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A Critical Review of Ayurvedic Management of Breast Cancer with Special Reference to Rasayana and Bhasma Therapy

Dr Trupti Naikare

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HIGHLIGHTS

- Breast cancer in Ayurveda is classifiable under Granthi and Arbuda — pathological accumulations involving vitiation of Mamsa, Rakta, and Meda Dhatu with progressive Tridosha involvement — offering a clinically nuanced explanatory model beyond simplistic neoplastic description.
- Classical Rasayana formulations including Ashwagandha (Withania somnifera), Shatavari (Asparagus racemosus), and Triphala demonstrate reproducible in vitro and in vivo anti-proliferative, pro-apoptotic, and immunomodulatory actions substantiated by contemporary preclinical research.
- Metallic Bhasma preparations, particularly Swarna, Rajata, and Trivanga Bhasma, contain nanostructured metallic particles (10–56 nm range) produced through classical Shodhana-Marana procedures, lending biological plausibility to their cytotoxic and immunostimulatory effects in breast cancer models.
- Significant gaps persist in high-quality clinical trial evidence for Ayurvedic oncology; future research must prioritise rigorous randomized controlled trials integrating validated classical protocols with modern oncological endpoints, standardised Bhasma characterisation, and mechanistic pathway studies.

ABSTRACT

Background: Breast cancer remains the most prevalent malignancy among women globally, with conventional therapies incurring significant morbidity. Ayurveda, the classical Indian medical system, offers a conceptually rich understanding of neoplastic disease through the frameworks of Granthi and Arbuda, implicating disordered Dosha dynamics, Dhatu kshaya, and compromised Agni. Rasayana (bio-rejuvenative) therapies and Bhasma (calcined metallic or mineral preparations) represent two distinct but complementary therapeutic traditions within Ayurvedic oncology that have attracted growing scientific scrutiny.

Objective: This review critically synthesises available classical textual evidence and contemporary preclinical and clinical research concerning the role of Rasayana drugs and Bhasma preparations in the management of breast cancer, with the aim of identifying mechanistic pathways, evaluating evidence quality, and delineating future research priorities.

Methods: A systematic narrative review of classical Ayurvedic texts (Charaka Samhita, Sushruta Samhita, Ashtanga Hridayam) and contemporary peer-reviewed publications (PubMed, Scopus, AYUSH Research Portal, Google Scholar; 2000–2024) was conducted. Studies involving in vitro cytotoxicity, in vivo animal models, and human clinical trials pertaining to Rasayana drugs and Bhasma in breast cancer or cancer cell lines (primarily MCF-7, MDA-MB-231) were included. Articles from predatory journals, non-peer-reviewed sources, and those lacking methodological transparency were excluded.

Results: Twenty-two Rasayana drugs and six principal Bhasma preparations were identified with documented anti-cancer activity. Key mechanisms include induction of intrinsic and extrinsic apoptotic pathways, inhibition of NF-kB and PI3K/Akt/mTOR signalling, immunomodulation via natural killer cell activation, anti-angiogenic effects mediated through VEGF suppression, and nanoparticle-mediated cytotoxicity from Bhasma. Trivanga Bhasma demonstrated notable cytotoxicity against MCF-7 cells (IC50 data reported). However, the majority of evidence remains preclinical; robust phase I/II clinical trials are critically lacking.

Conclusion: Ayurvedic Rasayana and Bhasma therapies hold evidence-supported promise as integrative oncological interventions. Translation from preclinical promise to clinical validation requires standardised preparation methods, rigorous trial design, and interdisciplinary collaboration. Mechanistic nano-pharmacology of Bhasma offers a particularly compelling frontier for future investigation.

Keywords: *breast cancer; Rasayana; Bhasma therapy; Ayurvedic oncology; nano-pharmacology; integrative oncology*

1. Introduction

Breast cancer occupies a uniquely burdensome position in global oncology. According to the Global Cancer Observatory (GLOBOCAN 2022), breast cancer accounts for approximately 2.3 million new cases annually, constituting the leading cancer diagnosis in women worldwide, with an estimated 670,000 deaths per year.[1] In India, breast cancer has overtaken cervical cancer as the most prevalent female malignancy, with age-standardised incidence rates rising across urban and semi-urban populations alike.[2] Conventional therapeutic modalities — surgery, cytotoxic chemotherapy, targeted hormone therapy, and radiotherapy — while effective in many settings, are associated with substantial morbidity including immunosuppression, cardiomyopathy, peripheral neuropathy, and endocrine disruption. Chemoresistance and disease recurrence remain persistent clinical challenges, particularly in triple-negative breast cancer (TNBC) subtypes.[3]

It is against this backdrop that traditional medical systems have attracted renewed scientific and clinical attention. Ayurveda — the codified classical medical system of the Indian subcontinent, its foundational texts dating to approximately 600 BCE — offers a framework for understanding and managing neoplastic disease that is fundamentally different from, yet not necessarily incompatible with, contemporary biomedical oncology.[4] Unlike reductionist molecular models, Ayurveda situates disease within a dynamic interplay of constitution (Prakriti), environmental factors (Nidana), organismic intelligence (Agni), and tissular integrity (Dhatu Sara). Tumour formation, within this paradigm, is not a discrete cellular event but a systemic breakdown in the governance of growth, metabolism, and immunity.

Two therapeutic traditions within Ayurvedic oncology deserve particular scrutiny: Rasayana chikitsa — a class of bio-rejuvenative interventions aimed at restoring Dhatu integrity, enhancing Ojas, and modulating immune surveillance — and Bhasma therapy, which employs elaborately processed metallic and mineral preparations to achieve therapeutic effects at submicroscopic, and arguably nano-scale, levels.[5] Both traditions are supported by a growing, if still nascent, body of experimental evidence. Yet critical reviews integrating classical textual authority with contemporary mechanistic data remain sparse. This article aims to address that lacuna by providing a rigorous critical synthesis of available evidence, identifying mechanistic pathways, and mapping priorities for future translational research.

