International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 15-23 www.ijdmsrjournal.com ISSN: 2582-6018

Ayurvedic Perspective of Purification Therapy on Gut Microbiota in Rheumatoid Arthritis

Bikash Kumar Tripathy[1], Sayantan Bera[2], Srikanta Pandit[3], Tapas Kumar Sur[3]
[1]*Department of Panchakarma, Raghunath Ayurved Mahavidyalaya and Hospital, Contai, India*
[2]*Department of Panchakarma, J. B. Roy State Ayurvedic Medical College & Hospital, Kolkata, India*
[3]*Department of Kayachikitsa, J. B. Roy State Ayurvedic Medical College & Hospital, Kolkata, India*

Date of Submission: 01-11-2025 Date of Acceptance: 08-11-2025

ABSTRACT: The etiology of Rheumatoid Arthritis (RA) is not completely understood. However, it is known that a complex interaction between genetic and environmental factors induces a pathological activation of the immune system leading to the clinical onset of RA. The interplay between myeloid cells and T cells can exert profound effects on monocyte and macrophage function and may contribute to the immunopathology of RA. Dysbiosis of the gut microbiota exacerbated the severity of RA. In Ayurveda, Vaitarana Basti has been used for managing Amavata (RA) from ancient times. Vaitarana Basti is one of the most effective treatments (shodhana and aushadhi). However, its significance in RA, specifically how gut microbiota influences the underlying cellular and molecular processes during this therapy is still being elucidated. In this context we tried to emphasis the effective role of Ayurvedic purification therapy for modulating the gut microbial colonies during treatment management in RA patients.

Keywords- Rheumatoid Arthritis, Gut microbiota, Ayurveda, Panchakarma, Vaitarana basti, Amabata

I. INTRODUCTION

Rheumatoid arthritis (RA) is the most frequent systemic autoimmune disease. It affects nearly 1% of the general population and is characterized by a chronic inflammatory disease that primarily distresses multiple joints and causes irreversible bone and cartilage destruction (McInnes and Schett, 2017). Recently, a thorough analysis of the Global Burden of Diseases, Injuries and Risk Factors Study (GBD) reported that 17.6 million people globally have been diagnosed with RA, with a mortality rate of 0.47 per 100,000 populations, while years lived with disability (YLDs) constituted 76.4% (GBD, 2023). North America (1.46%) had the greatest reported mean point prevalence followed by Africa (0.80%), Europe (0.53%), South America (0.46%) and Asia (0.34%) (Almutairi et al, 2021). In RA, many immune cell types and signaling networks malfunction resulting in a maladaptive tissue repair process that causes organ damage, mostly in the joints (Weyand and Goronzy, 2021). It has been reported that the combination of genetic, epigenetic and environmental factors are responsible for the onset and development of RA. An elevated risk of RA is linked to older age, female sex and a family history of the condition. However, the sex difference is less pronounced in older individuals (Edilova et al, 2021). Furthermore, a clinical prediction rule for RA has been created and verified by researchers in the Netherlands. This guideline is meant to assist in identifying individuals with undifferentiated arthritis who are most likely to develop into RA and to direct referral and follow-up care (Mochan and Ebell et al, 2008).

Macrophages are central in perpetuating arthritis development by stimulating apoptotic immune cells and promoting the proliferation of fibroblasts (Martinez et al, 2009). Patients suffering from RA often exhibit discomfort, stiffness in several joints, swelling in peripheral joints, localized osteoporosis, constriction of the synovial space and fibrous ankylosis. The most often afflicted joints are the wrists, proximal inter-phalangeal joints and meta-carpophalangeal joints (NICE guideline, 2020). The gradual degeneration of synovial-lined joints is a hallmark of RA. A variety of innate and adaptive immune cells secrets different chemokines, inflammatory mediators, pro- and anti-inflammatory agents and other chemicals that affect the patient's synovial tissue and joints are involved in the pathophysiology of RA (Fang et al, 2020). The two most important cellular players in RA pathogenesis are monocytes and macrophages. It has been observed that RA monocytes possess the ability to generate pro-inflammatory cytokines such TNF-α, IL-1β, and IL-6 (Zhao et al, 2021).

Recent experimental and clinical researches have revealed the important role of the microbiome in RA (Tong et al, 2020). Increasing evidence suggests that immune abnormalities such as changes in the quantity and function of T and B cells, excess production of TNF-α, increased

proinflammatory cytokines and the presence of anti-citrullinated protein antibodies (ACPAs) may occur at the mucosal level due to a high load of microbial antigens suggesting a causal role of the microbiome in the pathogenesis of RA (Tsetseri et al, 2023). Moreover, studies have demonstrated that microbial differences in the gastrointestinal tract of RA patients may partially determine the bioavailability and subsequent clinical outcome of anti-arthritic medicines such as methotrexate, sulfasalazine and etanercept (Zhao et al, 2022). In addition, dietary nutrients also influence the composition and function of the gut microbiota, which may have a significant impact on the prevention and treatment of RA (Yang et al, 2024). Hence, the dysbiosis gut microbiota, inflammatory factors and immunological responses are all linked and influence the development of RA.

