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A Comprehensive Review On Medicinal Plants Targeting Breast Cancer Cells And Their Mechanisms Of Action

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Abstract
This review focuses on medicinal plants with demonstrated anticancer activity against breast cancer cells. These plants exhibit a wide range of biological properties, including anti-inflammatory, antioxidant, antitumorigenic, antimutagenic, antineoplastic, and antiproliferative effects, all of which play a crucial role in suppressing cancer cell growth. Several plants have also been shown to enhance the efficacy of conventional chemotherapeutic agents through synergistic interactions. For example, flaxseed exhibits synergistic effects with tamoxifen and trastuzumab; curcumin enhances the activity of paclitaxel and docetaxel; garlic shows synergism with tamoxifen and doxorubicin; and ginseng improves the effectiveness of paclitaxel, doxorubicin, and cisplatin. This review systematically discusses the medicinal plants, their pharmacological activities, chemical constituents, target cell and underlying mechanisms of action in breast cancer treatment.

Keywords: Breast cancer, Medicinal plants, Anticancer activity, Target cell, Mechanism of action.

Background
According to the World Health Organization (WHO), breast cancer is defined as "a disease in which abnormal breast cells grow uncontrollably and form tumors. If left untreated, these tumors can spread throughout the body and become fatal." Breast cancer most commonly originates in the milk ducts or lobules. It is one of the most prevalent cancers among women, with risk factors including increasing age (especially over 40 years), obesity, alcohol consumption, inherited gene mutations such as BRCA1 and BRCA2, and a history of radiation exposure. Early detection through screening methods, such as mammography, is essential for improving clinical outcomes[1].
Breast cancer is the most common cancer in women and the second most common cancer overall[2]. Approximately 99% of breast cancers occur in women and 0.5–1% of breast cancers occur in men. In 2022, there were an estimated 2.3 million new cases and 670,000 deaths from breast cancer occurred worldwide of breast cancer globally[1]. Every 14 seconds, somewhere in the world, a woman is diagnosed with breast cancer[3]. It is the most frequently diagnosed cancer among women in 157 of 185 countries in 2022[1]. Globally, breast cancer now represents one in four of all cancers in women. Since 2008, worldwide breast cancer incidence has increased by more than 20 percent. Mortality has increased by 14 percent[3]. For instance, in countries with a very high Human Development Index (HDI), 1 in 12 women will be diagnosed with breast cancer in their lifetime and 1 in 71 women die of it. In contrast, in countries with a low HDI; while only 1 in 27 women is diagnosed with breast cancer in their lifetime, 1 in 48 women will die from it. Approximately 99% of breast cancers occur in women and 0.5–1% of breast cancers occur in men[1]. 1 in 20 women worldwide will be diagnosed with breast cancer in their lifetime, and that if current rates continue, by 2050 there will be 3.2 million new breast cancer case[4].
With being the most common cancer in women, breast cancer accounts for 14% of Indian women[5]. 27.7% of all new cancers detected in women in India in the year 2018, were breast cancers and in the same year 1,62,468 incidences and 87,090 deaths were recorded. Every two women newly diagnosed with breast cancer one woman dies of it in India[6]. It was accounting for 28.2% of all female cancers, with an estimated 216,108 cases by 2022[7]. Overall, 1 in 28 women is likely to develop breast cancer during her lifetime. In urban areas, 1 in 22 women develops breast cancer during her lifetime as compared to rural areas where 1 in 60 women develops breast cancer in her lifetime[8].One woman is diagnosed with breast cancer, in India, every 4 minutes. One woman dies of Breast cancer, in India, every 8 minutes[9]. In 2025, India is expected to see a continued rise in

breast cancer cases, with some projections indicating around 232,832 new diagnoses and an annual increase of approximately 50,000 cases this decade[10].

Signs and symptoms of breast cancer

According to American Cancer Society (ACS), the most common symptom of breast cancer is a new lump or mass (although most breast lumps are not cancer). A painless, hard mass that has irregular edges is more likely to be cancer, but breast cancers can also be soft, round, tender, or even painful. Other possible symptoms of breast cancer include,

- Swelling of all or part of a breast (even if no lump is felt)
- Skin dimpling (sometimes looking like an orange peel)
- Breast or nipple pain
- Nipple retraction (turning inward)
- Nipple or breast skin that is red, dry, flaking, or thickened
- Nipple discharge (other than breast milk)
- Swollen lymph nodes under the arm or near the collar bone (Sometimes this can be a sign of breast cancer spread even before the original tumor in the breast is large enough to be felt).

Types

Breast cancers are broadly categorized as, Invasive and Non-invasive.

Non-Invasive (In Situ)

The cancer cells are confined to the ducts (like DCIS) and have not spread to surrounding breast tissue or other parts of the body.

Invasive (Infiltrating)

The cancer cells have spread from the ducts into the surrounding breast tissue and have the potential to spread to other parts of the body.

Sub-types

The most common and widely accepted classification of breast cancer is from an immunohistochemical perspective, based on the expression of the following hormone receptors: estrogen (ER), progesterone (PR) and human epidermal growth factor (HER2). Accordingly, the following four subtypes of breast cancer are widely recognized: luminal A, luminal B, HER2-positive, and triple-negative[11] given in Table 1.

Table 1: **Subtypes of breast cancer**

SUB Type	Prevalence (%)	ER	PR	HER	Ki-67 marker expression	Histological grade	Prognosis	Survival rate (%)
Luminal - A	73	+	+	-	Low – less than 20%	Low grade	Best	94.4
Luminal-B	11	+	-		High – greater than 20%	Intermediate/ Higher grade	Worst compared to A	90.7
HER-2	4					Higher	Worst compared to luminal tumors	84.8
	i) luminal HER-2	+	+	+	15-30%			
	ii) HER-2 enriched	+	-	-	> 30%			
TNBC	12	-	-	-	≥ 30%	Higher	Worst in all	77.1

Other specific types

Beyond the molecular subtypes and the invasive/non-invasive distinction, there are other named types.