2. Conceptual Ayurvedic Understanding of Breast Cancer

2.1 Granthi and Arbuda: The Classical Pathological Constructs

Ayurvedic classical texts do not employ a direct equivalent to the biomedical term 'cancer.' However, the constructs of *Granthi* (a knotted, circumscribed, firm swelling) and *Arbuda* (a deep-seated, rapidly growing, and structurally aberrant mass) together approximate the spectrum of neoplastic pathology. The Sushruta Samhita, in the *Nidanasthana*, provides a taxonomic account: Granthi are generally superficial, slow-growing, and manageable; Arbuda are characterised by greater depth of penetration, atypical tissue composition, and relative resistance to conventional management.[6]

Sushruta classifies Arbuda by the predominant Dosha involved — Vataja, Pittaja, Kaphaja, Raktaja, Mamsaja, and Medaja — as well as the compound category of Sannipata Arbuda arising from simultaneous vitiation of all three Dosha.[7] The Mamsaja and Raktaja varieties, involving pathological proliferation of Mamsa Dhatu (muscular/connective tissue) and Rakta Dhatu (haematopoietic and circulatory tissue) respectively, bear closest structural and behavioural resemblance to malignant epithelial neoplasms. The Sushruta Samhita further distinguishes *Stana Vidradhi* (breast abscess), *Stana Roga* (diseases of the breast), and implicitly references neoplastic change in mammary tissue within the context of Mamsaja Granthi arising from the chest wall region.

2.2 Dosha Dynamics and Samprapti (Pathogenesis)

The Samprapti — the classical account of pathogenetic unfolding — of Arbuda is initiated by the accumulation of vitiated Dosha within a susceptible Srotas (physiological channel). In the context of breast tissue, the relevant Srotas are the *Stanya Vaha Srotas* (lactiferous channels) and the *Rakta Vaha Srotas*. Prolonged inappropriate Ahara (dietary indiscretion), Vihara (lifestyle factors including sedentary behaviour, day sleeping, suppression of natural urges), Manasika Nidana (psychological stressors including sustained grief — Shoka — and anxiety — Bhaya), and exposure to external toxins (Agantuja Nidana) collectively disturb Agni — the organismic intelligence responsible for metabolic transformation at every level from cellular to systemic.[8]

Compromised Agni generates metabolic waste products — Ama — which are qualitatively different from optimally processed tissue. Ama, lodging within Srotas and interfering with Dhatu formation, initiates a cascade: Rasa and Rakta Dhatu are compromised first, followed by progressive vitiation of Mamsa and Meda. The classical text characterises this accumulation as occurring specifically at *Sanga* — a site of pathological stagnation — within the channels. Over time, this concentrated Ama-Dosha complex undergoes morbid transformation (*Dushya-Dosha Sammurchhana*) to form a Granthi, and if the constitutional resilience (Bala) of the individual is inadequate to reverse the process, the Granthi progresses to Arbuda.[9]

2.3 The Role of Ojas and Immune Surveillance

Perhaps the most conceptually sophisticated element of the Ayurvedic understanding of Arbuda is its account of immune surveillance failure. Ojas — described in the Charaka Samhita as the supreme essence of all Dhatu, residing ultimately in the heart and responsible for maintaining life, immunity, and psychological stability — is specifically identified as being depleted in the patient with Arbuda.[10] Charaka's description of Oja-kshaya (depletion of Ojas) includes features directly analogous to immunosuppression: the individual becomes vulnerable to opportunistic illness, demonstrates poor wound healing, and exhibits progressive tissue wasting. This conceptual framework maps remarkably well to contemporary understanding of tumour immunoevasion, wherein cancer cells down-regulate immunogenic surface antigens and create immunosuppressive tumour microenvironments.

The implication for therapy is consequential: Rasayana interventions, which are primarily aimed at restoring Ojas and Dhatu integrity, are not merely symptomatic treatments but address a root dimension of the pathogenetic process. This conceptual alignment between Rasayana's Ojovardhanaka (Ojas-augmenting) properties and modern immunotherapy's goal of restoring tumour immunosurveillance is one of the more compelling areas of convergent understanding between the two medical traditions.

2.4 Sthanika Vishesha: Breast-Specific Considerations

Charaka Samhita (Chikitsasthana, Chapter 12) and the Ashtanga Hridayam of Vagbhata provide additional detail on the specific vulnerabilities of the breast region. The Stanya Vaha Srotas, being particularly susceptible to Kapha accumulation (as Kapha governs tissue proliferation, fluid metabolism, and structural density), and to Pitta-mediated Rakta vitiation (governing inflammatory and metabolic activity), creates the precise Doshic milieu most conducive to neoplastic Granthi formation.[11] The Ashtanga Hridayam further notes that prolonged Shoka — sustained emotional distress — specifically affects Hridaya (the cardiac-psychological locus) and secondarily the Stanya Vaha Srotas, providing a classically grounded mechanism for the epidemiological observation that psychological stress is a risk factor in breast cancer.

Critically, Ayurveda does not restrict its pathological model to local tissue changes. It understands malignant Arbuda as a systemic condition that merely manifests locally. This has direct implications for treatment design: local interventions (Shastra karma, surgical excision) are considered insufficient without systemic Shodhana (purification of the internal milieu) and Rasayana (restoration of Dhatu and Ojas), a framework that resonates with modern recognition that locoregional surgery without systemic oncological management fails to address the systemic nature of metastatic disease.

3. Modern Understanding of Breast Cancer: A Brief Oncological Overview

Breast cancer is a heterogeneous group of malignancies arising from the ductal or lobular epithelium of the mammary gland. Molecular characterisation has yielded several clinically relevant subtypes: Luminal A (ER+/PR+, HER2-, low Ki-67), Luminal B (ER+/PR+, HER2+/-, high Ki-67), HER2-enriched (ER-, PR-, HER2+), and triple-negative breast cancer (TNBC: ER-, PR-, HER2-).[12] TNBC, which represents approximately 15–20% of all breast cancers, poses the greatest therapeutic challenge due to the absence of targetable hormone receptors and relatively poor prognosis.[13]

The molecular hallmarks of breast cancer include sustained proliferative signalling (via PI3K/Akt/mTOR, RAS/MAPK pathways), evasion of growth suppressors (p53, RB inactivation), resistance to apoptosis (Bcl-2 overexpression), angiogenesis (VEGF upregulation), epithelial-mesenchymal transition (EMT) facilitating invasion and metastasis, and immune evasion through PD-L1 expression and regulatory T-cell recruitment.[14] It is across precisely these molecular targets that Rasayana phytoconstituents and Bhasma-derived nanoparticles are increasingly demonstrating activity — a convergence that provides mechanistic grounding for the empirical Ayurvedic therapeutic tradition.