Numerous studies have shown that Indian Ayurvedic remedies provide considerable therapeutic outcomes in RA (Amavata). Aside from oral medicines, Ayurveda includes specialized therapies for noncommunicable and degenerative diseases such as Panchakarma (Verma et al, 2020). Basti karma, or an Ayurvedic medicated enema is an important treatment module described in Panchakarma. Basti remedies are beneficial in detoxifying vitiated pitta, kapha, rakta, samsarga and sannipataja doshas while also balancing and nourishing vata dosha (Banu and Banu, 2023). Vaitarana Basti is one of the most effective therapies (shodhana and aushadhi) for Amavata (RA). It has been shown in multiple studies to relieve clinical symptoms of RA. Additionally, it is thought to enhance the bioavailability of Ayurvedic medications (Baria et al, 2011; Kumar, 2011; Mhatre et al, 2018; Kaushik et al, 2021). Nevertheless, there are limited scientific reports that describe its specific mode of actions. Therefore, in this context we investigated the possible role of Vaitarana Basti therapy within the Ayurvedic treatment framework for Amabata (RA) focusing on its potential effects on gut microbiota regulation.

II. METHODOLOGY

This narrative review focuses on Amabata, an Ayurvedic purifying therapy for gut microbiota and its use to RA. The review includes material from both Ayurvedic literatures and recent scientific studies. A comprehensive search was undertaken utilizing electronic databases such as PubMed, Scopus and Google Scholar. The search terms were "gut microbiota," "Ayurveda," "panchakarma," "vaitarana basti," "amabata," "rheumatoid arthritis" and other similar topics published in last 10 years.

III. RESULTS

Based on the current investigation, the findings emphasize the traditional applications of Vaitarana Basti on Amabata and gut microbiota. These findings provide a comprehensive understanding of the role of Vaitarana Basti therapy in treating Amabata (RA) and its possible impact on gut microbiota.

Pathogenesis in Rheumatoid Arthritis

The relationship between genetic, epigenetic, metabolic and microbial factors and RA has been widely reviewed (Plant et al, 2014). There are two main types of RA based on the presence or the absence of ACPAs. The ACPA-positive group with RA exhibits a more aggressive clinical appearance than the ACPA-negative subset (England et al, 2023). 50–80% of RA patients have autoantibodies depending on the duration of the disease. Autoantibodies can activate inflammatory effector pathways in chondrocytes and cartilage, resulting in the release of extracellular matrix components (Wei et al, 2015; Fang et al, 2020). Numerous proinflammatory cytokines such as TNF-α, IL-1 and IL-6, chemokines like CCL5, CXCL-1 and CXCL-10 and different matrix lysing enzymes are secreted by macrophages. These factors then activate osteoclasts and fibroblasts assist in drawing in neutrophils, monocytes and lymphocytes and set off a chain of events that accelerate inflammation and destroy articular cartilage (McInnes et al, 2016; Zhao et al, 2021). The effective reduction of arthritic symptoms and signs in patients treated with TNF-α-neutralizing drugs has revolutionized RA therapy approaches and encouraged more research aimed at targeting additional inflammatory cytokines such as IL-1, IL-6, and IL-17 (Plant et al, 2014; Wei et al, 2015; Malmstrom et al, 2017). IL-17 expression is elevated and contributes to joint injury, while Wnt antagonist Dkk-1 is overexpressed and associated with bone erosions. Wnt10b expression is also upregulated in RA bone and correlates positively with IL-17 expression (Nanke, 2023). IL-6 primarily affects CD4+T-cell development, namely stimulating the Th17 pathway and suppressing T-reg differentiation. Increasing evidences demonstrate that RA development results due to an imbalance between CD4+T cell subsets (Zhao et al, 2021).

