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The Gut-Eye Axis and *Mandagni*: An Integrated Pathophysiological and Therapeutic Framework for Dry Eye Disease Management- A Review article

Sharma Adarsh Ramkalap*[1] and More Ravidas[2]

1. PhD Scholar, APM's Ayurved Mahavidyalaya, Sion, Mumbai; Assistant Professor, Department of Shalakyatantra, YMT Ayurvedic Medical College, Kharghar, Navi Mumbai, Maharashtra.Contact no: 8082229002, email Id: sharmaadarsh808@gmail.com

2. PhD Guide, Professor and HOD, Department of Shalakyatantra, APM's Ayurved Mahavidyalaya, Sion, Mumbai.Contact no: 9967483930, email Id: ravimore67@gmail.com.

***Corresponding Author**: Dr. Sharma Adarsh Ramkalap

Phd Scholar, APM's Ayurved Mahavidyalaya, Sion, Mumbai; Assistant Professor, Department of Shalakyatantra, YMT Ayurvedic Medical College, Kharghar, Navi Mumbai, Maharashtra. Contact no: 8082229002, email Id: sharmaadarsh808@gmail.com.

Abstract

Background: The Gut-Eye Axis (GEA) defines a crucial bidirectional link where intestinal integrity and microbial balance significantly modulate systemic inflammatory status, impacting ocular health. Dry Eye Disease (DED) is a widespread, chronic inflammatory disorder [1] frequently managed only symptomatically by conventional methods. The identification of systemic drivers is critical for achieving therapeutic breakthroughs.

Aim: To synthesize contemporary GEA research, focusing on DED pathogenesis via cytokine and metabolite pathways, with the classical *Ayurvedic* concepts of *Agni* (digestive fire), *Ama* (toxins), and *Pitta* vitiation. This integration aims to propose a unified, evidence-based diagnostic protocol and a three-phase integrated management strategy.

Methods: A narrative review was conducted, synthesizing clinical and preclinical studies on the GEA mechanism in ocular inflammation with classical *Ayurvedic* texts (*Charaka Samhita*[4] and *Astanga Hrdayam*[5]). The synthesis was established through the evaluation of functional homology between modern immunological markers and traditional *Ayurvedic* pathological processes.

Proposed Observation/Results: Gut Dysbiosis is demonstrated to be functionally homologous to *Mandagni* (impaired *Agni*),

leading to the formation of systemically active endotoxins and inflammatory signaling molecules (*Ama,* such as LPS and pro-inflammatory cytokines). The subsequent DED-specific ocular surface damage and inflammation are primarily mediated by *Pitta* vitiation.

Conclusion: The integrated GEA-Agni-Ama framework provides a sophisticated, personalized model for understanding DED etiology. A phased management protocol focusing sequentially on Agni restoration, gut barrier repair, and systemic *Pitta* expulsion promises a more holistic and effective approach to managing chronic DED.

Keywords: Gut-eye-axis, *Agni, Ama, Mandagni*, Dry Eye Disease, *Shushkakshipaka*.

1. Introduction

1.1. The Global Burden and Etiological Complexity of Dry Eye Disease (DED)

Dry Eye Disease (DED) is a highly pervasive, chronic, multifactorial disorder of the ocular surface and lacrimal gland, characterized by a loss of tear film homeostasis accompanied by ocular symptoms. The pathogenesis involves a complex interplay of environmental stressors, age-related changes, and underlying immune dysregulation[1]. Conventional ocular therapies, primarily reliant on artificial lubricants and anti-inflammatory agents like topical corticosteroids, often target local symptomatic relief without addressing the fundamental systemic drivers of the disease. The chronic and relapsing nature of DED(*Shushkakshipaka*) underscores the limitations of purely topical treatments and necessitates a shift toward identifying and neutralizing systemic, root-cause inflammatory pathways.

1.2. Emergence of the Gut-Eye Axis (GEA) as a Systemic Driver of Ocular Inflammation

The concept of the Gut-Eye Axis (GEA) has emerged as a fundamental physiological connection, establishing that the health and integrity of the intestinal microbiome significantly influence the immune and inflammatory status of distant sites, including the eye [2]. The GEA operates as a critical bidirectional communication pathway mediated primarily by the systemic circulation of microbial metabolites and immune cells. The hypothesis specifically defining the **gut dysbiosis–ocular surface–lacrimal gland axis** provides the modern mechanistic foundation linking intestinal pathology directly to DED pathogenesis, suggesting that alterations in the intestinal microbiota contribute significantly to the onset and progression of DED[3].

