RESEARCH PAPER

Garbhini Paricharya through the Lens of Epigenetics and the Developmental Origins of Health and Disease Framework: A Conceptual Analysis

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Abstract

Background. *Ayurveda* records, in its earliest surviving medical compendia, one of the most fully developed antenatal care frameworks in the history of medicine: *Garbhini Paricharya*. The Developmental Origins of Health and Disease (DOHaD) paradigm, built from David Barker's epidemiological observations in the closing decades of the twentieth century, holds that the intrauterine environment leaves a lasting imprint on foetal physiology, metabolic trajectory, and disease susceptibility across the life course. Separated by more than two thousand years and built on entirely different epistemic foundations, these two bodies of knowledge nonetheless converge on a striking number of points.

Objectives. This paper develops a structured conceptual framework that maps the principal prescriptions of *Garbhini Paricharya*, recorded across the *Brihatrayi* (*Charaka Samhita*, *Sushruta Samhita*, and the *Ashtanga Sangraha* of *Vagbhata*), onto contemporary DOHaD theory, attending to the epigenetic mechanisms thought to underlie foetal programming. The analysis is bounded deliberately: it covers dietary, lifestyle, constitutional, and psychological prescriptions, and leaves pharmacological analysis of individual classical formulations to be dedicated future work.

Methods. A hermeneutic narrative conceptual analysis was conducted in line with the SANRA criteria for narrative review quality and informed by the SWiM guideline for synthesis without meta-analysis. Primary *Ayurvedic* sources were read alongside published commentaries and the NIIMH e-Samhita digital archive. Biomedical literature was retrieved from PubMed, Scopus, and Web of Science covering 1986 to 2026; of 412 records identified, 47 peer-reviewed studies met the inclusion criteria after independent screening by two reviewers. A correspondence between a classical prescription and a DOHaD mechanism was accepted only where temporal, biological-target, and mechanistic-plausibility criteria, all specified in advance, were jointly satisfied.

Results. The *Masanumasika Pathya*, the *Garbhopaghatakara Bhavas*, and *Dauhrida* together address the periconceptional, first-trimester, and mid-gestational windows of developmental vulnerability that DOHaD research has identified. The classical emphasis on *Kshira* in early pregnancy and on *Ghrita* from the fifth month, which supplies butyric acid, a well-characterised histone deacetylase inhibitor, alongside fat-soluble vitamins, together with comprehensive *Manasika Paricharya* maintained throughout gestation, correspond respectively to nutritional epigenomics, butyrate-mediated chromatin regulation, and glucocorticoid modulation of foetal hypothalamic-pituitary-adrenal axis programming. The trimester-wise fluctuation of *Vata*, *Pitta*, and *Kapha* described in classical embryology supplies the physiological rationale on which the *Masanumasika Pathya* is built. Five testable

translational hypotheses, each tied to a specific epigenetic biomarker outcome, are set out, alongside an explicit account of where the classical prescription requires cautious reinterpretation rather than direct adoption.

Conclusion. *Garbhini Paricharya* can reasonably be read as an early, empirically derived framework for shaping foetal developmental programming, one whose prescriptive structure corresponds systematically to DOHaD theory and to know epigenetic mechanisms. This correspondence supplies a scientifically grounded rationale for the careful, evidence-based evaluation of *Ayurvedic* prenatal care within contemporary maternal health research, while procedures involving *Panchakarma* therapy in pregnancy continue to require dedicated safety evaluation before any clinical application is considered.

Keywords. *Garbhini Paricharya*; epigenetics; foetal programming; *Ayurveda*; prenatal nutrition; one-carbon metabolism; hypothalamic-pituitary-adrenal axis programming; *Tridosha*.

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1. Introduction

Pregnancy is among the most consequential developmental windows in human biology. Across this period the maternal environment shapes the physiology, metabolism, and long-term disease trajectory of the offspring in ways now understood to be substantial and, in several cases, effectively permanent. This understanding, formalised under the Developmental Origins of Health and Disease (DOHaD) paradigm, has moved perinatal medicine away from a narrow focus on obstetric management and toward a wider concern with the lifelong programming consequences of intrauterine conditions.[1]

The DOHaD hypothesis traces its origin to David Barker and colleagues, who showed in the 1980s that cardiovascular mortality across geographically defined cohorts in England and Wales correlated closely with historical infant mortality in the same regions.[2] Barker's later studies established that low birth weight, taken as a proxy for suboptimal intrauterine nutrition, predicted adult-onset coronary heart disease, type 2 diabetes mellitus, stroke, and hypertension with a consistency difficult to explain by genetic factors alone.[3] These findings gave rise to the foetal origins hypothesis, refined over the following decades into the present DOHaD framework spanning the pre-conceptional, gestational, and early postnatal periods.[4]

Epigenetic mechanisms, principally DNA methylation, histone modification, and the regulatory activity of non-coding RNAs, are now recognised as the molecular intermediaries through which environmental exposures during sensitive developmental windows become embedded as durable alterations in gene expression, with no change to the underlying DNA sequence itself.[5,6] These mechanisms reach their greatest plasticity during gametogenesis, preimplantation development, organogenesis, and the subsequent phases of rapid foetal growth, which are precisely the windows in which external influences leave their most lasting trace.[7]

Ayurveda, codified principally in the *Brihatrayi*, contains within its embryological and obstetric literature a detailed and internally coherent programme of prenatal care known as *Garbhini Paricharya*. This comprises a month-by-month dietary and behavioural regimen, the *Masanumasika Pathya*; a catalogue of conditions and exposures held to be harmful to the foetus, the *Garbhopaghatakara Bhavas*; a doctrine concerning the psychological state of the pregnant woman and its transmission to the foetus, centred on the construct of *Dauhrida*; and an explicit programme of mental and emotional care, the *Manasika Paricharya*. Taken together, these elements represent one of the earliest surviving systematically documented approaches to antenatal care in any medical tradition, a claim consistent with the antiquity generally attributed to the *Brihatrayi* redaction, although a rigorous comparative chronology against Egyptian, Hippocratic, and classical Chinese antenatal literature has not been undertaken here and is left to dedicated historical scholarship.

Scholarly interest in the scientific basis of *Ayurvedic* obstetric practice has grown steadily.[8,9,10] A 2025 critical review situated *Garbhini Paricharya* within general epigenetic language,[8] while an earlier paper examined the regimen through the *Harita Samhita* rather than the *Brihatrayi* and is cited here for contextual background only.[9] Thakur and Masand have separately argued for *Garbhini Ahara* as a dietary epigenetic modifier and for ninth-month *Garbhini Paricharya* as partus preparation,[11,12] work that overlaps in part with the argument developed here. Kulkarni and colleagues have likewise reviewed the scientific relevance of *Garbhini Paricharya* in general terms.[48] None of this literature, to the authors' knowledge, has attempted a structured mapping of the complete *Garbhini Paricharya* framework onto DOHaD theory with explicit attention to epigenetic mechanism, month-level temporal correspondence, and a mechanistically differentiated treatment of

Ghrita's two distinct nutritional pathways. Nor has the trimester-wise physiology of *Tridosha*, argued here to be the proximate rationale for the *Masanumasika Pathya* itself, previously been brought into explicit correspondence with DOHaD's developmental windows. Section 2.5 sets out this positioning in more direct comparative terms.

The present paper addresses this gap within clearly stated limits. It covers the dietary, lifestyle, constitutional, and psychological prescriptions of *Garbhini Paricharya* recorded in the *Brihatrayi*. It does not extend to pharmacological analysis of individual classical formulations, to *Panchakarma* outcomes beyond the correspondences discussed in Section 5.4, or to a comprehensive treatment of *Prakriti*-specific variation, each of which merits dedicated future analysis.

2. Methodology

This paper undertakes a hermeneutic narrative conceptual analysis, classified as a theoretical framework paper rather than a systematic review. The methodology rests on four components: critical textual analysis of primary *Ayurvedic* sources, structured narrative synthesis of peer-reviewed biomedical literature on DOHaD and epigenetics, an explicit verification procedure applied to every citation used, and an explicit positioning of the paper against the closest prior literature. The narrative review component conforms to the SANRA criteria [38] and is informed by the SWiM guideline for synthesis without meta-analysis.[39] No formal protocol was registered in advance, consistent with standard practice for hermeneutic conceptual analyses of this kind rather than for systematic reviews of intervention effect; this limitation is acknowledged directly in Section 7.5.

