* together and individually exhibited potent PL inhibitory activity except ethanoic extract of *Parisha (Thespesia populnea (L.) Sol.ex Correa)*. Individually *Ashwatha- Ficus religiosa L.* EE has shown potent pancreatic lipase inhibition with an IC_{50} value of 18.2 µg/ml and *Udumbara -Ficus racemosa L.* AE shown IC_{50} value of 29.47µg/ml when compared to orlistat the IC_{50} value is 30.59µg/ml. **Conclusion:** The inhibitory activity was demonstrated by PVK drugs together can play a beneficial role in the treatment of hyperlipidemia and obesity.

Keywords – PVK-*Panchavalkala*, AE-Aqueous extract, EE- Ethanolic extract, Obesity, PL-Pancreatic lipase, IC-inhibitory concentration

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I. INTRODUCTION:

Panchavalkalas are group of trees belong to Moraceae family found all over India. Their barks have ethno pharmacological uses for the treatment of diarrhea, hemorrhage, coughs, wound healing properties and also used as an antiseptic, antidiabetic, antioxidant and anti hyperlipidemic agent. [1] Several works have demonstrated the anti hyperlipedemic, antioxidant and antidiabetic activity. [2] The bark of PVK contain phytoconstituents like tannins, flavonoids, polyphenols such as gallic acid, ellagic acid, caffeic acid, burgeptol, beta sitosterol stegmasterol and etc. [3,4,5]

Medodusti (dyslipidemia*)*, *Medovriddi* (hyperlipidemia) *Sthouyla* (obesity), is excessive accumulation of fat in *Asthayiroopa* (unstable form) and *Sthayi roopa* (stable form) within the body that may have deleterious health effects. One of the approached methods of treating obesity and impaired fat metabolism is inhibition of pancreatic lipase activity. Pancreatic lipase breaks down triglycerides into fatty acids which then get absorbed through the duodenal mucosa. A pancreatic lipase inhibitor prevents the formation of fatty acids and hence prevents any accumulation of fats in the body.

Human pancreatic lipase (triacylglycerol acyl hydrolase EC3.1.1.3) is the most widely studied member of the human lipase family related to carboxyl esterase. It is secreted from the acinar cell of pancreas and has strong preference for triacylglycerides over cholesterol esters, phospholipids, and galactolipids. Apart from the hydrolysis of triacylglycerides, pancreatic lipase may cause the hydrolysis of retinyl esters *in vivo*. So, it is very much evidenced that pancreatic lipase with its cofactor colipase has prominent role in efficient digestion of dietary fat. Hence, the modulation of human pancreatic lipase may represent a new insight in the discovery of a number of therapeutics that can inhibit the absorption of fat in body and can be used in obesity and other related metabolic disorders. [9]

Orlistat is the drug of choice for inhibition of pancreatic lipase enzyme. However, the utility of orlistat is limited due to its toxicity to several internal organs, including, kidney and liver. Therefore, the use of synthetic drugs is limited by fatal adverse reactions, resulting in poor tolerability and patient compliance. This prompts an urgent need to search agents that are less toxic and thereby provide better safety and efficacy even on long term usage. Apart from the usual ways of dieting, exercising and yoga, people include Ayurveda products into their daily meal courses and regularly intake powders or juices of herbal plants as a remedy. Many extracts from plants,

fungi, bacteria or algae are tested for their inhibition of pancreatic lipase activity. Many such extracts or powders are reported in literatures that show high levels of inhibitory activity. Polyphenols, flavonoids and saponins are known to show a promising inhibiting effect. Since lipids from the ingesta represent the prime source of unwanted calories, inhibition of fat digestion is a preferable approach for reducing absorption of fats .[6,7]

The aim of the present study is to determine inhibitory potentials of *Panchavalkala dravyas* on pancreatic lipase activity *in vitro* as a combination and individually both in ethanoic and aqueous extracts.