1.3. Historical Context: The *Ayurvedic* Primacy of *Agni* (Digestive Fire) in Systemic Health

Ayurveda, the traditional medical science of India, has long asserted the centrality of the digestive system (*Koshtha*) to overall systemic health and disease pathogenesis [4]. This principle is encapsulated by the concept of *Agni* (digestive fire), which governs all metabolic and transformation processes[5].

According to classical texts, the state of *Agni* dictates systemic health, with *Mandagni* (impaired or weak *Agni*) leading to improper digestion and the subsequent formation of *Ama*[6]. *Ama* is defined as undigested, reactive material that permeates systemic channels (*Srotas Vaigunya*), initiating chronic inflammatory and disease processes[6]. This classical framework offers a personalized, holistic model for understanding chronic inflammatory conditions, aligning functionally with contemporary GEA discoveries by positing that the health of the digestive system is the primary determinant of systemic inflammation.

1.4. Aim and Objectives:

This paper aims to bridge modern immunological understanding of the GEA, particularly its role in DED, with the classical *Ayurvedic Agni-Ama* paradigm. By establishing functional homology between these two systems, the review seeks to propose a unified, evidence-based approach encompassing both objective modern diagnostic markers and personalized *Ayurvedic* assessments, culminating in a detailed, three-phase integrated management strategy for DED.

2. Modern Pathogenesis: The Gut-Eye Axis and Inflammatory Cascade in DED

The GEA operates through a defined inflammatory cascade, where compromised gut integrity leads to systemic inflammation that targets the vulnerable tissues of the eye[1]. The specific pathology linking gut health to DED involves three sequential steps: dysbiosis, barrier failure, and immune skewing [15].

Methods and materials: A narrative review was conducted, synthesizing clinical and preclinical studies on the GEA mechanism in ocular inflammation with classical *Ayurvedic* texts (*Charaka Samhita*[4] and *Astanga Hrdayam*[5]). The synthesis was established through the evaluation of functional homology between modern immunological markers and traditional *Ayurvedic* pathological processes.

Observations:

2.1. Gut Dysbiosis and Intestinal Barrier Dysfunction (*Leaky Gut*)

The process begins with **Gut Dysbiosis**, characterized by an imbalance in the intestinal microbiota composition, often involving a reduction in beneficial bacteria populations. This microbial imbalance directly impairs the production of crucial microbial metabolites, particularly Short-Chain Fatty Acids (SCFAs). A critical consequence of dysbiosis is the compromise of the structural integrity of the intestinal epithelium, specifically the tight junctions. This failure leads to increased intestinal permeability, commonly termed **"Leaky Gut"**.

The compromised barrier facilitates the **Translocation** of Pathogen-Associated Molecular Patterns (PAMPs), such as **Lipopolysaccharide (LPS)**, into the systemic circulation. LPS, an endotoxin derived from the cell walls of Gram-negative bacteria, is a potent molecular signal that triggers and sustains chronic systemic inflammation[7, 10].

2.2. Systemic Inflammation and Immune Skewing

Once LPS and other PAMPs translocate into the blood, they activate systemic immune cells, initiating a widespread inflammatory response. This results in the robust production of pro-inflammatory signaling molecules, known as **Cytokines**, including Tumor Necrosis Factor-alpha (TNF-α), Interleukin-6 (IL-6), and Interleukin-17 (IL-17)[8, 11]. These molecules are the primary systemic mediators carrying the inflammatory message from the gut to the eye.

A profound consequence of gut dysbiosis is the resultant skewing of the systemic adaptive immune response. Dysbiosis is recognized to drive an imbalance between regulatory T cells (Treg) and pro-inflammatory T-helper 17 cells (Th17)[9]. This shift promotes a **Th17-driven pro-inflammatory response**, characterized by the high production of IL-17[9]. This polarization acts as a systemic amplifier of ocular inflammation; the translocated LPS (analogous to initial *Ama*) does not merely act as a temporary trigger but fundamentally programs the systemic immune response toward a chronic autoreactive state[10]. This sustained inflammatory drive is necessary to perpetuate the severe inflammation observed in chronic ocular diseases, validating the need for therapies that target this fundamental immune reprogramming[7].

2.3. The Gut-Ocular Surface-Lacrimal Gland Axis in DED

The specific GEA hypothesis relevant to DED proposes that gut microbiota alterations affect the lacrimal glands and corneas via neural, endocrine, or immune pathways, ultimately causing ocular inflammation, reduced tear secretion, and diminished tear quality[3].