Ductal Carcinoma In Situ (DCIS)

A non-invasive type where cancer cells are in the milk ducts but haven't spread.

Invasive Ductal Carcinoma (IDC)

The most common type of invasive breast cancer, where cells start in the milk ducts and spread. It accounts for 70 -80% of all breast cancers.

Invasive Lobular Carcinoma (ILC)
Another common invasive type that starts in the milk-producing glands (lobules).

Rare types
1) Paget disease of the breast – it accounts only 1-3% of all breast cancer
2) Angiosarcoma
3) Phyllodes tumor

Inflammatory Breast Cancer (IBC)
A rare and aggressive form where cancer cells block the lymph vessels in the breast skin, causing redness, swelling, and a dimpled texture. It is rare and accounts for 1% to 5% of all breast cancers.

Treatment protocol
In early stages: Surgery + radiation ± systemic therapy (goal: cure).
In late stages: Combination of chemo, hormone, targeted, and supportive therapies (goal: control & prolong survival).

Drugs used for treatment
Common breast cancer drugs include hormone therapies like Tamoxifen and Aromatase Inhibitors (e.g., Anastrozole, Letrozole), chemotherapies such as Taxanes (e.g., Paclitaxel, Docetaxel), Anthracyclines (e.g., Doxorubicin), and Capecitabine. There are also targeted therapies that block specific proteins, including HER2-targeted drugs like Trastuzumab (Herceptin) and CDK 4/6 inhibitors like Palbociclib (ibrance). The choice of drug depends on the type and stage of breast cancer.
List of all drugs approved by Food and Drug Administration (FDA)[12]

Drugs Approved to Prevent Breast Cancer
1. Evista (Raloxifene Hydrochloride)
2. Raloxifene Hydrochloride
3. Soltamox (Tamoxifen Citrate)
4. Tamoxifen Citrate

Need for herbal medicine
Side Effects of Allopathic (Conventional) Cancer Drugs is important reason for patients switching to herbal medicine[13]. Cancer medicines (chemotherapy, targeted therapy, immunotherapy, hormone therapy) are effective, but they often damage normal healthy cells along with cancer cells. This causes side effects as mentioned in Table 2. Because of these side effects, many patients turn to herbal or natural therapies as supportive care. The reasons are, to reduce side effects (like nausea, fatigue, low immunity), to improve quality of life and to support immunity and overall health. Some herbs enhance the effectiveness and bio-availability of cancer drugs. For ex. Flaxseed enhances tamoxifen & trastuzumab effects (in animals) and Curcumin, Piperine, Quercetin, EGCG increases tamoxifen bioavailability via enzyme/transporter inhibition. Herbal medicines are not replacements for chemotherapy or targeted therapy. Some herbs have anticancer properties (lab/animal studies show potential. Table 3 shows the list of plants that have anti - cancerous effect against breast cancer.

Table 2: **Modern therapies and its side effects**

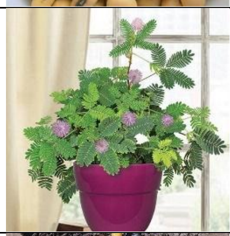
Therapy	Side effects
Chemotherapy	Hair loss, nausea, vomiting, loss of appetite, mouth ulcers, fatigue, weakness, low blood counts → infections, bleeding, anemia, Neuropathy (numbness/tingling in hands & feet), fertility issues.
Radiotherapy	Skin redness, breast swelling, fatigue, breast fibrosis, lymphedema, lymph/ heart effects, rare second cancers.
Targeted Therapy (e.g., Trastuzumab, CDK inhibitors)	Diarrhea, skin rashes, heart problems (some HER2 drugs), liver function abnormalities.
Hormone Therapy (Tamoxifen, Aromatase inhibitors)	Hot flashes, night sweats, bone thinning (osteoporosis), weight gain, mood changes, risk of blood clots (Tamoxifen).

Immunotherapy	Inflammation in lungs, liver, thyroid, skin, flu-like symptoms, fatigue.

Methodology
To collect data from various journals, the keywords medicinal plants, chemical compounds, target cells/cell lines, mechanisms, anticancer activity, and anti-proliferative properties were searched in international databases such as ScienceDirect, PubMed, Scopus, Google Scholar, and Research gate. The searches were restricted to articles published in English, covering the period from 1990 to 2025. A total of 71 articles were included in this survey.

Table 3: Botanical description of medicinal plants

S.no	Botanical name	Tamil name	Common name	Family	Part used	Images
1	Linum usitatissimum	Aali vithai	Linseed/ Flax seed	Linaceae	Seed	
2	Curcuma longa	Manjal	Tumeric	Zingiberaceae	Rhizome	
3	Nigella sativa	karunjeeragam	Black cumin	Rananculaceae	Seed	
4	Allium sativum	Pondu	Garlic	Liliaceae	Bulb	
5	Tinospora cordifolia	seenthil	Heart leaved moon seed	Menispermaceae	Stem	
6	Foeniculam vulgare	Perunjeeragam	Fennel	Apiaceae	Seeds	
7	Boerhavia diffusa	Mukkirattai	Hogweed	Nyctanginaceae	Leaf, Root, whole plant	