II. Materials & Methods

2.1 Plant material:

The barks of *Panchavalkala* trees i.e. *Nyagrodha (Ficus benghalensis L.), Udumbara (Ficus racemosa L.), Ashwatha (Ficus religiosa L.), Plaksha (Ficus virens Aiton.), Parisha (Thespesiapopulnea (L.) Sol.ex Correa* were collected in *Sharad Rutu* i.e. Late Autumn season (November to January) from their natural habitat, Dhanvantari Vana Bangalore University Campus and Reserve Forest, Jaraka Bande Kaval, Bangalore North during the year 2019-20. The specimens were authenticated by Dr. Ravikumar K. Taxonomist and Senior Botanist's from FRLHT TDU, Yelahanka Bangalore – 560064. Quantity sufficient matured barks of *Panchavalkalas* were cut in to small pieces separately and were dried under mild sun light covering thin cloth for 2 days and later shade dried for 10 days. Then the dried barks were powdered using pulverizer at Sanjeevini Pharma Kengeri, Bangalore. The analytical study and phytochemical analysis is carried out at Skanda Life Sciences Pvt. Ltd. DSIR recognized R & D center, Bangalore-560091 for the presence of marker compounds as mentioned in the Quality standards of Indian medicinal plants published by ICMR and API, CSIR

2.2 Preparation of the extract: The bark powders of PVK dravyas were extracted using ethanol and water as solvents individually and together. Accurately weighed 5g of powdered drug into 250ml conical flask with stopper. 100ml of solvent was added to it. Flask was frequently shacked during first 6hrs. Kept without disturbing for 18 hrs and then filtered. 25ml of filtrate was pipetted and evaporated to complete dryness. Then dry residue at105oC to constant weight. This semi solid sample was used for the study.

2.3 Preparation of Extract for In Vitro Assay: The tested extracts were initially dissolved in DMSO to give five different stock solutions with different concentration range. Subsequently, 20 µL aliquot of each stock solution was used in the reaction mixture to

give a final concentration range of 3.125- 200 µg/mL (3.125, 6.25, 12.5 25, 25, 50, 100 and 200 µg/mL).

2.4 Reagents:

Lipase (EC. 3.1.1.3) from Tri Acyl Glycerol Lipase, 20,000 units/g [100 units/5mg]; store at 2- 8°C. (Cas.No.9001-62-1, Catalogue No.62309-100mg), 4-Nitrophenyl palmitate (Mol. Wt. 377.52), store at 2-8°C (Sigma – Aldrich N9876-1G), Caffeine (Flavonoid) Sigma – Aldrich, Tris-HCl buffer (pH 7.6) (Thomas Baker, Catalogue No.159505), Orlistat [Biocon, Bangalore, India]

2.5 Enzyme Preparation The enzyme solutions were prepared immediately before use. Lipase (EC. 3.1.1.3) from Tri Acyl Glycerol Lipase, 20,000 units/g [100 units/5mg]; store at 2-8°C. (Cas.No.9001-62-1, Catalogue No.62309-100mg) was suspended in tris-HCl buffer (2.5 mmol, pH 7.6 with 2.5 mmol NaCl) to give a concentration of 200 unit/ml [8].

2.6 Preparation of working solution:

Tris-HCl buffer (pH 7.6)

(A) 50mM Tris – 6.057g in 1000mL of de-ionized water.

(B) 150mM NaCl – 8.766g in 1000mL of de-ionized water.

(C) 1mM EDTA – 0.372mg in 1000mL of de-ionized water.

(D) 10mM MOPS (3-morpholinopropane-1-sulfonic acid) – 2.09g in 1000mL of de-ionized water.

Dissolve all the chemicals in 800ml of de-ionized water and adjust the pH to 7.6 by using NaOH and HCL make up the volume to 1000ml using de-ionized water. Incubator, boiling water bath, Plate Reader equipment's were used to carry out the procedure.

2.7 Procedure.

Pre-incubate 100 µL Tris-HCL buffer; pH 7.6 along with test sample/reference standard of various concentrations and 10 µL of enzyme and incubate at 37°C for 10 minutes. Add 100 µL substrate (10mM 4-Nitrophenyl palmitate) and the total reaction volume was made to 1mL using the Tris-HCl buffer before measuring the absorbance at 410nm against blank using denatured enzyme. Incubate at 37°C for 8 minutes. Measure the absorbance at 410 nm. A control reaction is carried out without the test sample. The pancreatic lipase activity is defined as an increase of the rate of p-nitrophenol release which can be estimated from the slope of the linear segment of (absorbance vs time) profiles [9, 10].