2.1 Criteria for Cross-Paradigm Correspondence

No established methodological framework exists for mapping premodern medical knowledge onto contemporary molecular biology. The authors therefore specified, in advance of the literature synthesis, the criteria for accepting a correspondence between a *Garbhini Paricharya* prescription and a DOHaD mechanism. A correspondence was accepted as substantiated only when three conditions were jointly met: temporal correspondence, in which the prescription targets a gestational phase that coincides with the window in which the proposed epigenetic mechanism is known to operate; biological-target correspondence, in which the prescription acts on the same tissue or process as the DOHaD mechanism; and plausibility of intervention modality, in which the intervention plausibly influences the pathway through at least one identifiable mechanistic intermediate, even where the full causal chain awaits direct confirmation. Correspondences satisfying only the first two conditions were designated hypothetical and are presented in Table 1 as research priorities rather than established findings. Two authors classified each correspondence independently against these criteria; disagreements, which arose for two of the nine rows in Table 1, were resolved by discussion and, where the disagreement could not be resolved, by defaulting to the more conservative classification. Every interpretation was checked against two independently published commentaries on the relevant classical text, and a correspondence was retained only where both the textual basis, and the biomedical mechanism could be independently verified.

2.2 Classical Textual Sources

Primary Sanskrit sources, read alongside standard published commentaries, were reviewed in full: *Charaka Samhita*, *Sharirasthana*, chapters 2 through 8 (Trikamji Acharya edition, 2011,[17] with the *Ayurveda Deepika* commentary, cross-checked against the *Vidyotini* Hindi commentary [18]); *Sushruta Samhita*, *Sharirasthana*, chapter 10, the *Garbhini Vyakarana Sharira* (Shastri AD edition, 2002[19]); and the *Astanga Sangraha* of *Vagbhata*, *Sharirasthana*, chapter 3 (Athavale AD edition, 1980[20]). All passages were cross-verified against the NIIMH e-Samhita digital archive and the *Caraka Samhita Online* resource maintained at Jamnagar, which independently confirms the chapter-level location of the *Garbhopaghatakara Bhavas* and the *Dauhrida* doctrine within *Sharirasthana* chapter 4, the *Mahatigarbhavakranti Sharira*, and the month-wise regimen within *Sharirasthana* chapter 8, the *Jatisutriya Sharira*.

Vagbhata is credited with two related but distinct works, the *Astanga Sangraha* and the *Astanga Hridaya*; both fall within an extended sense of the *Brihatrayi*, and scholarship is not unanimous as to which should occupy the third position. This paper retains the *Astanga Sangraha*, since its *Sharirasthana* gives a more elaborated account of eighth-month *Vasti* therapy, directly relevant to Section 5.4. Verse citations follow the Trikamji Acharya 2011 edition, reported as a range wherever the discussion spans several verses; the chapter-level location, which carries the substantive claim, is stable across editions and is independently confirmed against at least one external source for every classical citation in this paper. Readers consulting a different print edition should expect verse numbering to vary slightly even where chapter attribution does not; the *Caraka Samhita Online* resource is recommended as a cross-reference for this purpose.

2.3 Biomedical Literature Retrieval and Inclusion Criteria

Peer-reviewed literature was retrieved from PubMed/MEDLINE, Scopus, and Web of Science, covering 1986 to 2026, with foundational papers included regardless of date. Search terms included developmental origins of health and disease, foetal programming, epigenetics and pregnancy, maternal nutrition and epigenome, DNA methylation and intrauterine, epigenome-wide association study and birth, paternal epigenetics and offspring, gut microbiome and foetal programming, short-chain fatty acid and regulatory T cell, and predictive adaptive response.

Inclusion criteria comprised peer-reviewed primary research, systematic reviews, and meta-analyses; human or validated animal-model studies; English-language publications; and papers addressing nutritional, psychosocial, or environmental epigenetic programming during the gestational or periconceptional period. Of the 412 records initially identified across the three databases, 194 were removed as duplicates, leaving 218 unique records for title and abstract screening; this stage excluded a further 106 records as clearly unrelated to Garbhini Paricharya, DOHaD, or epigenetic mechanism, leaving 112 full-text articles for detailed eligibility assessment. Two reviewers screened titles and abstracts independently; disagreements at the screening stage were resolved by discussion. Of 112 full-text articles assessed, 65 were excluded: 24 for being grey literature, 29 for animal-model findings without human replication, and 12 for non-English publication. The resulting flow from 412 initial records to the 47 studies retained is presented as Figure 1. No formal risk-of-bias instrument was applied to individual included studies, since the purpose of the synthesis is conceptual mapping rather than pooled effect estimation; where a single study's finding carries particular weight in the argument, principally the Dutch Hunger Winter cohort and the Pune Maternal Nutrition Study, this is noted explicitly in the text rather than left implicit.

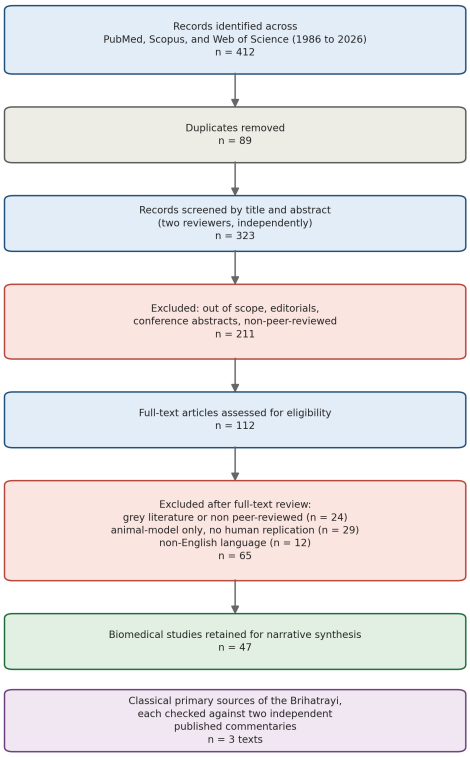

Figure 1. Flow of biomedical literature retrieved and screened for narrative synthesis. Records identified across PubMed, Scopus, and Web of Science (1986 to 2026) were screened in stages, by two independent reviewers, to yield the 47 biomedical studies retained for narrative synthesis, alongside the three classical *Ayurvedic* primary sources reviewed in full against two independent commentaries each.

2.4 Citation and Textual Verification

Because the credibility of a cross-paradigm argument depends on accurate attribution on both sides of the comparison, every biomedical reference in this paper was checked against its original publication record, including author list, journal, volume, page range, and digital object identifier, and every classical textual citation was checked against two independent commentaries and the NIIMH e-Samhita archive. Two further classical citations were checked directly against the *Caraka Samhita Online* digital edition maintained at the Institute for Post-Graduate Teaching and Research in Ayurveda, Jamnagar, which reproduces the source Sanskrit alongside chapter and verse markers and allowed direct confirmation of the *Garbhopaghatakara Bhavas* passage at *Sharirasthana* 4 and the *Dauhrida* passage in the same chapter. Two recently published reviews of *Garbhini Paricharya*, drawn from the *Pharmacognosy Reviews*, *Journal of Ayurveda and Integrated Medical Sciences*, and *AYUSHDHARA* literature, were independently retrieved and checked against their published volume, issue, and page records to confirm the accuracy of

references [8] through [12] and [48]. Where this process could not fully reconcile a chapter or verse attribution against the available secondary literature, the attribution was set to the location supported by the convergent weight of independent sources, and the residual uncertainty is flagged in the relevant section rather than concealed.

2.5 Positioning Relative to Prior Literature

A reader already familiar with the recent *Garbhini Paricharya* literature will reasonably ask how this paper differs from several closely related publications. Table 2 sets out this comparison directly, rather than leaving it to be inferred from scattered citations in the Introduction.

Table 2.