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 15-23 www.ijdmsrjournal.com ISSN: 2582-6018

Diagnosis and treatment management in Rheumatoid Arthritis

The American College of Rheumatology (ACR) has issued a set of guidelines aimed at promoting early and successful RA treatment and improving diagnosis. The 2010 criteria include the number of painful and swollen joints, CRP and ESR, ACPA or RF (rheumatoid factor) and the duration of symptoms. In addition to medicine the most recent update recommends exercise, rehabilitation, diet and functional evaluation (England et al, 2023). The Rheumatoid Arthritis Severity Scale (RASS) measures disease activity, functional disability and physical damage (Anderson et al, 2012). According to current study, up to 40% of RA cases may be prevented through a number of lifestyle factors such as maintaining a healthy weight and balanced diet, practicing good oral hygiene and limiting occupational risk exposures (Alwarith et al, 2019; Koller-Smith et al, 2022). Early detection and access to effective disease-modifying anti-rheumatic drug (DMARD) therapy improve RA outcomes, including the severity of disability and mortality rates. The two main pillars of modern RA treatment are disease modification and symptomatic management. Patients on delayed DMARD therapy were found to have an increased risk of radiographic joint space narrowing and bony erosions (Guo et al, 2018). Conventional synthetic DMARDs include methotrexate (folic acid analog), leflunomide (pyrimidine synthesis inhibitor), sulfasalazine (immunosuppression) and hydroxychloroquine (immunomodulator). Biological DMARDs include Etanercept (TNF-α inhibitor), Rituximab (B-cell depleting), Tocilizumab (IL-6 inhibitor), Anakinra (IL-1 inhibitpr), Secukinumab (IL-17 inhibitor), Denosumab (RANKL inhibitor), Mavrilimumab (GM-CSF inhibitor) and Tofacitinib (JAK1 and JAK3 inhibitor) (Jameson et al, 2022). However, their use is limited to RA patients due to substantial side effects, drug interactions and high price. As a result, the potential utility of plant-based diets and nutritional therapies in RA has been extensively reported around the world as an alternative to synthetic medications (Alwarith et al, 2019).

Gut microbiota in Rheumatoid Arthritis

The human microbiome project and the MetaHIT project have generated massive genomic databases (over 70 million 16S rRNA gene sequences) − accounting for many variables and significantly enhancing the possibility for future microbiome research (Alpızar-Rodrıguez et al, 2020). Numerous investigations revealed a key involvement of gut microbiota in RA pathogenesis, including the formation of proinflammatory metabolites, disruption of the intestinal mucosal barrier and molecular mimicry of autoantigens (Konig, 2020). These immunological abnormalities may occur at the mucosal level due to a high load of microbial antigens, implicating a causal role for the microbiome in the pathogenesis of RA (Yang et al, 2024). Studies have revealed that an elevated Th17/Treg ratio is closely connected to RA and that Th17/Treg balance is strongly influenced by gut microbiota and their metabolites (Kelly and Mulder IE, 2012). Individual microbiota varies substantially due to a variety of factors such as vaginal delivery, nutrition and fecal microbiota transplantation. It also fluctuates within the same individual regionally, depending on the local environment or temporarily due to exposure to environmental factors such as the use of antibiotics (Hasan and Yang, 2019).

Gene sequencing revealed 2172 species from humans and categorized them into 12 separate phyla, with Proteobacteria, Firmicutes, Actinobacteria and Bacteroidetes. Lactobacillaceae family dominates the small intestine, whereas the colon is dominated by anaerobic species from Prevotellaceae, Lachnospiraceae and Rikenellaceae families (Zhao et al, 2022). The role of microbiome in RA was investigated in sixteen clinical trials with both early-onset and established patients using 16S rRNA and shotgun metagenomics sequencing (Ermencheva et al, 2024). Kishikawa et al (2020) showed gut microbiome of RA patients contained high amounts of Prevotella copri in Japanese population. Th17 cytokines and ACPAs were found to be linked with P. copri antibodies (Kishikawa et al, 2020). Furthermore, a subset of RA patients exhibited P. copri 16S rDNA in their synovial fluid (Alpızar-Rodrıguez et al, 2020). Pianta et al (2017) hypothesized that intestinal T-cell activation caused by the identification of Prevotella-derived epitopes in genetically predisposed individuals migrates to joints. RA has also been linked to a number of different bacterial species, including Actinomyces, Collinsella, Lactobacillus and Eggerthella (HI et al, 2019). Collinsela aerofaciens elevated the incidence and severity of RA as well as a number of inflammatory cytokines and chemokines such as IL-17A, CXCL1, CXCL5 and NF-$_K$B1 (Forbes et al, 2018). Sun et al (2019) found that Alloprevotella associated positively with RF, ESR and CRP. Furthermore, investigations have revealed a reduction in the taxa Roseburia and Faecalibacterium in RA patients (Chen et al, 2016).

