

Integrating Ayurveda and Herbal Medicines into Cancer care- An Evidence based Review

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Abstract

Background: Cancer is one of the major causes of morbidity and mortality worldwide. Though there is greater advancement in diagnostics and cancer therapeutics, high treatment costs, limited accessibility, and long-term adverse effects of conventional therapies have urged the integration of various complementary approaches. Ayurveda, a holistic traditional medical system, has no clear description of cancer but describes conditions with similar clinical manifestations such as *Granthi*, *Arbuda*, *Apachi*, and employs herbal and herbo-mineral formulations aimed at restoring metabolic balance, immunity, and quality of life.

Objective: : To critically summarize Ayurvedic concepts relevant to cancer and evaluate contemporary experimental evidence supporting the role of Ayurvedic herbal medicines as complementary interventions in cancer care.

Methods: A narrative evidence-based literature review focusing on the Peer-reviewed experimental studies (in-vitro) published in the last five years was conducted. The search was done from the databases like PubMed and Scopus using predefined keywords and MeSH terms related to anticancer activity and herbal medicines. Classical Ayurvedic textbooks (*Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*) were also referred to identify herbs traditionally indicated for *Arbuda*, *Granthi*, and related disorders. Priority was given to peer-reviewed experimental studies, translational research, clinical investigations, and recent review articles.

Results: This review provides a strong experimental evidence for the anticancer activity of various herbs found in classical Ayurvedic texts. These herbs exhibited cytotoxic, anti-proliferative, pro-apoptotic, anti-inflammatory, anti-metastatic and immunomodulatory activities in a wide spectrum of cancer cell lines and animal models. Key mechanisms included apoptosis mediated by mitochondria, activation of caspases, cell-cycle arrest, modulation of oncogenes and tumor suppressor gene genes (TP53), inhibition of NF-κB signaling and reduction of oxidative stress.

Conclusion:

The findings provide scientific validation for several Ayurvedic herbs that traditionally support their role as complementary or adjuvant agents in the management of cancer. The use of established modern cancer therapies in combination with traditional herbal medicines may enhance therapeutic efficacy, decrease treatment-related toxicity and improve quality of life. Detailed clinical research is needed to translate these experimental findings into evidence-based practices of integrative oncology.

Key words: Cancer, Herbal medicines, Complementary therapy, Cytotoxic effect, Anti cancer

How to cite this article: Sarika A. Patil^{1*} Dr. Madhuri Pawar². Integrating Ayurveda and Herbal Medicines into Cancer care- An Evidence based Review. Int J Drug Deliv Technol. 2026;16(7):466-479. DOI: 10.25258/ijddt.16.7.50.

1. Introduction

Cancer is one of the major health concern and contributes substantially to the disease burden and mortality. Reports from GLOBOCAN 2025 indicate around 9.96 million deaths due to cancer have occurred in the year 2025 and the burden of cancer will continue to increase in the upcoming decades due to aging populations, urbanization, and alteration of lifestyles.(1) As per reports from ICMR (Indian Council of Medical Research), cancer affects about 2.5 million individuals in India and 7,00,000 new cancer cases are diagnosed every year in India.(2) The burden of cancer is continuously increasing and creating substantial pressure on the health care system, calling for affordable, efficacious and patient-oriented therapeutic approaches.(3)

During the recent decades, there has been tremendous progress in diagnosis and treatment modalities of cancer that mainly include precision

medicine, molecularly-targeted therapy, immunotherapy, radiotherapy, and drug delivery using nanotechnology. These varied treatments have improved the prognosis for many cancers.(4,5)However, these treatments are associated with major limitations in terms of treatment toxicity, multidrug resistance, disease recurrence, high cost of treatment and unequal access to health care facilities, especially in under developed and developing countries.(4) Consequently, increasing focus has been laid upon complementary treatment modalities that can help in symptom management, treatment-related side effects reduction, and improvement in quality of life of patients when used alongside oncologic treatment.(6)

Integrative oncology has emerged as a patient-oriented strategy of integrating conventional cancer treatments with complementary interventions that have a basis in scientific evidence. Herbal medicines

have gained significant popularity among complementary intervention strategies due to presence of various bioactive chemicals that have the ability to target multiple pathways associated with carcinogenesis like oxidative stress, inflammation, apoptosis, immune regulation, apoptosis and metastasis.

Ayurveda, an age old medicine being practiced in India since more than two epochs, takes into account a holistic approach to treat any ailment. Ayurveda lays emphasis on achieving physiological balance by adopting personalized approach, including diet, lifestyle modifications, herbal medicines and rejuvenation therapies. Though cancer is not mentioned as a different disease entity in Ayurvedic classics, but diseases like *Arbuda*, *Granthi*, and *Apachi* are characterized by symptoms and features that match with benign and malignant conditions in terms of Ayurvedic classics. It is crucial to understand that these represent conceptual similarities and not biomedical equivalents. In Ayurveda, therapeutic intervention is done to restore the systemic imbalance, build up host resilience, and maintain the tissue homeostasis through multimodal interventions including herbal preparations with immunomodulatory, anti-inflammatory, antioxidant and possibly anticancer properties.

Increasing research has taken place during recent decades to explore the anticancer potential of medicinal plants mentioned in Ayurvedic literature. Many studies have found that herbs impact the diverse cellular and molecular pathways associated with cancer progression, including induction of apoptosis, cell-cycle arrest, prevention of metastasis and angiogenesis, regulation of inflammatory signaling, and immune modulation.

The aim of the present evidence-based narrative review is to correlate the classical concepts of Ayurveda related to cancer with modern scientific evidence concerning Ayurvedic herbal medicines.

