SKM VAIDHYA AMIRTHAM

News Letter of SKM in Siddha, Ayurveda and Unani

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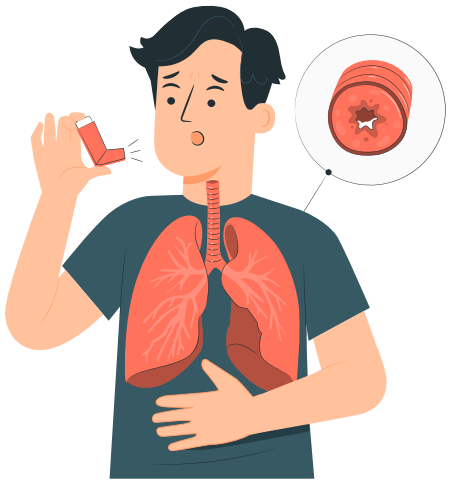
Effective for ALLERGIC & BRONCHIAL ASTHMA



- Alleviates allergic respiratory disorders
- Best medicine for Wheezing
- Effective for bronchitis

Bonus Habit: Healthy Drink Tips!

"भोजनान्ते पिबेत्तक्रं वासरान्ते पिबेत्पयः ।
निशान्ते च पिबेद्वारि त्रिभिर्रोगो न जायते ॥"

Drinking buttermilk at the end of a meal, milk at the end of the day, and water in the morning upon waking helps maintain good health and prevents diseases.

Articles are invited in Siddha, Ayurveda and Unani fields about clinical experience, rare medicinal preparations, successful treatments, Herbal informations and AYUSH Foods for our "SKM Vaidhya Amirtham" News letter which has around 10000 copies of circulation.

Please send your Articles/Suggestions to:
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Evaluation of Chronic toxicity of Urai mathirai - Siddha Herbal Formulation

INTRODUCTION:

Traditional Systems of medicines are playing a key role in meeting the global health care needs. India has different recognized systems of medicine. They are Ayurveda, Siddha, Unani, Yoga, Naturopathy Ho moeopathy and Sowa Rigpa. Among them Siddha is the unique system of medicine which is originated from Tamil nadu and has its origins in Tamil language. Literally the word "Siddha" means "established truth". Siddha system of medicine was claimed to alleviate the root cause of the diseases by maintaining the ratio of Vatham, Pitham and Kapham. The origin of the Siddha system of medicine is attributed from ancient saint called Siddhars[1].

Urai Mathirai is a drug used for the past three decades in the form of long finger size bullets which are rubbed and administered through breast milk with children's to improve immunity to get free from health hazards such as frequent respiratory infections /gastrointestinal infections and anorexia. For global acceptance, this system of medicine should undergo scientific validation, i.e., upgrading the one of the levels is safety of the dose[2].

The acute and sub acute toxicity of the Siddha herbal formulation urai mathirai was administered orally at a dose of 10, 50 and100 mg/kg.b.wt for 28 days does not showed any toxicity effect in wistar rats.

Hence the present study was performed to evaluate the chronic toxicity of the Siddha herbal formulation Urai Mathirai in experimental wistar rats. Through this study the safety of this herbal drug can be established for the clinical use of this traditional formulation among the children's to improve immunity.

MATERIAL AND METHODS:

Test animal

Wistar albino rats of both sex (Male and female) weighing about130–250 g were used in the chronic toxicology studies. The rats were housed in the Department of Pharmacology, Siddha central research institute, Chennai. The rats were kept in sanitized polypropylene cages housed with sterile corn cob as bedding materials at animal house, in an air conditioned environment with four rats in each cage and maintained at room temperature of (23 ± 2)ºC with relativehumidity (60% ± 10%) under 12 hourdark and light cycle(3). Rats were given free access to standard pellet diet and water ad libitum(4). All experimental procedures were in compliance with the Committee for the Purpose of Control and Supervision of Experiments on animals (CPCSEA) and were approved by Institutional Animal Ethical Committee, with an approval number **162/Pharma/SCRI/2017.**

Dose calculation

The clinical dose of Urai mathirai in children is 50 mg (HED = 2.5mg/kg body weight). The animal (rat) doses are calculated as per the FDA guidelines and the calculated therapeutic dose (TD) was found to be 10mg/kg of body weight. In this study to determine the dose correlated effects, 5 times TD and 10 times TD i.e. 50 mg/kg & 100 mg/kg of bodyweight were chosen correspondingly.