2.8 PL inhibition by test extract:

The lipase activity of PL was quantified by a colorimetric assay that measures the release of p-nitrophenol as previously described [11- 14]

however, with minor modification. The inhibition of pancreatic lipase activity by the prepared plant extracts was measured using the spectrophotometric assay described above. PL was pre-incubated with each particular extract for at least 8 minutes at 37 °C before adding the substrate. The final concentration of DMSO (Dimethyl Sulfoxide) was fixed and did not exceed 2.0%. The percentage of residual activity of PL was determined for each extract by comparing the lipase activity of PL with and without the extract. The concentration required to give 50% inhibition (IC_{50}) was determined for each tested extract. PL was pre-incubated with different concentrations (3.125-200 µg/mL) of each extract and the percentages of residual activity of PL data were used to evaluate the IC_{50} values. All assays were triplicated and the calculated inhibition percentages were the mean of 3 observations. Orlistat, a known inhibitor of PL, was used as a positive control in the assay mixture and Caffeine a flavonoid is used as a natural positive control [8, 9].

The relative pancreatic lipase activity (%) was calculated as follows:

% inhibition activity (I) =A
(absorbance control) – B
(absorbance test) / A (absorbance
control) X 100

Where, I is percentage of lipase inhibitory activity, A is the blank lipase activity (reaction solution without the sample addition), and B is the lipase activity of the sample (reaction solution containing the sample with various concentrations). The IC_{50} value was determined by plotting the percentage inhibition versus the sample concentration. Ionic concentration of buffer, Incubation temperature and time and Storage conditions of the reagents are the critical control points for the accurate results.

2.7 Statistical analysis: The data was collected by OD (optical density) metric method based on the concentration of test drug from 3.125, 6.25, 12.5, 25, 50, 100 and 200 µg/ml. Half maximal inhibitory concentration IC_{50} is the concentration of the substance required to inhibit a biological process such as enzyme ,cell, cell receptor by half. Percentage of inhibition and IC_{50} value is calculated using Graph pad prism software version 8.0 by nonlinear regression analysis of % inhibition recorded for different concentrations of test substance/ standard. For compounds showing <50% inhibition, IC_{50} value is not calculated.

III. Results:

Orlistat is the only commercial drug in the market affective against pancreatic lipase. The aim of this study is to evaluate the pancreatic lipase inhibition potential of *Panchavalkala*

aqueous and ethanoic extract in combination and individually. Orlistat is used as standard and caffeic acid is used as natural positive control to evaluate *in vitro* pancreatic lipase inhibition activity of five bark powders individually and in combination at various concentrations (3.125-100 µg/ml) as shown in the table-1. The concentration related inhibition activity is measured using the spectrophotometric assay calculating the IC_{50} µg/ml value of the samples and is represented in a graphical form in graphs 1to 13. IC_{50} value is half maximal inhibitory concentration indicate how much drug µg/ml is needed to inhibit a biological process by half, thus providing a measure of potency of an agonist drug.

Table 1: Lipase inhibition activity of standard (*Orlistat*) and Natural positive control (*Caffeine*)

Compound name	Conc. µg/ml	OD at 410nm	% Inhibition	IC_{50} µg/ml
Control	0	0.8132	0.00	
Orlistat	3.125	0.706	13.22	**30.59**
	6.25	0.631	22.36	
	12.5	0.572	29.60	
	25	0.421	48.19	
	50	0.313	61.52	
	100	0.198	75.68	
Control	0	1.078	0	34.64
Caffeine	3.125	1.007	6.62	
	6.25	0.904	16.20	
	12.5	0.864	19.85	
	25	0.691	35.95	
	50	0.276	74.39	
	100	0.110	89.82	

Table 2: Lipase inhibition activity of PVK Ethanolic extracts

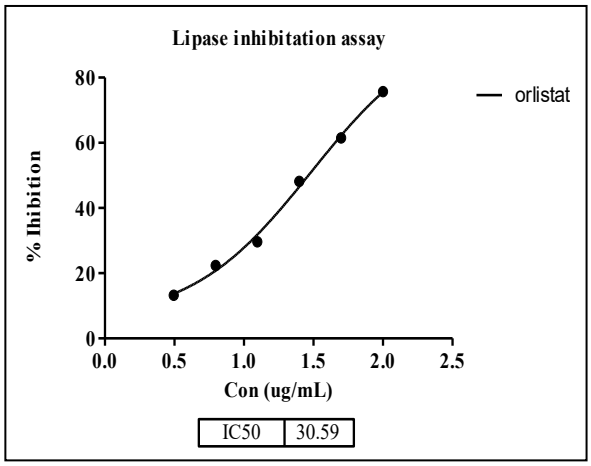
Compound name	Conc. µg/ml	OD at 410nm	% Inhibition	IC_{50} µg/ml
Control	0	1.078	0	
EE F. racemose	3.125	0.935	13.27	60.21
	6.25	0.875	18.89	
	12.5	0.819	24.02	
	25	0.753	30.15	
	50	0.588	45.50	

