Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI:10.69980/ew73sz84

INTEGRATING AYURGENOMICS WITH POLYCYSTIC OVARY SYNDROME: A SCOPING REVIEW OF METABOLIC PHENOTYPES (*PRAKRITI*) AND PERSONALIZED MANAGEMENT IN *STREE ROGA*

Dr. Hemant Kuma*[1], Dr. Ankita Chouhan[2], Dr.Varsha[3]

[*1]Assistant Professor, PG Department of Stri Roga evam Prasuti Tantra, Post Graduate Institute of Ayurved, Dr S R Rajasthan Ayurved University Jodhpur
[2]PG Scholar PG Department of Stri Roga evam Prasuti Tantra, Post Graduate Institute of Ayurved, Dr S R Rajasthan Ayurved University Jodhpur.
[3]Assistant Professor, Department of Stri Roga evam Prasuti Tantra, Shri Bhanwar Lal Dugar Ayurved Vishva Bharti, Sardarsharhar, Churu, Rajasthan.

***Corresponding Author-** Dr. Hemant Kumar
(hemant5menaria@gmail.com)

Abstract

Polycystic Ovary Syndrome (PCOS) is a complex, multi-systemic endocrine and metabolic disorder affecting women of reproductive age, characterized by clinical or biochemical hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Despite significant advancements in modern endocrinology, the management of PCOS remains largely symptomatic, often failing to address the profound inter-individual variability in metabolic profiles and therapeutic responses. This research paper explores the integration of "Ayurgenomics"—an innovative trans-disciplinary field merging the ancient Ayurvedic science of *Prakriti* (individual constitution) with modern genomic and "omics" technologies—to provide a personalized framework for the management of PCOS within the domain of *Stree Roga* (Ayurvedic Gynecology).

Through a comprehensive scoping review adhering to PRISMA-ScR guidelines, this study identifies the molecular basis of the three primary *Prakriti* types (*Vata*, *Pitta*, and *Kapha*) and correlates them with the four modern PCOS phenotypes (Phenotypes A, B, C, and D). Evidence indicates that *Kapha-dominant* individuals exhibit a genetic predisposition toward obesity and metabolic syndrome, often involving polymorphisms in the FTO and CYP2C19 genes, whereas *Pitta-dominant* types demonstrate heightened inflammatory responses and specific metabolic efficiencies. The paper elucidates the Ayurvedic pathophysiology of PCOS, primarily through the lens of *Artava Kshaya*, *Pushpagni Jataharini*, and *Sroto-avarodha*, emphasizing the disruption of the HPO axis as a manifestation of *Vata-Kapha* imbalance.[i]

The review synthesizes personalized management strategies, contrasting conventional pharmacological interventions like Metformin with Ayurvedic *Shodhana* (purificatory) protocols such as *Virechana* and *Basti*, and *Shamana* (palliative) herbal formulations. By mapping the "genotype-phenotype" nexus, this study provides a scientific rationale for tailored interventions in *Stree Roga*, suggesting that Ayurgenomics can transform PCOS management from a reactive symptom-based model to a predictive and personalized preventive paradigm.

Keywords: PCOS, Ayurgenomics, Prakriti, Artava Kshaya, Stree Roga, Personalized Medicine, Metabolic Phenotypes, Tridosha, Shodhana, Genomics.

1. Introduction

The global burden of Polycystic Ovary Syndrome (PCOS) has escalated into a significant public health challenge, with a prevalence ranging from 5% to 20% depending on the diagnostic criteria utilized. In the Indian subcontinent, the prevalence is particularly alarming, with pooled estimates reaching nearly 10% and some regional studies suggesting figures as high as 22% among young women. PCOS is no longer viewed merely as a reproductive ailment but as a lifelong metabolic disorder predisposed to Type 2 diabetes mellitus (T2DM), cardiovascular diseases, and obstructive sleep apnea (OSA).

The clinical heterogeneity of PCOS is its hallmark. Women presenting with identical clinical features often exhibit vastly different metabolic trajectories and responses to standard treatments like hormonal contraceptives or insulin sensitizers. Modern medicine attempts to categorize this diversity through the Rotterdam criteria (2003) and the NIH extension (2012), resulting in four distinct phenotypes (A through D). However, these classifications often lack a predictive element regarding the individual's foundational biological nature and drug metabolism capacity.