Standard treatment modalities include surgery (lumpectomy, modified radical mastectomy), adjuvant chemotherapy (anthracycline and taxane-based regimens), hormonal therapies (tamoxifen, aromatase inhibitors), targeted biologics (trastuzumab for HER2+ disease), and immunotherapy (pembrolizumab for PD-L1+ TNBC). Despite these advances, five-year survival for metastatic breast cancer remains approximately 29%, underscoring the imperative for adjunctive and novel therapeutic strategies.[15]

4. Rasayana in Cancer Management: Classical Principles and Contemporary Evidence

4.1 The Classical Doctrine of Rasayana

Rasayana is defined in the Charaka Samhita (Chikitsasthana 1.1.7-8) as that which confers optimal quality upon Rasa and other Dhatu, promotes longevity, enhances memory and intellect, preserves youthfulness, and augments the body's resistance to disease.[16] The term itself — Rasa (primordial nutrient fluid) + Ayana (pathway or vehicle) — denotes a class of interventions that optimise the transformation, transport, and quality of Dhatu at every level of tissue elaboration. Charaka identifies two principal modes of Rasayana administration: Kutipraveshika (intensive in-patient residential regimen conducted in specifically constructed chambers) and Vatatapika (outpatient ambulatory regimen). The former is considered superior in efficacy but requires complete dedication of the patient; the latter is practical for most contemporary clinical applications.

Rasayana drugs are further subdivided by their primary target: Medhya Rasayana (cognitive rejuvenation), Balya Rasayana (strength-promoting), Ayushkara Rasayana (lifespan-extending), and Vyadhihara Rasayana (disease-specific). In the context of Arbuda management, the relevant categories are Vyadhihara (targeting the specific pathology), Balya (correcting systemic weakness and Dhatu kshaya), and the Dhatu-specific interventions targeting Mamsa, Rakta, and Meda. Acharya Charaka specifically mentions that Rasayana therapies are contraindicated in active Ama states without prior Shodhana — an important clinical caution that has direct relevance when considering Rasayana in the context of actively proliferating neoplastic disease.

4.2 Mechanisms of Action: Phytochemical and Pharmacological Perspectives

Contemporary research has identified multiple pharmacologically active phytoconstituents within Rasayana drugs that account for their anti-cancer activities. These include alkaloids (berberine, withanine), withanolides, saponins (shatavarins), tannins (ellagic acid, chebulic acid), polyphenols (gallic acid, emblicanin A and B), and terpenoids (ursolic acid, oleanolic acid).[17] The mechanisms by which these constituents operate in cancer biology include:

• Apoptosis induction: Multiple Rasayana phytoconstituents activate both intrinsic (mitochondrial, caspase-9 dependent) and extrinsic (death receptor, caspase-8 dependent) apoptotic pathways. Withaferin A, a steroidal lactone from Ashwagandha, is among the most thoroughly characterised, demonstrating induction of caspase-3 and PARP cleavage in MCF-7 cells at concentrations achievable in vivo.[18]

• PI3K/Akt/mTOR pathway inhibition: Berberine from Guduchi, ellagic acid from Amalaki, and bacoside A from Brahmi have independently demonstrated inhibitory effects on the PI3K/Akt/mTOR axis, a master regulator of cell survival and proliferation that is constitutively activated in many breast cancer subtypes.[1920]

• Immunomodulation: Ashwagandha, Guduchi, and Shatavari stimulate natural killer (NK) cell activity, macrophage phagocytic function, and cytokine secretion (particularly TNF-alpha and IL-12). These effects partially recapitulate the Ojovardhanaka action attributed to Rasayana in classical texts, and align mechanistically with the goals of modern cancer immunotherapy.[21]

• Anti-angiogenesis: Curcumin (from Haridra, a frequent Rasayana adjunct), ursolic acid, and crocin from Kumkuma suppress VEGF expression and inhibit endothelial tube formation, thereby limiting the neovascularisation that sustains solid tumour growth.[22]

• NF-kB pathway suppression: The transcription factor NF-kB governs inflammatory gene expression, anti-apoptotic proteins (Bcl-xL, IAPs), and metastatic mediators (MMP-2, MMP-9). Multiple Rasayana constituents — notably withaferin A, berberine, and gallic acid — demonstrably suppress NF-kB activation, linking them to both anti-proliferative and anti-metastatic outcomes.[23]

• Chemosensitisation: Amalaki extracts and specific Rasayana combinations have demonstrated synergistic cytotoxicity with conventional chemotherapeutic agents (doxorubicin, paclitaxel) in breast cancer cell lines, suggesting potential as adjuvants that reduce effective chemotherapy dosage and thereby mitigate toxicity.[24]

4.3 Evidence Synthesis: Key Rasayana Formulations

Ashwagandha (Withania somnifera) is perhaps the most extensively studied Rasayana in oncological contexts. Devi and colleagues demonstrated that withaferin A and withanolide D induced dose-dependent apoptosis in MCF-7 cells through mitochondrial pathway activation.[25] In vivo studies using murine breast cancer xenograft models have confirmed significant tumour volume reduction with oral Ashwagandha root extract standardised for withanolide content.[26] Importantly, Ashwagandha has also demonstrated protective effects on normal haematopoietic cells during chemotherapy — an Ayurvedically expected Rasayana property (Dhatu-raksha) that translates clinically to potential myeloprotection during cytotoxic therapy.

Triphala — the classical tridoshic formulation comprising Amalaki, Haritaki, and Bibhitaki — has been assessed in multiple cancer cell lines. Its active constituent gallic acid demonstrated IC50 values in the 40–80 microM range in MCF-7 cells in multiple independent studies.[27] A clinical pilot study (n=32) employing Triphala as a Rasayana adjunct to chemotherapy in breast cancer patients reported improved quality of life scores and reduced grade III mucositis compared to controls; however, this study's small sample size and non-standardised Triphala preparation limit generalisability.[28]

Guduchi (Tinospora cordifolia) has demonstrated broad-spectrum immunomodulatory activity, with standardised aqueous extracts significantly increasing NK cell cytotoxicity, macrophage activation, and peripheral lymphocyte counts in both preclinical and limited clinical settings.[29] Jagetia and Rao documented significant regression of Ehrlich ascites carcinoma in mouse models with oral Guduchi administration, with the mechanism attributed to enhanced host immune response rather than direct cytotoxicity.[30] In breast cancer cell lines, NF-kB pathway inhibition by Tinospora berberine has been reported by Singh and colleagues.[31]

Table 1. Important Rasayana Drugs, Classical Properties, and Modern Anticancer Evidence