8	Vitis vinifera	Thiratchai	Grapevine	Vitaceae	Seeds of white grape	
9	Centella asiatica	Vallarai keerai	Indian pennywort	Apiaceae	Leaves (dry)	
10	Zingiber officinale	Inji	Ginger	Zingiberaceae	Rhizome	
11	Brassica oleracea	Pachai pookose	Broccoli	Brassicaceae	Florets/ Stalks/ leaves	
12	Aegle marmelos	Vilvam	Bael, Holy fruit	Rutaceae	Fruits/Leaf/ Bark	
13	Glycine max	Oota avarai	Soya bean	Fabaceae	Seeds	
14	Mimosa pudica	Thotta sinungi	Tough-me-not	Fabaceae	Whole plant	
15	Semecarpus anacardium	Serankottai	Marking nut	Anacardiaceae	Leaves, nuts, fruits	
16	Hemidesmus indicus	Nannari	Indian sarsaparilla	Asclepiadaceae	Root/Leaf/Flower	
17	Smilax zeylanica	Kal thamarai	Ceylon sarsaparilla	Smilacaceae	Root	

18	Thespesia populnea	Poovarasu	Indian tulip tree	Malvaceae	Stem	
19	Phyllanthus emblica	Nellikai	Indian goose berry/ Amla	Phyllanthaceae	Fruit	
20	Piper nigrum	Milagu	Black pepper	Piperaceae	Fruit	
21	Piper longum	Thippili	Long pepper	Piperaceae	Fruit	
22	Withania somnifera	Amukkira kizhangu	Indian ginseng	Solanaceae	Root/leaf	
23	Aloe barbadensis	Sotru katrazhai	Aloe vera	Liliaceae	Jelly	
24	Capsicum annum	Kudai milagai	Bell pepper	Solanaceae	Fruit	
25	Glycyrrhiza glabra	Adhimadhuram	Liquorice	Fabaceae	Root	
26	Moringa oleifera	Murungai	Drumstick tree	Moringaceae	Leaf/bark	

27	Ferula assa-foetida	Perungayam	Hing	Apiaceae	Oleo- gum resin	
28	Mangifera indica	Mangai maram	Mango tree	Anacardiaceae	Seed kernel, peels, fruit pulp	
29	Bacopa monnieri	Neerpirammi or Nirbrahmi	Brahmi or Indian Pennywort	Plantaginaceae	Whole plant	
30	Camellia sinensis	Theyilai / Tey	Tea Plant, Tea Shrub, Tea Tree	Theaceae	Leaves	

Table 4: chemical constituents, target cell, mechanism and activity of medicinal plant

S. no	Botanical name	Chemical compounds	Cell lines (in vitro) & xenografts (in vivo)	Molecular mechanism	Activity	Reference
1	Linum usitatissimum	1. Omega-3 fatty acid, α-linolenic acid, Lignan [SDG (95%)- Seciosolariciresinol diglucoside], and the remaining 5% consist of lariciresinol, pinoresinol, and matairesinol	I. In vitro 1.**ER+ (Estrogen Receptor positive**) human cancer cell **(MCF-7)** 2. It also reduced metastasis of **ER- breast** tumor 3. It increases effectiveness of trastuzumab in reducing the growth of **HER2-** overexpressing human breast tumors **(BT-474)**	1.It acts through modulation of estrogen metabolism and estrogen receptor, growth factor receptor signaling pathway 2. Reduces the expression of c-erb2 level [also known as HER2—an oncogene associated with the development and progression of breast cancer] 3. SDG Lignan decreases the level of **Ki-67** – A biomarker that signals the increase of cell proliferation. 4.Downregulation of Akt and MAPK pathways	Anti cancerous, cytotoxicity to ER+ cell lines, anti-inflammatory reduced cell proliferation, reduced angiogenesis, increased apoptosis, decreased tumor volume, anti-oxidant, anti-metastatic, anti-tumor effect, hormone modulating effects.	14, 15
		2.PFH-G9 [Purified flaxseed hydrolysate]	II. In vitro 1. MCF-7 cell 2.T47D cell In vivo	5. It reduces metastasis markers [MMPs-2 &9], reduce VEGF, insulin growth factor, progesterone, but		16

			1.Mice with Ehrlich carcinoma	enhanced expression of caspase-3. 6. Flaxseed oil was found to reduce MCF-7 tumor growth partially through IGF-1 signaling pathway		
2	Curcu ma longa	Curcumin	1. In vitro i. MCF-7 ii.MCF-10A iii.BT-474 iv.T47D v. MCF7 [LCC2, LCC9] vi. MDA-MB-231 vii. MDA-MB-435 viii. MDA-MB-468 2. In vivo i. MDA-MB-231 cells in nude BALB/c mice ii.4T1 mouse	Curcumin inhibits breast cancer cell proliferation by the following mechanisms: 1. Inducing cell cycle arrest and p53-dependent apoptosis, which occurs only in G2 phase. 2. Altering expression of signaling proteins, including Ras, phosphatidylinositol-3-kinase (PI3K), protein kinase B (Akt), mammalian target of rapamycin (mTOR) and Wnt/β-catenin, NF-κB, nuclear factor-κB 3. Downregulating transcription factors-MMP, matrix metalloproteinase; human epidermal growth factor receptor 2; VEGFR, vascular endothelial growth factor receptor; VEGF, vascular endothelial growth factor [inhibiting tumor growth and angiogenesis]. 4. Bcl-2 & Bcl-xL downregulation/ inhibition and upregulation of Bax	Antioxidant, antitumor, free radical quenching process, anti-inflammator y, induction of apoptosis, inducing angiogenesis , immunomod ulatory property, anti-proliferative, anti-metastatic,	17,18
3	Nigella sativa	Thymoquinone, carvacrol, Trans-anethole	1. In vitro i. BT-549 and ii. MDA-MB-231 iii. TNBC iv. MCF-7 cells 2. In vivo v. Breast cancer xenograft mouse model vi. Intracardiac injection of MDA-MB-231-luc cells in mice	1.Downregulation of NF-κB regulated CXCR4 expression [MDA-MB-231& BT-549, MCF-7] 2. TQ abrogates CXCL12- induced over expression of CXCR4 and inhibits CXCR4- induced cellular invasion and migration in TNBC cells. 3. TQ also inhibited the protein expression of anti-apoptotic genes, such as XIAP, survivin, Bcl-xL and Bcl-2, in breast	Antioxidant, anti-inflammator y, anti-proliferative, Inducing apoptosis. Anticancer activity, Anti-metastatic activity.	19, 20