Experimental design

The oral chronic toxicity study was evaluated accordance with to Organization for Economic Cooperation and Development (OECD) guideline 408 and Central Council for Research in Ayurvedic Sciences (CCRAS) guideline (6, 7). Wistar albino Rats of both sexes (over night fasting free access to water), aged 8–12 weeks old were used. In 80 rats (40Male and 40females) were randomized into groups separately based on bodyweight. After the randomization process, each study animal was assigned a unique number and identified by a picric acid mark. Group-I served with vehicle as normal control, Group-II, Group-III and Group-IV administered with test drug orally at a dose of 10 mg/kg, 50 mg/kg and 100 mg/kg respectively for 90 days. Before drug administration, the body weight of each animal was determined and the dose was calculated according to the body weight for every week consecutively for 90 days (8). Rat general health, and signs of toxicity, body weight, mortality, food and water intake was monitored. At 90th day 50% of the experimental animals (40 animals form both sex) in each group were subjected to euthanasia.

The necropsy was carried out on all euthanized animals and the organs were isolated and observed macroscopically for abnormalities. After 30days of post treatment the remaining half animals (40animals) were euthanized and organs were collected and observed macroscopically for abnormalities. The collected organs during the treatment and post treatment were preserved in 10% formaldehyde solution for histopathological examination.

Relative organ weight

The internal organs (brain, heart, thymus, lungs, liver, stomach, spleen, kidney, adrenal gland, pancreas and sex organs) excised from all the experimental rats after 90th day and 120th day.Organ-to-body weight ratio Posted on Authorea 23 Apr 2020 — CC BY 4.0 — https://doi.org/10.22541/au.158765852.23761913 — This a preprint and has not been peer reviewed. Data may be preliminary. was calculated by dividing the weight (g) of each organ by the weight (g) of rats before sacrifice.

Biochemical parameters

The biochemical analysis were done on serum after centrifugation of collected blood and the following parameters like Blood glucose, Total Cholesterol, Triglyceride, HDL, LDL, SGOT, SGPT, ALP, GGT, Total Protein, Albumin, LDH, CRP, Creatinine Kinase levels, Creatinine Kinase – MB, Urea, Serum creatinine, Total Bilirubin, Uric acid, Serum Calcium were determined for both control and Urai mathirai treated groups by using standard biochemical method.

Haematological parameters

The hematological parameters such as white blood cell (WBC), red blood cell (RBC), lymphocyte (LYMP), monocyte (MON), granulocyte (GRAN), hemoglobin (HGB), and Hematocrit (Hct) Levels were evaluated.

Histopathological examination

The organs (brain, heart, thymus, lungs, liver, stomach, spleen, kidney, adrenal gland, pancreas and sex organs) excised from all the experimental rats were fixed in 10% formalin in labeled bottles, and processed for histological examination. Tissues embedded in paraffin wax were sectioned 5 mm thick and stained with haematoxylin and eosin, mounted on glass slides and examined under a standard light microscope[10]

Statistical analysis

All the data was expressed as Mean ± SEM. Statistical analysis was tested by using One-way ANOVA followed by (Dunnett's test) using Graph pad prism version-8.The significance level was set at $P>0.05$ for all tests. Group II, III, and IV will be statistically compared with Group I to find the treatment related effects.

RESULTS:

Chronic toxicity study

All the treatment group ratswere administered with Urai mathiraidrug solution at a dose of (10, 50 and 100mg/kg b.wt) throughout the 90 days found to be no clinical toxicity signs such as physical observations, behavioral changes and other parameters such as body weight, food intake, water intake, respiration, convulsion, tremor, changes in eye and skin colors, etc were observed in the treated group compared to the control group. The observations were measured and summarized in Table 1.

Effect of Urai mathirai on relative organ body weight

The average and relative organ weight of Urai mathirai orally treated group of animals (at dose of 10, 50 and 100mg/kg b.wt) and control groups showed statistically non-significant differences ($P > 0.05$). The results revealed that, the internal organs of rats were not adversely affected throughout the treatment. The effect ofUrai mathirai on principal organ weights relative to body weight were presented in Table 2 .

Effect of Urai mathirai on biochemical parameters

The results of the various biochemical parameters on the experimentally treated rats with the oral administration of the Urai mathirai at a dose of (10, 50 and 100mg/kg b.wt) and normal groups showed statistically non-significant differences ($P > 0.05$). The results revealed that no abnormal changes in serum biochemical parameters such as albumin, total protein, globulin, Total bilirubin, urea, sodium, creatinine and uric acid levels etc., when compared to control group. The effect of Urai mathirai on biochemical parameters measured and summarized in Table 3.