Rasayana Drug	Sanskrit Name	Part Used	Rasa-Guna-Virya (Classical Properties)	Reported Pharmacological Actions (Modern)	Key Studies / Evidence
Ashwagandha	Withania somnifera	Root	Tikta, Katu rasa; Laghu, Snigdha guna; Ushna virya; Rasayana, Balya, Vataghna	Anti-proliferative, immunomodulatory, pro-apoptotic, anti-angiogenic; active constituents: withanolides	Devi et al. (2015) - withanolide D induces apoptosis in MCF-7; Mandal et al. (2020) - tumor growth inhibition in xenograft models
Shatavari	Asparagus racemosus	Root tuber	Madhura, Tikta rasa; Guru, Snigdha guna; Sheeta virya;	Cytotoxic to breast cancer cells (MDA-MB-231, MCF-7), antioxidant, estrogenic modulation; active constituents:	Bhutya et al. (2010) - antioxidant potential; Mitra et al. (2012) - cytotoxicity in hormone-receptor-positive lines

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Rasayana Drug	Sanskrit Name	Part Used	Rasa-Guna-Virya (Classical Properties)	Reported Pharmacological Actions (Modern)	Key Studies / Evidence
			Rasayana, Stanyajanana, Vatapitta shamaka	shatavarins, asparagine	
Guduchi	Tinospora cordifolia	Stem, leaves	Tikta, Kashaya rasa; Guru, Snigdha guna; Ushna virya; Tridosha shamaka, Rasayana, Medhya	Immunomodulatory, anti-tumor, anti-inflammatory, anti-oxidant; active: berberine, tinosporin, palmatine	Jagetia & Rao (2006) - significant tumor regression; Singh et al. (2019) - NF-kB pathway inhibition
Amalaki	Phyllanthus emblica	Fruit	Pancha rasa (except Lavana); Laghu, Ruksha guna; Sheeta virya; Rasayana, Vayasthapana, Tridoshaghna	Anti-proliferative, anti-angiogenic, antioxidant, chemosensitizer; active: emblicanin A&B, gallic acid, ellagic acid	Madhuri & Pandey (2009) - synergistic cytotoxicity with doxorubicin; Iranshahy et al. (2021) - review of anti-cancer mechanisms

Rasayana Drug	Sanskrit Name	Part Used	Rasa-Guna-Virya (Classical Properties)	Reported Pharmacological Actions (Modern)	Key Studies / Evidence
Haritaki	Terminalia chebula	Fruit rind	Pancha rasa; Laghu, Ruksha guna; Ushna virya; Rasayana, Deepaniya, Tridoshaghna	Apoptosis induction, anti-proliferative, MMP inhibition, anti-metastatic; active: chebulinic acid, chebulagic acid	Saleem et al. (2002) - growth inhibition in MCF-7; Patel et al. (2021) - downregulation of VEGF expression
Saffron (Kumkuma)	Crocus sativus	Stigma	Tikta, Katu rasa; Laghu guna; Ushna virya; Hridya, Varna prasadana, Rasayana	Anti-proliferative, pro-apoptotic, anti-angiogenic; active: crocin, crocetin, safranal	Aung et al. (2007) - crocin-induced apoptosis; Zhao et al. (2020) - inhibition of breast cancer metastasis
Brahmi	Bacopa monnieri	Whole plant	Tikta, Kashaya rasa; Laghu, Snigdha guna; Sheeta virya; Medhya Rasayan	Cytotoxic, anti-proliferative, pro-apoptotic, anti-inflammatory; active: bacoside A & B	Vollala et al. (2011) - neuroprotection + anti-tumor potential; Hota et al. (2021) - breast cancer cell line inhibition

Rasayana Drug	Sanskrit Name	Part Used	Rasa-Guna-Virya (Classical Properties)	Reported Pharmacological Actions (Modern)	Key Studies / Evidence
			a, Vata-Pitta shamaka		
Punarnava	Boerhavia diffusa	Root	Tikta, Kashaya, Madhura rasa; Laghu, Ruksha guna; Ushna virya; Rasayana, Shothahara, Mutrala	Anti-proliferative, anti-inflammatory, antioxidant; active: punarnavine, boeravinone	Manu & Kuttan (2009) - anti-metastatic activity; Agrawal et al. (2017) - cytotoxicity in triple-negative breast cancer (TNBC) lines

Note: Rasa = taste; Guna = quality; Virya = potency. All pharmacological data pertain to preclinical studies unless otherwise stated. IC50 values vary substantially by cell line, preparation method, and extraction solvent. Classical properties sourced from Charaka Samhita, Sushruta Samhita, and Dravyaguna Vijnana (Acharya P.V. Sharma). References cited in section 4.

4.4 Critical Appraisal of Rasayana Evidence

A critical assessment of the Rasayana literature reveals both promise and significant limitations. The majority of studies employ single-compound isolates rather than classical multi-drug formulations (Yoga), creating an epistemological tension: Ayurveda's therapeutic rationale is founded on synergistic multi-constituent interactions, yet modern pharmacology evaluates single actives. Studies employing whole plant extracts or classical formulations (e.g., Ashwagandharishta, Chyavanaprasha, Brahma Rasayana) remain comparatively rare and are often characterised by variable standardisation.[32]

Furthermore, the biological matrices used in most studies (MCF-7, a hormone-receptor-positive, non-invasive model) may not adequately represent the complexity of clinical breast cancer, particularly TNBC. Bioavailability of oral Rasayana phytoconstituents remains incompletely characterised, and the classical understanding of Deepana-Pachana (optimisation of metabolic capacity before Rasayana administration) as a prerequisite for efficacy has no systematic modern analogue. These methodological gaps must be addressed before preclinical evidence can meaningfully inform clinical practice.[33]

5. Role of Bhasma Therapy in Breast Cancer Management

5.1 Classical Principles of Bhasma Preparation

Bhasma — derived from the Sanskrit root 'bhasma' meaning ash or calcined residue — are preparations obtained through the elaborate, multi-step pharmaceutical processing of metals, minerals, or gems. The process involves two principal stages: Shodhana (purification, which removes toxic surface impurities and alters the physicochemical state of the raw material) and Marana (incineration or transformation through a series of precisely controlled trituration and heating cycles, typically employing plant-based media including herbal juices, specific acids, and alkalis as processing agents).[34] The Rasa Shastra texts — particularly the Rasa Tarangini, Rasa Ratna Samucchaya, and Ayurveda Prakasha — provide exhaustive detail on Shodhana and Marana procedures specific to each metal or mineral, with the number of Puta (incineration cycles) determining the fineness and therapeutic efficacy of the final product.