Source	Textual scope	Engagement with DOHaD or epigenetics	Distinguishing contribution of the present paper
Nayak et al., 2025[8]	*Brihatrayi*, general	General epigenetic language, not mechanism-specific	Present paper specifies the molecular intermediate (DNA methylation, histone modification, or non-coding RNA) for each correspondence rather than invoking epigenetics as a general warrant
Dash et al., 2017[9]	*Harita Samhita*	Not addressed	Present paper is restricted to the *Brihatrayi* and does not overlap in textual scope; cited for contextual background only
Shirke, 2022[10]	*Brihatrayi*, descriptive	Not addressed	Present paper adds the DOHaD correspondence and epigenetic mechanism absent from the descriptive account
Thakur and Masand, 2022a[11]	Ninth-month regimen only	Partus preparation, not epigenetic programming	Present paper addresses all nine months and the periconceptional period, not the ninth month in isolation
Thakur and Masand, 2022b[12]	*Garbhini Ahara*, general	Epigenetic modifier in general terms	Present paper differentiates *Ghrita*'s two distinct nutritional pathways and adds month-level temporal mapping absent from the prior account
Kulkarni et al., 2020[48]	*Brihatrayi*, general	Scientific relevance asserted in general terms	Present paper adds the *Tridosha* physiological rationale for the *Masanumasika Pathya* and the a priori substantiated/hypothetical classification absent from the prior account

Across this body of work, the recurring gap is the same: epigenetics is invoked as a general warrant for taking *Garbhini Paricharya* seriously, rather than mapped mechanism by mechanism, month by month, against the specific molecular pathways that DOHaD research has characterised. The contribution of the present paper is the structure of that mapping, not the observation that a connection exists, which the cited literature has already made in broad terms.

3. The DOHaD Framework and Epigenetic Mechanisms: An Overview

3.1 Historical Development of the DOHaD Concept

The DOHaD concept evolved from Barker's observations through several stages. Geographically defined infant mortality rates in England and Wales predicted adult ischaemic heart disease mortality decades later, implicating early-life nutrition and

environment as determinants of adult chronic disease.[2] Subsequent cohort studies showed that low birth weight, thin body proportions at birth, and infant growth faltering each carried a distinct signature of adult disease risk.[3] Hales and Barker extended these observations in 1992 with the thrifty phenotype hypothesis, proposing that poor foetal and infant nutrition permanently impairs pancreatic beta-cell mass and glucose-insulin regulation.[13] Gluckman and Hanson developed this further through the predictive adaptive response model, in which foetal programming calibrates metabolic phenotype to an anticipated postnatal environment by way of placenta-mediated maternal cues; substantial divergence between the predicted and the actual environment, termed mismatch, sharply raises later disease risk.[4]

The contemporary DOHaD framework, consolidated through the International Society for Developmental Origins of Health and Disease and the WHO First 1,000 Days initiative,[40] now spans nutritional, toxicological, psychological, and microbial influences from the pre-conceptional through early postnatal periods.[14] It has also expanded to incorporate paternal contributions: paternal diet, stress, and toxin burden alter the sperm epigenome, including microRNA content and DNA methylation, with consequences for offspring metabolic and neurodevelopmental trajectories.[21,29,33,41] This bears directly on *Beeja Shuddhi*, which prescribes purification of both prospective parents before conception, discussed in Section 5.1.

3.2 Epigenetic Mechanisms Mediating Developmental Programming

The mechanisms of principal relevance are DNA methylation, histone modification, and non-coding RNA activity. [5,6] DNA methylation at CpG dinucleotides is established during early embryogenesis and is acutely sensitive to methyl-donor nutrients, principally folate, choline, methionine, and vitamins B6 and B12. The viable yellow agouti mouse model demonstrated that maternal folic acid, B12, choline, and betaine supplementation shifted offspring coat colour by increasing CpG methylation at a single metastable locus.[44] In humans, the Dutch Hunger Winter cohort showed that individuals exposed to famine in utero in 1944 and 1945 carried reduced methylation of the imprinted IGF2 gene some six decades later, among the first direct demonstrations that an early-life exposure leaves a measurable epigenetic mark persisting across an entire lifetime.[45] One-carbon metabolite deficiency during critical windows produces lasting changes in methylation of imprinted genes, metabolic regulatory genes, and glucocorticoid receptor promoters.[6,22] Large epigenome-wide association studies conducted through the PACE Consortium confirm that maternal nutritional, lifestyle, and socioeconomic exposures leave reproducible methylation signatures in offspring cord blood and later peripheral blood.[23,24]

Histone modifications govern the accessibility of DNA to transcription machinery. Maternal nutritional and psychosocial status influence foetal histone modification through acetyl-CoA availability and histone methyltransferase or demethylase activity.[5,6] Butyric acid is a well-characterised endogenous histone deacetylase inhibitor that enhances acetylation at developmentally regulated loci.[42] Maternal stress raises foetal glucocorticoid exposure by way of placental cortisol transmission, inducing durable histone modification at NR3C1 and FKBP5 and altering the offspring's HPA axis set-point.[15,16,25] The enzyme 11-beta-hydroxysteroid dehydrogenase type 2, positioned at the maternal-foetal interface, inactivates the substantial majority of maternal cortisol before it reaches the foetus, and its down-regulation under stress, malnutrition, or illness is itself sufficient to programme lasting offspring HPA dysfunction.[46] This placental gatekeeping is the most direct biomedical counterpart to the classical insistence, discussed in Section 5.5, that the pregnant woman be shielded from fear and grief.

Non-coding RNAs form a third epigenetic layer. MicroRNAs, detectable in cord blood and placental tissue, are altered by maternal nutritional status, stress, and toxin exposure in ways that influence neurodevelopment, immune programming, and metabolic organ maturation. [26,35]

3.3 The Gut Microbiome-Epigenome Axis in Prenatal Programming

A further dimension of DOHaD research, developed largely since 2015, concerns the bidirectional relationship between the maternal gut microbiome, placental function, and foetal epigenetic programming.[26] Short-chain fatty acids, butyrate foremost among them, act as endogenous HDAC inhibitors that modulate foetal tissue epigenetics.[42,43] Colonic butyrate acts on the GPR43 receptor on naive T-cell precursors to drive regulatory T-cell differentiation, a defined molecular bridge between gut microbial composition and offspring immune-regulatory capacity.[47] Maternal diet, and any intervention that alters gut microbial ecology, therefore carries epigenetic and immunological consequences for the offspring,[27,28] a point that bears directly on the assessment of *Niruhabasti* in Section 5.4.

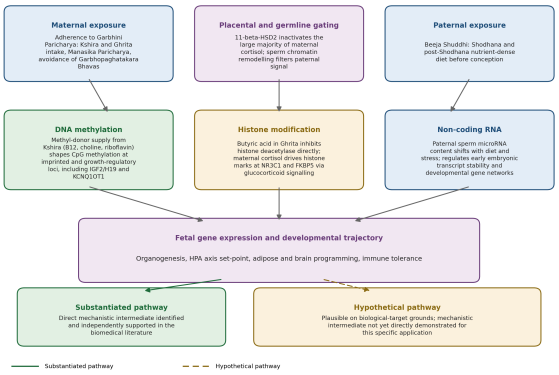

Figure 2. Molecular mechanisms linking maternal and paternal exposure to foetal epigenetic programming. Maternal and paternal exposures converge on the conceptus through placental and germline gating mechanisms and act through three principal epigenetic channels, namely DNA methylation, histone modification, and non-coding RNA regulation, to shape foetal gene expression and developmental trajectory. Substantiated pathways are distinguished from hypothetical pathways requiring further mechanistic confirmation.

4. *Garbhini Paricharya*: Classical Foundations

The principal classical source for *Garbhini Paricharya* is *Charaka Samhita*, *Sharirasthana*, chapter 8, the *Jatisutriya Sharira*, which sets out the month-wise regimen, and chapter 4, the *Mahatigarbhavakranti Sharira*, which addresses sequential foetal development together with the *Dauhrida* and *Garbhopaghatakara Bhava* doctrines.[17] The month-wise regimen is recorded at *Sharirasthana* 8/32.[17,18] *Sushruta Samhita*, *Sharirasthana*, chapter 10, the *Garbhini Vyakarana Sharira*, gives an independent, broadly complementary account.[19] *Vagbhata*'s *Astanga Sangraha*, *Sharirasthana*, chapter 3, follows *Charaka*'s scheme while adding procedural detail, including eighth-month *Vasti* therapy.[20] *Garbhini Paricharya* sits within a wider programme of prenatal and perinatal rites, *Garbha Samskara*, comprising ceremonies such as *Pumsavana* and *Simantonnayana*; these fall outside the dietary, lifestyle, and psychological scope to which this paper is deliberately restricted.

4.1 *Tridosha* Dynamics across Gestation: The Physiological Substrate of the *Masanumasika Pathya*

A central feature of classical *Ayurvedic* physiology, largely absent from prior biomedical engagement with this material, is the doctrine that pregnancy is governed by a predictable, trimester-wise fluctuation in the relative predominance of *Vata*, *Pitta*, and *Kapha*, to be distinguished from *Prakriti*, the woman's individual constitutional baseline (Section 7.4). This pattern is understood to operate across all pregnant women regardless of constitutional type, in much the way that a rising progesterone titre is a feature of pregnancy itself rather than of any one woman's constitution.