				cancer cells and breast tumor xenograft.		
4	Allium sativum	It has 33 organosulfur compounds. **DADS** (Diallyl-disulfide), Allin, Allicin, Ajoene, Allyl-propyl -disulfide, Diallyl-trisulfide **(DATS)**, S-allyl-cysteine, S- allo-mercapto-cysteine **(SAMC),** Vinyldithiins	Chemical compounds act on different cell lines: **1. DATS** i. MCF-7 ii.T47D iii.MDA-MB-231 **2. SAMC** i. MCF-7 **3. DADS** i. MDA-MB-231 ii. Female rat with mammary cancer growth **4. Ajoene** i.MDA-MB-231	1. Derivatives of garlic and their nano formulations resulted in altered expression levels of numerous re-ported genes associated with the regulation of several important signaling pathways, including the JNK, Akt/PI3K, p38, MAPK, EMT, Wnt, p53, ERK1/2, NF-κB, Nrf2, STAT3 and Chk1/cyclin B1 in human breast carcinoma.	Anti proliferative, anti-oxidant, anti-inflammatory, anti-metastatic, Mutagenesis inhibition, Anti-tumorigenic, inducing apoptosis, Anti-cancer activity, Inhibition of cell cycle progression.	21
5	Tinospora cordifolia	Bis (2- ethyl hexyl) 1H-pyrrole-3,4-dicarboxylate (TCCP), carnosol, phenolics, glycosides, terpenoid steroids, polysaccharides, aliphatic compound, and alkaloids, rutin, quercetin	I. In vitro 1. MDA-MB-231 2.T47D [TC stem extract] II. In vivo Murine mammary carcinoma: Ehrlich ascites tumor [EAT]	Through apoptotic pathway activation: 1.Extrinsic or death receptor pathway 2. Intrinsic or mitochondrial pathway 3. HSP90 was downregulated by TCCP by inactivation of HSF-1 resulting in inhibition of tumor cell proliferation, VEGF-induced cell migration, and concomitant decrease in tumor burden and neo-angiogenesis in vivo. The mechanism of suppression of HSPs involves inactivation of PI3K/Akt and phosphorylation on serine 307 of HSF-1 by the activation of ERK1. 4.Restoration of p^{53}	Cytotoxicity, induced apoptosis, antiproliferative, ROS enhancement, cell cycle inhibition. Anti-neoplastic, anti-metastatic, anti-angiogenic activities. Anti-cancer property.	22, 23, 24
		TcCF [chloroform fraction of TC]	II. In vitro 1. MDA-MB-231 2. MCF-7	1. It induced apoptosis through modulating the expression of pro- and anti-apoptotic proteins.		25
6	Foeniculam vulgare	Trans-anethole, fenchone, alpha-phellandrene, and estragole	1.HER2 ER+ breast cancer type 2. Female BALB/c mice with 4T1 cells	1.Decrease the protein expression of HSP 70&90. HSP90 inhibitors suppress many oncogenic signaling pathways simultaneously, and reduce the possibility of	Anti-metastatic, anti-tumor activity, anti-cancer activity, decreased	26

				mutations which lead to tumor resistance.	tumor volume, antioxidant, ROS scavenging.	
7	Boerha via diffusa	Boeravinone A-O, flavonoids glycosides, phenolic glycosides, lignan glycosides, punarnavine, rutin, caffeic acid, kaempferol, gallic acid, ferulic acid, isoquercitrin, chlorogenic acid, traumatic acid, phenolic acid	1. TNBC i. MDA-MB-231[methanolic aerial part extract] ii. MDA-MB-453 2. Hormone dependent MCF-7 cells (Root decoction, methanolic whole plant extract) 3. Murine 4T1	1. **Caffeic acid** i.It has been found to induce cell cycle arrest and apoptosis in MDA-MB-231 breast cancer cells and also reduce cell survival and activate apoptosis in MCF-7 breast cancer cells. 2. **Ferulic acid** The cytotoxic activity of ferulic acid has been shown on three different breast cancer cell lines. i. In MDA-MB-231 cells, ferulic acid has decreased cell viability by inducing apoptosis and suppressed metastasis by reversing epithelial-mesenchymal transition. ii. In the MCF-7 cells, ferulic acid has reduced viable cell numbers and new DNA synthesis by inhibiting the EGFR receptor. iii. It has also inhibited the growth of 4T1 mouse breast cancer cells. 3. **Isoquercitrin** i. Inducing the mitochondrial-mediated apoptosis pathway via the inhibition of LSD 1 in MDA-MB-231 cells. 4. **Rutin** i. It enhances chemosensitivity to cyclophosphamide and methotrexate in MDA-MB-231and MCF-7 breast cancer cell lines by reversing multidrug resistance via inhibition of P-gp and BCRP pumps. 5. **Chlorogenic acid** i. It has been shown to inhibit proliferation, induce apoptosis, and	Antioxidant, anticancer activity, anti-enzymatic activity, cytotoxic activity [methanolic aerial part extract], antiproliferative [methanolic whole plant extract], antiapoptotic, anti-inflammatory, immunomodulatory properties, inducing apoptosis, suppress migration.	27, 28