Effect of Urai mathirai on Hematological parameters

The hematological parameters white blood cell (WBC), red blood cell (RBC),lymphocyte (LYMP), monocyte (MON), granulocyte (GRAN),hemoglobin (HGB), andHematocrit (Hct) Levelswere within normal limits compared to control group. No significant differences ($P > 0.05$) between treated animals with the Urai mathirai orally at dose of 10 mg/kg, 50 mg/kg and 100 mg/kg and control group rats were found. The hematological parameters were measured and summarized in Table 4.

Effect of Urai mathirai in Histopathological Study

Sections of lung, liver, kidney, spleen, heart, and brain tissues were perfused with 10% formalin and stored in the same andused for histopathological studies. The tissues were then embedded in molten paraffin wax. Sections were cut at 5µm thickness and stained with haematoxylin and eosin. The sections were then viewed under light microscope. The macroscopic examination of organs of treated rats revealed no abnormalities in the colour or texture when compared with the organs of the control group. Although some differences have been observed in the histopathological slides were presented in Figure 1-8.

Histopathology

Histopathological findings of lungs showed mild pulmonary oedema, multi focal mild mononuclear cell infiltration in peribronchiolar and alveolar region of control rats (Group I) and also treated rats groups (Group II to Group IV) of Urai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg). It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure1.

A. Group I B. Group II
C. Group III D. Group IV

Figure 1 (A-D) Histopathological findings of Lungs in different groups of rats.

Histopathological findings of liver showed sinusoidal congestion, very mild hepatocellular degeneration and focal hepatocellular necrosis in both control rats (Group I) and also in treated rats groups (Group II to Group IV) of Urai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg). It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 2 .

A. Group I B. Group II
C. Group III D. Group IV

Figure 2 (A-D) Histopathological findings of Liver in different groups of rats.

Histopathological findings of kidney showed mild tubular epithelial cell degeneration and focal mild interstitial mononuclear cell infiltration in both control rats (Group I) and also in treated rats groups (Group II to Group IV) of Urai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg).It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 3.

A. Group I B. Group II
C. Group III D. Group IV

Figure 3 (A-D) Histopathological findings of Kidney in different groups of rats.

Histopathological findingsof spleen showed no abnormality in both control rats and also in treated rats groups (Group II to Group IV) ofUrai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg). It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 4.

A. Group I B. Group II
C. Group III D. Group IV

Figure 4 (A-D) Histopathological findings of Spleen in different groups of rats.

Histopathological findingsof heart showed no abnormality in both control rats and also in treated rats groups (Group II to Group IV) ofUrai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg). It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 5.

A. Group I B. Group II
C. Group III D. Group IV

Figure 5 (A-D) Histopathological findings of heart in different groups of rats.

Histopathological findingsof brain showed no abnormality in both control rats and also in treated rats groups (Group II to Group IV) of Urai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg).It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in theFigure 6.

A. Group I B. Group II
C. Group III D. Group IV

Figure 6 (A-D) Histopathological findings of brain in different groups of rats.

Histopathological findings of glandular and non glandular stomach showed no abnormality in both control rats and also in treated rats groups (Group II to Group IV) of Urai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg).It revealed that no significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 7.

A. Group I B. Group II
C. Group III D. Group IV

Figure 7 (A-D) Histopathological findings of stomach in different groups of rats.

Histopathological findings of pancreas showed no abnormality in both control rats and also in treated rats groups (Group II to Group IV) ofUrai mathirai orally at a dose level of(10 mg/kg, 50 mg/kg and 100 mg/kg). It revealed thatno significant changes were observed related to drug toxicity effect and pictures were presented in the Figure 8 .

A. Group I B. Group II
C. Group III D. Group IV

Figure 8 (A-D) Histopathological findings of pancreas in different groups of rats

CONCLUSION:

Urai mathirai administered in 3 doses by oral route (at therapeutic dose of 10 mg/kg, average dose 50 mg/kg and high dose 100mg/kg) regularly for 90 days did not produce any toxicity in animal models. There were no statistically significant alterations found in behavior, biochemistry, hematological parameters, organ weight and histopathology during the experimental period.

Table 1: General appearance and behavioral observations of control and treated groups.

