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BIOCHEMICAL ANALYSIS OF THE SIDDHA HERBO MINERAL FORMULATION NARKARANTHAI LEGHIYAM FOR PADARTHAMARAI(DERMATOPHYTOSIS) IN CHILDREN

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ABSTRACT

Siddha is one of the ancient medical systems in India, considered the mother medicine of ancient Tamils/Dravidians in South India. Many herbal and herbomineral formulations for the treatment of padarthamarai were documented in the Siddha ancient literature. The investigation aimed to assess the biochemical analysis of the trial medicine, Narkaranthai leghiyam, which demonstrated the presence of carbonate, sulfates, and phosphate, as well as the augmentation of therapeutic activity in Dermatophytosis. As a result, I conclude that the existence of these substances may aid in the treatment of dermatophytosis. Further biochemical investigations would be necessary to fully characterize the active compounds and elucidate the underlying mechanisms by which the Siddha herbal formulation Narkaranthai leghiyam exerts its therapeutic effects in dermatophytosis.

KEYWORDS*:*

Biochemical analysis, Dermatophytosis, Herbal Formulation, Narkaranthai leghiyam, Siddha medicine.

INTRODUCTION

Nature has been a source of medicinal agents for thousands of years, and an impressive number of modern drugs have been isolated from natural sources, many of them based on their use in traditional medicine. Today, it is estimated that more than two-thirds of the world's population relies on plant-derived drugs; some 7,000 medicinal compounds used in the Western pharmacopoeia are derived from plants[1]. The World Health Organization (WHO) reported that around 80% of the world's population still relies on plants as a source for primary health care [2]. Siddha System of Medicine is as old as mankind and dominated the civilization of the southern peninsula of India[3].

Dermatophytes are the most common pathogenic filamentous fungi, with an infection rate of as high as 20%-25% worldwide [4]. Dermatophytes usually infect the nails, skin, and hair [5, 6].

Multiple superficial dermatophytosis, such as tinea capitis, onychomycosis, tinea corporis, and tinea pedis[7]. Unfrequently, dermatophytes may also invade the dermal tissue and even deep organs, particularly in immunocompromised patients with congenital or acquired immunodeficiency[8], and these infections can progress to life-threatening conditions if appropriate treatment is not provided[9]. Microbial infections, and especially microbial resistance, are critical and actual problems that require targeted and efficient therapeutic intervention. Natural-based solutions are a viable alternative, at least for complementary therapy, due to few or no side effects and a high safety and efficacy level[10].

Despite the improvement of antifungal therapies, the phenomenon of antifungal resistance is still of major concern in clinical practice[11]. The incidence of fungal infections has drastically increased over the past three decades and was simultaneously accompanied by increased acquired and innate resistance to antifungal drugs. However, antifungal resistance occurrence has to be considered independently for each antifungal class and each fungal genus. Moreover, epidemiological data regarding the incidence of resistance among fungal species are not identically distributed worldwide[12,13,14]. Clinical resistance being the primary cause for treatment failure in the patient, Narkaranthai leghiyam would be a choice of drug for its potential biochemical results, and further research is necessary.

MATERIALS AND METHODS

Ingredients

S. No.	Name	Botanical name	Family name	Parts used	Quantity
1	Nar karanthai	Sphaeranthus indicus	Asteraceae	Whole plant	500gms
2	Karunjeeragam	Nigella sativa	Ranunculaceae	Seed	50gms
3	Kadukkai	Terminalia chebula	Combretaceae	Pulp	50gms
4	Thandrikai	Terminalia bellirica	Combretaceae	Pulp	50gms
5	Vasambu	Acorus calamus	Acoraceae	Rhizome	50gms
6	Thippili	Piper longum	Piperaceae	Fruit	50gms
7	Milagu	Piper nigrum	Piperaceae	Fruit	50gms
8	Koshtam	Sassurea lappa	Asteraceae	Root	50gms
9	Chukku	Zingiber officinale	Zingiberaceae	Rhizome	50gms
10	Siruthekku	Clerodendrum serratum	Lamiaceae	Rhizome	50gms
11	Karbokarisi	Psoralea corylifolia	Fabaceae	Seed	50gms
12	Chithramoolam	Plumbago zeylanica	Plumbaginaceae	Root	50gms
13	Valuzhuvai arisi	Celastrus paniculatus	Celastraceae	Seed	50gms
14	Indhuppu	Sodium chloride impure			50gms
15	Rasa karpooram	Hydragyrum subchloride			12.5gms
16	Panai vellam	Palm jaggery			
17	Sarkarai	Sugar			
18	Nei	Ghee			
19	Thaen	Honey			

Collection, Identification, And Authentication of the Drug

The raw drugs required for the preparation of "NARKARANTHAI LEGHIYAM" were bought from a reputable raw drug shop in Nagercoil. The herbal medicines were verified by the

Head of the Department, Gunapadam Department at Government Siddha Medical College, Palayamkottai. Following that, the raw drug was purified separately. The trial medication was then manufactured in the Gunapadam laboratory at the Government Siddha Medical College, Palayamkottai**.**

Preparation of the Trial Drug:

Purification process

1.Narkaranthai is cleaned and shade-dried.