				suppress migration of human MDA-MB-231 and MDA-MB-453 cells, and murine 4T1 breast cancer cells by impairing the NF-κB/EMT signaling pathway. ii. In 4T1 breast cancer cells, it has been revealed that chlorogenic acid induces apoptosis via p53, Bax, Bcl-2, and caspase-3 signaling pathways. iii. Impairing NF-κB/EMT signaling pathway. 6. **Traumatic acid** i. It has shown to decrease cell proliferation and viability and induce apoptosis by influencing lipid peroxidation in MCF-7 breast cancer cells.		
8	Vitis vinifer a	1.Alkaloids, flavonoids, terpenoid, steroids and quinine, Polyphenol, Resveratrol, Proanthocyanidins , Anthocyanin, flavanols, flavonols, stilbenes, phenolic acids, catechins, epicatechin, gallic acid, p-coumaric. 2. Methanolic extract of seed: Dotriacontane, Linoleic acid and Decanoic acid ethyl ester, 1,2,3, propanetriol, monoacetate, and Dichloro methyl propane sulfone	1.MCF-7 (ER+) cells (Dry seed extract) 2.MDA-MB-231 (Methanolic extract of grape seed)	1. Inhibition of proteins Bcl-2, Bcl-xL, and survivin, and induction of apoptosis, and downregulation of p^{53}. 2. Downregulation of interlukin-1 alpha (IL-1α) 3.The LDH Lactate Dehydrogenase leakage in MDA MB 231 cell line may be due to the cytotoxic nature of the Vitis vinifera seed which confirms the anti-tumor activity.	Antioxidant, anticancer, antitumor activity, antiproliferat ive, chemo-preventive, suppressing metastasis.	29, 30
9	Centell a asiatica	Asiaticoside (AC), MECA [Methanolic extract of centella asiatica]: Asiatic acid, high concentration of	**In vitro** 1.MCF-7 cells (methanolic extract) 2. MDA-MB-231 **In vivo**	1.Induces apoptosis in MCF-7 cells by induction of nuclear condensation, flip-flop movement of the membrane, loss of mitochondrial membrane	Induce apoptosis, anti- tumor activity, antiproliferat ive, antioxidant,	31, 32

		phenolic and flavonoid constituents.	2.Subcutaneous injection of MDA-MB-231 into female BALB/c nude mice	potential and by inducing DNA strand breaks. 2. AC downregulates YAP1 & VEGFA signaling pathways in both cells 3. Increased levels of cleaved caspase 3 and Bax after AC treatment, while the levels of anti-apoptotic protein Bcl-2 were decreased. 4. AC inhibits tumorigenesis and angiogenesis in nude mice by dampening the YAP1/VEGFA signaling axis.	cytotoxic activity, inhibiting angiogenesis and tumorigenesis in nude mice. Suppress cell growth, migration, invasion in breast cancer cells.	
10	Zingiber officinale	1.**Monoterpenes** Cineole, citral, limonene, linalool, β-pinene 2.**Sesquiterpenes** Elemene, farnesene, zerumbone. 3**. Phenolics** [6]-gingerol High phenolic compound. 8-gingerol, 10-gingerol, zingerone, shogaols [6,8,10], paradol.	In vitro 1. MCF-7 2.MDA-MB-231	1.It reduced clonogenicity and cell migration in breast cancer cell lines (MDA-MB-231 and AT1) by decreasing matrix metalloproteinase (MMP-9). Terpenoid compounds in the Zingiberaceae group can inhibit tumor development by stopping the cell cycle in apoptosis. 10-gingerol can inhibit cell proliferation through protein downregulation by regulating the cell cycle in MCF-7 and MDA-MB-231 cell lines.	Anticancer, anti-metastatic, anti-proliferative, cell cycle arrest, antioxidant, antiangiogenic, anti-inflammatory, induce apoptosis and autophagy, anti-emetic in chemotherapy, chemo-prevention, immunomodulatory activity.	33
		[6]-gingerol nano-encapsulated with κ-carrageenan (Zo-NPS)	In vivo 1.Female *Mus musculus* Balb/c mice induced with benzo[α]pyrene	2.Reduces/blocking TNF-α level 3. Boost antioxidant defenses (SOD, GSH, CAT) in blood serum.		34
11	Brassica oleracea	1.Aliphatic glucosinolates include progoitrin, glucoerucin, glucoraphanin, glucoiberin,	**In vitro** 1. Hormone dependent i) MCF-7 ii) T-47D 2.Triple negative	1.It inhibits the histone deacetylases (HDAC3) activity which is responsible for tumor expression.	Antioxidant, anticancer, anti-inflammatory, anti-metastatic,	35, 36