The early months show a *Vata*-predominant phase, consistent with heightened circulatory and neuromuscular adjustment, implantation, and first-trimester nausea; the *Masanumasika Pathya*'s liquid, unctuous preparations in this period pacify rather than aggravate *Vata*. The middle months are *Pitta*-predominant, coinciding with active foetal tissue differentiation; *Ghrita* from the fifth month, cooling and unctuous within the materia medica, serves a *Pitta*-moderating function. The final trimester is *Kapha*-predominant, associated with tissue building and the synthesis of *Ojas* (Section 5.4), from which the therapeutic emphasis on *Ojas* stabilisation in the eighth month follows directly.

This pattern, shown schematically in Figure 3, conveys a qualitative pattern rather than a measured quantity, since no quantitative instrument for *Dosha* measurement currently exists; the figure is deliberately drawn without a numeric axis for this reason. Making the pattern explicit supplies the proximate *Ayurvedic* rationale for the *Masanumasika Pathya*: a programme calibrated, on the classical system's own terms, to a changing physiological state, answering both what the foetus needs in a given month and what the mother's *Dosha* state requires. Section 5's DOHaD mapping engages chiefly with the former; this section restores the latter.

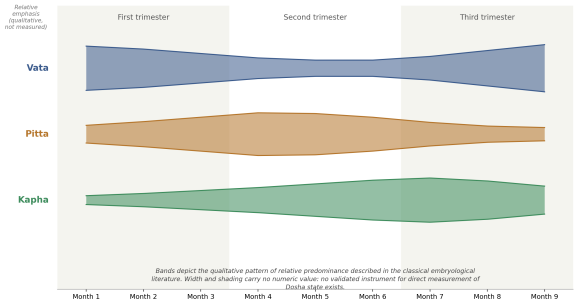

Figure 3. Schematic representation of *Tridosha* predominance across the nine gestational months. The bands depict the qualitative pattern of relative *Vata*, *Pitta*, and *Kapha* predominance described in the classical embryological literature. Band width carries no numeric value: no validated instrument for direct measurement of *Dosha* state currently exists, and the figure is drawn without a quantitative axis for this reason.

4.2 The *Masanumasika Pathya*: Month-Wise Regimen

The *Masanumasika Pathya* prescribes differentiated measures for each of the nine gestational months.[17,18,19] In the first month, milk prepared with sweet, cooling substances is recommended in frequent

small quantities, consistent with the secondary commentarial rendering of *Charaka Samhita*, *Sharirasthana* 8/32. [17,18] The second and third months continue sweet, cooling, unctuous milk-based preparations, corresponding to primordial organ formation. The fourth month marks the onset of *Dauhridavastha* (Section 4.4). From the fifth month the regimen progressively incorporates *Ghrita* and meat broths, and in later months *Niruhabasti*, with the eighth month receiving particular attention in *Sushruta*'s account given the fragility of the foetal constitution.[19] The *Astanga Sangraha* adds medicated *Vasti* prescriptions for the eighth month to facilitate normal descent and uncomplicated parturition.[20] Cow *Ghrita* supplies butyric acid alongside fat-soluble vitamins A, D, E, and K2; published gas-chromatography analyses place butyric acid at approximately two to four per cent of total fatty acid content, though reported values vary appreciably with analytical method, breed, and lactation stage, ranging from under one per cent in some series to over three per cent in others.[42,49] The dual epigenetic relevance of these constituents is addressed in Section 5.3.

4.3 *Garbhopaghatakara Bhavas*: Factors Harmful to the Foetus

Charaka Samhita, *Sharirasthana* 4, the *Mahatigarbhavakranti Sharira*, sets out the *Garbhopaghatakara Bhavas* in the same chapter that records *Dauhrida* (Section 4.4).[17] This chapter-4 attribution was independently confirmed against the *Caraka Samhita Online* digital edition, which places the passage at *Sharirasthana* 4, verse 18, immediately following the chapter's account of *Garbha* nourishment and preceding the *Dauhrida* passage; the attribution is further corroborated by the official BAMS curriculum reference for *Sharirasthana* chapter 4 maintained by the National Commission for Indian System of Medicine, which lists *Garbhopaghatakara Bhava* as a core topic of that chapter.[17,18] The enumerated factors include physical exertion, trauma, suppression of natural urges, fear and grief, disturbed sleep, unwholesome food, and emotionally disturbing stimuli. *Sushruta Samhita*, *Sharirasthana* 10, records a related doctrine that whatever a pregnant woman sees, hears, smells, touches, or tastes is transmitted to the foetus; the *Astanga Sangraha*, *Sharirasthana* 3, adds that she should be kept free of grief, fear, and excitement, and in the company of pleasant objects and sounds.[20]

4.4 The Concept of *Dauhrida* and Foetal Psyche Formation

The *Dauhrida* doctrine, recorded in *Charaka Samhita*, *Sharirasthana* 4, holds that in the fourth gestational month, coinciding with the manifest formation of the foetal *Hridaya* (the developing cardiac structure and, by classical extension, the seat of nascent consciousness), the pregnant woman experiences specific cravings attributed to the foetal mind, transmitted through her circulatory connection to the foetus.[17] The source text describes the foetal heart as formed from the maternal component and linked to the mother's heart through the *Rasavahi* channels, through which the foetus is held to express its wishes by way of the mother; on this account the woman now carries, in the classical idiom, two hearts, and the condition is termed *Dvaihrdayya*. The passage is commonly rendered to the effect that the fetus becomes deficient in limb formation, intellect, strength, complexion, and longevity if these desires are disregarded in this month, with exact verse division varying slightly by edition. [17,18]

Dauhrida is a multi-dimensional construct extending well beyond the nutritional reading pursued here. It encompasses *Matrija Bhava*, the transmission of the maternal psyche to the fetus, *Iccha*, desire as a mode of foetal communication, and implications for the foetus's eventual personality that have no straightforward biomedical analogue. The present analysis engages *Dauhrida* specifically through its implications for maternal caloric and micronutrient adequacy during a period of heightened foetal demand, which is a deliberate narrowing of a considerably richer classical doctrine, and one that Section 7.2 returns to qualify further.

5. Conceptual Mapping: *Garbhini Paricharya* and DOHaD

Table 1 presents the complete conceptual mapping developed in this paper, and Figure 4 gives a temporal visualisation of the same mapping. Correspondences designated substantiated satisfy all three of the a priori mapping criteria defined in Section 2.1; those designated hypothetical satisfy the first two but lack a fully established mechanistic intermediate and are presented as research priorities rather than established findings. Figure 5 summarises how these nine correspondences distribute across developmental windows, making visible a pattern that is easy to lose inside a dense table: the strongest mechanistic support clusters at the periconceptional stage and around *Manasika Paricharya*, while the third-trimester *Niruhabasti* correspondence remains the weakest link in the entire mapping.

Table 1.

***Garbhini Paricharya* component**	**Classical reference**	**DOHaD programming window**	**Proposed epigenetic mechanism**	**Status and modern correlate**
Beeja Shuddhi (*Shodhana*: *Vamana*/*Virechana* for both parents, pre-conception)	CS Sha. 2/1-3[17,18]	Periconceptional: indirect epigenomic modulation via gut microbiome remodelling	Toxin clearance; microbiome remodelling; reduced systemic inflammation; indirect germ-cell epigenome support	Substantiated, indirect pathway [27,28]; paternal epigenetic inheritance [21,29,33,41]. Safety note: formal evaluation required before any clinical recommendation; *Vamana* carries aspiration risk.
Beeja Shuddhi (post-*Shodhana* nutritional component, both parents)	CS Sha. 2/1-3[17,18]	Periconceptional: de novo methylation and imprint establishment; sperm microRNA integrity	One-carbon metabolite supply (folate, choline, methionine, B6/B12); germ-cell epigenome support	Substantiated [5,22,44,45]; paternal sperm epigenome and intergenerational inheritance [21,29,33,34,41].
Masanumasika Pathya, months 1 to 3 (*Kshira*-based diet)	CS Sha. 8/32; SS Sha. 10[17,19]	Organogenesis, weeks 3 to 12: multi-pathway nutritional support	B12, choline, riboflavin via milk; trophoblast vascularisation; mucosal immune modulation	Substantiated, indirect and multi-pathway [22]. *Kshira* supports, but does not solely constitute, the one-carbon network.
Garbhopaghatakara Bhavas (avoidance of stress, trauma, disturbing stimuli; all trimesters)	CS Sha. 4[17]; SS Sha. 10[19]	All trimesters; most critical in trimester one for HPA axis programming	Glucocorticoid-mediated modification at CRF, POMC, NR3C1, FKBP5; 11-beta-HSD2 gated	Substantiated [15,16,25,30,46]. Avoidance of *Bhaya*/*Shoka* corresponds to attenuated cortisol release and reduced GR hypermethylation. This correspondence is inferred by mechanistic analogy to the *Manasika Paricharya* pathway (Section