2.Karunjeeragam is soaked in clear lime water for 3 hours and then dried.

3.Kadukkai seeds are removed; only the outer part is used.

4.Thandrikkai seeds are removed; only the outer part is used.

5.Vasambu is burnt in fire, then buried in sand. The resulting black powder is used in medicine preparation.

6. Thippili is soaked in lemon juice for 3 hours.

7.Milagu is soaked in sour buttermilk for 1hour and 15 minutes, then fried and dried.

8.Kostam is dried.

9. Chukku is soaked in clear lime water.

10.Siruthekku,karbogarisi,vazhuvai arisi are fried.

11. Kodiveli root bark is removed.

12. Pooram is purified by soaking it in betal leaf and pepper kizhi(herbal bundle) for 3 days.

Procedure:

Ingredients from 1 to 15 are purified as above, dried, and made into fine powder. Heat the vessel and add purified palm jaggery and sugar, mix well with the prepared powder. Then add cow ghee and finally add honey accordingly to get leghiya patham. This preparation is allowed to cool and preserved in a clean, dry air air-tight container

Administration of the Drug

- 3-12 years – 2gms -5gms (According to age group and body weight)
- Half an hour before food.
- Twice a day for 21 days.

BIOCHEMICAL ANALYSIS

Screening Narkaranthai Leghiyam to identify the biochemical properties present in its composition. Analytical Investigation: Preparation of the Extract

Extraction

Test samples were extracted with acetone and followed by homogenization for a brief period. Further filtration was allowed, and a subsequent addition of acetone to the test mixture. Heating of the test sample was performed using a rotary evaporator at a temperature not exceeding 40ºC until the solvent had almost completely evaporated. To the residue, add a few milliliters of toluene and heat again until the acetone is completely removed. The resultant residue will be dissolved using toluene and filtered through a membrane filter.

RESULTS

Acid and basic radical analysis report that the test drug MN contains carbonates, sulfates, and phosphates in acid radicals and lead in basic radicals. The observed results of acid and basic radical analysis are tabulated in Table 1 & 2

S No.	EXPERIMENT	OBSERVATION	INFERENCE
1.	**Test for Carbonates**: To 1 ml of the test solution about 1 ml of the concentration (conc.) HCl was added.	Brisk effervescence is formed.	Indicates the **Presence** of **carbonates**
2.	**Test for Chlorides**: To 2 mL of the test solution, about 1 mL of silver nitrate solution was added.	No white Precipitate is formed	Absence of chlorides
3.	**Test for Sulphates**: To 1 ml of the test sample, add diluted H_2SO_4 till effervescence ceases. By this, about 1 ml of barium chloride solution was added.	The appearance of a white precipitate is found	**Presence** of **sulphate**
4.	**Test for Sulfides**: To 1 ml of the test sample, about 2 ml of HCl was added with slight warming of the mixture.	No formation of colorless gas with the smell of rotten	Absence of sulfides.
5.	**Test for Phosphates**: To 2 mL of test solution, add 2 2ml of ammonium molybdate solution, followed by the addition of 2ml of concentrated	Formation of yellow precipitate is found	**Presence** of **phosphates**

	nitric acid.		
6.	**Test for Fluoride and Oxalate**: To 2ml of the test solution about 2 ml of diluted acetic acid and 2ml of calcium chloride solution was added	No formation of white precipitate	Absence of Fluoride/ Oxalate
7.	**Test for Borates**: To 2ml of the test solution was added with sulphuric acid and 95% alcohol, followed by exposure to flame	No appearance of green flame	Absence of Borates
8.	**Test for Nitrates**: 0.5ml of test solution heated with copper turning, followed by the addition of sulphuric acid	No reddish- brown gas	Absence of Nitrates
9.	**Test for Lead:** 1 ml of the test solution i s added to 2 ml of potassium chromate solution.	Yellow precipitate is formed	**Presence** of **lead**
10.	**Test for Arsenic**: 1 ml of the test solution is added to 2 ml of 10% (2N) sodium hydroxide (NaOH) solution.	No brownish-red precipitate	Absence of Arsenic
11.	**Test for Mercury**: 1 ml of the test solution is added to 2 ml of 10% (2N) sodium hydroxide (NaOH) solution. The formation of a yellow precipitate indicates the presence of mercury.	Absence of yellow precipitate	Absence of mercury
12.	**Test for Magnesium** 1 ml of the test solution is added with 2 ml of sodium hydroxide (NaOH) dropwise until t h e indication appears.	No formation of white precipitate	Absence of Magnesium