Ayurvedic Views on Cancer

Cancer cannot be identified as a separate and unique entity in the classical Ayurvedic texts. Instead of the identifying diseases solely on the basis of nomenclature, Ayurveda focuses on understanding the pathophysiologic process (*samprapti*) of diseases in accordance with *Dosha*, *Dhatu*, *Mala*, *Agni*, *Srotas*, and *Prakriti*. In the view of *Acharya Charaka*, the number of diseases is countless because of their varied manifestations depending on causative factors and patient's personal characters. Thus, the management of the disease is based not on its label but on the nature of the pathophysiological process.(7)

From the list of diseases described in *Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*, *Arbuda*, *Granthi*, and *Apachi* have gained much attention regarding the neoplastic disorder. While *Granthi* is defined as a localized swelling due to the derangement of the *Dosha* in *Mamsa* (muscle),

Meda (adipose), or *Rakta* (blood) tissues, *Arbuda* represents a big, deep-seated, slowly-growing, hard, painless mass with gradual increasing in size.(8) The description of *Apachi* involves a glandular swelling mainly in the neck region.(9) Although these conditions show some clinical similarities with benign and malignant tumors, they cannot be considered as biomedical equivalents of cancer in the present days. However, they serve as a conceptual model for abnormal tissue growth and progression of diseases in Ayurveda.

In terms of the disease development, Ayurveda assumes disturbances in the system's homeostasis. The improper eating habits (*Aharaja Nidana*), unhealthy lifestyle (*Viharaja Nidana*), environmental factors, and psychological stress (*Manasika nidana*) impair the process of *Agni* (digestive fire) and result in *Ama*. This represents a pathological condition indicating the existence of unprocessed metabolic products. The prolonged presence of *Ama* leads to the derangement of *Tridosha* (*Vata*, *Pitta*, and *Kapha*) that causes dysfunction of *Dhatus* and *Srotas*, which in turn causes progression of the tissue pathology.(10) These principles are not similar to any particular biological mechanism, however, can be regarded as conceptual counterparts to the mechanisms of chronic inflammation, metabolic disorder, oxidative stress, immune dysfunction, and changes in the tissue microenvironment known to promote cancer development in modern biomedical science.

Among *tridoshas*, *Kapha* has a relation to the excessive growth and proliferation of the cells, *Pitta* – to the metabolism and inflammation, and *Vata* – to the invasion, dissemination, and progression of diseases. This results in the disturbance of the tissue homeostasis and physiology. However, these associations are merely interpretative and intended for interdisciplinary communication rather than establishment of biological equivalence.(11)

Thus the management of the conditions resembling neoplastic diseases in Ayurvedic medicine includes a comprehensive and individualized approach based on the restoration of physiological homeostasis instead of the targeted tumour therapy. According to classical treatment principles, the approaches include *Nidana Parivarjana* (elimination of causative factors), *Shodhana* (bio-purificatory therapies), *Shamana* (palliative measures), *Rasayana* (rejuvenation therapy aimed at the enhancement of the host resistance), and alteration in the diet and lifestyle.(12) Herbal medicines play a central part in the Ayurvedic treatment as many of them have anti-inflammatory, antioxidant, immunomodulatory, and tissue protective effects. The medicinal plants described in the Ayurvedic literature are studied for its various anti cancer pathways like induction of apoptosis, inhibition of cell multiplication and metastasis, modulation of various inflammatory signalling pathways,

managing oxidative stress and immune response in the body .

Ayurveda offers a holistic and personalized approach in accordance with the current integrative oncology approach, which can act as an adjuvant to conventional cancer therapy by reducing the symptom burden , improving the quality of life, and immunity of the body and reducing the untoward effects of cancer therapy. Nevertheless, scientific research in support of many Ayurvedic medicinal herbs is expanding, although clinical confirmation is still lacking. Thus, the integration of ayurvedic treatments in conventional oncology needs to be supported by scientific evaluations, standardization of herbal products, pharmacovigilance and rigorous clinical research.

Materials and Methods

3.1 Design of the study

This paper presents a narrative evidence-based review that meticulously analyses and synthesises available scientific data on Ayurvedic herbal treatments potentially applicable in integrated cancer therapy. This analysis combines traditional Ayurvedic references with modern biological research to provide an in-depth exploration of the mechanisms, therapeutic potential, and evidential foundation of medicinal herbs in cancer treatment.

3.2 Strategy for literature search

An extensive literature search was performed utilizing the databases PubMed, Scopus and Google scholar for publications published in the last five years. Further publications were identified by manual screening of references of included articles. For the study of medicinal plants and therapeutic principles of their use in the treatment of *Arbuda*, *Granthi* and *Apachi*, classical Ayurvedic texts such as *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga hridaya* were examined.

The literature search was done using MeSH terms and free-text keywords like “Cancer”, “Neoplasms”, “Integrated Oncology”, “Ayurveda”, “Herbal Medicine”, “Medicinal Plants”, “Phytochemicals”, “Apoptosis”, “Cell Cycle Arrest”, “Anti-inflammatory”, “Immunomodulation”, “Metastasis”, “Angiogenesis”, “Oxidative Stress”, “*Arbuda*”, “*Granthi*”, and “*Apachi*”.

3.3 Literature selection

Relevant articles were picked by screening of title, abstract and full text. The publications included in this study were priority peer reviewed original research studies which assessed the anticancer effects of Ayurvedic medicinal herbs in vitro.

3.4. Eligibility Criteria

Studies were included if they:

- Studied medicinal plants mentioned in classical Ayurvedic scriptures;
- evaluated anticancer efficacy of plants in experimental or clinical environment;
- studied molecular or cellular pathways related to cancer biology;

- published in English in peer-reviewed journals; and

- released in the years 2020-2026.

Removed were:

- conference abstracts without full-text papers;
- articles and preprints not published;
- studies on non-Ayurvedic herbal remedies that were not relevant to the objective of this study; and
- duplicated articles.

3.5 Data extraction and evidence syntheses

Data retrieved included: botanical name of medicinal plant, plant part used, bioactive chemicals of plant, experimental model employed, kind of cancer, molecular pathways and main outcomes . The evidence was narratively assembled and organized by biological mechanisms: apoptotic induction, cell cycle arrest, anti-inflammatory action, suppression of angiogenesis and metastasis, control of oxidative stress and immunomodulation.