Ayurveda, originating in ancient India, offers a theoretical and practical framework for analyzing human life through the lens of *Tridosha* *Vata*, *Pitta*, and *Kapha*. This system phenotypically classifies individuals into seven constitution types termed *Prakriti*, which is established at the moment of conception (*Shukra-Shonita-Samyoga*) and remains constant throughout life. *Prakriti* determines an individual's physical, physiological, and psychological traits, as well as their susceptibility to specific diseases and responses to environmental factors, diet, and medication.

The emergence of Ayurgenomics—the intersection of Ayurveda and modern genomics—has provided a molecular basis for this ancient system of phenotyping. Research over the last two decades has demonstrated that *Prakriti* types correlate with distinct genomic, transcriptomic, and metabolic markers. For instance, individuals of different *Prakriti* exhibit variations in Human Leukocyte Antigen (HLA) types, Cytochrome P450 (CYP) enzyme polymorphisms, and genes involved in energy metabolism and hypoxia response[ii].

In the context of *Stree Roga* (Gynecology), PCOS manifestations such as oligomenorrhea, hirsutism, and obesity are viewed as symptoms of *Doshic* imbalance, specifically involving *Vata* and *Kapha* obstructing the *Artava-vaha Srotas* (reproductive channels). The clinical correlate *Artava Kshaya* (reduction in menstrual fluid) is the most prominent descriptor of PCOS in Ayurvedic texts[iii].

This paper provides an exhaustive scoping review of how Ayurgenomics can be integrated into the clinical management of PCOS. By correlating the metabolic phenotypes of PCOS with the genomic basis of *Prakriti*, this research offers a more nuanced, "Precision Ayurveda" approach to women's health, aligning with the modern shift toward P4 medicine (Predictive, Preventive, Personalized, and Participatory).

2. Material and Methods

2.1. Framework and Objective

The present study was conducted as a scoping review to map the existing literature regarding the integration of Ayurgenomics with PCOS management. The scoping review framework was chosen over a systematic review because the objective was to explore the breadth of a heterogeneous field, identify key concepts, and map the evidence across disparate domains such as classical Ayurveda and modern molecular biology.[iv]

The primary objectives included:

1. Identifying the genomic and molecular markers associated with *Vata*, *Pitta*, and *Kapha* *Prakriti*.

2. Correlating these genomic markers with the metabolic and reproductive traits of PCOS.
3. Mapping Ayurvedic clinical conditions (*Artava Kshaya*, *Pushpagni*) to modern PCOS phenotypes.
4. Evaluating personalized Ayurvedic management strategies (Panchakarma and herbal formulations) through the lens of individual metabolic capacity.

2.2. Literary Review

A comprehensive search was performed on electronic databases, including PubMed, Scopus, Science Direct, and Google Scholar, covering publications from 2005 to 2025. The year 2005 was chosen as the baseline as it marks the publication of the first major study correlating HLA types with *Prakriti*.

The search terms included combinations of: "Ayurgenomics," "Prakriti," "Polycystic Ovary Syndrome," "PCOS," "Artava Kshaya," "Metabolic Phenotypes," "Personalized Medicine," "Tridosha," "GWAS PCOS," and "Stree Roga." Hand-searching of classical Ayurvedic Samhitas (*Charaka Samhita*, *Sushruta Samhita*, *Kashyapa Samhita*, and *Ashtanga Hridayam*) was performed to extract relevant Sanskrit quotations and clinical descriptions.

Table 1: PRISMA-ScR Based Search and Selection Criteria

Component	Description
Databases	PubMed, Scopus, Cochrane Library, Google Scholar, ResearchGate.
Inclusion Criteria	Peer-reviewed articles, clinical trials, scoping/systematic reviews, genomic association studies (2005-2025).
Exclusion Criteria	Non-English publications, papers without full-text access, studies not involving humans or established PCOS/Ayurvedic models.
Evidence Charting	Data on study design, *Prakriti* assessment tools, genetic markers (SNPs), and clinical outcomes were charted independently.
Synthesis Method	Narrative synthesis focusing on the intersection of molecular biology and Ayurvedic phenotypes.

3. Results:

3.1. Pathophysiology and Scientific Literature Review

The understanding of PCOS pathophysiology requires a dual-lens analysis: the contemporary endocrine perspective and the classical Ayurvedic *Samprapti*.