The inflammatory cytokines that reach the ocular surface (e.g., IL-beta, IL-6) induce tissue damage by upregulating the expression of Matrix Metalloproteinase-9 (MMP-9) in the conjunctival epithelium[11]. Elevated MMP-9 levels are a well-established clinical biomarker for ocular surface inflammation, signifying conjunctival epithelial damage and goblet cell loss, which are hallmarks of DED pathology[12].

Concurrently, a deficit in protective metabolites exacerbates the damage. Due to dysbiosis, the production of beneficial SCFAs (like butyrate) decreases, leading to lower systemic SCFA concentrations. Butyrate plays a critical, direct protective role: studies demonstrate that gut-derived butyrate, reaching the ocular surface via systemic circulation, interacts with local ocular epithelial cells (via SLC5A8 receptors) to suppress pro-inflammatory gene expression[13]. In DED mouse models, intragastrically delivered butyrate successfully ameliorates ocular surface disease[13]. Thus, the failure of *Agni* (resulting in low SCFA production) constitutes a dual failure: it permits the escape of pro-inflammatory endotoxins (*Ama*) from the gut while simultaneously removing the body's primary protective anti-inflammatory agent from the ocular surface, critically predisposing the eye to chronic inflammatory damage.

3. *Ayurvedic* Correlation: *Mandagni*, *Ama*,

and *Pitta* Vitiation

The Ayurvedic paradigm offers a sophisticated conceptual framework that aligns precisely with the molecular events described by the GEA, facilitating an integrated understanding of root-cause pathology.

3.1. Functional Parallelism: Establishing the *Agni-Ama*-GEA Core Pathology

A direct conceptual parallel exists between the GEA inflammatory cascade and classical Ayurvedic pathology concerning disease initiation. This correlation confirms that the traditional understanding of health originating in digestion is scientifically validated by modern microbiome research.

***Mandagni* and SCFA Production:** The concept of *Mandagni* (Impaired *Agni*) is functionally homologous to modern gut dysbiosis[16]. When *Agni* is robust, it produces healthy metabolites (equivalent to SCFAs), which are crucial for immune modulation and gut barrier integrity. Conversely, when *Agni* is weak, it results in the generation of toxic, pro-inflammatory byproducts, which is the definition of *Ama*[6]. This correlation scientifically validates the centrality of proper digestive metabolism to preventing systemic inflammatory disease.

Table 1: Correlation Between Gut-Eye Axis Pathology and *Ayurvedic* Principles

Modern Concept (GEA)	**Functional Description**	***Ayurvedic* Counterpart**	**Pathological Link**
Gut Dysbiosis	Imbalance of microbiota; reduced SCFA production.	***Mandagni*** (Impaired *Agni*)	Leads to poor digestion and assimilation.
Increased Intestinal Permeability	Compromised tight junctions ("Leaky Gut").	***Srotas Vaigunya*** (Channel Vitiation)	Allows the passage of toxins/*Ama* into circulation.
LPS / Inflammatory Cytokines	Endotoxins and signaling molecules that drive systemic inflammation.	***Ama*** (Toxins)	Undigested, reactive material that causes pathological inflammation.
Ocular Inflammation (DED, Uveitis)	Vasculitis, neovascularization, tissue damage.	***Pitta* and *Rakta Dushthi***	*Ama* and heat (*Pitta*) travel via *Rakta Dhatu* (Blood) causing tissue (*Dhatu*) damage.

Ama

and Systemic LPS/Cytokines: The *Ayurvedic* concept of *Ama*—undigested, reactive material causing pathological inflammation —corresponds precisely to the functional role of translocated bacterial endotoxins (LPS) and the ensuing systemic cytokine burden (TNF-alpha, IL-6). Furthermore, the increased intestinal permeability (Leaky Gut) mirrors ***Srotas Vaigunya*** (vitiation of channels), which allows *Ama* to enter the systemic circulation.

3.2. Ocular Inflammation and *Pitta* Vitiation

The ultimate manifestation of GEA pathology in the eye, including the ocular surface damage seen in DED, is attributed primarily to the vitiation of *Pitta Dosha*[5]. *Pitta* governs heat, metabolism, and transformation. The destructive and inflammatory characteristics of pro-inflammatory cytokines, specifically TNF-alpha and IL-6, mirror the pathological effects of *Prakupita Pitta* (aggravated *Pitta*) on ocular tissues (*Dhatu Dushthi*)[7, 14]. The heat and catabolic nature of these cytokines cause tissue breakdown (*Daha*) and redness (*Raga*), which are the hallmarks of *Pitta* vitiation.