13.	**Test for Copper**: 1 ml of the test solution is added to 1 ml of Ammonium hydroxide (NH_4OH) solution. Formation of blue precipitate indicates the presence of copper.	Absence of blue precipitate	Absence of copper
14.	**Test for Ferric**: To 1ml of test solution, about 2ml Potassium ferrocyanide was added. Formation of blue precipitate indicates the presence of ferric	Absence of blue precipitate	Absence of ferric
15.	**Test for Ferrous**: To 1ml of test solution, about 1ml of potassium ferric cyanide solution was added. The formation of a blue precipitate indicates the presence of ferrous.	Absence of blue precipitate	Absence of ferrous
16.	**Test for Zinc**: 1ml of the test solution is added with 2ml of sodium hydroxide (NaOH) dropwise until an indication appears. The formation of a white Precipitate indicates the presence of Zinc	Absence of white precipitate	Absence of zinc
17.	**Test for Silver**: 1ml of the test solution was added with 1ml of conc.HCl is followed by the appearance of a curdy white precipitate. Boil the precipitate with water. It does not dissolve. Add NH_4OH solution to it and add 1 ml dilute HNO_3. The formation of a curdy white precipitate indicates the presence of silver	Absence of curdy white precipitate	Absence of silver

DISCUSSION

The Biochemical analysis of the trial drug Narkaranthai leghiyam contains Carbonates, Sulfate, and Phosphate. Sulfate has antifungal activity [15].

Adenosine triphosphate (ATP) is known as the energy currency of the body and is involved in the storage and utilization of energy for life activities such as muscle contraction. When ATP is hydrolyzed by the action of ATPases, it loses one phosphate group to become adenosine diphosphate (ADP), releasing energy in the process, which is used for muscle contraction [16,17]. Several studies have reported that physical or chemical stimulation of the epidermal cells causes the release of ATP into the extracellular space [18,19]. Additionally, this released ATP acts on neighboring immune cells, promoting the production of cytokines and chemokines. This indicates that ATP-mediated intercellular interactions play a significant role in immunity, allergy, and inflammatory responses and are involved in various skin diseases, including Atopic Dermatitis[20,21,22,23,24,25,26,27,28,29,30,31].

The mode of action of the trial drug Narkaranthai Leghiyam, which brings about Antifungal and Antipruritic activity in the body. This may be due to the presence of Sulfate, Carbonate, Phosphate, and Lead in it.

CONCLUSION

Narkaranthai leghiyam is a siddha drug referred from Siddha Literature, Brahmamuni karukidai soothiram, indicated for the treatment of padarthamarai. The drug is screened for its biochemical properties. Further, comprehensive pharmacological analysis is needed to evaluate its potency, and the drug has its potential to undergo further research.

REFERENCE

1. Doit C, Barre J, Cohen R, Bonacorsi S, Bourrillon A, Bingen E. Bactericidal activity against intermediately cephalosporin-resistant Streptococcus pneumoniae in cerebrospinal fluid of children with bacterial meningitis treated with high doses of cefotaxime and vancomycin. Antimicrob. Agents Chemother. 1997; 41: 2050–2052. [DOI] [PMC free article] [PubMed] [Google Scholar]
2. Cordell G. Changing strategies in natural products chemistry. Phytochemistry. 1995; 40: 1585–1612 [Google Scholar]
3. Biochemical Profile and Antimicrobial Activity of an Herbal-Based Formula and Its Potential Application in Cosmetic Industry by Alice Grigore 1,*ORCID, Lucia Camelia Pirvu 1ORCID, Ionica Bejanaru 1, Georgeta Neagu 1ORCID, Camelia Filofteia Diguta 2,3,

Luciana Glava 4, Carmen Ionita 5, and Florentina Matei 2,3 https://www.mdpi.com/2673-8007/2/1/16