International Journal Dental and Medical Sciences Research
Volume 7, Issue 6, Nov – Dec 2025 pp 15-23 www.ijdmsrjournal.com ISSN: 2582-6018

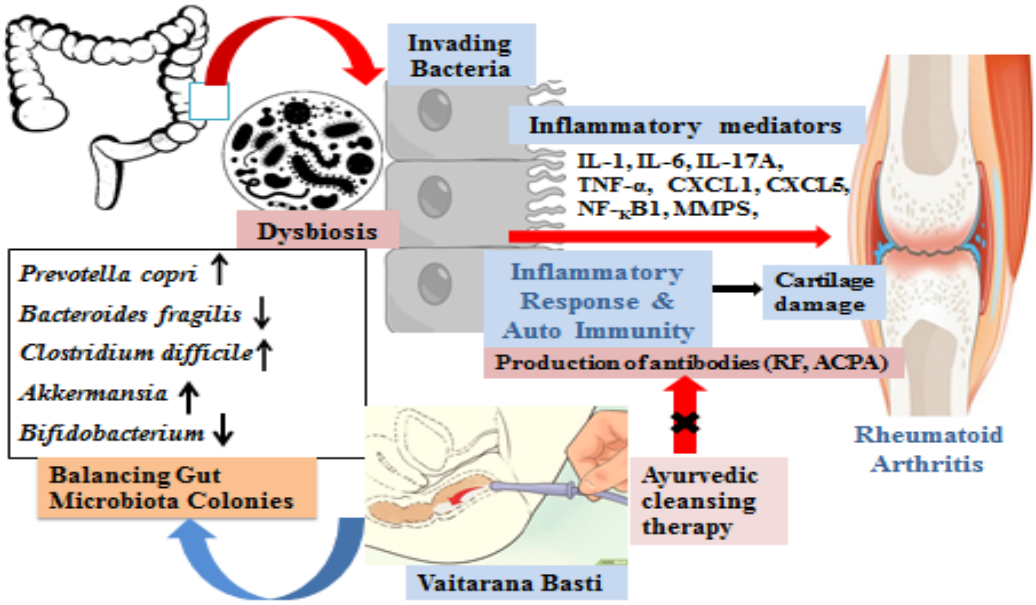

Fig. 1: Gut microbiome in the pathogenesis of Rheumatoid Arthritis (Amavata)

Additionally, studies have demonstrated that gut microbiota of patients with RA may affect the bioavailability and final clinical outcome of methotrexate, sulfasalazine, and etanercept (Zhao et al., 2022). Probiotics, herbal remedies and dietary nutrients all have an impact on the growth and functionality of pathogenic gut microbiota. Mei et al (2021) demonstrated Huayu-Qiangshen-Tongbi mixture a traditional Chinese medicine (TCM) reverse the reduction of Clostridium celatum in individuals with RA.

Table 1: Role of gut microbiota in Rheumatoid Arthritis and its relationship with Prakiti

Gut Microbiota	Function in RA	Relationship with Prakiti
Eggerthella lenta Clostridium difficile (Pathogenic)	• Regulate cartilage formation • Presentation of arthritogenic self-peptides ○ E. lenta growth stimulate CXCL5 and CD4 T-cells, as well as B-cells that produce IL-17 and IFN-γ ○ C. difficile can trigger inflammation through alterations in hormone levels or via infections ○ It can rise in RA patients	**Vata Prakriti** : Higher levels of – • Bacteroides fragilis • Prevotella copri • Ruminococcus bromii **Pitta Prakriti** : Higher levels of – • Escherichia coli • Enterobacter spp. • Clostridium perfringens **Kapha Prakriti:** Higher levels of – • Bifidobacterium adolescentis • Faecalibacterium prausnitzii
Bacteroides fragilis Bacteroides uniformis Bacteroides plebeius (Beneficial)	• Primary structures of articular cartilage • A decline in these bacteria may contribute to the development of RA ○ B. fragilis is important for the therapeutic effect of methotrexate ○ Bacteroides species may be needed to strengthen the intestinal barrier	
Roseburia intestinalis (Beneficial)	• Autoantigen of antiphospholipid syndrome • Lower levels of R. intestinalis cause less butyrate synthesis, which	

	reduces the protective benefits ○ R. intestinalis is a major producer of butyrate, a short-chain fatty acid (SCFA) with strong anti-inflammatory effects ○ The reduced abundance of R. intestinalis in RA patients contributes to an imbalance in the Treg/Th17 ratio, which favors RA's pro-inflammatory state.	• Akkermansia muciniphila
Prevotella copri Prevotella spp. (Pathogenic)	• Autoantigen react to T cells and autoantibodies from RA patient • P. copri may contribute to RA pathogenesis by the activation of inflammatory responses and molecular mimicry between microbial and host epitopes ○ Enrich in RA patients' inflamed joints ○ Prevotella spp. are common in BAL of RA patients	

Ayurvedic Purification Therapy in Rheumatoid Arthritis

Panchakarma is regarded as the backbone of Ayurvedic treatment (chikitsa). Because it attempts to eliminate the underlying cause of disease (vyadhi) at a cellular level thus avoiding recurrence (Conboy et al, 2009). Five types of therapeutic applications described in Panchakarma (Sanshodhana Chikitsa). These are Vamana, Virechana, Basti, Raktamokshana and Nasya. These techniques were selected due to their importance in Ayurvedic medicine and because the start and conclusion of therapy allow for clear observations of pre- and post-test differences (Sharma et al, 2007).