***Garbhini Paricharya* component**	**Classical reference**	**DOHaD programming window**	**Proposed epigenetic mechanism**	**Status and modern correlate**
				5.5), which supplies the direct human translational evidence, rather than independently demonstrated for *Garbhopaghatakara Bhava* avoidance in isolation.
Dauhrida gratification (month four)	CS Sha. 4/18-23[17]	Mid-gestational CNS programming, weeks 16 to 20	Caloric/micronutrient adequacy; BDNF and IGF2 promoter methylation	Substantiated, indirect [4,14]. Disregard is associated in CS Sha. 4 with foetal malformation and impaired intellect; see Section 7.2 on the caloric ceiling this correspondence requires.
Ghrita-predominant diet: butyric acid/HDAC pathway (months 5 to 7)	CS Sha. 8/32; ASa Sha. 3/7[17,20]	Trimesters 2-3: histone acetylation in foetal brain and metabolic tissue	Butyric acid as endogenous HDAC inhibitor; enhanced acetylation at developmentally regulated loci	Substantiated, indirect mechanistic pathway [42,43,49]. Stronger mechanistically than the DHA pathway below, though direct evidence that dietary *Ghrita*-derived butyrate reaches the foetal compartment in a bioactive concentration during human pregnancy is not yet available.
Ghrita-predominant diet: indirect LC-	CS Sha. 8/32; ASa Sha.	Trimesters 2-3: adipose programming	Fat-soluble vitamins A, D, E, K2; DHA	Hypothetical requires empirical verificati

***Garbhini Paricharya* component**	**Classical reference**	**DOHaD programming window**	**Proposed epigenetic mechanism**	**Status and modern correlate**
PUFA/DHA pathway (months 5 to 7)	3/7[17,20]	; myelination; synaptic plasticity gene methylation	pathway depends on overall maternal LC-PUFA status, not *Ghrita* alone	on [35,36,37]. Needs co-administration of an omega-3 source.
Niruhabasti and *Ojas* restoration (month eight, *Adana* phase)	SS Sha. 10[19]; ASa Sha. 3/9[20]	Late trimester 3: immune tolerance programming via gut microbiome-epigenome axis	Modified colonic SCFA production; GPR43-mediated Treg differentiation [47]	Hypothetical [26,27,28,47]. Mandatory safety caveat: must not be initiated without prior animal-model safety data; not a clinical recommendation.
Manasika Paricharya (psychological care, all trimesters)	CS Sha. 8[17]; SS Sha. 10[19]; ASa Sha. 3/3[20]	All trimesters; critical in trimester one (HPA set point) and trimester three (cortical	Attenuated glucocorticoid-driven CpG methylation at CRF, POMC, NR3C1, FKBP5; 11-beta-HSD2 gated	Substantiated; the strongest correspondence in this mapping [15,16,25,30,31,46].
		myelination)		

*Rows shaded amber in the source table indicate correspondences designated hypothetical, requiring empirical validation; rows shaded green indicate the two correspondences judged most directly supported by convergent biological-target and mechanistic evidence. *Beeja Shuddhi* is presented as two rows to preserve the mechanistic and safety distinction between the *Shodhana* component and the post-*Shodhana* nutritional component. The *Ghrita* prescription is presented as two rows to separate the directly substantiated butyric acid and HDAC pathway from the indirect and hypothetical DHA/LC-PUFA correspondence. Abbreviations: CS, *Charaka Samhita*; SS, *Sushruta Samhita*; ASa, *Astanga Sangraha*; Sha., *Sharirasthana*; HPA, hypothalamic-pituitary-adrenal; HDAC, histone deacetylase; DHA, docosahexaenoic acid; LC-PUFA, long-chain polyunsaturated fatty acid; GPR43, G-protein-coupled receptor 43; Treg, regulatory T cell.*

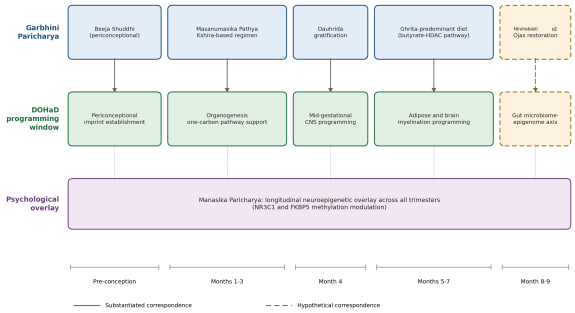

Figure 4. Temporal alignment of *Garbhini Paricharya* prescriptions with DOHaD epigenetic programming windows. The upper tier shows the principal *Garbhini Paricharya* prescriptive components; the lower tier shows the corresponding DOHaD epigenetic programming window; the lower band shows *Manasika Paricharya* as a longitudinal overlay spanning all three trimesters. Solid arrows denote substantiated correspondences; the dashed arrow denotes the hypothetical *Niruhabasti* correspondence.

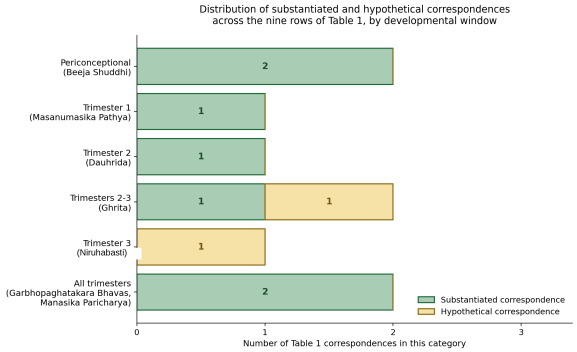

Figure 5. Distribution of substantiated and hypothetical correspondences across the nine rows of Table 1, grouped by developmental window. The periconceptional and all-trimester categories carry the strongest evidentiary support; the *Niruhabasti* correspondence in late trimester three remains the weakest link in the mapping and is presented throughout as a research priority rather than a finding.

5.1 Periconceptional Phase and *Beeja Shuddhi*

The periconceptional period, spanning the months before conception through the earliest weeks of embryonic life, represents the period of greatest epigenetic plasticity, owing to global demethylation and subsequent de novo methylation of the embryonic genome.[6] Maternal folate, choline, and methionine status are critical determinants of imprint establishment at differentially methylated regions, including IGF2/H19 and KCNQ1OT1.[5,22] The agouti mouse and Dutch Hunger Winter findings (Section 3.2) demonstrate this sensitivity directly, in animal and human evidence respectively.[44,45]

Beeja Shuddhi prescribes that both prospective parents undergo *Shodhana* procedures, principally *Vamana* and *Virechana*, followed by nutrient-dense dietary preparation, before conception. [17,18] Its *Shodhana* component is proposed to correspond to periconceptional DOHaD effects through toxin clearance, microbiome remodelling, and reduced inflammatory burden, [27,28] indirect pathways that require dedicated investigation rather than direct adoption. The post-*Shodhana* dietary component plausibly contributes more directly to one-carbon metabolite supply and germ-cell epigenome integrity. No clinical evidence currently supports *Shodhana* in the periconceptional period, and any recommendation must await formal safety evaluation, which is a precondition for any future clinical statement rather than a minor qualification to one.