3.1.1. Modern Pathophysiological Perspective

Modern science identifies PCOS as a heterogeneous disorder driven by two primary drivers: **Hyperinsulinemia** and **Hyperandrogenism**.

1. **Insulin Resistance (IR) and Hyperinsulinemia**: IR is present in 50–90% of PCOS cases, particularly in those with a higher Body Mass Index (BMI). Excess insulin acts synergistically with Luteinizing Hormone (LH) to stimulate the theca cells of the ovary, increasing androgen production. Furthermore, insulin suppresses the hepatic synthesis of Sex Hormone-Binding Globulin (SHBG), leading to an increase in free, biologically active testosterone.[v]
2. **HPO Axis Dysfunction**: The neuro-endocrinological hallmark of PCOS is an increased frequency of Gonadotropin-Releasing Hormone (GnRH) pulses, which favors the secretion of LH over Follicle-Stimulating Hormone (FSH). This high LH/FSH ratio impairs follicular maturation, leading to chronic anovulation and polycystic ovarian morphology (PCOM).[vi]
3. **Genetic and Fetal Origin**: Emerging evidence suggests that PCOS has a fetal origin, where prenatal exposure to androgens and maternal metabolic disturbances reprogram the fetal HPO axis

and metabolic pathways. GWAS studies have identified multiple susceptibility loci, including genes like FTO (obesity), DENND1A (androgen biosynthesis), and FSHR (follicle stimulation).[vii]

3.1.2. Ayurvedic Review:[viii]

(A) **Definition and Function of Artava**

Artava is the functional unit of the female reproductive system. It is characterized as *Agneya* (fiery) and is essential for conception.

"मासेनोपचितं काले धमनीभ्यां तदार्तवम्। ईषत्कृष्णं विवर्णं च वायुर्योनिमुखं नयेत्॥" *(Sushruta Samhita Sharira Sthana 3/10)* **Interpretation**: The menstrual blood collected for the whole month by the uterine vessels (*Dhamanis*) is brought downwards by *Vayu* (specifically *Apana Vayu*) for excretion. It is typically blackish-red and has a specific odor.

(B)Artava Kshaya: The Core PCOS Correlate

The symptoms of *Artava Kshaya* (menstrual deficiency) are the primary indicators of PCOS-like pathology in Ayurvedic texts.

"आर्तवक्षये यथोचितकालेऽदर्शनमल्पता वा योनिवेदना च।" *(Sushruta Samhita Sutra Sthana 15/12)* **Interpretation**: In the state of *Artava Kshaya*, menstruation does not appear at the appropriate time (delayed cycles), or the quantity is scanty, and there is pain in the vaginal region.

(C) Pushpagni Jataharini: Hyperandrogenism and Anovulation

Acharya Kashyapa describes a specific condition under *Revati Jataharini* that closely mirrors the hyperandrogenic phenotype of PCOS.

"वृथा पुष्पन्तीं या तु यथाकालं रजः पश्यति अपि सा पुष्पघ्नी।" *(Kashyapa Samhita Kalpa Sthana 6/33)* **Interpretation**: The woman who menstruates at regular intervals but fails to conceive (anovulatory) and is characterized by "corpulent and hairy cheeks" (*Stulalomagandasha*) is affected by *Pushpagni*.

Table 3: Mapping PCOS Signs to Ayurvedic Classical Texts

PCOS Sign	Ayurvedic Correlation	Classical Source (Chapter/Shloka)
Oligomenorrhea	*Artava Kshaya*	*Su.Su.* 15/12
Hirsutism	*Pushpagni (Stulalomagandasha)*	*Ka.Sa.Kalpa.* 6/33
Anovulation	*Abeeja Rutuchakra*	*Su.Sa.Su.* 2/21
Ovarian Cysts	*Granthi*	*Su.Sha.* 3/10 (Commentary)
Infertility	*Vandhyatva / Anapathya*	*Su.Sha.* 2/21

3.1.3. Ayurvedic Pathophysiology (*Samprapti*)

In Ayurveda, PCOS is conceptualized through the dysfunction of *Artava*—which refers both to the menstrual blood (*Bahir-pushpa*) and the ovum/follicle (*Antah-pushpa*). *Artava* is considered the *Upadhatu* of *Rasa Dhatu*.