The origins of Basti Karma can be traced back to the Vedic period, specifically the Rigveda and Atharvaveda (Verma et al, 2020). Basti Karma is most important in Ayurvedic treatment since it effectively eliminates unhealthy Vata, the only dosha (disorders) responsible for the movement of all doshas, dhatus and mala throughout the body (Banu and Banu, 2023). Rishi Charaka elegantly emphasized "**Basti Vataharanam Shreshtha**." Concisely, Basti (enema therapy) is the best treatment for alleviating Vata dosha (disorders). It alone constitutes "ardhachikitsa" (half of all treatments) because of its complete ability to treat a wide range of ailments, particularly those caused by Vata imbalance (Charaka Samhita, 2019).

Basti is a detoxifying and nourishing procedure that is administered straight into the Pakvashaya (colon) and Aantra (intestines) avoiding the stomach and upper the GI system. Moreover, four chapters of the Sushruta Samhita are devoted exclusively to the description of Bastikarma in Chikitsasthana (Sushruta Samhita, 2012). The two main varieties of rectal Basti are (a) **Anuvasana Basti** which is nourishing and strengthening in character and is typically done with herbal oils; and (b) **Niruha Basti** which is purifying and cleansing in nature and is done with herbal decoctions (Banthanal and Kulkarni, 2024). The efficacy of Basti dravya (enematic medications) is dictated by four pharmacodynamic principles: absorption, system biology, neural stimulation and excretion (Bera and Mukherjee, 2021).

Acharya Chakradatta mentioned Vaitarana Basti in the Niruha adhikara (Dwivedy, 2019). It is one of the most effective therapies (shodhana and aushadhi) for Amavata (RA). Vaitarana Basti is a cleansing and nourishing therapy based on similar principles that penetrates directly into the colon and intestines without being digested by the stomach or upper GI tract (Agalcha et al, 2022). It is suggested that Basti dravya (enematic medications), mainly anti-arthritic herbal medicines are rapidly absorbed and delivered to their target organs (Bera and Mukherjee, 2021). A clinical investigation found that Triphaladi decoction used as Basti dravya includes gallic acid, as the biomarker was subsequently identified and quantified in the blood (Auti, 2011). Numerous studies have also reported Vaitarana Basti improves clinical and functional status including joint pain, edema and stiffness and also lowers the prognostic indicators including ESR, CRP and ACPA in individuals with Amavata

or RA (Baria et al, 2011; Mhatre and Yadav, 2018; Kaushik et al, 2021; Banu and Banu 2024). However, there are very limited scientific reports to claim its definite mode of action.

Preliminary research suggests that individuals with different Prakriti types (Ayurvedic classification) have varied microbial fingerprints, implying a possible relationship between constitution and gut ecology. The functional fingerprints were strongly linked with Prakriti group characteristics and disease predispositions (Mobeen et al, 2019). The gut microbiome of the three different Prakriti samples showed a difference in the abundance of Bacteroides, Desulfovibrio, Parabacteroides, Slackia and Succinivibrio (Chaudhari et al, 2019). Individuals with Vata Prakriti had higher levels of carbohydrate-metabolizing bacteria such as Bacteroides fragilis, Prevotella copri and Ruminococcus bromii. But Pitta Prakriti individuals had higher amounts of Escherichia coli, Enterobacter spp. and Clostridium perfringens. Kapha Prakriti individuals revealed an increased abundance of Bifidobacterium adolescentis, Faecalibacterium prausnitzii and Akkermansia muciniphila (Yadav et al, 2025). Despite the paucity of studies on the effects of Vaitarana Basti on gut microbiota, it is highly probable to regulate the gut microbiota colonies during this procedure in Amavata. The constituents of Vaitarana Basti dravya are lipids, carbohydrates and minerals that have a nutritional effect on the gut microbiota, perhaps improving or regulating colonization (Alwarith et al, 2019; Agalcha et al, 2022). Apart from Vaitarana Basti, Simhanad guggulu combined with Sunthi kwath is a regularly employed oral medication during Amavata Basti therapy (Pandey et al, 2012; Mishra et al, 2021; Kalita et al, 2022; Kumar et al, 2024). There is ample pharmaceutical, pharmacological and clinical evidence on the effects of these medications on Amavata or RA, but their involvement in gut microbiota is still being explored (Agrawal and Prased, 2020).