	100	0.443	58.91	
	200	0.346	67.91	
EE F. benghalensis	3.125	0.905	16.12	46.12
	6.25	0.857	20.49	
	12.5	0.733	32.08	
	25	0.629	41.66	
	50	0.485	55.02	
	100	0.330	69.45	
	200	0.193	82.07	
EE F. religiosa	3.125	0.921	14.58	18.2
	6.25	0.792	26.59	
	12.5	0.6534	39.41	
	25	0.502	53.49	
	50	0.394	63.50	
	100	0.281	73.95	
	200	0.172	84.02	
EE F. virens	3.125	0.931	13.64	49.07
	6.25	0.865	19.75	
	12.5	0.797	26.08	
	25	0.704	34.75	
	50	0.537	50.19	
	100	0.405	62.46	
	200	0.295	72.62	
EE T. populnea	3.125	1.061	1.60	IC50 was not calculated due to lesser % of Inhibition
	6.25	1.055	2.16	
	12.5	1.034	4.13	
	25	0.994	7.82	
	50	0.906	15.96	
	100	0.834	22.66	
	200	0.764	29.19	
EE PVK	3.125	0.910	15.60	24.12
	6.25	0.767	28.91	
	12.5	0.621	42.41	
	25	0.523	51.50	
	50	0.463	57.03	
	100	0.320	70.36	
	200	0.160	85.20	

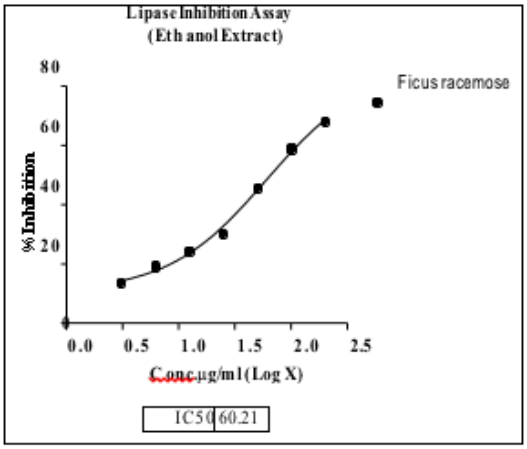
Table 3: Lipase inhibition activity of PVK aqueous extracts

Compound name	Conc. µg/ml	OD at 410nm	% Inhibition	IC_{50} µg/ml
Control	0	1.078	0	
AE F. racemose	3.125	0.908	15.83	29.47
	6.25	0.851	21.10	
	12.5	0.672	37.70	
	25	0.578	46.39	
	50	0.440	59.23	
	100	0.307	71.51	
	200	0.181	83.25	
AE F. benghalensis	3.125	1.036	3.96	185.70
	6.25	0.998	7.45	
	12.5	0.951	11.83	
	25	0.884	18.02	
	50	0.815	24.40	
	100	0.696	35.44	
	200	0.494	54.15	
AE F. religiosa	3.125	1.012	6.16	114.2
	6.25	0.945	12.34	
	12.5	0.901	16.44	
	25	0.826	23.40	
	50	0.735	31.86	
	100	0.583	45.96	
	200	0.407	62.23	
AE F. virens	3.125	0.980	9.11	88.32
	6.25	0.890	17.49	
	12.5	0.823	23.66	
	25	0.754	30.06	
	50	0.668	38.02	
	100	0.517	52.07	
	200	0.343	68.19	
AE T. populnea	3.125	0.992	7.98	178.5
	6.25	0.945	12.36	
	12.5	0.884	17.99	
	25	0.844	21.74	

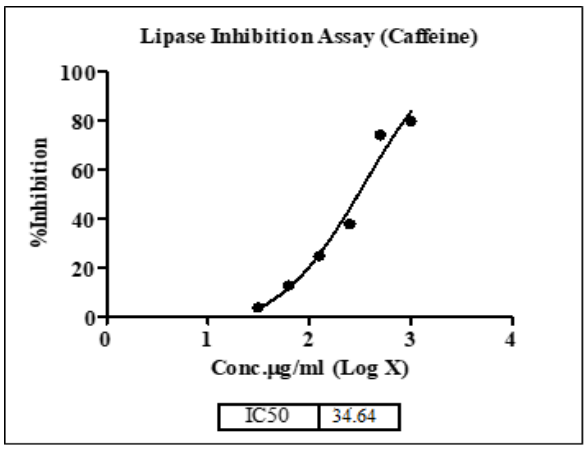
	50	0.787	26.99	
	100	0.692	35.79	
	200	0.510	52.71	
AE PVK	3.125	0.924	14.36	41.36
	6.25	0.846	21.56	
	12.5	0.720	33.26	
	25	0.643	40.34	
	50	0.492	54.35	
	100	0.333	69.12	
	200	0.219	79.66	