Results

Herbal remedies play an inevitable role in Ayurvedic therapies and have long been used to treat illnesses characterized by abnormal tissue growth, persistent inflammation, glandular enlargement, and localized masses. *Charaka Samhita*, *Sushruta Samhita*, and, *Ashtanga Hridaya* describes a variety of medicinal plants used in the treatment of *Arbuda*, *Granthi*, *Apachi*, and other related illnesses. The therapeutic interventions were administered topically (ointments, compresses, and medicinal applications) or orally (infusions, powders, and supplementary formulations) based on the progression and manifestation of the ailment.

Although these classical descriptions should not be viewed as direct equivalents of modern cancer, they offer a valuable traditional framework for understanding abnormal tissue growth and serve as a basis for contemporary research into the anticancer potential of Ayurvedic medicinal plants

Table 1 summarizes the medicinal plants described in the classical texts together with their traditional therapeutic applications and current pharmacological relevance.

Table 1 Medicinal plants described in Classical Ayurvedic texts along with their pharmacological relevance

S . N o.	Sansk rit name	Accept ed botanic al name	Class ical indic ation	Classi cal therap eutic use	Clas sical refer ence
1.	Arjuna	<i>Termin alia arjuna</i> (Roxb.) Wight & Arn.	Grant hi	Bark of Arjuna is used as <i>Prade ha</i>	Su. Chi. 18/1 0 (13)

				along with Madhūka, Jambu and Vetasa .	
2.	Arka	<i>Calotropis</i> spp.	Granthi; chronic Arbud	Used in external applications for Granthi; Arka leaves are also included with Sudhā, Saindhava, Guḍa and Kāñjika in the management of chronic Arbuda.	Su. Chi. 18/13; A.H. Utt. 30/1-3 (14, 15)
3.	Citrakā	<i>Plumbago zeylanica</i> L.	Granthi	Included in the therapeutic regimen for Granthi in Aṣṭāṅga Hṛdaya.	A.H. Utt. 30/1 (15)

4.	Drākṣā	<i>Vitis vinifera</i> L.	Granthi; Pittārbuda	Drākṣā-rasa is used with Harīta kī in Granthi; Drākṣā is also included in a formulation indicated for Pittārbuda.	Su. Chi. 18/9; A.H. Utt. 30/34 (16, 17)
5.	Haridrā	<i>Curcuma longa</i> L.	Medo-Arbuda	Haridrā is included in the local treatment following management of Medo-Arbuda.	Su. Chi. 18/41 (18)
6.	Harīta kī	<i>Terminalia chebula</i> Retz.	Granthi	Harīta kī is administered with Drākṣā-rasa or Ikṣu-rasa in the treatment sequence.	Su. Chi. 18/9 (19)

7.	Jambu	<i>Syzygium cumini</i> (L.) Skeels	Granthi	Bark is used in <i>Pradeha</i> along with Madhuka, Arjuna and Vetasa.	Su. Chi. 18/10(20)
8.	Ketakī *	<i>Pandanus odorifer</i> (Forssk.) Kuntze	Granthi	<i>Tṛṇaśūnya</i> is mentioned in the external application for Granthi; identification with Ketakī should be treated cautiously.	Su. Chi. 18/10(20)
9.	Muchakunda	<i>Pterospermum acerifolium</i> (L.) Willd.	Granthi	<i>Muchakunda</i> is included in an external application for Granthi.	Su. Chi. 18/10(20)
10.	Śigru	<i>Moringa oleifera</i> Lam.	Apachi; Arbuda	Śigru is mentioned in Apachimanagement in	Su. Chi. 18/23; A.H. Utt. 30/1

				Suśrutā; Aṣṭāṅga Hṛdaya includes it in the treatment of Arbuda.	–3 (21)
11.	Śyōṇāka	<i>Oroxylum indicum</i> (L.) Kurz	Vātajā Granthi	Included in <i>Pralepa</i> indicated for Vātajā Granthi.	Su. Chi. 18/5(22)
12.	Yaṣṭimadhu	<i>Glycyrrhiza glabra</i> L.	Granthi-treatment context	Included in the therapeutic formulation described in the Granthi treatment.	Su. Chi. 18/8(23)

Results

A total of 34 Ayurvedic medicinal plants belonging to 24 botanical groups were found in the studies published in the years 2020-2026 which fulfilled the eligibility requirements. The assembled evidence comprised experimental studies on crude extracts, refined phytochemicals and standardized herbal formulations. Most of the evidence was preclinical: 80% of the investigations were in vitro, 15% in vivo with experimental models. (Table 2).

Table 2- Experimental evidences showed anticancer effect of ayurvedic medicinal herbs.

S. No.	Ayurvedic Drug	Accepted Botanical	Active Constituent / Extract	Cancer Model / Cell Line	Dose / IC50	Major Anticancer Finding
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		Name				
1	Aamra	Mangifera indica L.	Aqueous fruit extract	Chronic lymphocytic leukemia B-lymphocytes	NR	Increased cytotoxicity and caspase-3 activation through ROS-mediated mitochondrial apoptosis; effect was selective towards CLL cells. (24)
2	Arka	Calotropis gigantea L.	95% ethanolic stem-bark extract and fractions	Colorectal cancer – HC T116, HT-29	IC50 : 32.8 ± 0.83 µg/mL (HC T116); 60.6 ± 3.61 µg/mL (HT-29)	Dose-dependent cytotoxicity; apoptosis associated with ROS generation, ATP depletion and mitochondrial pathway activation. (25)
3	Ataśī Bīja	Linum usitatissimum	Enterolactone	AML – KG-1	40–100 µM	Increased PAR

		atis simum L.	e/enterodiol	1, Monomac-1		P cleavage and activation of apoptotic pathways. (26)
4	Bilva	Aegle marmelos (L.) Correa	Ethanol fruit-pulp extract	DMBA-induced mammary carcinoma in rats	200 mg/kg/day × 5 weeks	Significantly reduced mammary tumor volume and tumor-associated biochemical abnormalities. (27)
5	Curcuma raktakanda	Curcuma raktakanda	Acetone, hexane and ethyl-acetate extracts	Glioma and other cancer-cell models	IC50 approximately 32.97–51.65 µg/mL	Cytotoxicity, apoptosis and suppression of cancer-cell migration. (28)
6	Dāḍima	Punica granatum L.	Hydroalcoholic pomegranate extract (PPE); ellagic acid	Gastric adenocarcinoma – AGS	PPE: 265, 214, 169 µg/mL; ellagic acid: 60, 41,	Concentration/time-dependent inhibition of proliferation