		sinigrin, and gluconapin. Sulforaphane, Indole-3-carbinol, diindolylmethane, **Flavonoids:** quercetin, kaempferol, rich in selenium, Isothiocyanate	i)MDA-MB-231 ii)MDA-MB-468 iii) SUM159 **In vivo** 3. Nonobese diabetic/severe combined immunodeficient mouse model exhibited that sulforaphane eliminated breast cancer stem cells in vivo.	2. kaempferol induces phagocytic activity & increases the production of NK cells. 3. SFN acts in further ways i) It acts as a Nrf2 inducer, has the ability to prevent the metastasis of implanted breast cancer cells in mice and the growth of breast cancer cells in humans. ii. Modulate epigenetics (modulates various p^{450} cytochromes CYPs 19, 1A1, 1A2 &1B1 in MCF-7 ER+, MDA-MB-231 ER-. It reduces cytochrome p450. ii) Sulforaphane downregulated the Wnt/β-catenin self-renewal pathway. sulforaphane decreased the protein levels of β-catenin and cyclin D1 in both SUM159 and MCF7 cell lines iii) Sulforaphane was also shown to suppress angiogenesis and metastasis by downregulating vascular endothelial growth factor, HIF-1α, matrix metalloproteinase-2 and matrix metalloproteinase-9.	anti-angiogenesis, anti-carcinogenic, immunomodulatory. Chemo preventive action, induce apoptosis, cell cycle arrest, antiproliferative.	
12	Aegle marmelos	1.Fruit bulb Aegeline, aegelenine, marmelin, o-methyl halfordinol, alloimperatorin, furocoumarins, psoralen, o-isopentenyl halfordinol, marmelosin, umbelliferone and scopoletin are the coumarins. 2.Alcoholic extract of fruit	1.DMBA induced breast cancer in rats [bark extract] 2.MCF-7 & MDA-MB-231 cells [leaf extract] 3.Cytotoxic activity against SKBR3 human breast adenocarcinoma cell line [ethanolic fruit extract] 4. DMBA induced mammary cancer in Charles Foster rats [ethanolic fruit pulp extract]	1.Tumor volume reduced 2. Serum TNF-α level reduced 3. suppression of NF-κB activation and subsequent reduction in the p-AKT levels and thus decreases cell survival, proliferation, and invasiveness. 4. The expression of vascular endothelial growth factor (VEGF) and interleukin-8 (IL-8) that regulates capillary growth in the tumor have	Antiproliferative, anticancer, immunomodulatory, cytoprotective, antineoplastic effect, antitumor activity, cytotoxic activity, antioxidant, anti-inflammatory,	38, 39

		bulb: carotenoids, phenolics, flavonoids, alkaloids, tannins, terpenoids, coumarins, steroids, saponins, lignins, phlobatannins, inulin and cardiac glycosides.		also been found to be reduced by marmelin. 5. Modulating wnt/beta catenin pathway (decreased m-RNA expression of MCF-7.	anti-angiogenic, antiapoptotic, free radicals scavenging.	
13	Glycine max	Genistein [phytoestrogen]	**1.Triple negative** MDA-468, MDA-MB-231, BT20 2. **Hormone dependent** i) MCF-7, MCF-7-D-40 & T47D, MCF-7 HER2 cells. ii) BT-474 - synergistic effects with tamoxifen.	1. **EGF → Akt → NF-κB pathway** Genistein blocks EGF signaling → suppresses Akt → downregulates NF-κB → induces apoptosis. 2. **MEK5 → ERK5 → NF-κB pathway** Genistein inhibits MEK5/ERK5 → reduces NF-κB activity → suppresses cell growth & triggers apoptosis. 3. **Akt → HIF1α → VEGF pathway** (angiogenesis-related) In silico studies: genistein inhibits this cascade → ↓ VEGF expression → suppresses angiogenesis. 4. Genistein addition in T47D cells results in a decrease in the expression of MMPs 2, 3, 3, and 15, preventing angiogenesis and metastasis. 5. Genistein controls MAPK (ERK1/2) and PI3K/AKT signaling pathways, epigenetic regulation, inhibits angiogenesis, invasion, and cell migration, and modifies the expression of numerous miRNAs. 6. Genistein raises the Bax/Bcl-2 ratio, causing apoptosis through autophagy-dependent pathways.	Inhibiting angiogenesis, anti-tumor activity, inducing apoptosis, anti-oxidant, immunity modulation, anticancer effect, antiproliferative, chemo-preventive activity. It also inhibits invasion, and cell migration.	40, 41
14	Mimosa pudica	L- mimosine, Methanol extract of whole plant [MMP]	1.Triple negative i)MDA-MB-453 ii)MDA-MB- 231	1.It significantly reduces the level of anti-apoptotic protein Bcl-2.	Free radicals scavenging activity, antioxidant,	42, 43

			2. Hormone dependent i) MCF-7 cell	2.Induce mitochondrial dependent intrinsic pathway apoptosis by increasing intracellular ROS. 3. 1. NF-κB, COX & p-AKT pathway inhibiton. 4. It inhibits the cell cycle traverse in the late G1phase prior to the onset of DNA synthesis that leads to cell cycle arrest.	anticancer activity, cytotoxic effect, anti-tumor activity, inducing apoptosis, anti-mutagenic effect.	
15	Semecarpus anacardium	1.BLEA [Ethyl acetate extract of S.anacardium leaves]-17 phytochemical compounds- (E)-octadec-9-enoic acid, palmitic acid, (E)-3,7,11,15-tetramethylhexadec-2-en-1-ol, methyl3-(3,5-di-tert-butyl-4-hydroxyphenyl) propanoate 2.The nut contains bhilawanols, cardol, anacardic acid, semecarpol and anacardol	**In vitro** 1. **Hormone dependent** i. MCF-7 cells [leaf] ii. T47D [nut] 2.**Triple negative** i. MDA-MB-231[leaf] **In vivo** 1.EAC cell-induced tumor-bearing mice	1. The nut extract is cytotoxic and induces cell death through apoptosis in breast cancer (T47D) cells by arresting the transition of the G2/M phase 2. BLEA exhibited cell cycle arrest in the G1 phase and apoptotic cell death in MCF-7 cells.	Cytotoxicity, anti-tumor, inducing apoptosis, inhibits cell migration, anticancer activity, antioxidant, anti-inflammatory, suppressed cancer cell migration, dna synthesis inhibition, inhibition in tumor growth.	45
16	Hemidesmus indicus	1.Root: Tannins, sterols, saponin, terpenoid, lupeol, amyrins, resin acid, desinine, sitosterol, and fatty acids, hemidesmol, 2-hydroxy-4-methoxy benzaldehyde (MBALD) 2.Flower: Isoquercetin, rutin, hyperoside 3.Leaf: tannins, hemidesmin 1&2.	1.MCF-7 cell	1. Modified several intracellular signaling pathways [STAT-3, HIF-1, NF-κB, IL-1, Cox-2, TNF-α, and oncogenic Kinase (P13k, IKK, IRAK1/4, JAK, MARK/ERK, and Syk/Src] involved in cell survival and proliferation, and ultimately caused tumor cell death via reduction in mitochondrial transmembrane potential and enhanced the Bax/Bcl-2 ratio. The mitochondrial depolarization is caused by H. indicus roots. By releasing intracellular Ca2 + reserves, H. indicus caused a large Ca2+ increase.	Anti-inflammatory, anticancer, antioxidant activity, chemo-preventive agent, cytotoxicity, anti-angiogenic effect, anti-carcinogenic, antiproliferative.	46-50