The specific *Pitta* subtype involved in DED is *Alochaka Pitta*, which resides in the retina and is responsible for visual function and ocular metabolism. The integrity of *Alochaka Pitta* is highly susceptible to metabolic disturbances and heat. Elevated Reactive Oxygen Species (ROS) and free radicals, which are central to the pathogenesis of DED and other chronic ocular conditions, represent the molecular expression of excessive *Pitta* heat. This fundamental correlation provides scientific justification for the traditional use of anti-*Pitta* therapies, as they functionally act as targeted cytokine and ROS neutralizers.

4. Integrated Diagnostic Framework and Therapeutic Synthesis

An integrated approach utilizes the strengths of both systems: modern markers for quantitative molecular definition of the root cause, and *Ayurvedic* diagnostics for personalized assessment and therapeutic selection.

4.1. Triangulated Diagnostic Framework

Effective root-cause management relies on triangulating data from both fields:

1. **Modern Markers:** These objective tests quantify the GEA pathology. They include Stool Microbiome Analysis (to map dysbiosis), the Lactulose/Mannitol Test (to quantify intestinal permeability), and Systemic Cytokine Profiles (to measure the load of inflammatory mediators like IL-17 and TNF-alpha). These markers quantify the GEA root cause.
2. ***Ayurvedic* Markers:** Personalized assessments like *Nadi Pariksha* (Pulse diagnosis) and *Jivha Pariksha* (Tongue diagnosis) are used to qualitatively assess the functional state of *Agni* and the presence of *Ama*. This provides the clinician with a personalized *Dosha*

imbalance profile, typically showing *Pitta* and *Vata* vitiation in DED, which guides the appropriate intensity and sequence of management phases.

4.2. The Three-Phase Integrated Management Protocol for DED

The integrated management strategy moves beyond mere symptomatic relief to a sequenced, root-cause resolution strategy, targeting *Mandagni* and *Ama*.

4.2.1. Phase I: *Deepana* and *Pachana* (Restoring *Agni*)

This initial phase focuses on igniting the digestive fire (*Agni*) and clearing initial, superficial *Ama*. The goal is to restore metabolic efficiency, which is functionally analogous to eliminating inflammatory dietary triggers and correcting fundamental digestive deficits.

- **Key Agents:** Ayurvedic herbs such as *Trikatu* (a blend of ginger, black pepper, and long pepper) and *Chitrak* are utilized to enhance *Agni*, aiding the body's natural ability to digest food and resolve latent *Ama* (LPS)[17].

4.2.2. Phase II: Gut Barrier Modulation and Microbiome Restoration

Once *Agni* is stabilized, the focus shifts to repairing the intestinal mucosal barrier and correcting the dysbiosis. This phase directly addresses intestinal permeability (*Srotas Vaigunya*).

- **Targeted Probiotics:** Modern research supports the use of targeted probiotic strains. Oral intake of specific strains, such as Lactobacillus fermentum HY7302, has been shown to significantly improve DED symptoms in mouse models by alleviating pro-inflammatory cytokines (IL-beta, IL-6) and regulating MMP-9 production[3]. Other clinical trials show that combined oral probiotics and prebiotics significantly improve dry eye symptoms in patients[9].
- ***Triphala* (Probiotic and Mucosal Support):** This classic *Ayurvedic* formulation, derived from three fruits (*Amalaki, Bibhitaki, Haritaki*), is used alongside probiotics. *Triphala* possesses proven probiotic effects, supporting beneficial gut flora, and improves mucosal integrity, directly addressing intestinal permeability. Furthermore, *Triphala* has immunomodulator and anti-inflammatory properties that help inhibit T-cells from releasing the cytokines (primarily IL-6) that incite the inflammatory component of dry eyes[12].

4.2.3. Phase III: Systemic *Pitta Shamana* and Detoxification

The final phase focuses on neutralizing the accumulated systemic inflammatory burden (*Prakupita Pitta* and entrenched *Ama*) that has reached the ocular tissues.