Response	Group I (Normal)	Group II (10mg/kgb.w)	Group III (50mg/kgb.w)	Group IV (100mg/kgb.w)
Colour	**Colour**	**Colour**	**Colour**	**Colour**
Fur	N	N	N	N
Eyes	N	N	N	N
Mucous Membrane	N	N	N	N
Urine	N	N	N	N
Behavioral observations	**Behavioral observations**	**Behavioral observations**	**Behavioral observations**	**Behavioral observations**
Mood	N	N	N	N
CNS Excitation	NO	NO	NO	NO
CNSDepression	NO	NO	NO	NO
Motor Indication	**Motor Indication**	**Motor Indication**	**Motor Indication**	**Motor Indication**
Abnormal gait	NO	NO	NO	NO
Righting reflex	N	N	N	N
Posture	N	N	N	N
Sensory Responses	**Sensory Responses**	**Sensory Responses**	**Sensory Responses**	**Sensory Responses**
Touch& pain response	N	N	N	N
Straube"s phenomenon	NO	NO	NO	NO
Reflexes	**Reflexes**	**Reflexes**	**Reflexes**	**Reflexes**
Pinnna& corneal	N	N	N	N
Autonomic effects	**Autonomic effects**	**Autonomic effects**	**Autonomic effects**	**Autonomic effects**

Response	Group I (Normal)	Group II (10mg/kgb.w)	Group III (50mg/kgb.w)	Group IV (100mg/kgb.w)
Defecation &Lacrimation	N	N	N	N
Urination &Salivation	N	N	N	N
Piloerection	N	N	N	N
Miosis & Mydriasis	NO	NO	NO	NO
Diarrhoea	NO	NO	NO	NO
Respiratory effect	**Respiratory effect**	**Respiratory effect**	**Respiratory effect**	**Respiratory effect**
Apnoea & dysponea	NO	NO	NO	NO
Death	NO	NO	NO	NO

N-Normal No-No Abnormality Observed

Table 2: Effect of Oral administrationof Urai mathirai on Relative organs weight (g) of rats.

Organs	Group I (Normal)	Group II (10mg/kgb.w)	Group III (50mg/kgb.w)	Group IV (100mg/kgb.w)
Brain	6.88±1.11	7.14±0.59	6.58±0.58	7.17±0.49
Heart	3.90±3.73	3.81±0.72	3.75±0.12	3.80±0.45
Thymus	3.41±1.83	2.23±1.11	0.75±0.14	0.89±0.19
Lungs	7.53±0.47	6.61±0.36	7.01±0.44	7.88±1.22
Liver	39.60±4.11	34.43±1.46	36.04±1.32	39.06±5.04
Stomach	6.14±0.86	6.48±0.42	5.73±0.18	6.60±0.45
Spleen	2.42±0.18	2.91±0.29	2.94±0.16	2.56±0.47
Pancreas	1.64±0.95	3.30±0.36	4.35±1.64	3.19±0.38
Kidney	8.26±1.05	8.55±0.96	7.92±0.19	8.53±0.78
Adrenal gland	0.34±1.20	0.27±0.07	0.20±0.04	0.22±0.05

All values are expressed as mean ± SEM (n=8). No significant difference since $p > 0.05$, as compared to control group .

Table 3: Effect of oral administrationUrai mathirai on biochemical parameters at the end of the treatment period

Organs	Group I (Normal)	Group II (10mg/kgb.w)	Group III (50mg/kgb.w)	Group IV (100mg/kgb.w)
Blood glucose(mg/dl)	72.47±6.83	71.00±5.97	75.36±6.88	72.71±6.12
Total Cholesterol levels (mg/dl)	73.13±3.78	71.64±3.04	68.86±4.49	70.65±3.57
Triglyceride levels (mg/dl)	103.00±10.05	121.64±14.94	109.93±9.90	101.29±13.96
HDL levels (mg/dl)	30.87±6.70	25.07±1.41	25.36±1.76	23.47±1.26
LDL levels (mg/dl)	28.27±3.65	23.00±3.36	21.43±3.41	27.56±2.89
SGOT levels (U/L)	164.40±12.31	190.43±14.45	194.29±12.72	162.53±4.64
SGPT levels (U/L)	62.60±2.07	64.79±4.10	64.50±3.17	67.76±9.74
ALPlevels (U/L)	200.60±19.15	183.21±16.86	220.36±20.74	165.47±14.62
GGTlevels (U/L)	4.60±0.46	4.92±0.47	4.64±0.37	4.71±0.32
Total Protein levels (g/dl)	7.67±0.11	7.50±0.10	7.59±0.17	7.64±0.18
Albumin levels (g/dl)	3.41±0.07	3.44±0.10	3.37±0.13	3.45±0.06
LDHlevels (U/L)	1234.13±65.36	1184.50±82.60	1232.50±91.74	14090.12±18.76
CRP levels (mg/L)	0.48±0.06	0.54±0.09	0.50±0.08	0.58±0.11
CreatinineKinase levels (U/L)	814.87±70.65	904.79±124.68	887.07±139.57	651.65±41.45
CreatinineKinase –MBlevels (U/L)	507.80±43.57	496.50±50.51	446.14±36.92	369.76±18.04
Urea levels (mg/dl)	30.67±1.68	32.57±2.26	32.57±1.25	34.71±1.57
Serum creatinine levels (mg/dl)	0.53±0.02	0.53±0.02	0.56±0.02	0.54±0.03
Total Bilirubin levels (mg/dl)	0.12±0.01	0.11±0.01	0.11±0.01	0.10±0.00
Uricacid levels (mg/dl)	0.13±0.02	0.12±0.01	0.13±0.02	0.12±0.01
SerumCalcium levels (mg/dl)	9.98±0.12	9.89±0.12	9.12±0.68	14.22±4.49