				2. The angiogenesis inhibition induced in hypoxia was regulated by a more complex mechanism involving firstly HIF-1α inhibition, and then VEGF and VEGFR-2 down-regulation.		
17	Smilax zeylanica	Diosgenin, smilagenin, β-sitosterol, hydroxytyrosol, squalene and sarsapogenin.	1.MCF-7 & MDA-MB-231cells	Diosgenin is able to inhibit proliferation of MCF-7 & MDA-MB-231 breast cancer by upregulation of p53 tumor suppressor gene and downregulation of Bcl-2 which promotes cell survival.	Anti-inflammatory, antioxidant, cytotoxic activity.	50
18	Thespesia populnea	Populene- A-H, mansonone-E, mansonone -D, gossypol, thespone, thespesone.	1.Bark methanol extract: MCF-7 & MDA-MB-231cells 2.Wood & dark heartwood dichloromethane extract: MCF-7 cell	1. It may act via estrogen receptor pathways or through alternative signaling pathways such as MMP, Akt, NF-κB, and MAPK.	Anti-tumor, antioxidant activity, cytotoxic activity, wound healing properties.	50, 51
19	Phyllanthus emblica	PEFeNPs, polyphenols, Tannins: Ellagic acid, corilagin, pyrogallol, chebulic acid, gallic acid **Flavonoid**: Quercetin **Others:**geraniin, norsesquiterpenoids, elaeocarpusin, and prodelphinidins B1 and B2.	**In vitro** 1.MDA-MB-435 2.MDA-MB-468 3.MDA-MB-231 It doesn't act against MCF10A (normal breast cancer cell) 4.MCF-7 **In vivo** 1.DMBA induced breast cancer in rats	1.Alteration of mTOR/Maf-1/PTEN signaling pathway 2. Inhibition of NF-κB 3. inhibition of growth factor signaling pathway i. EFGR pathway ii.PI3K pathway	Anticancer & antioxidant activity, anti-inflammatory, antitumor, free radicals scavenging activities, tumor repressive property, induce apoptosis, cancer preventive, cytotoxic activity, anti-proliferative, autophagy, anti-neoplastic activity.	52-54
20	Piper nigrum	Piperine	**In vitro** 1.**Hormone dependent**. i. HER-2 overexpressing breast cancer cell ii. MCF-7	1.Piperine strongly suppressed EGF-induced MMP-9 expression through inhibition of AP-1 and NF-κB activation by interfering with ERK1/2, p38 MAPK, and	Immunomodulatory, antioxidant, anti-inflammatory, induce cytotoxicity,	55-57

			2.**Triple Negative** Treated by piperine potentiated TRAIL [tumor necrosis factor related apoptosis-inducing lignand] i. MDA-MB-468 ii. MDA-MB-231 **In vivo** i.EMT6/P derived from BALB/c mouse. ii. Murine 4T1	Akt signaling pathways resulting in a reduction in migration. It blocks ERK1/2 signaling pathway which leads to significant reduction in SREBP-1 level and FAS [fatty acid synthase] expression. 2.Induce apoptosis through activation of caspase-3 and PARP [poly ADP-ribose polymerase (PARP] cleavage. 2. Piperine enhances TRAIL responsiveness along with suppression of survivin synthesis and p65 phosphorylation. 3. It inhibits tumor necrosis factor-α (TNF-α)-induced activation of NF-κB 4.It inhibited human X-linked IAP(XIAP), a protein that prevents apoptosis. 5. Cytotoxic effect through cell cycle arrest in G2/M phase. 6. It controls a lot of proteins in the Bcl-2 family, which includes Bax and Bcl-2. 7. It suppresses the tumor growth through downregulation of Ki-67 expression.	antimetastatic, anticancer, chemo-preventive effect, anti-proliferative, inducing apoptosis, anti-angiogenic property.	
21	Piper longum	Piperlongumine	1.MCF-7 2. TNBC 3.MDA-MB-231	1. Induced cell cycle arrest in G2/M phase 2.Modulates Cell Cycle-Regulatory Proteins such as cyclins and CDKs. 3. Induce intracellular ROS accumulation & GSH depletion. 4. Decreases nuclear translocation of NF-κB p^{65} 5. IKKβ suppression 6. PL Increases the Expression of p21 mRNA 7. PL directly interacts with Keap1, which leads to Nrf2 activation and	Anticancer, antioxidant, antiproliferative, suppress migration	58