					27 µg/mL (198, 136, 90 µM) at 24, 48, 72 h	n; antic ancer activi ty with limite d cytot otoxici ty to norm al derm al fibro blasts . (29)
7	Dev adā ru	Ced rus deo dar a Rox b.	Total lignan s	Lun g canc er – A54 9	IC50 : 39.8 2 ± 1.74 µg/mL	Inhib ited prolif eration, incre ased G2/ M- phase popul ation and induc ed apopt osis. (30)
8	Dhā mār gav a	Luf fa cyli ndri ca L.	Aqueo us- ethano lic leaf extract	Acu te lym pho blast ic leuk emi a stem cells	IC50 : 3 µg/µ L	Redu ced viabil ity and clono genic ity; incre ased G2/ M popul ation and induc ed apopt osis. (31)
9	Dhā nya ka	Cor ian dru m	Corian der extract	Hep G2 hepa toce	5–10 mg/ mL in	Redu ced MMP -2

		sati vu m L.		llula r carc ino ma; B16 F10 mel ano ma	migr ation /inva sion expe rime nts	and u-PA activi ty/ex pressi on and inhibi ted cance r-cell migr ation and invas ion. (32)
1 0	Elā	Elet tari a car da mo mu m (L.) Mat on	Carda mom extract	Ehrl ich ascit es/s olid tum or mod el	100 mg/k g	Redu ced tumo r growt h and modu lated oxida tive stress and apopt otic respo nses. (33)
1 1	Era nda	Rici nus co mm unis L.	Fruit extract ; active ethyl- acetate fractio n	Brea st canc er – MC F-7, MD A- MB- 231; 4T1 mou se mod el	In vitro: 0.05 –1 µg/mL; in vivo: 0.5 mg/k g	Inhib ited migr ation, adhes ion, invas ion and MMP -2/9; decre ased Bcl-2 and incre ased Bax, caspa se-7 and PAR P cleav age. (34)

12	Eraṇḍakar kaṭī	Carica papaya L.	Aqueous seed extract	Colorectal cancer – Caco-2	IC50 : 9.734 µg/mL	Upregulated p53, Cysc and caspase-3; down regulated Bcl-2; induced G2/M arrest and apoptosis. (35)
13	Gokṣura	Tribulus terrestris L.	Methanolic/ethanolic extracts	Hep G2	0.1 mg/mL (100 µg/mL); methanolic extract	Dow nregulation of TNF-α, MMP-9, Bcl-2 and AFP, with reduced IL-1β and TNF-α protein expression. (36)
14	Guḍūcī	Tinospora cordifolia (Willd.) Hook. f. & Thomson	Chloroform and hexane extracts	Glioblastoma – U87 MG; neuroblastoma – IMR-32	IC50 : 25 and 50 µg/mL (U87 MG); 32.5 and 35 µg/mL (IMR-32)	Inhibited proliferation and migration; induced differentiation, senescence and

					R-32)	GFA P/M AP-2 expression. (37)
15	Curcuma aeruginosa	Curcuma aeruginosa Roxb.	Methanolic rhizome extract	Lung – A549; cervical – HeLa	IC50 : 15.42 ± 1.50 and 25.40 ± 0.13 µg/mL	Induced DNA damage and increased caspase-8 and caspase-3 activity. (38)
16	Hashtidaṇṭī	Baliospermum montanum	Ethanollic crude root extract	Liver cancer – Hep G2, KKU-M156	IC50 : 0.06 ± 0.02 and 0.16 ± 0.02 µg/mL	Strong cytotoxicity against liver cancer cells compared with normal HaCaT cells. (39)
17	Kañcāra	Bauhinia variegata L.	Methanolic bark extract / isolated phytochemicals	C6 glioma, MCF-7, HCT-15	IC50 approximately 119–420 µg/mL	Cytotoxicity associated with isolated phytochemicals including kaempferol; apoptosis/c

						ell-cycle effects reported. (40)
18	Met hika	Trigonella foenum-graecum L.	Methanolic seed extract	Breast cancer – MC F-7, SK-BR3	IC50 : 150 and 40 µg/mL, respectively	Dose - dependent cytotoxicity, inhibition of migration/adhesion and p53-dependent mitotic catastrophe leading to apoptosis. (41)
19	Mustaka	Cyperus rotundus L.	n-Hexane rhizome fraction	Breast cancer – MC F-7	IC50 : 120.82 µg/mL	Cytotoxicity with cell-cycle alteration and apoptosis. (42)
20	Nimba	Azadirachta indica A. Juss.	Methanolic stem-bark extract	Cervical cancer – HeLa, SiHa, ME-180	IC50 approximately 95–145 µg/mL	Cell-cycle arrest, apoptosis and inhibition of migration; modulation

						of CDK 1, cyclins, Bcl-2/survivin and caspase-3. (43)
21	Nimbaphala	Citrus limetta Risso	Peel essential oil	Breast cancer – MC F-7, MD A-MB-231	IC50 approximately 47.31 and 55.11 µg/mL	Increased ROS and cytochrome-c release, consistent with mitochondrial apoptosis. (44)
22	Punarnava	Boerhaavia diffusa L.	Methanolic extract	Breast cancer – MD A-MB-231	582.9 µg/mL (24 h); 304.7 µg/mL (48 h)	Inhibited proliferation and induced apoptosis/cell-cycle changes. (45)
23	Rasana	Allium sativum L.	Ethanol bulb extract	ND EA-induced hepatocellular carcinoma in rats	50–500 mg/kg	Improved liver architecture and antioxidant defense and increased TP53 expression

						ssion. (46)
24	Sarpagandhā	Rauvolfia serpentina	Leaf extract	Cervical cancer – HeLa	IC50 : 171.5 µg/mL	Cytotoxicity accompanied by cell clumping and nuclear condensation consistent with apoptosis. (47)
25	Sitāphala	Annona squamosa L.	Petroleum ether, acetone, ethanol/methanol seed extracts	MC F-7, Hep G2 and HEK-293	Extract-dependent IC50 values	Selective cytotoxicity towards cancer cells compared with normal HEK-293 cells. (48)
26	Svargīyavṛkṣa	Simarouba glauca DC.	Chloroform/ethyl-acetate fractions; tricaproin	Colorectal cancer – HCT-116/HCT-115; breast – MDA-MB-	Extract-dependent	Inhibition of proliferation; tricaproin reported to inhibit class-I HDA Cs