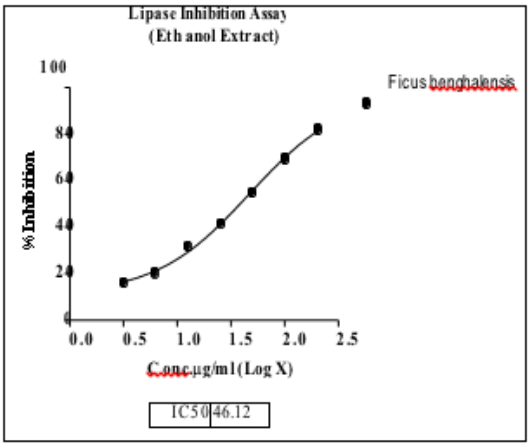
Graph 1: Lipase Inhibition activity of Standard (*Orlistat*)



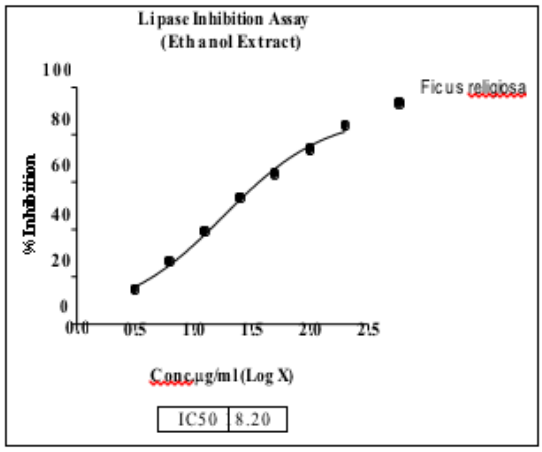
Graph 3: Lipase Inhibition activity of EE F. racemose



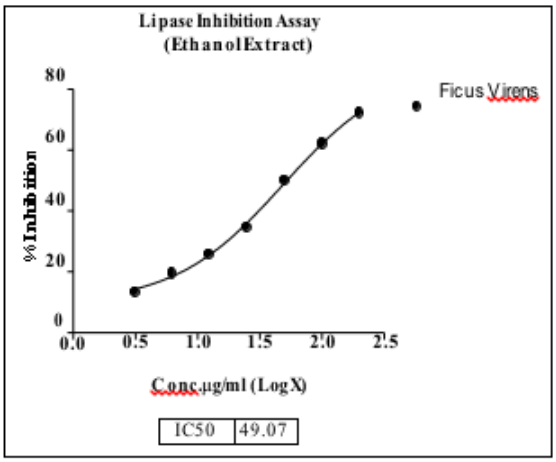
Graph 2: Lipase Inhibition activity of Natural Positive Control *Caffeine*



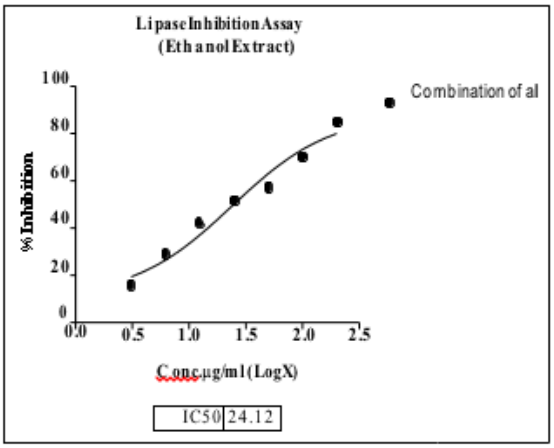
Graph 4: Lipase Inhibition activity of EE F. benghalensis



