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TRIPHALA AS A MULTI-TARGET ANTICANCER CANDIDATE: AN INTEGRATIVE NARRATIVE REVIEW FROM CLASSICAL AYURVEDIC LITERATURE TO CONTEMPORARY EXPERIMENTAL ONCOLOGY

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ABSTRACT **Background:** Triphala a classical Ayurvedic polyherbal formulation composed of Emblica officinalis (Phyllanthus emblica), Terminalia bellirica, and Terminalia chebula, is traditionally classified as a rasayana with rejuvenative, restorative, and disease-modifying properties. Although not described in classical Ayurvedic texts as a modern "anticancer drug," its traditional indications in tissue restoration, wound healing, ophthalmic disorders, metabolic dysfunction, and constitutional strengthening have stimulated modern interest in its antineoplastic potential. [4,5] **Objective:** To critically review the anticancer effectiveness of Triphala by integrating evidence from Ayurvedic texts and contemporary biomedical literature. **Methods:** This narrative review synthesizes evidence from Ayurveda sources, including Charaka Samhita, Sushruta Samhita, and Astanga Hridaya, together with peer-reviewed studies published in biomedical and integrative medicine journals indexed in major scholarly databases. [1-5] **Results:** Preclinical studies consistently show that Triphala exerts selective cytotoxicity against malignant cells while sparing normal cells, principally through reactive oxygen species–mediated apoptosis, p53/ERK signaling, modulation of Bax/Bcl-2 balance, suppression of c-Myc/cyclin D1, inhibition of VEGF/VEGFR-2–mediated angiogenesis, perturbation of microtubule dynamics, and downregulation of PI3K/Akt/mTOR and NF-kB–related pathways. Activity has been reported in breast, pancreatic, colon, cervical, ovarian, endometrial, and lymphoma models. However, direct human evidence for anticancer efficacy remains insufficient. Clinical evidence currently supports safety and adjunctive supportive-care benefits, such as mitigation of radiation-induced oral mucositis, rather than tumor regression. [6-12,14,15] **Conclusion:** Triphala is a biologically plausible, multi-target phytopharmaceutical candidate with substantial preclinical anticancer promise and strong classical rasayana foundations. Nevertheless, current evidence does not justify its recommendation as a stand-alone anticancer therapy. Standardized preparations, pharmacokinetic work, herb–drug interaction studies, and rigorously designed clinical trials are required before translational oncology use can be endorsed. [1,4-15]

KEYWORDS : Triphala; Ayurveda; Rasayana; Cancer; Integrative Oncology

INTRODUCTION

Triphala is most widely used polyherbal formulations in Ayurveda and Triphala Churna prepared from the dried fruits of *Haritaki* (*Terminalia chebula*), *Bibhitaki* (*Terminalia bellirica*), and *Amalaki* (*Phyllanthus emblica*) in equal proportion. Triphala is primarily as a *rasayana* rather than as a specific remedy for neoplastic disease. Modern oncology-oriented research has nonetheless become increasingly interested in Triphala because it demonstrates selective cytotoxicity in cancer cells, pleiotropic signaling effects, anti-inflammatory and antioxidant/pro-oxidant duality, and possible chemopreventive value. The central question is no longer whether Triphala is pharmacologically active, but whether its activity is sufficiently robust, reproducible, and clinically translatable to justify an evidence-based anticancer role. [4-6,14]

From an Ayurvedic perspective, modern cancer cannot be simplistically equated with a single classical disease entity, though concepts such as *arbuda* and *granthi* are often invoked in interpretive oncology literature. What is more directly relevant is that classical texts position Triphala at the interface of rejuvenation, tissue repair, metabolic regulation, and constitutional resilience—domains highly relevant to carcinogenesis, chronic inflammation, and host response. This makes Triphala especially suitable for integrative review: its traditional rationale supports restorative and preventive use, while modern studies explore direct tumoricidal mechanisms. [1-3]

Classical Ayurvedic Foundations

In *Charaka Samhita* (Chikitsa Sthana, Rasayana Adhyaya, Part III, verses 41–47), Triphala Rasayana is described in several formulations. The text claims that regular administration promotes longevity and freedom from *jara* (senility) and *vyadhi* (disease), while some variants are said to improve *medha* (intellect), *smriti* (memory), and *bala* (strength). These statements should not be misread as direct proof of anticancer efficacy; rather, they establish Triphala's classical status as a rejuvenative and disease-resisting formulation, which is conceptually important for contemporary chemopreventive and host-support paradigms. [1]