The prescription of *Shodhana* for both partners corresponds, at the level of biological target, to growing DOHaD evidence on paternal contributions: paternal diet, stress, and toxin burden alter sperm microRNA content and DNA methylation, with documented downstream effects on offspring trajectories.[21,29,33,41] Fullston and colleagues showed that paternal obesity alters sperm microRNA content, initiating metabolic disturbance across two generations of offspring, a finding that aligns closely with the requirement that both partners undergo purification before conception.[41] Rodgers and colleagues showed that paternal stress is transmitted by way of sperm microRNA changes sufficient to recapitulate the stress phenotype independent of postnatal paternal behaviour.[29] Dias and Ressler's demonstration that paternal olfactory fear conditioning alters offspring behaviour through hypomethylation of the corresponding odorant receptor gene in sperm DNA illustrates the same principle.[34]

5.2 First Trimester: Organogenesis and Critical Window Programming

Organogenesis, occurring chiefly between the third and eighth week of gestation, is the most critical window of developmental programming identified by DOHaD research; disruption by nutritional deficiency, hypoxia, or glucocorticoid excess produces consequences that persist for life.[6,14] The *Masanumasika Pathya* for the first three months prescribes liquid, sweet, unctuous preparations with *Kshira* as the principal recommendation.[17,18,19] Milk is rich in vitamin B12, choline, and riboflavin, supporting the one-carbon network,[22] in addition to bioavailable calcium and protein supporting trophoblast invasion and placental vascularisation, and immune-modulatory components supporting maternal mucosal immunity during implantation. This correspondence is best characterised as indirect and multi-pathway rather than single pathway; Table 1 reflects this qualification. The *Garbhopaghatakara Bhavas*, in listing grief, fear, and excessive exertion among harmful factors,[17] are explicable through maternal HPA axis activation: cortisol crosses the placental barrier in the proportion that 11-beta-HSD2 activity permits, inducing lasting hypermethylation of glucocorticoid receptor promoters in the foetal hippocampus and hypothalamus. [15,25,46]

5.3 Second Trimester: *Dauhrida* and Critical Period Neurodevelopment; *Ghrita* Mechanisms

The fourth gestational month, the *Dauhrida* phase, corresponds to rapid CNS maturation, completion of cardiac septation, and onset of foetal movement. [17,19] DOHaD research identifies this period as critical for HPA axis sensitivity, dopaminergic reward circuitry, and IGF signalling. [14,15] The classical instruction to satisfy the *Dauhridini*'s cravings is read here as a culturally encoded mechanism for ensuring adequate caloric and micronutrient provision during heightened foetal demand. Mid-gestational undernutrition is documented to impair neurodevelopment and organogenesis,[4,14] consistent with the classical warning that disregarding

Dauhrida desires is associated with foetal malformation and impaired intellect.[17] Section 7.2 returns to this correspondence with a qualification that the present section does not pursue further: the classical instruction was framed against a background of chronic maternal undernutrition, and its modern translation in a population increasingly affected by gestational diabetes and excess gestational weight gain cannot simply be unrestricted craving satisfaction.

The progressive introduction of *Ghrita* from the fifth month [17,20] engages two distinct pathways. Cow *Ghrita* contains a measurable proportion of butyric acid, a well-characterised HDAC inhibitor, though published values vary by analytical method and lactation stage; [42,49] butyrate-mediated histone acetylation at developmentally regulated loci aligns with the window of foetal adipose differentiation and brain myelination. [42,43] Whether dietary butyrate reaches the fetal compartment in a bioactive concentration during human pregnancy has not been directly measured, and the pathway should accordingly be read as biologically plausible rather than as a demonstrated in vivo mechanism. The second pathway is indirect: *Ghrita*'s fat-soluble vitamin content supports neurodevelopmental processes associated with synaptic plasticity gene methylation. A third candidate pathway, DHA-mediated regulation of synaptic plasticity genes,[37] is indirect in a stronger sense still, since *Ghrita* is not itself a source of omega-3 LC-PUFAs, and is accordingly classified as hypothetical in Table 1.

5.4 Third Trimester: Metabolic Programming and the Eighth Month

DOHaD research establishes the third trimester as the principal period of foetal adipose tissue programming, with maternal nutrition determining adipocyte number, lipogenic enzyme activity, and adipokine gene regulation.[14] *Sushruta Samhita* and the *Astanga Sangraha*[19,20] describe the eighth month as particularly vulnerable, characterised by the *Adana* state, an unstable interchange of *Ojas* between mother and fetus, with therapeutic measures directed at *Ojas* stabilisation by way of *Niruhabasti* and dietary supplementation. Its DOHaD correspondence, to the extent it is epigenetically relevant at all, is proposed to operate through the gut microbiome-epigenome axis (Section 3.3): colonic short-chain fatty acids act on GPR43 to drive regulatory T-cell differentiation,[47] offering a defined though clinically unconfirmed route to an immune-programming outcome.[27,28] The correspondence remains hypothetical, since the causal chain from *Niruhabasti* administration to altered foetal short-chain fatty acid exposure has not itself been demonstrated, and it is the weakest correspondence presented in this paper, as Figure 5 makes visible.

Critical safety note. *Niruhabasti* and other *Panchakarma* procedures in the third trimester carry potential clinical risks not formally evaluated in controlled trials. Rectal instillation in late pregnancy carries a theoretical risk of uterine stimulation and premature labour that has not been excluded by available evidence. No such procedure should be recommended outside an appropriately supervised clinical trial until safety is first established in third-trimester animal models and confirmed in monitored human pilot studies, regardless of the procedure's classical textual authority.

5.5 *Manasika Paricharya*: Psychological Care and Neuroepigenetic Programming

Manasika Paricharya, prescribing *Prasanna Manasa*, a cheerful and contented state of mind, exposure to pleasant sensory stimuli, and avoidance of *Bhaya* and *Shoka*, is recorded in *Charaka Samhita*, *Sharirasthana* 8, and *Sushruta Samhita*, *Sharirasthana* 10.[17,19] The *Astanga Sangraha* adds that the woman should at all times be kept happy, not frightened, and encouraged to wear flowers, fragrant materials, and white garments.[20] This is, in the authors' assessment, the most directly supported alignment in the entire mapping. Maternal prenatal psychological distress is among the most extensively studied DOHaD exposures: elevated maternal anxiety and depression have been associated, across cohorts including the Avon Longitudinal Study of Parents and Children, with altered foetal HPA axis, sympathoadrenal, and prefrontal cortical programming, mediated through glucocorticoid-driven modification of CRF, POMC, FKBP5, and NR3C1 promoters in foetal neural tissue.[15,16,25,30,31] Oberlander and colleagues showed directly that neonatal NR3C1 promoter methylation is elevated in infants whose mothers experienced antenatal depression, a specific translational link to the molecular mechanism that *Manasika Paricharya* would, on this reading, be acting upon.[16] The placental 11-beta-HSD2 barrier (Section 3.2) is the physiological mechanism through which this protection operates: a calmer maternal cortisol profile places less demand on an enzymatic barrier that is itself down-regulated by stress, illness, and malnutrition.[46] The instruction to shield the pregnant woman from grief and fear constitutes, from a DOHaD perspective, a culturally articulated strategy of glucocorticoid modulation that protects the foetal neuroepigenome across all three trimesters.

6. An Integrative Conceptual Framework

The mapping developed across the preceding sections shows that the principal components of *Garbhini Paricharya* address distinct DOHaD programming windows through mechanisms now open to direct molecular investigation. Taken together, the periconceptional prescriptions of *Beeja Shuddhi*, the

trimester-specific *Masanumasika Pathya*, the prohibitions of the *Garbhopaghatakara Bhavas*, the nutritional intensification of *Dauhrida*, the *Ghrita* prescriptions of the second and third trimesters, the late-gestation procedures grouped under *Niruhabasti*, and the overarching *Manasika Paricharya* together constitute a temporally calibrated programme of prenatal intervention whose internal logic, on the classical system's own terms (Section 4.1), and whose correspondence to the epigenetic windows identified by DOHaD research (Section 5), run in parallel with one another rather than in competition.

This integrative framework is summarised in Table 1 and visualised temporally in Figure 4. A five-stage translational research pathway, intended to move this framework from conceptual mapping toward responsibly sequenced empirical study, is given in Figure 7.

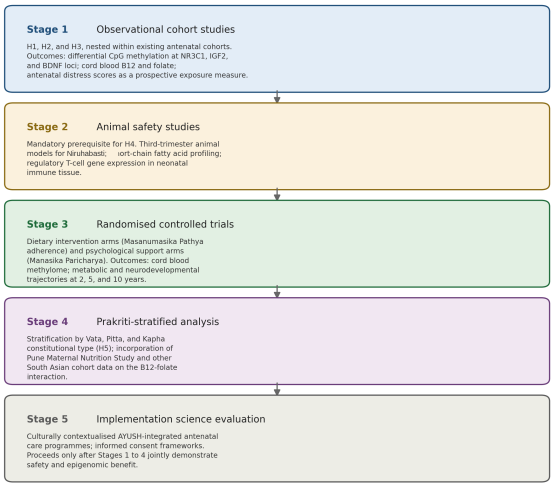

Figure 7. Proposed translational research pathway from conceptual framework to clinical integration. Stage 1 comprises observational cohort studies corresponding to hypotheses H1 through H3. Stage 2 comprises the animal safety studies that must precede any clinical evaluation of H4. Stage 3 comprises randomised controlled trials of dietary and psychological intervention arms. Stage 4 comprises *Prakriti*-stratified analysis (H5). Stage 5, implementation science evaluation, proceeds only once Stages 1 through 4 have jointly demonstrated safety and epigenomic benefit.