IV. CONCLUSION

As an outcome, it is possible that the human gut microbiota had specific bacterial taxa in abundance across three Prakriti categories, indicating their physiological and pathological significance. Ayurvedic cleansing therapy or Vaitarana Basti allows the biological system, including the specific colonies of gut microbiota to quickly return to normalcy and regenerate, while also facilitating the desirable pharmacokinetic effect of therapeutic medicines delivered simultaneously or later. Therefore, it is believed that using the unique Vaitarana Basti to reduce the colonies of detrimental gut microbiota responsible for Amabata pathogenesis could be beneficial in the medical management of Amabata (RA) patients.

Acknowledgements
The authors thank Mrs. Sreyashi Sur for help in art and designing.

Conflict of Interest
Authors declare no conflicts of interest.

Sources of Funding
This study received no grants from public, commercial or non-profit funding entities.

Author Contribution
Conceptualization: Tapas Kumar Sur, Srikanta Pandit. Formal analysis: Tapas Kumar Sur, Bikash Tripathy, Sayantan Bera. Supervision: Tapas Kumar Sur, Srikanta Pandit. Writing–original draft: Tapas Kumar Sur. Writing–review & editing: Bikash Tripathy, Sayantan Bera, Srikanta Pandit, Tapas Kumar Sur.

REFERENCES

[1] Agalcha R, Rajoria K, Kumar S, Singh SK, and Sitpal N. Critical analysis on action of Basti: a review. Research & Reviews: A Journal of Ayurvedic Science, Yoga & Naturopathy, 9(3), 2022, 32-36.
[2] Agrawal K, and Prased R. A systemic review of Amavata and its management. Mukt Shabd Journal, 9(6), 2020, 5234-5244.
[3] Almutairi KB, Nossent JC, Preen DB, Keen HI, and Inderjeeth CA. The prevalence of rheumatoid arthritis: a systematic review of population-based studies. J Rheumatol, 48(5), 2021, 669-676.
[4] Alpızar-Rodrıguez D, Finckh A, and Gilbert B. The role of nutritional factors and intestinal microbiota in rheumatoid arthritis development. Nutrients, 13, 2020, 96.
[5] Alwarith J, Kahleova H, Rembert E, Yonas W, Dort S, Calcagno M, Burgess N, Crosby L, and Barnard ND. Nutrition interventions in rheumatoid arthritis: the potential use of plant-based diets. A review. Front Nutr., 6, 2019, 141.
[6] Anderson J, Caplan L, Yazdany J, Robbins ML, Neogi T, Michaud K, Saag KG, O'Dell JR, and Kazi S. Rheumatoid arthritis disease activity measures: American College of Rheumatology recommendations for use in