In *Astanga Hridaya*, Triphala is explicitly described as "rasayanavara" and "akshyamayapaha", that is, an excellent rejuvenator and a remedy for diseases of the eyes; the same passage also attributes wound-healing and skin-disease–ameliorating properties to the formulation. In *Sushruta Samhita*, Triphala appears in formulations for ulcer purification, wound cleansing, healing oils, and local therapeutic washes. Collectively, these references frame Triphala as a formulation with restorative, detoxifying, and tissue-healing roles—properties that may underlie its present-day exploration in inflammation-associated carcinogenesis, mucosal toxicity, and cancer supportive care. [2,3]

Review Methodology

This manuscript is structured as a narrative review suitable for journal development rather than a PRISMA-compliant systematic review. Classical evidence was drawn from *Charaka Samhita*, *Sushruta Samhita*, and *Astanga Hridaya*. Modern evidence was synthesized from peer-reviewed review articles, PubMed-indexed primary studies, and oncology/integrative medicine journals reporting mechanistic, in vitro, in vivo, and clinical findings on Triphala. Emphasis was placed on studies addressing cancer-cell selectivity, apoptosis, angiogenesis, microtubule biology, colon cancer stem cells, pancreatic xenografts, gynecological cancer models, safety, and clinical supportive care. [1-15]

Phytochemical Rationale for Anticancer Activity

Triphala is chemically rich in tannins, polyphenols, flavonoids, phenolic acids, and related bioactives. Frequently cited constituents include gallic acid, ellagic acid, chebulinic acid, chebulagic acid, quercetin, naringin, homoorientin, and isorhamnetin. The anticancer interest in Triphala arises from its unusually broad mechanism profile. Unlike single-target cytotoxics, Triphala appears to exert systems-level pressure on malignant cells through oxidative stress modulation, apoptosis induction, antiangiogenic action, suppression of proliferative signaling, and interference with cytoskeletal dynamics. [4,5,14]

A recurrent mechanistic theme is selective pro-oxidant stress in tumor cells. In several models, Triphala elevates intracellular reactive oxygen species in malignant cells while sparing normal cells, leading to DNA damage responses and apoptosis. This is mechanistically attractive because malignant cells often operate near redox threshold limits, making them more vulnerable to additional oxidative burden. Other reported mechanisms include p53 activation, ERK phosphorylation, elevation of the Bax/Bcl-2 ratio, p53-independent apoptosis, suppression of c-Myc and cyclin D1, inhibition of VEGFR-2 phosphorylation, and persistent disruption of microtubule assembly dynamics. [6-12,14]

Preclinical Anticancer Evidence

a) Breast Cancer and Lymphoma models

Aqueous Triphala reduced viability in MCF-7 breast cancer cells and in a transplantable mouse thymic lymphoma model while sparing normal breast epithelial cells and normal murine tissues. Increased

annexin-V positivity, apoptotic DNA damage, and concentration-dependent reactive oxygen species generation supported apoptosis as a core mechanism. In vivo oral administration also reduced tumor volume in lymphoma-bearing mice, suggesting that Triphala's effects were not limited to cell culture systems. [6]

A breast-cancer study comparing MCF-7 and T47D cells demonstrated that MCF-7 cells were more sensitive to Triphala than p53-deficient T47D cells. Antioxidants such as glutathione and N-acetylcysteine attenuated the antiproliferative effect, strengthening the argument for ROS-dependent apoptosis. They suggest that tumor genotype, especially p53 status, may influence responsiveness to Triphala-based interventions. [7]

b) Pancreatic Cancer

Growth inhibition in pancreatic cancer cell lines and significant suppression of Capan-2 xenograft growth in nude mice after oral administration. Mechanistically, Triphala-induced apoptosis was linked to ROS generation and activation of ERK and p53 signaling; blockade of oxidative stress or pathway inhibition attenuated the effect. Notably, normal human pancreatic ductal epithelial cells were not similarly damaged, again supporting selective cytotoxicity. [8]