All values are expressed as mean ± SEM (n=15, 14, 15,17). No significant difference since $p > 0.05$, as compared to control group.

Table 4: Effect of oral Urai mathirai on haematological parameters at the end of the treatment period

Parameters	Group I (Normal)	Group II (10 mg/kg b.w)	Group III (50 mg/kg b.w)	GroupIV(10 mg/kg b.w)
WBC (10^9/L)	12.10±0.64	11.59±*0.99*	*10.94* ±*0.71*	*10.52*±*0.80*
RBC (10^{12}/L)	7.42±0.17	7.78±0.31	7.66 ±0.21	7.76 ±0.24
LYMP (10^9/L)	8.13±0.40	9.36±1.10	7.54 ±0.55	7.11 ±0.55
MON (10^9/L)	0.31±0.02	0.31±0.03	0.32 ±0.03	0.31 ±0.03
GRAN (10^9/L)	3.68±0.28	3.21±0.38	3.08 ±0.24	2.81 ±0.29
HGB (g/dL)	12.00±0.27	12.44±0.47	12.19±0.26	19.61±7.41
HCT (%)	38.44±0.87	39.36±1.56	38.17±0.87	39.32±1.37

All values are expressed as mean ± SEM (n=13, 14,15,17). No significant difference since $p > 0.05$, as compared to control group, except $p < 0.05$ in group,

Ref: Dayanand Reddy Gaddam[1], Sathiyarajeswaran P[2], Patturayan K[3], Ganesan K[2], Narasimha Kumar G .V[4], Pullaiah C.P[2], and Dhanaraj K[2]

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In Vitro A-amylase inhibitory assay and Anti-oxidant, phytochemical screening of polyherbal siddha formulation Madhumega chooranam

INTRODUCTION:

Siddha System of Medicine is one of the age old medical system is being practiced in India. This system provides better solutions to manage many life style disorders. Type 2 Diabetes mellitus is one of the major disorder which impacts important threat due to its prevalence, chronicity and disabling complications.DM (Diabetes mellitus) is a metabolic disorder characterized by chronic hyperglycemia or increased a blood glucose level which affects the carbohydrate, fat and protein metabolism resulting from complete or relative lack of insulin secretion [1].It prevalence increases globally and projected as 300 million by 2025. In India more than 10 million populations will suffer by this metabolic disorder [2]. Many therapeutic strategies are applied recently to observe the possible mechanism in type 2 diabetes mellitus [3]. The conventional method is α-amylase inhibition assay on cell lines. The objective of this study is to enumerate the basic pharmacological process of α-amylase inhibition assay on popularly known Siddha drug Madhumegachooranam[4] by cell line studies.

MATERIAL AND METHODS:

Siddha Medicine Madhumega chooranam(MMC) is purchased from a registered pharmacy. The chemicals used in the present study were of analytical grade and purchased from a reputed laboratory. The ethanolic extract was subjected into α-amylase inhibition by the method described by a standard method. The same extract has been tested for Anti oxidant activity involving different methods and phyto chemical analysis executed to assess its qualitative nature.

Preparation of MMC extract

The extracts are prepared by refluxing in ethanol for 72 hours followed by solvent recovery using rotary evaporator. The extracts are resuspended in 1% DMSO in a final concentration of 10mg/ml.