- **Systemic Anti-Pitta Agents:** Herbs like *Amalaki* (*Emblica officinalis*) and *Guduchi* are crucial systemic anti-inflammatory agents. *Amalaki* is a potent anti-*Pitta* agent due to its strong antioxidant capacity, directly neutralizing the 'hot' and damaging effects of ROS and systemic cytokines, thereby providing cytoprotection to

highly metabolic tissues like the RPE cells in the eye[14].

- **Therapeutic Purgation (*Virechana*):** For severe, chronic cases where *Pitta* and *Ama* are deeply rooted in the system (*Koshtha*), *Virechana* (therapeutic purgation) is recommended[5]. *Virechana* is considered the single most effective therapy for removing excess *Pitta* and *Ama* from the gastrointestinal tract[13].

***Virechana* as Functional Immune System Reset:** Clinical observations suggest that *Virechana* reduces systemic inflammatory markers such as C-reactive protein (CRP). Since *Pitta* is functionally correlated with these systemic cytokines, the detoxification achieved through *Virechana* provides a powerful, natural mechanism that effectively clears the GI tract of the reservoir of vitiated *Pitta* and *Ama*. This process results in profound systemic immune modulation, functionally mirroring the mechanism of Fecal Microbiota Transplantation (FMT) by fundamentally resetting the systemic inflammatory burden.

5. Discussion and Clinical Implications

5.1. Rationale for Personalization in GEA Treatment

The integrated GEA-*Agni-Ama* framework highlights that treatment success depends on personalization. While the GEA provides the molecular pathway (Gut Dysbiosis-Inflammation-DED), the specific status of the *Doshas* (*Pitta*, *Vata*) and the level of *Agni* deficiency dictate the necessary therapeutic intensity. By combining quantitative modern metrics (SCFA levels, cytokine profiles) with qualitative *Ayurvedic* assessment (*Nadi Pariksha*), clinicians can determine if a patient requires intensive *Shodhana* (purification, Phase III) or milder *Deepana* (restoration, Phase I). This personalized sequencing ensures that the therapeutic intervention is optimized for the patient's specific root-cause pathology, maximizing therapeutic efficacy.

5.2. Comparing Symptomatic Relief vs. Root-Cause Management

Conventional DED therapies primarily offer symptomatic relief by lubricating the ocular surface or suppressing local inflammation with corticosteroids. The integrated protocol represents a paradigm shift by targeting the initiating metabolic and inflammatory defects, *Mandagni* and *Ama*. By sequentially restoring the digestive ecology (Phase I), repairing the physical gut barrier (Phase II), and then clearing the systemic inflammatory heat (*Pitta Shamana*, Phase III), the integrated approach aims for a more durable and complete resolution of the underlying chronic inflammatory state, offering outcomes superior to those achieved through local treatments alone.

5.3. Future Directions in Clinical Trials and Biomarker Development

The functional homology established between the GEA and the *Agni-Ama* paradigm demands rigorous clinical validation. Future research must focus on double-blind, randomized controlled trials to assess the efficacy of integrated protocols utilizing both targeted probiotics (Lactobacillus fermentum[3]) and Ayurvedic formulations (*Triphala*, *Amalaki*).

Crucially, these trials should employ GEA-specific biomarkers to quantify treatment success:

1. **SCFA Profiling:** To objectively measure the restoration of *Agni* function.
2. **LPS/Cytokine Levels:** To quantify the clearance of *Ama* and reduction of *Pitta* heat.
3. **Ocular Markers:** To assess clinical endpoints, such as tear MMP-9 activity, which is a key inflammatory marker in DED[11, 12].
4. Specifically, the profound systemic effects of *Virechana* on inflammatory markers[13] warrant dedicated research to correlate its use with verifiable reductions in GEA and ocular inflammatory markers, thereby solidifying its place as a powerful root-cause intervention in chronic inflammatory ocular disease.

6. Conclusion

The Gut-Eye Axis provides the contemporary scientific basis for the ancient *Ayurvedic* principle that health and chronic disease originate in the digestive system. The concepts of *Mandagni* and *Ama* offer a clinically actionable framework that aligns perfectly with modern knowledge of gut dysbiosis, systemic endotoxemia (LPS), and cytokine-mediated inflammation. An integrated diagnostic approach, leveraging both microbiome profiling and personalized *Ayurvedic* assessment (*Ashthasthana parisksha-Nadi Pariksha*), followed by a targeted, three-phase management protocol focusing on *Agni* restoration, gut barrier repair, and systemic *Pitta* expulsion (*Virechana*), promises a holistic and highly effective therapeutic strategy for chronic Dry Eye Disease.

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