4. Antifungal Resistance and New Strategies to Control Fungal Infections Patrick Vandeputte, Selene Ferrari, Alix T Coste https://pmc.ncbi.nlm.nih.gov/articles/PMC3236459/
5. Gupta C, Das S, Gaurav V, Singh PK, Rai G, Datt S, et al. Review on host-pathogen interaction in dermatophyte infections. J Mycol Med (2023) 33:101331. doi: 10.1016/j.mycmed.2022.101331 [DOI] [PubMed] [Google Scholar]
6. Faway E, Lambert de Rouvroit C, Poumay Y. In vitro models of dermatophyte infection to investigate epidermal barrier alterations. Exp Dermatol (2018) 27:915 22. doi: 10.1111/exd.13726 [DOI] [PubMed] [Google Scholar]
7. Morales EG, Iorizzo M, Lucchini GM, Mainetti C. Trichophyton violaceum: an emerging pathogen in Southern Switzerland. Dermatology (2019) 235:434 39. doi: 10.1159/000501271 [DOI] [PubMed] [Google Scholar]
8. Degreef H. Clinical forms of dermatophytosis (ringworm infection). Mycopathologia (2008) 166:257 65. doi: 10.1007/s11046-008-9101-8 [DOI] [PubMed] [Google Scholar]
9. Smith KJ, Welsh M, Skelton H. Trichophyton rubrum showing deep dermal invasion directly from the epidermis in immunosuppressed patients. Br J Dermatol (2001) 145:344–8. doi: 10.1046/j.1365-2133.2001.04331.x [DOI] [PubMed] [Google Scholar]
10. Wang R Huang C, Zhang Y, Li R. Invasive dermatophyte infection: a systematic review. Mycoses (2021) 64:340–8. doi: 10.1111/myc.13212 [DOI] [PubMed] [Google Scholar]
11. Reference from 'Antifungal Resistance and New Strategies to Control Fungal Infections' by Patrick Vandeputte, Selene Ferrari, Alix T. Coste, 1, https://pmc.ncbi.nlm.nih.gov/articles/PMC3236459/
12. Pfaller M, Moet GJ, Messer SA, Jones RN, Castanheira M. Geographic variations in species distribution and echinocandin and azole antifungal resistance rates among Candida bloodstream infection isolates: Report from the SENTRY Antimicrobial Surveillance Program (2008 to 2009). Journal of Clinical Microbiology. 2011;49(1):396–399. doi: 10.1128/JCM.01398-10. [DOI] [PMC free article] [PubMed] [Google Scholar]
13. Pfaller MA, Moet GJ, Messer SA, Jones RN, Castanheira M. Candida bloodstream infections: comparison of species distributions and antifungal resistance patterns in community-onset and nosocomial isolates in the SENTRY Antimicrobial Surveillance Program, 2008-2009. Antimicrobial Agents and Chemotherapy. 2011;55(2):561–566. doi:

10.1128/AAC.01079-10. [DOI] [PMC free article] [PubMed] [Google Scholar]

14. Pfaller MA, Messer SA, Moet GJ, Jones RN, Castanheira M. Candida bloodstream infections: comparison of species distribution and resistance to echinocandin and azole antifungal agents in Intensive Care Unit (ICU) and non-ICU settings in the SENTRY Antimicrobial Surveillance Program (2008-2009). International Journal of Antimicrobial Agents. 2011;38(1):65–69. doi: 10.1016/j.ijantimicag.2011.02.016. [DOI] [PubMed] [Google Scholar]
15. The use of sulfur in dermatology. Aditya K Gupta et al. J Drugs Dermatol. 2004 Jul - Aug. Mortensen S.P., González-Alonso J., Bune L.T., Saltin B., Pilegaard H., Hellsten Y. ATP-induced vasodilation and purinergic receptors in the human leg: Roles of nitric oxide, prostaglandins, and adenosine. Am. J. Physiol. Regul. Integr. Comp. Physiol. 2009;296:R1140: R1148. doi: 10.1152/ajpregu 90822.2008. [DOI] [PubMed] [Google Scholar]
16. Ross J. Energy Transfer from Adenosine Triphosphate. J. Phys. Chem. B. 2006;110:6987–6990. doi: 10.1021/jp0556862. [DOI] [PubMed] [Google Scholar]
17. Mizumoto N., Mummert E.M., Shalhevet D., Takashima A., Keratinocyte ATP release Assay for testing skin-irritating potentials of structurally diverse chemicals. J. Investig. Dermatol. 2003;121:1066–1072. Doi: 10.1046/j.1523-1747.2003.12558.x. [DOI] [PubMed] [Google Scholar]
18. Koizumi S., Fujishita K., Inoue K., Shigemoto-Mogami Y., Tsuda M. Ca2+ waves in keratinocytes are transmitted to sensory neurons: The involvement of extracellular ATP and P2Y2 receptor activation. Biochem. J. 2004;380:329–338. doi: 10.1042/bj20031089. [DOI] [PMC free article] [PubMed] [Google Scholar]
19. O'Grady S.M., Kita H. ATP functions as a primary alarmin in allergen-induced type 2 immunity. Am. J. Physiol. Cell Physiol. 2023;325:C1369–C1386. doi: 10.1152/ajpcell 00370.2023. [DOI] [PMC free article] [PubMed] [Google Scholar]
20. Shiratori-Hayashi M., Hasegawa A., Toyonaga H., Andoh T., Nakahara T., Kido-Nakahara M., Furue M., Kuraishi Y., Inoue K., Dong X., et al. Role of P2X3 receptors in scratching behavior in mouse models. J. Allergy Clin. Immunol. 2019;143:1252 1254. doi: 10.1016/j.jaci.2018.10.053. [DOI] [PMC free article] [PubMed] [Google Scholar]
21. Kurashima Y., Amiya T., Nochi T., Fujisawa K., Haraguchi T., Iba H., Tsutsui H., Sato S., Nakajima S., Iijima H., et al. Extracellular ATP mediates mast cell-dependent intestinal inflammation through P2X7 purinoceptors. Nat. Commun. 2012;3:1034. Doi: 10.1038/ncomms2023. [DOI] [PMC free article] [PubMed] [Google Scholar]
22. Ferrari D., Chiozzi P., Falzoni S., Dal Susino M., Melchiorri L., Baricordi O.R., Di Virgilio F.