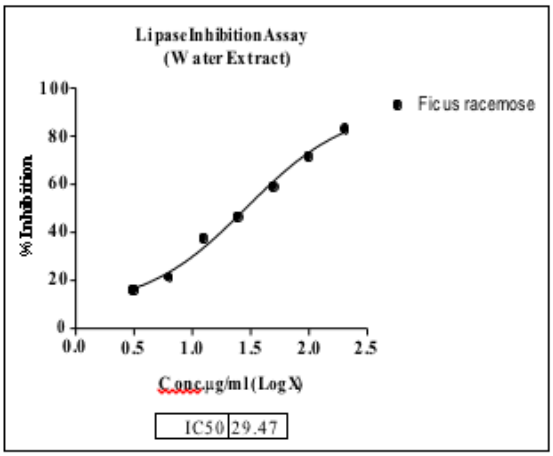
Graph 5: Lipase Inhibition activity of EE F. religiosa



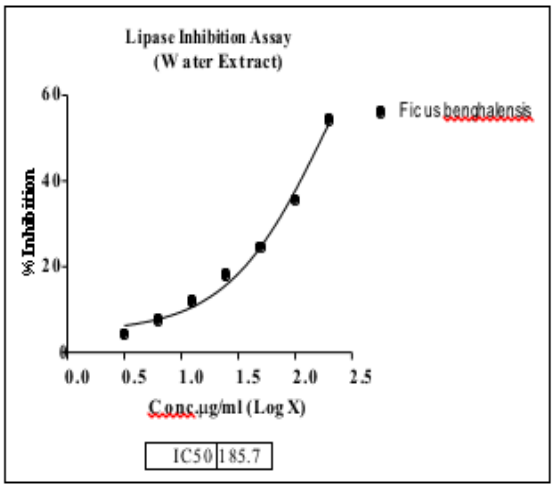
Graph 6: Lipase Inhibition activity of EE F. virens



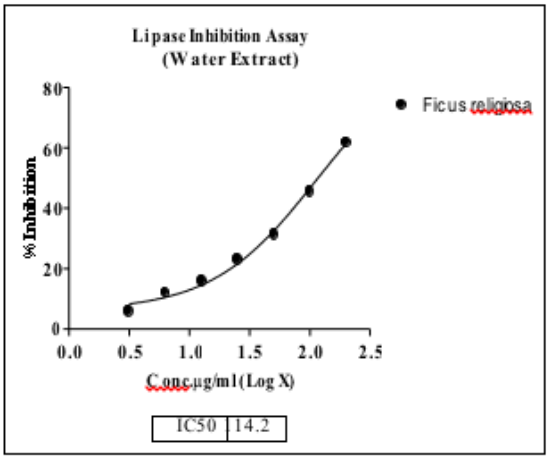
Graph 7: Lipase Inhibition activity of EE PVK Combination



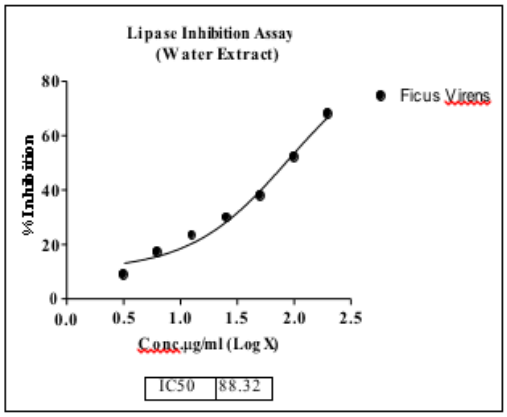
Graph 8: Lipase Inhibition activity of AE F. racemosa



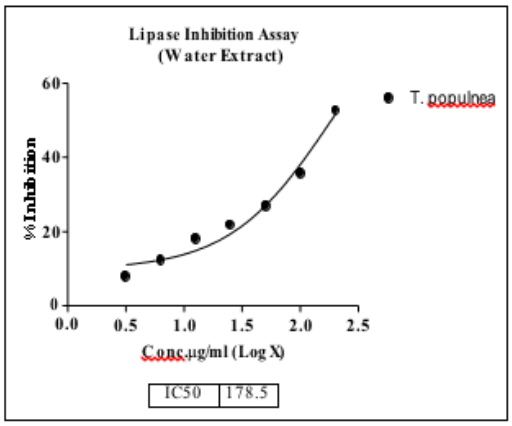
Graph 9: Lipase Inhibition activity of AE F. benghalensis