1. In vitro α-amylase inhibitory assay [5-6]

Principle:

The reducing sugars produced by the action of α-amylase react with dinitrosalicylic acid and reduce it to a brown coloured product, nitro amino salicylic acid.

Procedure:

Twenty five microlitres of 10 mg/ml extract and 25µl of 25mM phosphate buffer pH 6.9, containing porcine α-amylase at a concentration of 0.5 mg/ml were incubated at 25°c for 10 min. After pre incubation, 25µl of 0.5% starch solution in 25mM phosphate buffer pH 6.9 was added. The reaction mixtures were then incubated at 25°c for 10 min. The reaction was stopped with 50µl of 96mM 3, 5 dinitrosalicylic acid colour reagent. The micro plate was then incubated in a boiling water bath for 5 min and cooled to room temperature. Absorbance was measured at 540nm using a microplate reader.

Calculation:

$$\% \text{ inhibition} = \frac{\text{control test}}{\text{control}} \times 100$$

2. Determination of Anti oxidant activity

Anti oxidant activity of MMC extract is performed by using assay of Nitric oxide scavenging activity, Super oxide free radical scavenging activity and DPPH assay. The results were tabulated for further discussion.

a) DPPH Assay (2, 2-diphenyl -1-picrylhydrazyl) [7-8]

The radical scavenging activity of different extracts was determined by using DPPH assay according to a standard method. The decrease in the absorption of the DPPH solution after the addition of an antioxidant was measured at 517 nm. Ascorbic acid (10mg/ml DMSO) was used as reference.

Principle

1,1-diphenyl-2-picryl hydrazyl is a stable free radical with red colour which turns yellow when scavenged. The DPPH assay uses this character to show free radical scavenging activity. The scavenging reaction between (DPPH) and an antioxidant (H-A) can be written as,

DPPH + [H-A] →DPPH-H + (A)Antioxidants react with DPPH and reduce it to DPPH-H and as consequence the absorbance decreases. The degree of discolouration indicates the scavenging potential of the antioxidant compounds or extracts in terms of hydrogen donating ability.

Reagent Preparation

0.1mM DPPH solution was prepared by dissolving 4mg of DPPH in 100ml of ethanol.

Procedure

Different volumes (1.25-10µl) of plant extracts were made up to 40µl with DMSO and 2.96ml DPPH (0.1mM) solution was added. The reaction mixture incubated in dark condition at room temperature for 20 minutes. After 20 minutes, the absorbance of the mixture was read at 517nm.3ml of DPPH was taken as control.

Calculation:

$$\% \text{ inhibition} = \frac{\text{control test}}{\text{control}} \text{x}100$$

Nitric Oxide Scavenging Activity[9-11]

Nitric oxide (NO.) has also been involved in a variety of biological functions, including neurotransmission, vascular homeostasis, antimicrobial, and antitumor activities. Despite the possible beneficial effects of NO, its contribution to oxidative damage is also reported. This is due to the fact that NO can react with superoxide to form the peroxynitrite anion, which is a potential oxidant that can decompose to produce OH and NO. The procedure is based on the principle that, sodium nitro prusside in aqueous solution at physiological pH spontaneously generates nitric oxide which interacts with oxygen to produce nitrite ions that can be estimated using Griess reagent. Scavengers of nitric oxide compete with oxygen, leading to reduced production of nitrite ions. Large amounts of NO may lead to tissue damage.Nitric oxide scavenging activity was measured spectrophotometrically. Sodium nitro prusside (5mmolL-1)in phosphate buffered saline pH 7.4,was mixed with different concentration of the extract (250-2500µg mL1)prepared in methanol and incubated at 25°C for 30minutes .A control without the test compound, but an equivalent amount of methanol was taken. After 30minutes, 1.5mL of the incubated solution was removed and diluted with 1.5mL of Griess reagent (1% sulphanilamide, 2% phosphoric acid and 0.1% N-1-naphthyl ethylene diaminedihydrochloride). Absorbance of the chromophore formed during diazotization of the nitrate with sulphanilamide and subsequent coupling with N-1 naphathyl ethylene diaminedihydrochloride was measured at 546nm and the percentage scavenging activity was measured with reference to the standard.