c) Colon Cancer and Cancer Stem Cells

Methanolic Triphala extract suppressed proliferation in HCT116 colon cancer cells and human colon cancer stem cells, independent of p53 status. It inhibited colony formation, reduced c-Myc and cyclin D1 expression, increased the Bax/Bcl-2 ratio, and promoted PARP cleavage. Because cancer stem cells are implicated in recurrence, metastasis, and treatment resistance, these findings raise the possibility that Triphala may function as a chemopreventive or adjuvant rather than merely a nonspecific cytotoxin. Still, the lack of in vivo validation in this study remains a major limitation. [10]

d) Gynecological Cancers

In ovarian, cervical, and endometrial cancer cell lines, Triphala inhibited proliferation and induced apoptosis with associated downregulation of MAPK/ERK, PI3K/Akt/mTOR, and NF-kB/p53-related signaling. This work combined network pharmacology with experimental validation, reflecting the multi-component, multi-target behavior expected of a classical polyherbal formulation. Yet translation remains preliminary because the evidence is entirely preclinical. [11]

e) Antiangiogenic and Microtubule-targeting Effects

Triphala and its constituent chebulinic acid inhibit VEGF-induced angiogenesis by suppressing VEGFR-2 phosphorylation. Since angiogenesis is indispensable for tumor progression and metastasis, this finding provides a plausible systems-level explanation for Triphala's in vivo antitumor activity. [9]

Another mechanistic study showed that aqueous Triphala extract perturbs tubulin conformation, disrupts the microtubule network, stabilizes microtubule dynamics abnormally, and impairs microtubule reassembly in HeLa, PANC-1, and MDA-MB-231 cells, culminating in programmed cell death. This is especially interesting because microtubules remain one of the most clinically validated anticancer targets in modern chemotherapy. [12]

Human Evidence, Safety, and Translational Gaps

The major weakness in the Triphala-oncology literature is the absence of convincing human trials showing direct anticancer efficacy. A major 2020 review explicitly noted that, despite abundant experimental evidence, no clinical trial had yet established Triphala as an anticancer treatment. This remains the decisive barrier between pharmacological promise and clinical recommendation. [14]

In a phase I trial in healthy volunteers, oral aqueous Triphala extract at 2500 mg/day for 4 weeks produced no serious adverse effects. In cancer care, a retrospective study in head and neck cancer patients receiving radiotherapy found that Triphala combined with povidone iodine delayed and reduced oral mucositis, lessened weight loss, and reduced treatment breaks, but did not improve tumor response. That distinction matters: current clinical evidence supports Triphala better as a supportive integrative intervention than as a proven tumor-directed therapy. [13,15]

Additional translational concerns include variability in Triphala raw materials, lack of universally accepted extraction standards, uncertain oral bioavailability of multiple phytochemicals, limited pharmacokinetic data, possible herb–drug interactions during chemotherapy, and heterogeneity between churna, aqueous extract, methanolic extract, and constituent-focused experiments. The oncology field cannot assume that results from one extract are automatically generalizable to traditional churna preparations. [4,5,13,14]

DISCUSSION

The evidence reviewed here supports three conclusions. First, Triphala has a genuine and coherent classical Ayurvedic rationale: its established identity as a *rasayana*, wound-healing, and tissue-restorative formulation makes modern oncologic investigation intellectually defensible. Second, Triphala displays unusually broad preclinical anticancer plausibility, including selective apoptosis, antiangiogenesis, suppression of proliferative signaling, and microtubule disruption. Third, and most important, the current literature still falls short of proving clinical anticancer effectiveness in humans. [1-3,6-15]

A balanced interpretation, therefore, is that Triphala should presently be viewed as a promising adjuvant, chemopreventive, and supportive-care candidate, not as a validated stand-alone anticancer drug. The most rational development path would involve standardized Triphala preparations, mechanistically stratified animal studies, formal toxicology, drug-interaction work, biomarker-guided phase I/II oncology trials, and trials separating endpoints such as tumor regression, progression-free survival, mucosal protection, treatment tolerance, inflammatory markers, and quality of life. Without such rigor, Triphala risks being either overhyped by enthusiasts or prematurely dismissed by skeptics. [13-15]

CONCLUSION

Triphala occupies a rare position at the intersection of classical *rasayana* doctrine and contemporary multi-target oncology research. Ayurvedic consistently portray it as a rejuvenative, disease-mitigating, wound-healing, and constitutional strengthening formulation. Modern experimental studies support significant anticancer activity across several models, mediated by selective oxidative stress, apoptosis induction, antiangiogenesis, suppression of oncogenic signaling, and microtubule dysfunction. Yet clinical evidence required to claim direct anticancer effectiveness in humans is still lacking. At present, the most evidence-based position is that Triphala is a high-potential investigational adjunct whose preclinical strength justifies further translational and clinical study, but whose anticancer claims should remain cautious until confirmed in well-designed human trials. [1-3,6-15]