				231; cervical – SiHa; lung – A549		and colorectal cancer-cell growth. (49, 50)
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Extensive review of literature suggests that , experimental studies focused on the efficacy of various medicinal plants on different types of cancers such as breast, colon, cervical, liver, lung, gastric, prostate, ovarian, oral, glioma, neuroblastoma, leukaemia, liposarcoma, and hepatocellular cancers. The most widely examined experimental models were breast and colorectal malignancies, followed by cervical, liver and lung cancers. Conversely, pancreatic, ovarian, and hematologic cancers were quite infrequent. The most often reported anticancer pathways were apoptosis induction, cell proliferation inhibition via cell cycle arrest, decrease of chronic inflammation and oxidative stress, invasion and metastasis suppression, and regulation of tumour linked signaling pathways. These data suggest that the Ayurvedic medicinal plants may have multi-target pharmacological effects through simultaneous modulation of numerous hallmarks of cancer rather than through a single molecular target.

Apoptosis induction

Apoptosis is the main anticancer mechanism discussed in the evaluated papers. About two-thirds of the studied medicinal plants induced apoptosis through intrinsic mitochondrial pathways, including activation of caspase-3 and caspase-9, increased Bax/Bcl-2 ratio, cytochrome-c release, PARP cleavage and restoration of TP53-mediated signaling. Medicinal plants namely *Curcuma longa*, *Terminalia chebula*, *Punica granatum*, *Azadirachta indica*, *Carica papaya*, *Boerhavia diffusa*, *Calotropis gigantea*, *Cedrus deodara*, *Linum usitatissimum*, *Allium sativum*, *Rauvolfia serpentina*, *Annona squamosa*, *Cassia angustifolia*, *Capparis spinosa*, *Baliospermum montanum*, *Mangifera indica*, *Curcuma aeruginosa* and *Simarouba glauca* have been proved to activate apoptotic pathways consistently in different cancer models. The frequent detection of mitochondrial apoptosis in a range of phytoconstituents suggests that activation of programmed cell death is a common mechanism underlying the anticancer actions of Ayurvedic medicinal herbs.

Inhibition of cell proliferation and cell cycle progression.

Cell-cycle arrest was the second most frequently reported mechanism. In almost one-third of the investigated medicinal plants, the ability to limit tumor cell proliferation by inhibiting the cell cycle

at G0/G1, G2/M or S phases was observed. *Terminalia chebula*, *Cyperus rotundus*, *Azadirachta indica*, *Carica papaya*, *Cedrus deodara*, *Curcuma longa*, *Andrographis paniculata*, *Luffa cylindrica*, *Boerhavia diffusa*, *Cassia angustifolia* and *Bauhinia variegata* exhibited significant control over cyclins, cyclin-dependent kinases and checkpoint proteins. Further antiproliferative activity has been reported for *Mangifera indica*, *Aegle marmelos*, *Amomum subulatum*, *Coix lacryma-jobi*, *Saccharum officinarum*, *Vitis vinifera*, *Tinospora cordifolia* and *Tribulus terrestris*, suggesting a wide-ranging inhibition of tumour growth in various experimental models.

Modulating inflammation and oxidative stress

Management of chronic inflammation and oxidative stress is a major therapeutic strategy. Numerous medicinal herbs have shown to reduce inflammatory mediators and intracellular oxidative stress, and enhance endogenous antioxidant defense systems. Commonly studied molecular targets included NF- κ B, TNF- α , IL-8, inducible nitric oxide synthase (iNOS), reactive oxygen species (ROS) and antioxidant enzymes. The plant species *Curcuma longa*, *Punica granatum*, *Tinospora cordifolia*, *Saccharum officinarum*, *Elettaria cardamomum*, *Allium sativum*, *Trigonella foenum-graecum*, *Coriandrum sativum*, *Tribulus terrestris*, *Aegle marmelos* and *Capparis spinosa* have repeatedly indicated the presence of anti-inflammatory and antioxidant characteristics. These results suggest that modification of inflammatory tumor microenvironment has a major role in anticancer efficacy of Ayurvedic medicinal herbs. **Inhibition of migration, invasion and metastasis.**

The major cause of cancer death is metastasis. Many Ayurvedic medicinal plants have shown to suppress tumor migration, invasion, colony formation, and extracellular matrix disintegration by regulating key mediators involved in metastatic progression. *Boerhavia diffusa*, *Ricinus communis*, *Vitis vinifera*, *Coriandrum sativum*, *Tinospora cordifolia*, *Amomum subulatum*, *Curcuma aeruginosa*, *Andrographis paniculata*, *Tribulus terrestris* and *Simarouba glauca* have been shown to inhibit significantly matrix metalloproteinases (MMP-2 and MMP-9), vascular endothelial growth factor (VEGF), urokinase-type plasminogen activator (u-PA) and various other mediators associated with epithelial-mesenchymal transition. These results imply that suppression of metastatic signaling pathways is a common property of Ayurvedic therapeutic herbs.