Classical quality control parameters for Bhasma include: Varitara (floats on water surface without sinking), Rekhapurnatva (fills the lines of the fingerprint), Nishchandrata (absence of lustre), Slakshnata (smoothness to touch), Niruttha (inability to revert to metallic form on reheating), Apunarbhava (inability to regenerate), and Uttama (completely fine, without granularity).[35] These parameters, while empirically derived, have been retrospectively interpreted through modern analytical techniques as indicators of nanoparticle size reduction, surface passivation, and altered crystalline structure — all of which have direct implications for bioavailability and pharmacological activity.

5.2 Swarna Bhasma (Gold)

Swarna Bhasma, prepared from purified gold through approximately 100 Puta cycles with herbal media, has been the most extensively characterised Bhasma in modern analytical and pharmacological literature. Transmission electron microscopy (TEM) and X-ray diffraction (XRD) studies have confirmed the presence of gold nanoparticles in the 25–56 nm range within authentic Swarna Bhasma preparations.[36] Sharma and colleagues demonstrated that Swarna Bhasma-derived gold nanoparticles induced apoptosis in MCF-7 breast cancer cells through mitochondrial pathway activation, confirming the classical description of Swarna Bhasma as a Rasayana with Arbuda-nashana properties.[37] The immunomodulatory effects of Swarna Bhasma — documented in animal models as enhanced macrophage activation, lymphocyte proliferation, and NK cell cytotoxicity — are consistent with its classical designation as a Tridosha-hara and Ojas-vardhaka preparation.

5.3 Rajata Bhasma (Silver)

Silver nanoparticles have established anti-cancer credentials in contemporary nanomedicine, and the classical Bhasma preparation of Rajata employs a processing route that produces nanostructured silver with comparable physicochemical characteristics.[38] Lokina and colleagues reported significant cytotoxicity of Rajata Bhasma in MCF-7 cells (IC50 in the sub-50 microgram/ml range), with apoptosis confirmed by annexin V staining and flow cytometry.[39] The mechanism involves mitochondrial membrane potential disruption and activation of the caspase cascade. A critical consideration is the dual nature of silver nanoparticle activity: at low concentrations, immunomodulatory; at higher concentrations, directly cytotoxic. Classical texts implicitly encode this dose-dependence through their emphasis on precise dosage (Matra) determination based on Prakriti and Agni status.

5.4 Trivanga Bhasma (Tin-Lead-Zinc)

Trivanga Bhasma — a trimetallic preparation of Vanga (tin), Naga (lead), and Yasada (zinc) — is of particular interest in oncological contexts both for its complexity and for the emerging evidence specifically relating to its activity in breast cancer cell models. The most current characterisation study has confirmed its nanocrystalline structure through XRD and TEM analysis.[40] Naikare and colleagues reported cytotoxicity of Trivanga Bhasma against MCF-7 cells with demonstrable IC50 values, alongside apoptosis induction confirmed by multiple downstream markers.[41] The zinc component is of particular mechanistic significance: zinc deficiency is well-established as a factor in breast cancer susceptibility and progression, and the zinc-containing nanoparticles in Trivanga Bhasma may restore zinc-dependent tumour suppressor functions including p53 stabilisation and activation.

A note of caution is warranted regarding the lead (Naga) component of Trivanga Bhasma. Lead is a known systemic toxin, and while classical Shodhana and Marana procedures are claimed to render it non-toxic and therapeutically safe, the evidence for this claim from a modern toxicological standpoint is far from comprehensive. Patgiri and colleagues have reported safety in animal studies at classical therapeutic doses, but rigorous long-term human toxicity data remain absent.[42] This represents a critical gap that must be addressed before Trivanga Bhasma can be considered for integrative oncological protocols outside of well-supervised research settings.

5.5 Other Relevant Bhasma

Tamra Bhasma (copper-based), Lauha Bhasma (iron-based), and Abhraka Bhasma (mica-based) have each demonstrated pharmacological activities potentially relevant to breast cancer management, including pro-apoptotic cytotoxicity, ferroptosis induction, and immunostimulatory effects respectively.[43] Abhraka Bhasma, which undergoes the most elaborate preparation process among the metallic Bhasma (requiring hundreds of Puta cycles according to some classical recipes), has been used traditionally for the management of Rajayakshma (classically described systemic wasting, approximating a state of immunosuppression and cachexia) — a clinical picture not dissimilar to advanced cancer cachexia. Its hematopoietic stimulatory effects documented in animal models are of particular clinical relevance as an adjunct during myelosuppressive chemotherapy.

Table 2. Bhasma Preparations: Classical Properties, Preparation Processes, and Pharmacological Evidence Relevant to Breast Cancer

Bhasma Name	Source Material	Relevant Shodhana-Marana Process	Ayurvedic Properties & Indications	Reported Pharmacological / Nano-pharmacological Actions	Relevant Preclinical / Clinical Evidence
Swarna Bhasma	Pure gold (Au)	Shodhana: quenching in oils; Marana: trituration with herbal juices, 100+ Puta cycles	Tridoshaghna, Rasayana, Medhya, Balya; indicated in Arbuda, Grahani, Kshaya, neurological disorders	Immunomodulation, induction of caspase-mediated apoptosis; Au nanoparticles (~25-56 nm); ROS scavenging, anti-inflammatory via NF-kB suppression	Sharma et al. (2017) - gold nanoparticle-mediated apoptosis in MCF-7; Upadhyay et al. (2021) - FTIR/TEM characterization confirming nanostructure; Mukherjee et al. (2020) - in vitro anti-proliferative evidence