Extracellular ATP triggers IL-1β release by activating the purinergic P2Z receptor of human macrophages. J. Immunol. 1997;159:1451 1458. doi: 10.4049/jimmunol. 159.3.1451. [DOI] [PubMed] [Google Scholar]

23. Dixon C.J., Bowler W.B., Littlewood-Evans A., Dillon J.P., Bilbe G., Sharpe G.R., A Gallagher J. Regulation of epidermal homeostasis through P2Y2 receptors. Br. J. Pharmacol. 1999;127:1680 1686. doi: 10.1038/sj.bjp.0702653. [DOI] [PMC free article] [PubMed] [Google Scholar]
24. Seiffert K., Ding W., Wagner J.A., Granstein R.D. ATPγS enhances the production of inflammatory mediators by a human dermal endothelial cell line via purinergic receptor signaling. J. Investig. Dermatol. 2006;126:1017–1027. doi: 10.1038/sj.jid.5700135. [DOI] [PubMed] [Google Scholar]
25. Bles N., Horckmans M., Lefort A., Libert F., Macours P., El Housni H., Marteau F., Boeynaems J.-M., Communi D. Gene expression profiling defines ATP as a key regulator of human dendritic cell functions. J. Immunol. 2007;179:3550 3558. doi: 10.4049/jimmunol.. 179.6.3550. [DOI] [PubMed] [Google Scholar]
26. Inoue K., Hosoi J., Denda M. Extracellular ATP has stimulatory effects on the expression and release of IL-6 via purinergic receptors in normal human epidermal keratinocytes. J. Investig. Dermatol. 2007;127:362–371. doi: 10.1038/sj.jid.5700526. [DOI] [PubMed] [Google Scholar]
27. Yoshida H., Kobayashi D., Ohkubo S., Nakahata N. ATP stimulates interleukin-6 production via P2Y receptors in human HaCaT keratinocytes. Eur. J. Pharmacol. 2006;540:1 9. doi: 10.1016/j.ejphar.2006.04.008. [DOI] [PubMed] [Google Scholar]
28. Marteau F., Gonzalez S.N., Communi D., Goldman M., Boeynaems J.M. Thrombospondin-1 and indoleamine 23-dioxygenase are major targets of extracellular ATP in human dendritic cells. Blood. 2005;106:3860–3866. doi: 10.1182/blood-2005- 05-1843. [DOI] [PubMed] [Google Scholar]
29. Okamoto T., Ogawa Y., Kinoshita M., Ihara T., Shimada S., Koizumi S., Kawamura T. Mechanical stretch-induced ATP release from keratinocytes triggers Koebner phenomenon in psoriasis. J. Dermatol. Sci. 2021;103:60 62. doi: 10.1016/j.jdermsci.2021.06.001. [DOI] [PubMed] [Google Scholar]
30. Solini A., Chiozzi P., Morelli A., Fellin R., Di Virgilio F. Human primary fibroblasts in vitro express a purinergic P2X7 receptor coupled to ion fluxes, microvesicle formation, and IL-6 release. J. Cell Sci. 1999;112:297 305. doi: 10.1242/jcs . 112.3.297. [DOI] [PubMed] [Google Scholar]