4.5 Pharmacologic action on several targets

One of the most important conclusions to arise from this analysis was the multi-target character of the Ayurvedic therapeutic herbs. Rather of targeting specific signaling molecules, different herbs simultaneously altered pathways involved in apoptosis, oxidative stress, inflammation,

angiogenesis, and metastasis. *Curcuma longa*, *Tinospora cordifolia*, *Punica granatum*, *Boerhavia diffusa*, and *Azadirachta indica* have shown multitarget action in several experimental paradigms. These pleiotropic pharmacological effects are in agreement with the systems-based therapeutic philosophy of Ayurveda and may provide advantages over single-target treatments in complex conditions such as cancer. The common molecular targets studied in these research were caspase-3, caspase-9, Bax, Bcl-2, TP53, NF- κ B, cyclin-dependent kinases, matrix metalloproteinases (MMP-2/MMP-9), vascular endothelial growth factor (VEGF), and reactive oxygen species (ROS). Though chemically diverse, the phytoconstituents targeted a relatively narrow number of oncogenic signaling pathways. Thus, multitarget modulation rather than single-target suppression is the principal anticancer method of the Ayurvedic medicinal plants.

Discussion

This evidence-based narrative review demonstrates that Ayurvedic medicinal plants possess considerable experimental anticancer potential through the modulation of several molecular pathways involved in tumor development, and metastasis. Neoplastic diseases are mentioned in Ayurveda under the terms like *Arbuda*, *Granthi* and *Apachi*. Modern research has revealed many molecular processes supporting the therapeutic importance of these medicinal herbs in oncology. Classical ayurvedic knowledge may offer prospects for creation of complementary approaches to cancer care through integration of modern biomedical evidence. The present review throws light on the multitarget nature of the Ayurvedic therapeutic herbs. Most of the medicinal plants not only influence apoptosis and cell cycle progression, but also inflammation, oxidative stress, angiogenesis and metastasis simultaneously, compared to traditional anticancer medications that usually target a single pathway. Cancer is a disease that is defined by dysregulation of numerous signaling pathways. It is very advantageous to have such pleiotropic action. Induction of apoptosis was the most consistent mechanism across the analyzed studies. The enhanced Bax/Bcl-2 ratio, caspases activation, cytochrome-c release, PARP cleavage and the restoration of TP53 signalling in most of the plants promoted the activation of mitochondrial apoptotic pathways in cancer cells. Studies have also often shown the regulation of cyclins, cyclin-dependent kinases and checkpoint proteins, which indicated reduction of tumor proliferation via arrest of the cell cycle. Furthermore, anti-metastatic and anti-angiogenic actions were shown by inhibition of matrix metalloproteinases and VEGF signalling. The anti-inflammatory and antioxidant activities of these therapeutic herbs were likewise remarkable. Plants may alter the tumor microenvironment and

diminish chronic inflammation associated with tumor progression by downregulating NF- κ B, TNF- α , IL-6, IL-8, inducible nitric oxide synthase and reactive oxygen species together with the increase of endogenous antioxidant defense. The botanicals evaluated were *Tinospora cordifolia*, *Curcuma longa*, *Azadirachta indica*, *Boerhavia diffusa* and *Terminalia chebula*, and they displayed a wide spectrum of pharmacological actions like antioxidant, immunomodulatory, antiproliferative, anti-inflammatory and anti-metastatic activity. These results experimentally justify the classical concept of Rasayana in the maintenance of physiological homeostasis and increase of host resistance.

Current study has focused on only few pathways apoptosis and anti-proliferative effects. Emerging fields such as cancer stem cells, autophagy, ferroptosis, epigenetic regulation, tumor metabolism, immunological checkpoint signaling and microbiome interactions remain under-explored. Future studies in the domains of systems biology, network pharmacology, multi-omics technology and well-designed clinical trials may uncover the therapeutic potentials and mechanisms of Ayurvedic medicinal plants. These herbal formulations can improve symptom control, reduce medication-related toxicities, improve quality of life and, maybe, improve therapeutic efficacy. However, without robust multicentric randomized controlled studies with standardized formulations and confirmed treatment effects, no procedures can be offered to apply them in ordinary clinical practice. Recent reports suggest that anticancer efficacy of Ayurvedic herbal medicines is frequently associated with simultaneous modulation of various cellular pathways. While these data are mainly preclinical, the findings provide a strong scientific rationale to further investigate standardized Ayurvedic botanicals as supplemental therapy in evidence-based integrative cancer.

Conclusion.

One of the most important conclusions to arise from this analysis was the multi-target character of the Ayurvedic therapeutic herbs. Rather of targeting specific signaling molecules, different herbs simultaneously altered pathways involved in apoptosis, oxidative stress, inflammation, angiogenesis, and metastasis. *Curcuma longa*, *Tinospora cordifolia*, *Punica granatum*, *Boerhavia diffusa*, and *Azadirachta indica* have shown multitarget action in several experimental paradigms. These pleiotropic pharmacological effects are in agreement with the systems-based therapeutic philosophy of Ayurveda and may provide advantages over single-target treatments in complex conditions such as cancer. The common molecular targets studied in these research were caspase-3, caspase-9, Bax, Bcl-2, TP53, NF- κ B, cyclin-dependent kinases, matrix metalloproteinases

(MMP-2/MMP-9), vascular endothelial growth factor (VEGF), and reactive oxygen species (ROS). Though chemically diverse, the phytoconstituents targeted a relatively narrow number of oncogenic signaling pathways. Thus, multitarget modulation rather than single-target suppression is the principal anticancer method of the Ayurvedic medicinal plants.

Thus, the current review is suggestive of Integration of traditional ayurvedic principles with modern molecular oncology and empirical clinical research may be advantageous in the development of safe, effective and scientifically proved phytopharmaceuticals for holistic therapy of cancer. These efforts might enhance the status of Ayurveda in the emerging paradigm of evidence-based integrative oncology.