3. Phytochemical Analysis [12]

Phytochemicals, chemical compounds that occur naturally in plants (phyto means "plant" in Greek), are responsible for color and biological properties. The term is generally used to refer to those chemicals that may have biological significance but are not established as essential nutrients.The following tests are used for the analysis of phytochemicals as described by a standard method.The following tests are used for the analysis of phytochemicals present in the alcoholic extract of the tested drug. Some of the tests are done based on the standard procedure to assess the presence of alkaloid, flavanoid, phenols, glycdosides, terpenoids and tannins. The inferences were listed in table 1.

Test for Alkaloids

Dragandroff's test

8g of Bi(No3)3. 5H2O was dissolved in 20 ml HNO3 and 2.72g of potassium iodide in 50 ml H2O.These were mixed and allowed to stand. When KNO3 crystals out, the supernatant was discarded off and made up to 100 ml with distilled water. The alkaloids were regenerated from the precipitate by treating with Na2CO3 followed by extraction of the liberated base with ether.To 0.5ml of alcoholic solution of extract added to 2.0 ml of HCl. To this acidic medium 1.0 ml of reagent was added. An orange red precipitate produced immediately indicates the presence of alkaloids.

Test for Flavanoids
Shinoda's test

In a test tube containing 0.5 ml of alcoholic extract 5-10 drops of dilute HCl and a small piece of ZnCl2 or Mg were added and the solution was boiled for few minutes. In the presence of flavanoids reddish pink or dirty brown color was produced.

Test for Phenol
Ferric chloride test

To 2 ml of alcoholic solution of extract, 2 ml of distilled water followed by drops of 10% aqueous solution of FeCl3 solution were added. Formation of blue or green indicates the presence of phenols.

Test for Glycosides

A small amount of alcoholic extract was dissolved in 1 ml of H2O and the aqueous NaOH solution was added. Formation of yellow color indicates the presence of glycosides.

Test for Tannins
Ferric chloride test

To 1 -2 ml of aqueous extract, few drops of 5%aqueous ferric chloride solution was added. A bluish black colour,which disappears in addition of a few ml of sulfuric acid, formation of yellowish brown precipitate.

Test for Triterpenoids
LibermannBurchard test

The extract, 10 mg was dissolved in 1 mlof chloroform; 1ml of acetic anhydridewas added following the addition of 2 mlof Conc.H2SO4. Formation of reddishviolet colour indicates the presence of triterpenoids.

Table 1.Phytochemical Analysis of Madhumega chooranam

Test	Observation	Inference
Alkaloids	Orangeredprecipitatewasfound	Presenceofalkaloid(++)
Flavanoids	Pinkcolourwasformed	PresenceofFlavanoid(+)
Phenols	Nocharacteristicchangewasobserved	Absenceofphenols
Glycosides	Yellowcolourwas formed	Presenceofglycosides(++)
Terpenoids	Redcolourwasformedinthechloroformlayer	Presenceofterpenoids(++)
Tannins	Nocharacteristicchangewasobserved	Absenceoftannins(-)

Statistical Analysis

Statistical difference and linear regression analysis were performed using Graphpad prism 6 statistical software.

RESULTS AND DISCUSSION:

The results showed that there was a concentration dependent α-amylase inhibitory effect of crude extract of Madhumegachooranam. At the concentration increased from 12.5 to 100ug/ml, percentage of inhibition increased from 37.22 % to 61.65 %. At a concentration of 100 ug/ml there was a decrease in cell viability (Table 1, Figure 2&3). The IC50 value was obtained at 20.2ug/ml (Figure 3). The antioxidant activity observed in different methodologies showed that Madhumegachooranam is having significant anti oxidant activity (Figure.3) Phytochemical analysis revealed that the presence of alkaloid,flavanoid,glycosides and terpenoids (Table 1). They are frequently implicated as having anti diabetic effect[13].

Antioxidants are slow down the oxidative damage of our body. Antioxidants act as a free radical scavengers. Preventing and repairing damages. Health problems such as Heart diseases,cancer and degenerative disorders are all exacerbated by oxidative damage.The Antioxidant activity of the drug was tested by DPPH and Nitric oxide scavenging activity.