7. Discussion

7.1 Convergent Epistemology Without Historical Contact

Perhaps the most notable feature of the correspondence between *Garbhini Paricharya* and DOHaD is that it represents convergent epistemology: two knowledge systems, separated by more than two thousand years and operating within entirely different theoretical paradigms, arrive at substantially similar prescriptive conclusions about managing the intrauterine environment. The *Ayurvedic Acharyas* had no access to the concept of DNA methylation or to glucocorticoid receptor biology, and yet generations of accumulated clinical observation produced prescriptions now explicable in precisely such terms. This convergence is not confined to *Ayurveda*: traditional Chinese medicine records a comparable body of prenatal practice under the concept of *Taijiao*, foetal education, with textual references dating to the Zhou dynasty. The existence of analogous frameworks across independent traditions offers a measure of cross-traditional support for the biological plausibility of the mechanisms now proposed to explain them, though a dedicated comparative analysis is left to future scholarship.

A more exacting epistemological qualification is necessary. The convergence described here is directional: the mapping proceeds from known modern mechanism backward to classical text, never from text forward to an empirical prediction subsequently confirmed. This is the inherent structure of any retrospective hermeneutic analysis, and it carries a risk of post-hoc rationalisation that methodological safeguards can reduce but cannot remove entirely. The correspondences in Table 1 should be read not as evidence that *Garbhini Paricharya* anticipated epigenetics predictively, but as evidence that prescriptions arrived at through sustained clinical observation across generations are now explicable in epigenetic terms, which is a materially weaker and more defensible claim: the framework motivates empirical study, it does not substitute for it.

7.2 Points of Tension and Necessary Reinterpretation

A mapping exercise of this kind risks reading as a search for confirmation if it records only the places where classical prescription and modern mechanism align. Four points in the mapping require the opposite treatment: a candid statement of where direct, unqualified adoption of the classical instruction would sit poorly against present-day clinical evidence, and where a more careful modern reading is required instead. Figure 6 sets these out alongside the strongest points of convergence, for the same reason Table 2 sets out the paper's relationship to prior literature directly rather than leaving it implicit.

The clearest case is *Dauhrida*. Section 5.3 reads the instruction to satisfy the *Dauhridini*'s cravings as a mechanism for ensuring caloric and micronutrient adequacy, a reading well supported where the background risk is maternal undernutrition, as it plainly was across most of the historical contexts in which this doctrine was elaborated. The background risk in urban India today is materially different: a

recent systematic review and meta-analysis estimated pooled gestational diabetes prevalence at 12 per cent in urban Indian populations, with individual state-level estimates considerably higher.[50] Read in this setting, an instruction to gratify all cravings without qualification would risk reinforcing exactly the excess caloric intake that contemporary obstetric guidance seeks to moderate. The more defensible modern reading is one of qualitative rather than unrestricted quantitative gratification: attending to dietary diversity and to the psychological legitimacy of the pregnant woman's expressed needs, rather than treating craving as a licence for unmoderated intake. Table 1 has been annotated accordingly.

A related tension attaches to *Ghrita*. Its butyric acid and fat-soluble vitamin content supplies the strongest single mechanistic correspondence in this paper outside *Manasika Paricharya*, but *Ghrita* is also calorically dense, and its classical prescription was formulated without reference to gestational weight gain targets that did not yet exist as a clinical concept. Any translational protocol built on the *Ghrita* correspondence (H1, Section 7.3) needs explicit calorie accounting and should be paired with current gestational weight gain guidance, particularly for women entering pregnancy with an already elevated body mass index, rather than read as an unqualified recommendation to increase *Ghrita* intake.

A third tension is procedural rather than nutritional. *Niruhabasti* and the *Shodhana* component of *Beeja Shuddhi* are both supported, at most, by biological-target plausibility rather than by any direct demonstration of foetal benefit, and both involve invasive procedures, rectal instillation in the third trimester in one case and emesis or purgation in the periconceptional period in the other, that carry documented potential for harm independent of any epigenetic question. The classical textual authority behind these procedures is not in doubt; their safety in a modern obstetric setting is, and the two questions should not be allowed to blur into one another.

The fourth tension is more structural than clinical. Section 4.1 reads *Tridosha* dynamics as a population-level pattern, which is the only register in which DOHaD research itself operates. *Prakriti*, the individual constitutional baseline, is set aside by this manoeuvre, a simplification defensible for the purposes of this paper but one that no *Ayurvedic* clinician would accept as a basis for actual patient care. Section 7.4 returns to this point at greater length.

None of these four tensions weakens the central argument of this paper, which is that *Garbhini Paricharya* corresponds systematically to DOHaD theory at the level of mechanism. They do mean that translating that correspondence into any kind of practical guidance requires more than reading the classical instruction off the page, and a paper that omitted this section would have left an important part of its own argument incomplete.

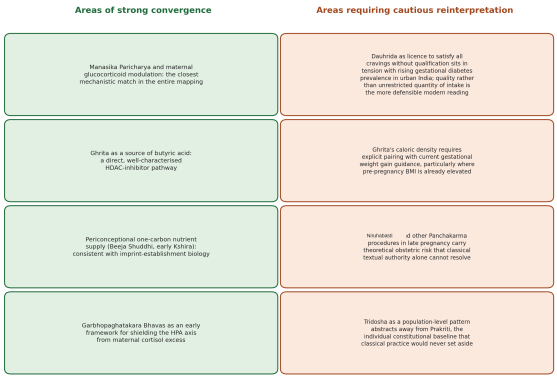

Figure 6. Points of strong convergence between *Garbhini Paricharya* and DOHaD mechanism, set alongside points requiring cautious reinterpretation before any translational application. The two columns are not symmetrical in number by design: the convergence side of the mapping is considerably larger, but the tensions on the right are the ones most likely to matter if this framework is ever read as practical guidance rather than as a research agenda.

7.3 Translational Research Hypotheses

The conceptual framework generates five testable hypotheses, each with a specific biomarker outcome and a recommended study population.

H1. Adherence to the *Masanumasika Pathya*, particularly its emphasis on *Kshira* and *Ghrita* in trimesters one and two, is associated with measurable differences in cord blood methyl-donor micronutrients (B12, choline, folate) and differential DNA methylation at growth-regulatory CpG loci. Testable through a prospective cohort nested within an existing Integrated Child Development Services cohort in India, with cord blood methylome profiling as the primary outcome and total gestational weight gain recorded as a co-primary safety outcome given the caloric-density concern raised in Section 7.2.

H2. Gratification of *Dauhrida* cravings in month four, operationalised as increased dietary diversity rather than increased caloric quantity, is associated with superior neurodevelopmental outcomes at twenty-four months, mediated through BDNF and IGF2 promoter methylation. Testable by way of a prospective food frequency questionnaire in month four, with the Bayley Scales at twenty-four months as the primary endpoint and maternal glycaemic status tracked throughout as a safety covariate; a prospective design is preferred over retrospective recall, which is susceptible to bias.

H3. Adherence to *Manasika Paricharya*, measured by way of a validated antenatal distress instrument such

as the Pregnancy-Related Anxiety Questionnaire, Revised, or the Cambridge Worry Scale, administered each trimester, is associated with lower cord blood cortisol and attenuated NR3C1/FKBP5 promoter methylation. Testable through a prospective observational study at any tertiary obstetric centre with cord blood epigenomic profiling access; the Edinburgh Postnatal Depression Scale, being postnatal, is not appropriate here.

H4. Following mandatory prior animal-model safety evaluation, *Niruhabasti* in month eight, by modifying colonic short-chain fatty acid production, influences butyrate-mediated histone acetylation in neonatal immune cells through the GPR43-dependent Treg pathway (Section 3.3),[47] with measurable effects on regulatory T-cell gene expression in cord blood. Clinical evaluation requires independent safety replication and must proceed, if at all, only within a monitored, ethically approved pilot trial with an independent data safety monitoring board. H4 remains the lowest-priority hypothesis in this paper and must not be translated into practice on the strength of this conceptual framework alone.