Reference

1. International Agency for Research on Cancer. Cancer Tomorrow [Internet]. Lyon: International Agency for Research on Cancer; [cited 2026 Jul 20]. Available from: https://gco.iarc.who.int/tomorrow/en/dataviz/isotype?type=1&single_unit=500000&populations=900&group_populations=0&multiple_populations=0&years=2025
2. National Institute of Cancer Prevention and Research. Statistics [Internet]. Noida: ICMR-National Institute of Cancer Prevention and Research; [cited 2026 Jul 20]. Available from: <https://cancerindia.org.in/statistics/>
3. Dede Z, Tumer K, Kan T, Yucel B. Current advances and future prospects in cancer immunotherapeutics. *Medeni Med J*. 2023;38(1):88-94. doi:10.4274/MMJ.GALENOS.2023.29599.
4. Kurhade Y, Arora K. Emerging therapeutic strategies in cancer: from cytotoxic agents to precision medicine. *Int J Pharm Sci*. 2026;4(4):4843-6.
5. Kurhade Y, Arora K. Emerging therapeutic strategies in cancer: from cytotoxic agents to precision medicine. *Int J Pharm Sci*. 2026;4(4):4843-6. doi:10.5281/zenodo.19909245.
6. Ansari S, Patel G, Malaviya A, Dehankar V, Rajeswari BS. Oncology treatment in India: a narrative exploration of patient engagement and care strategies. *Cureus*. 2025;17(7):e87329. doi:10.7759/cureus.87329.
7. National Institute of Indian Medical Heritage. e-Samhita [Internet]. Hyderabad: National Institute of Indian Medical Heritage; [cited 2026 Aug 5]. Available from: <https://niimh.nic.in/ebooks/ecaraka/?mod=read>
8. Chakrapanidatta. Charaka Samhita with Ayurvedadipika commentary. Yadavji Trikamji Acharya, editor. Varanasi: Chaukhambha Surbharati Prakashan; 2013. Chikitsa Sthana 12/81.
9. Vagbhatta. Ashtangahrudaya with Sarvangasundari commentary. Lalchandra Vaidya,

- editor. Varanasi: Motilal Banarsidass; 2004. Uttara Sthana 29/23-24.
10. Dhruva A, Hecht FM, Miaskowski C, Kaptchuk TJ, Bodeker G, Abrams D, et al. Correlating traditional Ayurvedic and modern medical perspectives on cancer: results of a qualitative study. *J Altern Complement Med.* 2014;20(5):364-70. doi:10.1089/acm.2013.0259.
11. Arnold JT. Integrating Ayurvedic medicine into cancer research programs part 1: Ayurveda background and applications. *J Ayurveda Integr Med.* 2023;14(2):100676. doi:10.1016/j.jaim.2022.100676.
12. Jain R, Kosta S, Tiwari A. Ayurveda and cancer. *Pharmacogn Res.* 2010;2(6):393-4. doi:10.4103/0974-8490.75463.
13. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/10.
14. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/13.
15. Vāgbhaṭa. Aṣṭāṅga Hṛdaya with Sarvāṅgasundarā commentary of Aruṇadatta and Āyurvedarasāyana commentary of Hemādri. Paradakara HS, editor. Varanasi: Chaukhambha Krishnadas Academy; 2014. Uttara Sthāna 30/1-3.
16. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/9.
17. Vāgbhaṭa. Aṣṭāṅga Hṛdaya with Sarvāṅgasundarā commentary of Aruṇadatta and Āyurvedarasāyana commentary of Hemādri. Paradakara HS, editor. Varanasi: Chaukhambha Krishnadas Academy; 2014. Uttara Sthāna 30/34.
18. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/41.
19. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/9.
20. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/10.
21. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/23.
22. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/5.
23. Suśruta. Suśruta Saṃhitā with Nibandhasaṅgraha commentary of Dalhaṇa. Yādavaji Trikamji Ācārya, editor. Varanasi: Chaukhambha Orientalia; 2012. Cikitsā Sthāna 18/8.
24. Ayatollahi A, Rahmati J, Salimi A, Pourahmad J. A comparison of cytotoxic effects of *Mangifera indica* L. and *Juglans regia* aqueous extract on human chronic lymphocytic leukemia. *Iran J Pharm Res.* 2019;18(4):1843-53. doi:10.22037/ijpr.2019.111977.13462.
25. Winitchaikul T, Sawong S, Surangkul D, Srikummool M, Somran J, Pekthong D, et al. *Calotropis gigantea* stem bark extract induced apoptosis related to ROS and ATP production in colon cancer cells. *PLoS One.* 2021;16(8):e0254392. doi:10.1371/journal.pone.0254392.
26. Tannous S, Haykal T, Dhaini J, Hodroj MH, Rizk S. The anti-cancer effect of flaxseed lignan derivatives on different acute myeloid leukemia cancer cells. *Biomed Pharmacother.* 2020;132:110884. doi:10.1016/j.biopha.2020.110884.
27. Akhouri V, Kumari M, Kumar A. Therapeutic effect of *Aegle marmelos* fruit extract against DMBA induced breast cancer in rats. *Sci Rep.* 2020;10:18016. doi:10.1038/s41598-020-72935-2.
28. Mishra S, Verma SS, Rai V, Awasthee N, Singh S, et al. Curcuma raktakanda induces apoptosis and suppresses migration in cancer cells: role of reactive oxygen species. *Biomolecules.* 2019;9(4):159. doi:10.3390/biom9040159.
29. Cheshomi H, Bahrami AR, Rafatpanah H, Matin MM. The effects of ellagic acid and other pomegranate (*Punica granatum* L.) derivatives on human gastric cancer AGS cells. *Hum Exp Toxicol.* 2022;41:09603271211064534. doi:10.1177/09603271211064534.
30. Shi X, Du R, Zhang J, Lei Y, Guo H. Evaluation of the anti-cancer potential of *Cedrus deodara* total lignans by inducing apoptosis of A549 cells. *BMC Complement Altern Med.* 2019;19(1):281. doi:10.1186/s12906-019-2682-6.
31. Yehia S, Abdel-Salam IM, Elgamal BM, El-Agamy B, Hamdy GM, Aldouski HM. Cytotoxic and apoptotic effects of *Luffa cylindrica* leaves extract against acute lymphoblastic leukemic stem cells. *Asian Pac J Cancer Prev.* 2020;21(12):3661-8. doi:10.31557/APJCP.2020.21.12.3661.
32. Huang H, Nakamura T, Yasuzawa T, Ueshima S. Effects of *Coriandrum sativum* on