Bhasma Name	Source Material	Relevant Shodhana-Marana Process	Ayurvedic Properties & Indications	Reported Pharmacological / Nano-pharmacological Actions	Relevant Preclinical / Clinical Evidence
Rajata Bhasma	Pure silver (Ag)	Shodhana: quenching in lemon juice/buttermilk; Marana: bhavana with herbal juices, multiple Puta	Tridoshaghna, Deepaniya, Rasayana; indicated in Arbuda, Prameha, Kshaya, Vishama Jwara	Ag nanoparticles (~10-30 nm); cytotoxic, anti-proliferative, pro-apoptotic in breast and cervical cancer lines; mitochondrial membrane disruption	Lokina et al. (2014) - significant cytotoxicity in MCF-7; Tripathi et al. (2017) - characterization confirming crystalline AgNP structure; Patra & Baek (2016) - systematic review of anticancer Ag nanoparticles
Trivanga Bhasma	Tin (Vanga), Lead (Naga), Zinc (Yasada)	Shodhana: each metal individually; Marana: combined trituration with Aloe vera bhavana, Gajaputa	Tridoshaghna, Rasayana; primarily indicated in Prameha, Shukra-dushti, Arbuda; balances Apana Vata	Synergistic multi-metal nanoparticle effects; anti-proliferative, immunomodulatory; zinc component activates p53-mediated apoptosis; tin and zinc regulate oxidative stress	Naikare et al. (2023) - cytotoxicity of Trivanga Bhasma in MCF-7 (IC50 data); Patgiri et al. (2015) - physico-chemical analytical profile; Pingale et al. (2022) - TEM/XRD confirmation of nanocrystalline structure
Tamra Bhasma	Copper (Cu)	Shodhana: successive quenching in	Kapha-Vata shamaka, Deepaniya, Lekhana,	Cu-based nanoparticles; anti-proliferative, induces	Krishnamachary et al. (2013) - copper nanoparticle

Bhasma Name	Source Material	Relevant Shodhana-Marana Process	Ayurvedic Properties & Indications	Reported Pharmacological / Nano-pharmacological Actions	Relevant Preclinical / Clinical Evidence
		oils/butter milk; Marana: bhavana with Nimbu swarasa, multiple Puta cycles	Rasayana; indicated in Gulma, Pandu, Arbuda, Kustha	mitochondrial pathway of apoptosis, anti-oxidant at low doses, pro-oxidant at higher doses in tumor cells	anti-tumor activity; Singh et al. (2020) - cytotoxic screening of Tamra Bhasma; Verma et al. (2016) - cell cycle arrest in cancer lines
Lauha Bhasma	Iron (Fe)	Shodhana: quenching in Triphala/ Kumari swarasa; Marana: bhavana with herbal acids, 100 Puta minimum	Pitta-Kapha shamaka, Deepaniya, Lekhana, Rasayana; indicated in Pandu, Arbuda, Gulma, Kshaya	Iron oxide nanoparticles; ferroptosis induction in cancer cells; anti-angiogenic, anti-proliferative; sensitizes tumor cells to oxidative stress	Kumari et al. (2018) - cytotoxic activity in cancer cell lines; Shukla et al. (2019) - characterization and standardization of Lauha Bhasma; emerging evidence for ferroptosis pathway
Abhraka Bhasma	Biotite mica	Elaborate multi-cycle Shodhana and Marana involving mica purification and hundreds of heat	Tridoshaghna, Rasayana, Medhya; indicated in Rajayakshma, Kshaya, Arbuda, Prameha; known for Vayasthapana effect	Silica and aluminum-based nanomaterials; immunomodulatory, anti-oxidant, hematopoietic stimulation; potential for sensitizing cancer cells to chemotherapy	Krishnamurthy et al. (2012) - physicochemical characterization; Halpern (2003) - traditional and modern review; Kar et al. (2021) -

Bhasma Name	Source Material	Relevant Shodhana-Marana Process	Ayurvedic Properties & Indications	Reported Pharmacological / Nano-pharmacological Actions	Relevant Preclinical / Clinical Evidence
		cycles with Kutki, Triphala bhavana			immunomodulatory screening

Note: Shodhana = purification; Marana = incineration/transformation; Puta = incineration cycle. Nanoparticle size ranges are approximate and vary with preparation batch. All pharmacological data are preclinical unless noted. FTIR = Fourier-transform infrared spectroscopy; TEM = transmission electron microscopy; XRD = X-ray diffraction. Lead (Naga) content in Trivanga Bhasma requires rigorous safety evaluation before clinical use. References cited in section 5.

6. Possible Mechanisms of Action

6.1 Nano-Pharmacological Mechanisms of Bhasma

The most compelling mechanistic explanation for Bhasma activity lies in contemporary nano-pharmacology. The Marana process — which subjects metals to repeated trituration with herbal juices (Bhavana) and incineration — is functionally equivalent to several aspects of 'top-down' nanoparticle synthesis. The resulting nanoparticles benefit from: (a) reduced particle size enabling enhanced cellular uptake via endocytosis; (b) increased surface area to volume ratio facilitating greater reactivity; (c) surface passivation by organic molecules from herbal processing media, which confer biocompatibility and may serve as targeting ligands; and (d) altered crystalline structure reducing inherent metal toxicity while preserving biological activity.[44]

The plant-derived coating on Bhasma nanoparticles — introduced during Bhavana steps — is particularly significant. These organic coatings may include polyphenols, flavonoids, alkaloids, and glycosides from the processing herbs, creating a functionalised nanoparticle surface that is chemically distinct from synthetic metallic nanoparticles produced in the laboratory. This phytochemical functionalization could explain the enhanced biocompatibility and differential toxicity (greater cytotoxicity to malignant cells, lower toxicity to normal cells) reported in some Bhasma studies — an observation that aligns with the classical claim that properly prepared Bhasma is Nishavisha (devoid of toxic properties).[45]

6.2 Apoptosis Induction

Both Rasayana phytoconstituents and Bhasma nanoparticles converge on apoptosis induction as a primary anti-cancer mechanism. The intrinsic (mitochondrial) pathway is activated through: (i) downregulation of anti-apoptotic Bcl-2 family proteins; (ii) upregulation of pro-apoptotic Bax and Bad; (iii) cytochrome c release into the cytoplasm; and (iv) activation of the Apaf-1 apoptosome, leading to caspase-9 and subsequently caspase-3 activation. The extrinsic pathway is engaged through TRAIL-R upregulation and FasL-Fas signalling. Withaferin A, silver nanoparticles from Rajata Bhasma, and gallic acid from Triphala have all been individually demonstrated to activate caspase-3, the final executioner caspase, in breast cancer cell lines.[46]

6.3 Immunomodulation

The immunomodulatory dimension of both Rasayana and Bhasma therapy is particularly important in the oncological context. Rasayana drugs classically described as Ojas-vardhaka — Ashwagandha, Shatavari, Guduchi — have been demonstrated to enhance adaptive immunity (T-lymphocyte proliferation, Th1/Th2 cytokine balance restoration) and innate immunity (NK cell activation, macrophage M1 polarisation, dendritic cell maturation) in experimental models.[47] These effects translate mechanistically to enhanced anti-tumour immune surveillance, potentially reversing the immunosuppressive tumour microenvironment that characterises breast cancer progression. Gold nanoparticles from Swarna Bhasma have been specifically reported to enhance dendritic cell maturation, a finding of direct relevance to the emerging field of nanoparticle-mediated cancer immunotherapy.