clinical practice. Arthritis Care Res (Hoboken), 64(5), 2012, 640-647.
[7] Auti S. Effect of Triphaladi basti in the management of dyslipidemia M.D.(Ayu.) dissertation, Jamnagar; 2011.
[8] Banthanal P, and Kulkarni M. Critical review on Niruha Basti w.s.r. to Charaka Samhita. J Ayurveda Integr Med Sci,10, 2024, 5964.
[9] Banu M, and Banu W. Management of Amavata through Vaitarana Basti w.s.r to Rheumatoid Arthritis. AYUSHDHARA, 10(6), 2023, 183-188.
[10] Baria R, Joshi N, and Pandya D. Clinical efficacy of Panchamuladi Kaala Basti (enema) in the management of Amavata (Rheumatoid Arthritis). Ayu, 32(1), 2011, 90-94.
[11] Bera S, and Mukherjee S. A pilot clinical study of Mashadibasti in the management of Pakshaghata (Hemiplegia). AYUHOM, 8, 2021, 82-89.
[12] Chakradatta Sri Chakrapanidatta with the Vaidayaprabha Hindi commentary Indradeva Tripathi. Editor Ramanath Dwivedy. (Chaukhambha Sanskrita Sansthan, Varanasi, 2019).
[13] Charaka Samhita of Agnivesha. Commentary by Chakrapanidatta. Edited by Pandith Kashinath Shastri, Vol 2, Siddhi sthana. (Choukhambha Sanskrit Sansthan, Varanasi, 2019).
[14] Chaudhari D, Dhotre D, Agarwal D, Gondhali A, Nagarkar A, Lad V, Patil U, Juvekar S, Sinkar V, and Shouche Y. Understanding the association between the human gut, oral and skin microbiome and the Ayurvedic concept of prakriti. J Biosci, 44(5), 2019, 112.
[15] Chen J, Wright K, and Davis JM et al. An expansion of rare lineage intestinal microbes characterizes rheumatoid arthritis. Genome Med, 8, 2016, 43.
[16] Conboy L, Edshteyn I, and Garivaltis H. Ayurveda and Panchakarma: measuring the effects of a holistic health intervention. Scientific World Journal, 9, 2019, 272-280.
[17] Edilova MI, Akram A, and Abdul-Sater AA. Innate immunity drives pathogenesis of rheumatoid arthritis. Biomed J. 44(2), 2021, 172-182.
[18] England BR, Smith BJ, and Baker NA, et al. 2022 American College of Rheumatology Guideline for Exercise, Rehabilitation, Diet, and Additional Integrative Interventions for Rheumatoid Arthritis. Arthritis Rheumatol. 75(8), 2023, 1299-1311.
[19] Ermencheva P, Kotov G, Shumnalieva R, Velikova T, and Monov S. Exploring the role of the microbiome in rheumatoid arthritis—a critical review. Microorganisms, 12, 2024, 1387.
[20] Fang Q, Zhou C, and Kutty SN. Molecular and cellular pathways contributing to joint damage in rheumatoid arthritis. Mediators of Inflammation, 3830212, 2020.
[21] Forbes JD, Chen C-Y, and Knox NC et al. A comparative study of the gut microbiota in immune-mediated inflammatory diseases—does a common dysbiosis exist? Microbiome, 6, 2018, 22.
[22] Global, regional, and national burden of rheumatoid arthritis, 1990–2020, and projections to 2050: a systematic analysis of the Global Burden of Disease Study 2021. Lancet Rheumatol, 5, 2023, e594–610.
[23] Guo Q, Wang Y, and Xu D. et al. Rheumatoid arthritis: pathological mechanisms and modern pharmacologic therapies. Bone Research, 6, 2018, 15.
[24] Harrison's Principles of Internal Medicine. Jameson J, Fauci AS, Kasper DL, Hauser SL, Longo DL, and Loscalzo J. Edtior, 21st Edition. (McGraw Hill, London, 2022).
[25] Hasan N, and Yang H. Factors affecting the composition of the gut microbiota, and its modulation. Peer J, 7, 2019, e7502.
[26] HI, Li JR, and Liu CC et al. An association of gut microbiota with different phenotypes in Chinese patients with rheumatoid arthritis. J Clin Med,8, 2019, 1770.
[27] Kalita D, Gupta OP, Prakash, and Sarma BP. Evaluate the efficacy of Kshara Basti and Sunthi-Guduchi Churna in amavata (rheumatoid arthritis) - a clinical study. AYUSHDHARA, 9, 2022, 68-73. https://doi.org/10.47070/ayushdhara.v9i4.998
[28] Kaushik J, Kumar A, and Yadav P. Role of Vaitarana Basti in the management of amavata w.s.r. to rheumatoid arthritis – a case study. Int Res J Ayu Yoga, 4 (12), 2021, 67-73.
[29] Kelly D, Mulder IE. Microbiome and immunological interactions. Nutritional Review, 70 Suppl 1, 2012, S18–30.
[30] Kishikawa T, Maeda Y, and Nii T et al. Metagenome-wide association study of gut microbiome revealed novel aetiology of rheumatoid arthritis in the Japanese

population. Ann Rheum Dis.,79, 2020, 103–111.

[31] Koller-Smith L, Mehdi AM, March L, Tooth L, Mishra GD, and Thomas R. Rheumatoid arthritis is a preventable disease: 11 ways to reduce your patients' risk. Intern Med J., 52, 2022, 711–716.

[32] Konig, MF. The microbiome in autoimmune rheumatic disease. Best Pract Res Clin Rheumatol., 34, 2020, 101473.

[33] Kumar KPR. The efficacy of Ayurvedic treatment for rheumatoid arthritis: Cross-sectional experiential profile of a longitudinal study. Int J Ayurveda Research., 2, 2011, 8-13.

[34] Kumar S, Pandey A, Prasad R, Singh AK, and Yadav P. A comparative study to assess the treatment cost of Ayurvedic medicine (ginger and castor oil) and modern medicine (DMARDs) in Amavata (rheumatoid arthritis). Int J Ayu Pharma Research., 12(6), 2024, 111-118.

[35] Malmstrom V, Catrina AI, and Klareskog L. The immunopathogenesis of seropositive rheumatoid arthritis: from triggering to targeting. Nat. Rev. Immunol., 17, 2017, 60–75.

[36] Martinez FO, Helming L, and Gordon S. Alternative activation of macrophages: an immunologic functional perspective. Annu Rev Immunol., 27, 2009, 451-483.

[37] McInnes IB, Buckley CD, and Isaacs JD. Cytokines in rheumatoid arthritis - shaping the immunological landscape. Nature Reviews Rheumatology, 12, 2016, 63–68.

[38] McInnes, I. B., G. Schett. Pathogenetic insights from the treatment of rheumatoid arthritis. Lancet, 389, 2017, 2328-2337.