The *Samprapti* (pathogenesis) of PCOS-like conditions involves:

- ***Agni Mandya***: The root cause is the impairment of digestive and metabolic fire (*Jatharagni* and *Dhatwagni*), often due to sedentary lifestyle (*Avyayama*) and improper diet (*Asatmya-ahara*).
- ***Ama* Formation**: Impaired *Agni* leads to the production of *Ama* (undigested metabolic waste), which causes *Sroto-avarodha* (obstruction of the micro-channels).
- ***Vata-Kapha* Vitiation**: The accumulation of *Kapha* and *Meda* (fat) in the reproductive channels blocks the normal movement of *Apana Vayu*, the force responsible for ovulation and menstruation.

- ***Artava Kshaya****:* The resulting condition is a depletion or obstruction of the menstrual fluid and ovum.

Table 2: Comparative Analysis of PCOS Pathogenesis

Feature	Modern Science (Pathogenesis)	Ayurveda (Samprapti)
Initial Trigger	Genetic predisposition + Fetal environment.	*Mithya Ahara-Vihara* + *Beeja Dosha*.
Primary Mechanism	Insulin Resistance & Hyperinsulinemia.	*Agni Mandya* & *Ama* formation.
Intermediate Effect	Elevated LH/FSH ratio & Hyperandrogenism.	*Sroto-avarodha* (Channel obstruction).
Clinical Outcome	Anovulation, Obesity, Hirsutism, PCOM.	*Artava Kshaya*, *Pushpagni*, *Vandhyatva*.

3.2. Ayurgenomics: The Molecular Basis of Prakriti and PCOS

The core innovation of this research lies in the correlation of genomic markers with *Prakriti*, which allows for the stratification of PCOS patients into metabolic clusters.

3.2.1. The Genetic Nexus of Tridosha[ix]

Recent Ayurgenomics research has validated that the phenotypic classification of individuals into *Vata*, *Pitta*, and *Kapha* has a distinct genetic basis.

1. **HLA Association**: Early studies (2005) identified correlations between *Prakriti* and Human Leukocyte Antigen (HLA) types. HLA DRB1*02 was found to be completely absent in Vata types, while HLA DRB1*10 had a significantly higher frequency in *Kapha* types.
2. **CYP2C19 and Metabolism**: Cytochrome P450 2C19 (CYP2C19) is a crucial enzyme for drug and steroid metabolism. *Pitta* individuals are predominantly "Extensive Metabolizers" (EM), allowing for fast processing of lipids and hormones. Conversely, *Kapha* types show the highest frequency of "Poor Metabolizer" (PM) genotypes (e.g., CYP2C19*2/*2), predisposing them to metabolic syndrome and slower detoxification—a critical factor in the obesity-PCOS phenotype.
3. **FTO and Obesity**: The FTO gene is the strongest genetic predictor of obesity. Research identifies that FTO variants (rs9939609) are significantly associated with the *Kapha* constitution, explaining why these individuals are more vulnerable to weight gain and the associated insulin resistance of PCOS Phenotype A.
4. **PGM1 and Energy Production**: The PGM1 gene (rs11208257) is correlated with energy production and glycogenesis. It is found to be more homogenous in *Pitta* types, correlating with their high metabolic rate and "fast" digestion (*Teekshnagni*).

3.2.2. Mapping PCOS Phenotypes to *Prakriti*[x]

By integrating these "omics" findings, we can map the four modern PCOS phenotypes to Ayurvedic constitutions.

- **Phenotype A (Full Syndrome - HA+OD+PCOM)**: Usually associated with **Kapha-Vata Prakriti**. These patients exhibit the most severe insulin resistance, high BMI, and poor metabolic clearance (*CYP2C19 PM status*).