6.4 Anti-Angiogenesis and Anti-Metastatic Effects

Tumour angiogenesis — the formation of new blood vessels from pre-existing vasculature — is a rate-limiting step in solid tumour growth beyond 1–2 mm diameter. Multiple Rasayana constituents (crocin, ursolic acid, withaferin A) suppress VEGF transcription and secretion, and inhibit endothelial cell proliferation and migration in tube formation assays.[48] Anti-metastatic activity, an equally important clinical consideration, has been documented through MMP inhibition (chebulagic acid), E-cadherin upregulation (gallic acid), and inhibition of EMT markers (withaferin A inhibits Vimentin expression). These mechanisms collectively address the two most clinically significant dimensions of advanced breast cancer behaviour: local invasion and distant dissemination.

6.5 Epigenetic Modulation

An emerging and underexplored area concerns the epigenetic modulatory effects of Rasayana compounds. Sulforaphane-like constituents in Brassica-group Ayurvedic vegetables used in Rasayana protocols, as well as ellagic acid and gallic acid from Triphala, have demonstrated inhibitory effects on histone deacetylases (HDACs) and DNA methyltransferases (DNMTs) — epigenetic enzymes frequently dysregulated in breast cancer to silence tumour suppressor gene expression.[49] The classical concept of *Prajna-aparadha* (intellectual transgression leading to disease) implies at some level a departure from inherent biological programming — a metaphor that maps, however loosely, to the modern understanding of epigenetic dysregulation as a driver of malignant transformation. This is admittedly a speculative connection, but one that invites systematic investigation.

6.6 The Agni-Metabolism Nexus

A mechanistic bridge between classical Ayurvedic pathogenesis (Agni mandya → Ama formation → Srotas blockade → Arbuda) and modern oncological metabolism is emerging through mitochondrial biology. Impaired mitochondrial function — characteristic of the Warburg effect (aerobic glycolysis) that defines cancer cell metabolism — parallels the classical description of compromised Jatharagni (primary digestive metabolism) leading to Ama (incompletely transformed, metabolically aberrant products) accumulation. Rasayana drugs that restore Agni — described classically as Deepana and Pachana — may mechanistically correspond to agents that restore mitochondrial oxidative phosphorylation, reduce lactate production, and modulate the Warburg effect, thereby targeting a fundamental metabolic vulnerability of breast cancer cells.[50]

7. Integrative Perspective: Ayurveda and Modern Oncology

7.1 Rationale for Integration

The integration of Ayurvedic therapeutics within modern oncological care is neither a new idea nor an unrealised aspiration. In India, an estimated 60–80% of cancer patients report concurrent use of traditional or complementary therapies alongside conventional treatment, a figure that underscores the practical reality clinicians must engage with regardless of academic position.[51] The rationale for formalised integrative oncology extends beyond patient preference: the mechanistic congruence between Rasayana and Bhasma activities and the molecular targets of breast cancer, the potential for chemosensitisation, the immunostimulatory properties, and the quality-of-life benefits documented in pilot studies together constitute a genuine scientific basis for structured integration.

7.2 Potential Clinical Applications

The clinical roles most logically suited to Ayurvedic intervention in the breast cancer treatment continuum include: (a) neo-adjuvant context — Shodhana therapies (Panchakarma) to optimise the internal milieu and reduce atherogenic lipid accumulation (Meda Dhatu dushti) before surgery; (b) concurrent adjuvant context — Rasayana preparations to mitigate chemotherapy-related myelosuppression, mucositis, fatigue, and immunosuppression; (c) maintenance context — Rasayana protocols during surveillance period to support immune reconstitution, prevent recurrence, and restore Dhatu integrity; and (d) palliative context — specific formulations to address pain (via Vedanasthapana drugs), cachexia (via Balya Rasayana), and psychological distress (via Medhya Rasayana and Satvavajaya chikitsa).[52]

7.3 Evidence Hierarchy and Current Clinical Trial Landscape

The evidence base for Ayurvedic interventions in breast cancer currently sits predominantly at the level of in vitro and animal studies (Level IV-V evidence by Oxford CEBM criteria), with a small number of open-label pilot trials and observational studies.[53] The Clinical Trials Registry of India (CTRI) lists several registered trials examining Ayurvedic adjuncts in cancer management, though completed, published trials with adequate power are limited. The AYUSH-sponsored clinical trial on Ashwagandha in chemotherapy-induced neutropenia represents a methodologically noteworthy effort, though its scope does not extend to survival outcomes.[54] International registries (ClinicalTrials.gov) list trials examining standardised Ashwagandha and Triphala preparations in oncological contexts, primarily as supportive care interventions.

A critical methodological challenge in Ayurvedic clinical trials is the impossibility of double-blinding when practitioner-specific individualised treatment (Vikriti-based prescription) is employed. Pragmatic trial designs acknowledging this constraint — while employing validated patient-reported outcome measures, biomarker endpoints, and independent adjudication — are more appropriate than attempting to force Ayurvedic practice into a framework it was not designed to fit.[55]

7.4 Safety Considerations and Herb-Drug Interactions

A responsible integrative oncological perspective must engage seriously with safety. Known concerns include: (a) CYP450 enzyme interactions — several Rasayana constituents (including piperine from Maricha and Pippalimula, commonly added to enhance bioavailability) inhibit CYP3A4 and CYP2D6, potentially elevating plasma levels of concurrently administered chemotherapeutic agents metabolised by these enzymes; (b) hepatotoxicity — rare cases of herb-induced liver injury associated with high-dose Ashwagandha have been documented, though causality is confounded by concurrent medication use; (c) Bhasma safety — particularly concerning heavy metal content in Trivanga Bhasma (lead), Tamra Bhasma (copper), and Lauha Bhasma (iron) in cases of improperly prepared or adulterated preparations.[56] The principle that all Bhasma must comply with pharmacopoeial quality standards as specified in the Ayurvedic Pharmacopoeia of India (API) before clinical use is non-negotiable.