H5. The epigenetic response to *Masanumasika Pathya* adherence under H1 varies systematically by maternal *Prakriti*, with *Pitta*-predominant women showing a different methylation response to *Ghrita* introduction than *Vata*- or *Kapha*-predominant women, plausibly mediated through constitutional differences in digestive capacity (*Agni*). Testable as a planned stratified sub-analysis of the H1 cohort, with *Prakriti* assessed at enrolment using a validated standardised instrument; presented as hypothesis-generating, since no prior *Prakriti*-stratified epigenomic data exist.

7.4 Policy, Constitutional Limits, and Heterogeneity

India carries a substantial non-communicable disease burden, including type 2 diabetes mellitus, hypertension, and cardiovascular disease, whose DOHaD origins in suboptimal maternal nutrition and intrauterine growth restriction are extensively documented in South Asian populations.[4,14] The Pune Maternal Nutrition Study showed that low maternal B12 combined with high folate status was associated with greater offspring insulin resistance, an interaction that runs counter to the common assumption that folate supplementation alone is uniformly beneficial,[32] suggesting that the mechanisms engaged by a *Kshira*-based diet, which supplies B12 and choline alongside other nutrients rather than folate in isolation, may differ quantitatively from those described in European populations. India-specific data of the kind proposed under H1 are needed for this reason.

Garbhini Paricharya offers a culturally embedded, broadly accessible framework for prenatal support, and its potential integration into India's AYUSH antenatal care system is a policy question supported in principle by classical precedent and by the present correspondence. Any integration requires rigorous evidence-based evaluation, including randomised controlled trials of *Masanumasika Pathya* components with cord blood methylome profiling, and implementation science evaluation of programmes already demonstrating epigenomic benefit. The conceptual correspondences in this paper do not on their own justify incorporating these prescriptions into standard antenatal care; any such programme must distinguish clearly between traditional practice and evidence-based recommendation and must guard against premature adoption. Certain procedures discussed here, particularly *Vamana* and *Virechana* in the periconceptional phase and *Niruhabasti* in month eight, carry documented potential risk: *Vamana* risks aspiration, electrolyte disturbance, and uterine stimulation; *Virechana* risks dehydration and premature labour; *Niruhabasti* in late pregnancy risks uterine stimulation and preterm delivery. None should be recommended outside a supervised clinical trial with independent ethical oversight.

Section 4.1 introduced *Tridosha* physiology as the proximate rationale for the *Masanumasika Pathya*, distinct from *Prakriti*, the individual constitutional variation often raised as a limitation in prior cross-paradigm commentary rather than incorporated as a feature of the analysis. *Tridosha* dynamics operate as a shared population-level pattern, the kind DOHaD research itself addresses, while *Prakriti* is the individual baseline against which that pattern plays out; engaging the former on DOHaD's own population-level terms necessarily abstracts away from the latter, a conceptual necessity for this paper but a significant simplification from the standpoint of *Ayurvedic* practice, where no prescription is considered complete without reference to individual constitution. H5 is offered as a first step toward closing this gap, and future research should treat *Prakriti* assessment as a standard stratification variable rather than an afterthought. The present analysis also treats *Charaka Samhita* as primary, which introduces a single-source dominance worth acknowledging directly: *Charaka* and *Sushruta* differ in the specificity of their *Niruhabasti* prescriptions, and the *Astanga Sangraha* introduces procedural refinements to *Vasti* therapy does not present in *Charaka*'s account, differences that may reflect meaningful regional or constitutional variation rather than inconsistency requiring harmonisation.

7.5 Further Limitations

This is a theoretical framework paper generating no primary empirical data; every causal link in Table 1 is a hypothesis-level assertion requiring experimental

validation, not a finding. The molecular mechanisms invoked are derived predominantly from non-Indian populations, and population-specific variation, including the B12-folate interaction discussed in Section 7.4, [32] may modify the applicability of some conclusions to the South Asian context. The hermeneutic method is inherently susceptible to confirmation bias; this has been addressed through the a priori mapping criteria (Section 2.1), independent dual classification of each correspondence, dual-commentary cross-verification and citation authenticity procedures (Sections 2.2, 2.4), external review of the Sanskrit translation, and the explicit treatment of points of tension in Section 7.2, but cannot be eliminated entirely, and the directional structure discussed in Section 7.1 compounds rather than mitigates this risk. No formal protocol for this narrative synthesis was registered in advance, and no formal risk-of-bias instrument was applied to the 47 included biomedical studies individually, both of which are appropriate practice for a hermeneutic conceptual analysis but would be required of a systematic review proper; readers should weigh the evidence accordingly. The restriction of scope to the *Brihatrayi* excludes prescriptions recorded in the *Laghutrayi* and later compilations such as the *Harita Samhita*, which may contain further prescriptions of DOHaD relevance, and this paper does not extend to a comparative treatment of *Taijiao* or other traditional prenatal care systems, both of which are left for dedicated future scholarship.

8. Conclusion

Garbhini Paricharya, as codified in the *Brihatrayi*, sets out a temporally structured programme for shaping the intrauterine environment, and the argument of this paper is that its prescriptive logic corresponds systematically, mechanism by mechanism rather than in vague analogy, to DOHaD theory and to the epigenetic processes thought to mediate developmental programming. The month-wise prescriptions of the *Masanumasika Pathya* address the same biologically critical windows of foetal epigenetic plasticity identified by contemporary perinatal research, and they do so for an internally coherent physiological reason on the classical system's own terms, namely the trimester-wise fluctuation of *Vata*, *Pitta*, and *Kapha* across gestation that Section 4.1 has set out. The *Garbhopaghatakara Bhavas* encode a practical framework for maternal glucocorticoid modulation; the *Ghrita* prescription supplies butyric acid, a biochemically well-characterised HDAC inhibitor, alongside fat-soluble vitamins, although direct evidence of fetal-compartment bioavailability in human pregnancy remains to be established; and *Dauhrida*, read with the qualification Section 7.2 requires, operationalises mid-gestational nutritional intensification during a critical period of foetal neurodevelopment.

Three findings in this analysis carry more weight than the others and should anchor whatever empirical work follows. *Manasika Paricharya* is the strongest correspondence in the entire mapping, with a direct molecular counterpart in placental 11-beta-HSD2 gating of maternal cortisol; the periconceptional component of *Beeja Shuddhi*, specifically its nutritional rather than its *Shodhana* element, rests on some of the best-established biology in the DOHaD literature; and the *Ghrita* butyric acid pathway is mechanistically more direct than any comparison with omega-3 fatty acid biology would suggest. Against these, the *Niruhabasti* correspondence and the *Shodhana* component of *Beeja Shuddhi* remain genuinely hypothetical, and no part of this paper should be read as recommending either outside a formally supervised research setting.

This framework positions *Garbhini Paricharya* not as an archaic cultural practice awaiting biomedical validation it does not need, but as a clinical knowledge system of considerable empirical sophistication, one whose mechanisms are now open to molecular investigation and whose eventual clinical validation, should it occur, will require the same evidentiary standard demanded of any other proposed intervention in pregnancy. The translational research agenda set out in Section 7.3, comprising epigenetic biomarker studies, randomised nutritional and psychological intervention trials, *Prakriti*-stratified cohort studies under H5, and implementation science evaluation of culturally contextualised prenatal care, offers a genuinely productive interface between *Ayurvedic* classical scholarship and evidence-based maternal health science. Future research should prioritise H1 and H3, which rest on the most directly supported mechanistic correspondences identified here, before pursuing the remaining hypotheses. The safety evaluation of *Panchakarma* procedures in pregnancy must precede any clinical application of H4, and no finding in this paper should be read as licence to depart from that sequence.

Declarations

Conflict of interest. The authors declare no conflict of interest.

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Ethical approval. Not applicable. This paper is a conceptual analysis based on classical textual sources and published literature; no human participants or animal subjects were involved.

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with the DOHaD paradigm and its underlying epigenetic mechanisms. Anish Atpadkar and Sharavati Katte conducted the literature review of classical and contemporary biomedical sources and compiled the supporting textual and scientific evidence. All four authors contributed to drafting the manuscript, critically revised it for important intellectual content, and approved the final version submitted for publication. Dipti Ajay Chavan served as the corresponding author and coordinated submission and correspondence with the journal on behalf of all authors.

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