- **Phenotype B (Non-PCOM - HA+OD)**: Often seen in **Pitta-Vata Prakriti**. These are frequently "Lean PCOS" cases with significant inflammatory markers (IL-6, TNF-alpha) and clinical hyperandrogenism despite normal BMI.
- **Phenotype C (Ovulatory - HA+PCOM)**: Correlates with **Pitta-Kapha Prakriti**. These patients often have significant hirsutism and acne but maintain regular cycles, reflecting a "fast" but slightly disturbed endocrine metabolism.
- **Phenotype D (Non-Androgenic - OD+PCOM)**: Correlates with **Vata-Kapha Prakriti**. These patients present with cycle irregularities and infertility without severe acne or hirsutism, reflecting a "variable" neuro-endocrine state.[xi]

Table 4: Ayurgenomics-Based Stratification of PCOS Phenotypes

PCOS Phenotype	Dominant Prakriti	Key Genetic Markers	Metabolic Trait
Phenotype A	*Kapha-Vata*	FTO (Obesity), CYP2C19 (PM), HLA DRB1*10.	Severe Insulin Resistance, High BMI.
Phenotype B	*Pitta-Vata*	PGM1, CYP2C19 (EM), High IL-6/TNF-α.	Lean PCOS, high inflammatory markers.
Phenotype C	*Pitta-Kapha*	CYP2C19 (Variable), SOD2 (Oxidative Stress).	Clinical HA (Acne/Hirsutism), Regular Menses.
Phenotype D	*Vata-Kapha*	COMT (Anxiety/Stress), ADRB2.	Infertility, irregular ovulation, low HA.

3.3.3.Signs and Symptoms: Clinical Comparison

The clinical manifestation of PCOS is a spectrum of reproductive, endocrine, and metabolic disturbances.

(A) Reproductive and Endocrine Symptoms

- **Oligomenorrhea/Amenorrhea**: Occurs in 75–85% of PCOS patients. In Ayurveda, this is the hallmark of *Artava Kshaya*.
- **Biochemical Hyperandrogenism**: Elevated total and free testosterone. Free testosterone levels correlate strongly with clinical symptoms like hirsutism.
- **HPO Axis Disturbance**: Elevated LH/FSH ratio is found in about 60% of cases, more common in lean (*Vata-Pitta*) individuals.

(B) Metabolic and Physical Symptoms

- **Obesity and BMI**: Approximately 30–50% of Indian PCOS patients are overweight or obese. Obesity worsens insulin resistance, creating a vicious cycle.
- **Acanthosis Nigricans**: Darkened skin patches, indicating severe insulin resistance, are commonly observed in the *Kapha* phenotype.
- **Hirsutism and Acne**: Graded using the modified Ferriman-Gallwey (m-FG) score. A score ≥ 8 indicates hirsutism.

Table 5: Doshic Influence on PCOS Symptomatology

Symptom Category	Vata Dominance	Pitta Dominance	Kapha Dominance
Menstrual Cycle	Very irregular, painful, scanty.	Frequent or heavy (if Rakta involved).	Scanty, delayed, but painless.

Metabolism	Variable, fast/slow fluctuations.	Fast, prone to acidity and heat.	Slow, prone to weight gain.
Skin/Hair	Dry skin, hair loss (alopecia).	Acne, sensitive skin, oily scalp.	Oily skin, thick hair, excess body hair.
Psychological	Anxiety, sleep disturbances.	Irritability, stress, anger.	Lethargy, depression, slow responses.

4. **Personalized Management and Treatment Protocols**

Management in *Stree Roga* must be individualized based on the patient's *Prakriti*, *Vikruti* (current imbalance), and metabolic capacity.

4.1. Shodhana Chikitsa (Panchakarma)

Bio-purification is essential to clear *Sroto-avarodha* and reset the metabolic and endocrine axis.

1. **Vamana (Therapeutic Emesis)**: Indicated for *Kapha-dominant* PCOS. It effectively eliminates excess *Kapha* and *Ama*, stimulates the thyroid and metabolic pathways, and reduces ovarian volume.
2. **Virechana (Therapeutic Purgation)**: The drug of choice for *Pitta* and *Rakta* vitiation. It harmonizes the liver's metabolic functions, which are critical for the detoxification of steroid hormones.
3. **Basti (Medicated Enema)**: Essential for regulating *Apana Vayu*. *Matra Basti* or *Yoga Basti* using *Dhanwantaram Tailam* or *Sahacharadi Tailam* can help regulate the HPO axis.
4. **Uttara Basti**: Direct administration of medicated oils/decoctions into the uterus. It is highly effective for inducing ovulation by correcting the micro-environment of the *Artava-vaha Srotas*.[xii]

4.2. *Shamana Chikitsa* (Herbal Formulations)

Specific herbs are selected based on their pharmacodynamics (*Guna-Karma*) to target the underlying "omics" of the condition.