8. Discussion

8.1 Critical Synthesis

This review has attempted to navigate two distinct but interacting knowledge systems — classical Ayurvedic epistemology and contemporary biomedical research — without reducing either to a mere corroborating footnote for the other. The classical Ayurvedic account of Arbuda offers a systems-level understanding of carcinogenesis rooted in metabolic dysfunction (Agni mandya), toxic accumulation (Ama), channellar pathology (Srotas dushti), and immune failure (Oja-kshaya) that, while not identical to the molecular hallmarks framework of Hanahan and Weinberg, shares conceptual architecture at a level that cannot be dismissed as coincidental.[57]

The evidence for Rasayana and Bhasma efficacy is real but currently insufficient for standalone therapeutic claims. The most productive framing is not whether Ayurveda 'cures' breast cancer — an epistemologically inappropriate question within both traditions — but whether specific, well-characterised Ayurvedic preparations can meaningfully contribute to defined outcomes within an integrative oncological framework: reducing toxicity of conventional treatment, enhancing immune function, improving quality of life, potentially extending disease-free survival. The evidence, while predominantly preclinical, is mechanistically coherent enough to justify rigorous clinical investigation.

8.2 Gaps in Current Research

Several critical gaps impede translation of the existing evidence base into clinical practice. First, the absence of pharmacopoeially standardised, authenticated Bhasma preparations in published studies makes cross-study comparison unreliable. Batch-to-batch variability in Bhasma — driven by differences in raw material quality, Shodhana procedure, Marana cycle number, and Bhavana media — may account for the wide variation in reported IC50 values and biological activities.[58]

Second, bioavailability and pharmacokinetic data for Bhasma in human subjects are essentially absent. The classical route of administration (oral, with anupana such as ghee, honey, or milk) and the proposed nano-pharmacological mechanism of action need to be reconciled within a rigorous pharmacokinetic framework to determine which form of nanoparticle reaches tumour tissue and at what concentration. Third, in vitro studies overwhelmingly employ immortalised cancer cell lines at supraphysiological concentrations, a methodological limitation that has undermined the translational value of much phytotherapy research broadly, and is equally applicable here.

Fourth, from a classical Ayurvedic standpoint, the current body of research entirely omits the individualised Prakriti-based prescriptive dimension of Ayurvedic therapy. Mounting evidence in pharmacogenomics suggests that individual genetic variation significantly modifies drug response — a biological substrate that may partially underpin the classical concept of Prakriti-specific therapeutic variation. Studies that stratify patients by validated Prakriti categories (Vata, Pitta, Kapha predominance) alongside molecular breast cancer subtypes would represent a genuinely novel and scientifically significant research paradigm.[59]

8.3 Future Research Directions

Based on the present review, the following research directions are identified as priorities. First, prospective, adequately powered, randomised clinical trials of standardised Rasayana preparations (particularly Ashwagandha, Triphala, and Guduchi) as adjuncts to standard chemotherapy in early breast cancer should be designed using patient-reported quality-of-life outcomes and validated biomarkers (NK cell activity, cytokine profiles, neutrophil-lymphocyte ratio) as primary endpoints.[60]

Second, a systematic nano-characterisation programme for the principal anticancer Bhasma (Swarna, Rajata, Trivanga) using standardised protocols (TEM, XRD, FTIR, ICP-MS for heavy metal quantification, dynamic light scattering for zeta potential) should be established to create a reference database enabling meaningful inter-study comparison. Third, mechanism-based studies using patient-derived organoids or three-dimensional tumour spheroid models — which better recapitulate the tumour microenvironment — would significantly enhance the translational validity of Bhasma cytotoxicity data.[61]

Fourth, Prakriti-stratified pharmacogenomic studies examining gene expression variation relevant to drug metabolism and immune function across Prakriti types could begin to build a scientific bridge between individualised Ayurvedic prescriptive logic and personalised oncology. Fifth, the safety and pharmacokinetics of the most promising Bhasma preparations in healthy volunteers and cancer patients must be rigorously characterised in Phase I trials before any efficacy claims can be advanced. Finally, computational approaches including molecular docking, systems biology network analysis, and multi-omics integration offer powerful tools for identifying the molecular targets of multi-constituent Rasayana formulations that have resisted single-compound analysis.[62]

9. Conclusion

Ayurvedic management of breast cancer, centred on Rasayana chikitsa and Bhasma therapy, represents a therapeutically coherent, mechanistically grounded, and historically deep tradition that merits serious engagement from the scientific and oncological communities. The classical conceptualisation of Arbuda as a systemic disease arising from Agni compromise, Ama accumulation, and Ojas depletion offers a genuinely distinct and potentially complementary explanatory framework to molecular oncology's hallmarks model. Rasayana drugs — operating through apoptosis induction, immunomodulation, NF-kB suppression, anti-angiogenesis, and epigenetic modulation — demonstrate pharmacological profiles that are mechanistically relevant to the specific vulnerabilities of breast cancer biology. Bhasma preparations, reinterpreted through the lens of nano-pharmacology, offer a uniquely Ayurvedic contribution to the expanding field of nanoparticle-mediated cancer therapy.

However, the current evidence base, while scientifically promising, remains predominantly preclinical and insufficient to support definitive clinical recommendations without further rigorous investigation. The path forward requires: rigorous pharmacopoeial standardisation of Bhasma preparations; prospective clinical trials employing appropriate pragmatic designs; mechanistic studies using clinically relevant cancer models; systematic safety and pharmacokinetic characterisation; and cross-disciplinary collaboration between Ayurvedic scholars, oncologists, pharmacologists, and nanoscientists. Only through this integrated research commitment can the promise of Rasayana and Bhasma therapy be responsibly and effectively translated from the classical text to the clinical oncology ward.

Ultimately, integrative oncology — practiced at its best — demands neither the abandonment of classical Ayurvedic epistemology nor uncritical acceptance of its claims. It demands precisely the kind of critical, respectful, and methodologically rigorous inquiry that this review has attempted to model.

CRediT Author Statement

The following statement uses CRediT (Contributor Roles Taxonomy) standard categories. Initials represent co-authors as designated in the title page (submitted separately). **TSN:** Conceptualisation, classical textual analysis, Ayurvedic clinical interpretation, manuscript writing (Sections 2, 4, 5, 9), critical revision. **[Co-author 2 initials]:** Modern oncological literature review, data curation, manuscript writing (Sections 3, 6, 7), table preparation. **[Co-author 3 initials]:** Nano-pharmacological analysis, Bhasma characterisation review, manuscript writing (Sections 5, 6.1), critical revision. **[Co-author 4 initials]:** Reference compilation, formal analysis, supplementary data management, final editing.

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