[39] Mei L, Yang Z, Zhang X, Liu Z, Wang M, Wu X, Chen X, Huang Q, and Huang R. Sustained Drug Treatment Alters the Gut Microbiota in Rheumatoid Arthritis. Front Immunol. 12, 2021, 704089.

[40] Mhatre A, and Yadav R. Clinical Study to evaluate efficacy of Erandamuladi Basti in the management of Amavata w.r.t. rheumatoid arthritis. J Ayurveda Integr Med Sci, 5, 2018, 45-48.

[41] Mishra M, Sharma C, and Sharma S et.al. Vivid action of simhanada guggulu in the management of Amavata (rheumatoid arthritis): a review. Int J Health Sci Res., 11(6), 2021, 126-129.

[42] Mobeen F, Sharma V, and Prakash T. Functional signature analysis of extreme Prakriti endophenotypes in gut microbiome of western Indian rural population. Bioinformation. 15(7), 2019, 490-505.

[43] Mochan E, and Ebell MH. Predicting rheumatoid arthritis risk in adults with undifferentiated arthritis. Am Fam Physician., 77(10), 2008, 1451-1453.

[44] Nanke Y. The Pathogenesis of rheumatoid arthritis breakthroughs in molecular mechanisms 1 and 2. Int. J. Mol. Sci., 24, 2023, 11060.

[45] NICE guideline 2020. Rheumatoid arthritis in adults: management. www.nice.org.uk/guidance/ng100

[46] Pandey SA, Joshi NP, and Pandya DM. Clinical efficacy of Shiva Guggulu and Simhanada Guggulu in Amavata (rheumatoid arthritis). Ayu, 33(2), 2012, 247-54.

[47] Pianta A, Arvikar S, and Strle K et al. Evidence of the immune relevance of Prevotella copri, a gut microbe, in patients with rheumatoid arthritis. Arthritis Rheumatol, 69, 2017; 964–975.

[48] Plant D, Wilson A, and Barton A. Genetic and epigenetic predictors of responsiveness to treatment in RA. Nat Rev Rheumatol., 10 (6), 2014, 329–337.

[49] Sharma H, Candela HM, Singh G, and Bassist G. Utilization of Ayurveda in health care: an approach for prevention, health promotion, and treatment of disease. Part 2-Ayurveda in primary health care. J Altern Complement Med., 13(10), 2007, 1135-1150.

[50] Sun Y, Chen Q, and Lin P et al. Characteristics of gut microbiota in patients with rheumatoid arthritis in Shanghai, China. Front Cell Infect Microbiol.,9, 2019, 369.

[51] Sushruta Samhita. Commentary by Ambikadutta Shastri. Chikitsa sthana, Part-I, (Chaukhambha Sanskrita Sansthan, Varanasi, 2012).

[52] Tong Y, Marion T, Schett G, Luo Y, and Liu Y. Microbiota and metabolites in rheumatic diseases. Autoimmun Rev., 19, 2020, 102530.

[53] Tsetseri MN, Silman AJ, Keene DJ, and Dakin SG. The role of the microbiome in rheumatoid arthritis: a review. Rheumatol Adv Prac., 7(2), 2023, :rkad034.

[54] Verma GK, Shakya N, Mishra DK, Yadav R, and Gupta P. An Ayurvedic approach on Basti: A Review. Int. J. Res. Ayurveda Pharm., 11(3), 2020, 128-142

[55] Wei ST, Sun YH, Zong SH, and Xiang YB. Serum levels of IL-6 and TNF-alpha may correlate with activity and severity of rheumatoid arthritis. Med Sci Monit., 21, 2015, 4030-4038.
[56] Weyand, C.M., and Goronzy, J.J. The immunology of rheumatoid arthritis. Nat Immunol., 22, 2021, 10–18.
[57] Yadav S, Yadav CR, and Deepshikha P. A scoping review of scholarly publications on association of Prakriti and gut microbiome. J Res Ayurvedic Sci., 9, 2025, 144-151.
[58] Yang Y, Hong Q, Zhang X, and Liu Z. Rheumatoid arthritis and the intestinal microbiome: probiotics as a potential therapy. Front. Immunol., 15, 2024, 1331486.
[59] Zhao J, Guo S, Schrodi SJ and He D. Molecular and cellular heterogeneity in rheumatoid arthritis: mechanisms and clinical implications. Front. Immunol., 12, 2021, 790122.
[60] Zhao T, Wei Y, Zhu Y, Xie Z, Hai Q, Li Z, and Qin D. Gut microbiota and rheumatoid arthritis: From pathogenesis to novel therapeutic opportunities. Front. Immunol., 13, 2022, 1007165.