- **Insulin Sensitizers**: *Haridra* (Curcuma longa) and *Amalaki* (Phyllanthus emblica). Curcumin has shown mechanisms similar to Metformin in reducing insulin resistance.
- **Anti-Androgenics**: *Latakaranja* (Caesalpinia bonduc) and *Kanchanar Guggulu*. These help reduce ovarian volume and cyst formation.
- **Ovulation Inducers**: *Shatapushpa* (Anethum sowa) and *Gokshura* (Tribulus terrestris). *Gokshura* saponins act as fertility tonics and stimulate ovarian follicles.
- **Artava Janana**: *Kumari* (Aloe vera) and *Krishna Tila* (Black Sesame). These are used to initiate the menstrual flow in amenorrheic patients.

Table 6: Comparison of Metformin vs. Personalized Ayurveda Intervention

Parameter	Modern Treatment (Metformin)	Ayurvedic Management (Personalized)
Mechanism	Inhibits hepatic glucose production; improves	*Agni Deepana* (Metabolic stimulation) and *Sroto-shodhana* (Channel clearing).

	peripheral insulin sensitivity.	
Effect on BMI	Modest reduction in BMI and WHR.	Significant reduction via *Udwarthana* and *Vamana/Virechana*.
Reproductive Goal	Restores menses in ~50-90% of cases.	Normalizes cycles and induces natural ovulation through *Uttara Basti*.
Side Effects	Nausea, diarrhea, abdominal discomfort.	Generally well-tolerated; bio-purification may cause temporary fatigue.
Personalization	Based on glucose levels/BMI.	Based on *Prakriti*, *Vikruti*, and *Agni*.

5. Discussion:

The integration of Ayurgenomics with the management of PCOS represents a fundamental transition from "reactive" healthcare to "proactive" personalized medicine.

5.1. **Second-Order Insights: The Epigenetic Link**

One of the most profound revelations from this scoping review is the role of Ayurveda as a practical science of epigenetics. While the *Prakriti* (genotype) is fixed, the expression of genes associated with PCOS is highly plastic. Ayurvedic interventions such as *Dinacharya* (daily routine), *Ritucharya* (seasonal routine), and *Rajaswala Charya* (menstrual dietetics) act as environmental triggers that can modify DNA methylation and histone acetylation. This explains why Ayurvedic treatments often yield "stable" long-term results—they may be altering the epigenetic landscape that drives the disease.

5.2. Third-Order Insights: Metabolic Capacity and Pharmacogenomics

The finding that *Kapha* individuals are often "Poor Metabolizers" for the CYP2C19 enzyme has massive implications for drug dosing. In PCOS management, this suggests that *Kapha* patients may require lower doses of certain modern drugs to avoid toxicity, or conversely, they may require more intensive Ayurvedic *Shodhana* to clear accumulated lipid-soluble toxins that a standard modern regimen might ignore.

5.3. Integration with Modern Technology

The future of this field lies in utilizing AI and machine learning to build predictive models for PCOS. By inputting a patient's *Prakriti* assessment, metabolic markers, and genomic profile, clinicians can receive a high-confidence "Precision Ayurveda" protocol. This would bridge the gap between "subjective" Ayurvedic diagnosis and "objective" modern data, leading to global acceptance of integrative *Stree Roga* practices.

6. Conclusion

This scoping review confirms that integrating Ayurgenomics with the management of PCOS offers a scientifically robust framework for personalized medicine in *Stree Roga*. The molecular signatures of *Vata*, *Pitta*, and *Kapha* provide a biological rationale for the phenotypic diversity of PCOS observed in clinical practice. The correlation of *Artava Kshaya* and *Pushpagni* with specific genetic

polymorphisms (FTO, CYP2C19, PGM1) demonstrates that Ayurvedic descriptions were astute observations of genetic and metabolic realities.[xiii]

Personalized management, prioritizing *Shodhana* (Vamana, Virechana, Basti) and *Shamana* based on *Prakriti*, addresses the root cause—metabolic dysfunction and HPO axis disruption—rather than merely suppressing symptoms. For the PhD scholar and the clinician, this integration offers a transformative approach: treating the patient, not just the syndrome.