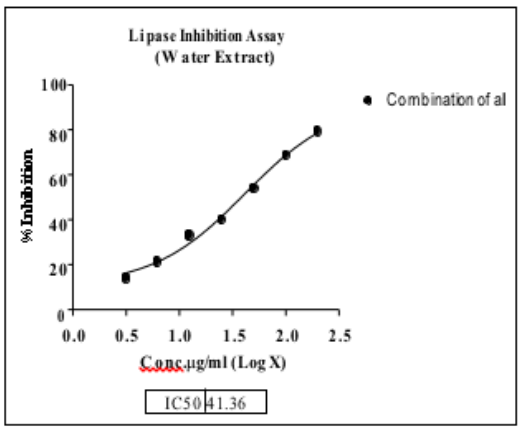
Graph 10: Lipase Inhibition activity of AE F. religiosa



Graph 11: Lipase Inhibition activity of AE F. virens



Graph12: Lipase Inhibition activity of AE T. populnea



Graph 13: Lipase Inhibition activity of AE PVK Combination

IV. DISCUSSION:

There has been huge growth in the incidence of obesity, dyslipidemia, hyperlipidemia and associated CVD over the last 25 years. Progress in the discovery and development of new drugs is rather limited and the available drugs are associated with certain unpleasant adverse and side effects. Pancreatic Lipase (PL), the principal lipolytic enzyme synthesized and secreted by the pancreas, plays a key role in the efficient digestion of triglycerides. PL is responsible for the hydrolysis of 50–70% of total dietary fats. [23] PL inhibition is one of the most widely studied mechanisms for the determination of the potential efficacy of natural products as antiobesity agents. Various studies identified new compounds and natural products for their PL inhibitory effect which are more potent compared to orlistat. Some of these plant extracts showed profound inhibition effects on fat digestion and are rich in polyphenols, saponins, and terpenes .[19]Many pancreatic lipase inhibitors from nature are under preclinical investigations; unfortunately, none of these have reached clinical level. In fact, it can sometimes be very challenging to extrapolate the results from *in vivo* or *in vitro* studies to human subjects, because they have not been found in many cases to be significantly effective.

Pancreatic lipase inhibitory properties have been extensively examined for the determination of the potential effect of natural products as anti hyperlipidemic agents. Due to the huge success of natural products for management of Hyperlipidemia and obesity, more research has been focused on the identification of newer pancreatic lipase inhibitors with less unpleasant adverse effects. So far, many natural products (plant extracts and isolated compounds) have been reported for their pancreatic lipase inhibition property including protamine, [20] ε-polylysine ,[21] polysaccharides like chitosan, [22] dietary fibers from wheat bran and cholestyramine, [19]soya proteins, [20]and synthetic compounds.

Pancreatic Lipase (PL), the principal lipolytic enzyme synthesized and secreted by the pancreas, plays a key role in the efficient digestion of triglycerides. PL is responsible for the hydrolysis of 50–70% of total dietary fats. PL inhibition is one of the most widely studied mechanisms for the determination of the potential efficacy of natural products as antiobesity agents. [21] The process of Fat digestion, assimilation, absorption and metabolism begins with hydrolysis of triglyceride into free fatty acids and monoglyceride. It is accomplished majorly by pancreatic lipase, a water soluble enzyme. PL is a lipolytic enzyme which catalyzes the hydrolysis of the ester bonds present in the triacylglycerols. The enzyme works to remove the fatty acids located at position 1 and position 3 of the triglyceride, hence leaving a 2-monoglyceride and two free fatty acids. [13] The enzyme is present in the gastrointestinal tract inside the adipocytes. Pancreatic lipase on entering the pancreatic duct mixes with bile salts and liquids following which it reaches the duodenal lumen in order to complete fat digestion. The enzyme is substrate specific. It gives preference to triglycerides over phospholipids. It does not act on water soluble substrates and prefers water insoluble substrates. Pancreatic lipase activity increases when it encounters a water-oil interface. This property is called interfacial activation. Even though lipase is secreted into the duodenum, it is inhibited by bile salts and hence requires a pancreatic protein called colipase for its activity. The property of hydrolyzing water-insoluble substrates and interfacial activation distinguishes pancreatic triglyceride lipase from the rest of the lipases. After the action of lipase, the fatty acids and monoglycerides hence formed are still associated with the bile salts and they complex with other lipids to form structures termed as micelles**.** Micelles reach the microvilli and the lipids along with fatty acids and the monoglycerides are absorbed into the epithelial cells. This is the

mechanism by which the fats are broken down and then absorbed into the cells of small intestine, eventually, entering the blood stream [22] .When an anti-obesity agent which inhibits pancreatic lipase is ingested, it reaches the intestine via the gastrointestinal tract. As soon as it reached the pancreas, it comes in contact with pancreatic triglyceride lipase enzyme. It acts on the active site of the enzyme and causes inhibition of the enzyme hence preventing the binding of lipids on to the active site of the enzyme. This in turn prevents the hydrolysis of the dietary lipids hence hindering their absorption through the intestinal membrane. These lipid molecules are hence excreted via the large intestine. This mechanism is targeted to reduce the amount of body fat and hence the body weight.