Figure 2. In vitroα-amylaseinhibition assay on Madhumega chooranam

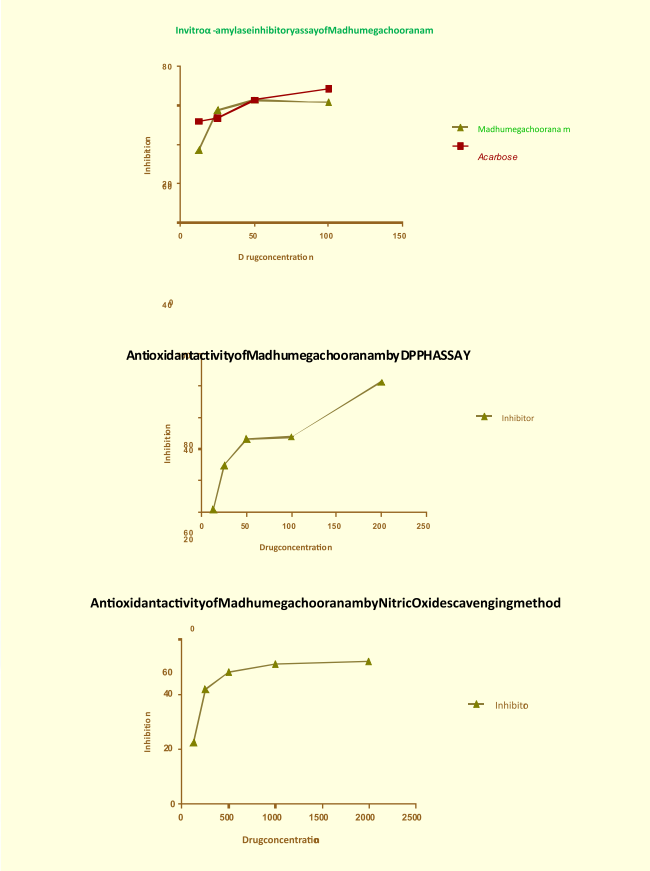

Figure 3. In vitroα-amylaseinhibition and anti oxidant activity on Madhumegachooranam

CONCLUSION

It is concluded that the tested drug Madhumegachooranam exhibited significant α-amylase inhibition on dose dependant manner and having marked anti oxidant activity. The phyto constituents may help in restoring normal health. Thus this drug may act as a combined way in controlling hyperglycemia with minimal untoward effects. The drug could be tested for the maximum limit of controlling hyperglycemia in clinical studies. Further studies are needed to distinguish the pharmacological pathway in the insulin metabolism.

Ref: Ravichandran.M[1], Mubarak.[H2]*

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TIP BITS: Guduchi (Tinospora cordifolia)

1. **Medhya** - The fresh juice of Gudūcī (20ml twice daily) may be used (C.S.Ci.1/1).
2. **Jvara** - (a) Gudūcī Svarasa (juice) + Satāvarī svarass equal parts (10ml each) are mixed together and given along with jaggary (Guda), in vāta jvara (S.S.Ut. 39/174)
   (b) Decoction prepared with Gudūcī, Parpati & Āmalakī (500- 100ml) may be administered in case of Pitta Jvara (Ha. Sam.3/ 2/71)
3. **Prameha** - Gudūcī Svarasa with honey (A.H.Ci.12/6)
4. **Slīpada** - Gudūcī Svarasa along with gingelly oil (Taila) is given orally (C.D.42/16)
5. **Amlapitta** - Leaves of Gudūcī, Nimba and Patola are made into juice and administered along with honey (B. P.Ci.10/16)
6. **Chardi** - Gudūci Hima Kaşāya may be given orally alongwith honey (Bha.Pra.Ci. 17/21).
7. **Vatarakta** - Chronic administration of Guduchi in either juice or paste or powder or decoction form will definitely cure Vātarakta (B.P.Ci. 29/41 & V M 23/10)

We had the privilege of participating as a proud sponsor in the 10th World Ayurveda Congress & Arogya Expo, held in Dehradun, Uttarakhand, from December 12 to 15, 2024. This prestigious event centered around the theme Digital Health: An Ayurveda Perspective.

As a leading manufacturer, SKM Siddha and Ayurveda showcased our latest innovations, including newly launched medicines such as Manyawin Forte, Katiwin, Gynedote, Ovorex, the Psorasiddh range, and more, at our vibrant Stall No. J2.

Our team of dedicated doctors and marketing professionals engaged with visitors, providing them with in-depth information about our products, ensuring they had direct access to the details they needed. It was a rewarding experience to connect with attendees and share our commitment to advancing health through Ayurveda.












National Siddha Day Celebration

On December 19, 2024, our company proudly celebrated National Siddha Day, commemorating the birth anniversary of Siddhar Agathiyar. The occasion was marked with traditional rituals, including a special pooja conducted to honor Siddhar Agathiyar's contributions to Siddha medicine. This celebration reflected our deep commitment to preserving and promoting the rich heritage of Siddha practices.




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