- migration and invasion abilities of cancer cells. *J Nutr Sci Vitaminol (Tokyo)*. 2020;66(5):468-77. doi:10.3177/jnsv.66.468.
33. Almeer RS, Alnasser M, Aljarba N, AlBasher GI. Effects of green cardamom (*Elettaria cardamomum* Maton) and its combination with cyclophosphamide on Ehrlich solid tumors. *BMC Complement Med Ther*. 2021;21:133. doi:10.1186/s12906-021-03305-2.
 34. Majumder M, Debnath S, Gajbhiye RL, Saikia R, Gogoi B, Samanta SK, et al. Ricinus communis L. fruit extract inhibits migration/invasion, induces apoptosis in breast cancer cells and arrests tumor progression in vivo. *Sci Rep*. 2019;9:14493. doi:10.1038/s41598-019-50769-x.
 35. Mahrous NS, Noseer EA. Anticancer potential of Carica papaya Linn black seed extract against human colon cancer cell line: in vitro study. *BMC Complement Med Ther*. 2023;23:271. doi:10.1186/s12906-023-04085-7.
 36. Khalid A, Nadeem T, Khan MA, Ali Q, Zubair M. In vitro evaluation of immunomodulatory, anti-diabetic, and anti-cancer molecular mechanisms of *Tribulus terrestris* extracts. *Sci Rep*. 2022;12:22478. doi:10.1038/s41598-022-26742-6
 37. Sharma A, Saggu SK, Mishra R, Kaur G. Anti-brain cancer activity of chloroform and hexane extracts of *Tinospora cordifolia* Miers: an in vitro perspective. *Ann Neurosci*. 2019;26(1):10-20. doi:10.5214/ans.0972.7531.260104.
 38. Zohmachhuana A, Vabeiryureilai M, Malsawmdawngliana, Kumar NS, Lalrinzuali K, Lalnunmawia F. Curcuma aeruginosa Roxb. exhibits cytotoxicity in A-549 and HeLa cells by inducing apoptosis through caspase-dependent pathways. *Biomed Pharmacother*. 2022;150:113039. doi:10.1016/j.biopha.2022.113039.
 39. Pipatrattanaseree W, Itharat A, Mukkasombut N, Saesiw U. Potential in vitro anti-allergic, anti-inflammatory and cytotoxic activities of ethanolic extract of *Baliospermum montanum* root, its major components and a validated HPLC method. *BMC Complement Altern Med*. 2019;19:45. doi:10.1186/s12906-019-2449-0.
 40. Sharma N, Sharma A, Bhatia G, Landi M, Brestic M, Singh B, et al. Isolation of phytochemicals from *Bauhinia variegata* L. bark and their in vitro antioxidant and cytotoxic potential. *Antioxidants (Basel)*. 2019;8(10):492. doi:10.3390/antiox8100492.
 41. Alrumaihi FA, Khan MA, Allemailem KS, Alsahli MA, Almatroudi A, Younus H, et al. Methanolic fenugreek seed extract induces p53-dependent mitotic catastrophe in breast cancer cells, leading to apoptosis. *J Inflamm Res*. 2021;14:1511-35. doi:10.2147/JIR.S300025.
 42. Simorangkir D, Masfria M, Harahap U, Satria D. Activity anticancer n-hexane fraction of *Cyperus rotundus* L. rhizome to breast cancer MCF-7 cell line. *Open Access Maced J Med Sci*. 2019;7(22):3904-6. doi:10.3889/oamjms.2019.530.
 43. Kumar S, Mulchandani V, Das Sarma J. Methanolic neem (*Azadirachta indica*) stem bark extract induces cell cycle arrest, apoptosis and inhibits the migration of cervical cancer cells in vitro. *BMC Complement Med Ther*. 2022;22:239. doi:10.1186/s12906-022-03718-7.
 44. Narayanankutty A, Visakh NU, Sasidharan A, Pathrose B, Olatunji OJ, Al-Ansari A, et al. Chemical composition, antioxidant, anti-bacterial, and anti-cancer activities of essential oils extracted from Citrus limetta Risso peel waste remains after commercial use. *Molecules*. 2022;27(23):8329. doi:10.3390/molecules27238329.
 45. Sinan KI, Akpulat U, Aldahish AA, Celik Altunoglu Y, Baloglu MC, Zheleva-Dimitrova D, et al. LC-MS/HRMS analysis, anti-cancer, anti-enzymatic and anti-oxidant effects of Boerhavia diffusa extracts: a potential raw material for functional applications. *Antioxidants (Basel)*. 2021;10(12):2003. doi:10.3390/antiox10122003.
 46. Ogar GO, Minari JB, Bello AJ, Chiwetelu J, Omogunwa OE, Oshikoya OS, et al. Influence of ethanolic extract of *Allium sativum* on TP53 gene and its anticancer potential in N-nitrosodiethylamine-induced hepatocellular carcinoma in male albino rats. *Iran J Basic Med Sci*. 2022;25(4):497-505. doi:10.22038/IJBMS.2022.62295.13787.
 47. Alshahrani MY, Rafi Z, Alabdallah NM, Shoaib A, Ahmad I, Asiri M, et al. A comparative antibacterial, antioxidant, and antineoplastic potential of *Rauwolfia serpentina* (L.) leaf extract with its biologically synthesized gold nanoparticles (R-AuNPs). *Plants (Basel)*. 2021;10(11):2278. doi:10.3390/plants10112278.
 48. Alaqeel NK, Almalki WH, Binothman N, Aljadani M, Al-Dhuayan IS, Alnamshan MM, et al. The inhibitory and anticancer properties of *Annona squamosa* L. seed extracts. *Braz J Biol*. 2023;82:e268250. doi:10.1590/1519-6984.268250.
 49. Jose A, Kannan E, Madhunapantula SV. Anti-proliferative potential of phytochemical fractions isolated from *Simarouba glauca* DC leaf. *Heliyon*. 2020;6(4):e03836. doi:10.1016/j.heliyon.2020.e03836.
 50. Jose A, Chaitanya MVNL, Kannan E, Madhunapantula SV. Tricaproin isolated from *Simarouba glauca* inhibits the growth of human colorectal carcinoma cell lines by targeting class-I histone deacetylases. *Front Pharmacol*. 2018;9:127. doi:10.3389/fphar.2018.00127.