The IC_{50} values are shown in tables. Half maximal inhibitory concentration (IC_{50}) was the concentration of the substance required to inhibit a biological process such as an enzyme, cell and cell receptor by half. IC_{50} value was calculated using Graph Prism software version 8.0 by non- linear regression analysis of % inhibition recorded for different concentrations of test substance/standard. The relative activity of test sample can be determined by comparing the IC_{50} value of sample with standard. Higher the IC_{50} value and lower was the relative activity in comparison to standard and vice-versa.

PVK in combination, the ethanol extract showed IC_{50} value of 24.12 µg/ml more significant than the aqueous extract which showed IC_{50} value of 41.36 µg/ml when compared to the Synthetic standard drug orlistat IC_{50} value of 30.59 µg/ml and a natural positive control caffeine IC_{50} value of 34.64 µg/ml as shown in table No.1&2 and graph No.1, 2, 7, 13.

PVK individually, ethanoic extract of Ficus religiosa showed very significant inhibition of in- vitro pancreatic lipase enzyme with IC_{50} value of 18.2 µg/ml followed by aqueous extract of Ficus racemose IC_{50} value of 29.47 µg/ml, ethanoic extract of Ficus benghalensis IC_{50} value of 46.12 µg/ml, ethanoic extract Ficus virens IC_{50} value of 49.07 µg/ml and ethanoic extract of Ficus racemose, IC_{50} value of 60.21 µg/ml. Similarly the aqueous extract of Ficus virens showed IC_{50} value of 88.32 µg/ml, aqueous extract Ficus religiosa IC_{50} value of 114.2 µg/ml, aqueous extract Thespasia populnea IC_{50} value of 178.5 µg/ml and aqueous extract Ficus benghalensis IC_{50} value of 185.70 µg/ml as summarized in table No.3 and graph No.5, 8, 4, 6, 3, 11, 10, 12 & 9.

Whereas the ethanoic extract of *Thespasia populnea* IC_{50} was not calculated due less percentage of inhibition.

The results of the study indicate that the PVK ethanoic and aqueous extracts both in combination and individually inhibit the activity of pancreatic lipase significantly. This can be attributed to the presence of Phenolic compounds, Flavonoids, Beta sitosterol and Tannins which is compared to Orlistat as synthetic positive control and Caffeine as natural positive control. This rationalize the effectiveness of this PVK combination in the treatment of Medho Vriddi which is specified as Asthayi Medovriddhi- Serum Hyperlipidemia as well as Sthayi Medo Vriddhi Viz. Obesity and other related disorders.

Among the Individual drugs of PVK, Ficus racemose, Ficus virens, Ficus religiosa, Ficus benghalensis and PVK combination water extract samples exhibited potent lipase inhibition as compared to standard orlistat and caffeine. The ethanolic extract of PVK combination, Ficus religiosa, Ficus virens, Ficus benghalensis and Ficus racemose exhibited very potent lipase inhibition activity as compared to standard drugs. Whereas Thespasia populnea ethanoic extract has not shown any significant activity though water extract has slight lipase inhibition activity.

In a way PVK may help in prevention of fat digestion and absorption by inactivating and inhibiting the PL enzyme by blocking the site of the enzyme due to the presence of Polyphenols, Flavonoids present in PVK. Tannins present in PVK may prevent the absorption of fats in to microvilli due to Kashaya rasa and eliminate the fats out of the GI tract without getting absorbed.

V. Conclusion:

The results of this study indicate that the PVK-Panchavalkala group barks together significantly inhibit the activity of pancreatic lipase which is comparable to orlistat can be attributed to the presence of high levels of phenolics like caffeic acid, gallic acid, chlorogenic acid, flavonoids, moderate levels of tannins and saponins. This rationalizes its effectiveness in the treatment of dyslipidemia, hyperlipidemia and obesity. Being a plant sourced natural drug these herbs will achieve the therapeutic result without any adverse effects. Further, in vivo studies are warranted to determine the usefulness of these plant sourced barks in treatment of hyperlipidemia and obesity.

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