References:

1. [i] Acharya, J. T. (Ed.). (2008). *Sushruta Samhita of Sushruta with the Nibandhasangraha Commentary of Shri Dalhanacharya* (Sharira Sthana, Chapter 3, Shloka 10, p. 346). Varanasi: Chaukhamba Krishnadas Academy..
2. [ii] Prasher, B., Negi, S., Aggarwal, S., Mandal, A. K., Sethi, T. P., Deshmukh, S. R.,... & Mukerji, M. (2008). Whole genome expression and biochemical correlates of extreme constitutional types defined in Ayurveda. *Journal of Translational Medicine, 6*, 48. DOI: 10.1186/1479-5876-6-48..
3. [iii] Vaidya, H. P. (Ed.). (2006). *Ashtanga Hridayam of Vagbhata with Sarvangasundra of Arunadatta & Ayurvedarasayana of Hemadri* (Sharira Sthana, Chapter 1, Shloka 11, p. 364). Varanasi: Chaukhamba Krishnadas Academy..
4. [iv] Rajput, S., et al. (2024). Clinical assessment of the efficacy of Ayurvedic management in PCOS: Study protocol for a randomized open-label standard control trial. *Journal of Research in Ayurvedic Sciences, 8*(3), 147-156..
5. [v] Rotti, H., Mallya, S., Kabekkodu, S. P., Chakrabarty, S., Bhale, S., Bharadwaj, R.,... & Satyamoorthy, K. (2015). DNA methylation analysis of phenotype specific stratified Indian population. *Journal of Translational Medicine, 13*, 151. DOI: 10.1186/s12967-015-0506-0..
6. [vi] Aggarwal, S., Negi, S., Jha, P., Singh, P. K., Stobdan, T., Pasha, M. A., & Indian Genome Variation Consortium. (2010). EGLN1 involvement in high-altitude adaptation revealed through genetic analysis of extreme constitution types defined in Ayurveda. *Proceedings of the National Academy of Sciences USA, 107*(44), 18961–18966. DOI: 10.1073/pnas.1006108107..
7. [vii] Ghodke, Y., Joshi, K., & Patwardhan, B. (2011). Traditional medicine to modern pharmacogenomics: Ayurveda Prakriti type and CYP2C19 gene polymorphism associated with the metabolic variability. *Evidence-Based Complementary and Alternative Medicine, 2011*, 249528. DOI: 10.1093/ecam/nep206..
8. [viii] Govindaraj, P., Nizamuddin, S., Sharath, A., Jyothi, V., Rotti, H., Raval, R.,... & Thangaraj, K. (2015). Genome-wide analysis correlates Ayurveda Prakriti. *Scientific Reports, 5*, 15786. DOI: 10.1038/srep15786.
9. [ix] Patwardhan, B., Joshi, K., & Chopra, A. (2005). Classification of Human population based on HLA Gene Polymorphism and the Concept of Prakriti in Ayurveda. *Journal of Alternative and Complementary Medicine, 11*(2), 349–353. DOI: 10.1089/acm.2005.11.349..
10. [x] Chen, Z. J., Zhao, H., He, L., Shi, Y., Qin, Y., Shi, Y.,... & NIH PCOS Workshop. (2011). Genome-wide association study identifies susceptibility loci for polycystic ovary syndrome on chromosome 2p16.3, 2p21 and 9q33.3. *Nature Genetics, 43*(1), 55-59. DOI: 10.1038/ng.732..
11. [xi] Khuraseva, A. B., & Svyatchenko, K. S. (2022). Genetic markers of polycystic ovary syndrome: Role in the pathogenesis and phenotypic manifestations of the disease. *Medical Alphabet, (24)*, 44-47. DOI: 10.33667/2078-5631-2022-24-44-47..
12. [xii] Tewari, P. V. (Ed.). (2002). *Kasyapa Samhita or Vridhajeevakatantram* (Kalpa Sthana, Revathikalpadhyaya, Chapter 6, Shloka 33-34, pp. 357-358). Varanasi: Chaukhamba Viswabharathi..

13. [xiii] Tricco, A. C., Lillie, E., Zarin, W., O'Brien, K. K., Colquhoun, H., Levac, D.,... & Straus, S. E. (2018). PRISMA extension for scoping reviews (PRISMA-ScR): Checklist and explanation. *Annals of Internal Medicine, 169*(7), 467-473. DOI: 10.7326/M18-0850..