REFERENCES

1. Agnivesha. *Charaka Samhita*. Chikitsa Sthana, Rasayana Adhyaya, Karaprachitiya Rasayana Pada, verses 41-47. Singh RH, Sodhi JS, Dixit U, translators. Charak Samhita Research, Training and Skill Development Centre; 2020.
2. Sushruta. *An English translation of the Sushruta Samhita*. Vol II. Bhishagratna KK, translator.
3. Vagbhata. *Astanga Hridaya Sutrasthan handbook*. https://www.planetayurveda.com/ayurveda-ebooks/astanga-hridaya-sutrasthan-handbook.pdf
4. Peterson CT, Denniston K, Chopra D. Therapeutic uses of Triphala in Ayurvedic medicine. *J Altern Complement Med*. 2017;23(8):607-14. doi:10.1089/acm.2017.0083
5. Baliga MS, Meera S, Mathai B, Rai MP, Pawar V, Palatty PL. Scientific validation of the ethnomedicinal properties of the Ayurvedic drug Triphala: a review. *Chin J Integr Med*. 2012;18(12):946-54. doi:10.1007/s11655-012-1299-x
6. Sandhya T, Lathika KM, Pandey BN, Mishra KP. Potential of traditional ayurvedic formulation, Triphala, as a novel anticancer drug. *Cancer Lett*. 2006;231(2):206-14. doi:10.1016/j.canlet.2005.01.035
7. Sandhya T, Mishra KP. Cytotoxic response of breast cancer cell lines, MCF 7 and T 47 D to Triphala and its modification by antioxidants. *Cancer Lett*. 2006;238(2):304-13. doi:10.1016/j.canlet.2005.07.013
8. Shi Y, Sahu RP, Srivastava SK. Triphala inhibits both in vitro and in vivo xenograft growth of pancreatic tumor cells by inducing apoptosis. *BMC Cancer*. 2008;8:294. doi:10.1186/1471-2407-8-294
9. Lu K, Chakroborty D, Sarkar C, Lu T, Xie Z, Liu Z, et al. Triphala and its active constituent chebulinic acid are natural inhibitors of vascular endothelial growth factor-A mediated angiogenesis. *PLoS One*. 2012;7(8):e43934. doi:10.1371/journal.pone.0043934
10. Vadde R, Radhakrishnan S, Reddivari L, Vanamala JKP. Triphala extract suppresses proliferation and induces apoptosis in human colon cancer stem cells via suppressing c-Myc/Cyclin D1 and elevation of Bax/Bcl-2 ratio. *Biomed Res Int*. 2015;2015:649263. doi:10.1155/2015/649263
11. Zhao Y, Wang M, Tsering J, Li H, Li S, Li Y, et al. An integrated study on the antitumor effect and mechanism of Triphala against gynecological cancers based on network pharmacological prediction and in vitro experimental validation. *Integr Cancer Ther*. 2018;17(3):894-901. doi:10.1177/1534735418774410
12. Cheriyamundath S, Mahaddalkar T, Save SN, Choudhary S, Lopus M. Aqueous extract of Triphala inhibits cancer cell proliferation through perturbation of microtubule assembly dynamics. *Biomed Pharmacother*. 2018;98:76-81. doi:10.1016/j.biopha.2017.12.022
13. Phetkate P, Kummalue T, Rinthong PO, Kietinun S, Sriyakul K. Study of the safety of oral Triphala aqueous extract on healthy volunteers. *J Integr Med*. 2020;18(1):35-40. doi:10.1016/j.joim.2019.10.002

14. Prasad S, Srivastava SK. Oxidative stress and cancer: chemopreventive and therapeutic role of Triphala. *Antioxidants (Basel)*. 2020;9(1):72. doi:10.3390/antiox9010072
15. Rao S, Kini V, Hegde SK, Meera S, Rao P, George T, Baliga MS. Ayurvedic drug Triphala in combination with providone iodine mitigates radiation-induced mucositis in head and neck cancer patients without affecting the tumor response. *Indian J Otolaryngol Head Neck Surg*. 2023;75(3):1480-9. doi:10.1007/s12070-023-03516-8