				upregulates HO-1 expression, thereby resulting in the selective killing of cancer cells in the breast [9]. In addition, PL induced apoptosis of breast cancer cells via activation of transcription 3 (STAT3) and phosphatidylinositol 3-kinase (PI3K)/Akt/mammalian target of the rapamycin (mTOR) signaling pathway.		
22	Withania somnifera	Withaferin-A, anaferine, isopelletierine, sitoindoside IX, somniferine, withanone	**In vitro** 1.ZR751 2.MCF-7 3.MDA-MB-231 4. SUM159 5.SK-BR3 6.T47D 7.MDA-MB-468 **In vivo** 1. MDA-MB-231 xenograft 2.SUM159 and MCF-7 xenograft	1.Withaferin A diacetate, a derivative of withaferin, significantly decreased the viability of breast cancer stem cells. Tubocapsenolide A (TA), a new withanolide derivative, can decrease the activity of the Hsp90-Hsp70 chaperone complex via diol oxidation, causing the instability of Hsp90 client proteins, and cell cycle arrest and apoptosis in human breast cancer MDA-MB-231 cells 2.inhibits STAT3 & STAT5 signaling and TNF. 3. Modulates MAPK pathway 4.tumor suppressor activation – upregulates p53 & upregulates p21 5.reduces estrogen receptor-α 6.decreases Bcl-2 &Bcl-XL / increases Bax 7.disruption of PI3/Akt signaling 8.NF-κB inhibition 9. inhibits MMP2-9 and VEGF expression. 10. It increased the expression of the breast cancer metastasis suppressor gene (BRMS1) in MDA-MB-231 breast cancer cells.	Anticancer, inducing apoptosis, inducing ROS level, anti-metastatic, anti-angiogenic, anti-inflammatory, antioxidant, chemo-preventive, immunomodulatory	59

23	Aloe barbadensis	Aloin, aloe-emodin, aloesin, emodin.	In vitro 1.MCF-7(ER +)	1.Aloe Emodin can be an effective inhibitor to the hormonal dependence MCF-7 breast cancer cells at low concentration without being cytotoxic to normal breast cells Targeting estrogen receptor protein stability through distinct mechanisms.	Anti-proliferative	60
			2. Her2/neu	1.Emodin Azide Methyl Anthraquinone Derivative (AMAD) was found to effectively block phosphorylation of Her2/neu, suppress growth, transformation and metastasis as a tyrosine kinase inhibitor, and increase the susceptibility of Her2/neu-over expressing cancer cells to standard cytotoxic therapeutic agents 2. Reduction of the level of Bcl-2 and increased levels of cleaved caspase-3, PARP, p53 and Bax 3. Disruption of the PI3K/Akt-dependent pathway	Apoptotic activity, anticancer, antioxidant, anti-inflammatory, wound healing, anti-tumor activity, anti-angiogenesis, suppress metastasis, anti-proliferative activity.	61
24	Capsicum annum	1.Pectic polysaccharides 2.Capsaicinoids: capsaicin, dihydrocapsaicin, nordihydrocapsaicin, homodihydrocapsaicin, homocapsaicin, norcapsaicin.	In vitro i.MCF-7 ii.BT-20	1. Capsaicin significantly decreased mitochondria membrane potential, induced the cleavage of PARP-1, and decreased procaspase-7 expression in both cells. Apoptosis-inducing factor (AIF) was distinctly released from mitochondria and translocated into the cytoplasm and nucleus in MCF-7 cells (52.9%), but not in BT-20 cells (2%) after treatment with 200μM of capsaicin for 24 hours and arrested cell cycle in S phase.	Inducing apoptosis and cell cycle arrest	62

		Capsaicin	In vitro ii.T47D iii.BT-474 2. Triple negative i.MDA-MB-231 ii.MDA-MB-436 iii.BT-20 iv.SKBR-3 In vivo i. Mouse xenograft model.	1. G0/G1 cell cycle arrest. 2. capsaicin is a novel modulator of the EGFR/HER-2 pathway in both ER-positive and -negative breast cancer cells with a potential role in the treatment and prevention of human breast cancer. 3. In vivo activation of ERK was clearly decreased, as well as expression of HER-2 and cyclin D1, whereas caspase activity and PARP cleavage products were increased in tumors of drug-treated mice.	Inducing apoptosis & cell cycle arrest, anti-proliferative	63
25	Glycyrrhiza glabra	Licochalcone A, glabridin, isoliquirtigenin, glycyrrhizin, glycyrrhetinic acid.	1.Hormonal dependent i.MCF-7 ii. T47D 2.Triple negative i.MDA-MB-231 ii.MDA-MB-361 In vivo i. 4T1 cell in BALD/c mice	1. Decrease Bcl-2, mTOR, p-mTOR, COX-2, N-cadherin & increases Bax, caspase 3 & 8, PARP, p62, CDK2, E-cadherin. 2. Decreases VEGF-A & HIF-1α.	Anti-inflammatory, antioxidant, anticancer, anti-proliferative, cell cycle arrest, activities, immune-stimulating activity, inducing autophagy, induce apoptosis, inhibiting angiogenesis.	64
26	Moringa oleifera	1.Chlorogenic acid, quercetin, ellagic acid, niazirin, kaempferol, eugenol.	1. Hormonal dependent In vitro i.MCF-7[leaf extract]	1. Its significant antiproliferative effect was observed at 200 μg/mL dose of fraction B (ethyl acetate) and cell viability was reduced to 40%. In conclusion, it causes cyclin dependent kinase-2 (CDK-2) inhibition while ellagic acid, chlorogenic acid and quercetin being the most stable and potent inhibitors to treat estrogen receptor positive breast cancer targeting CDK-2.	Antiproliferative, anti-cancer, anti-inflammatory activity, increase apoptosis, antioxidant, chemo-preventive.	65

		2.Niazimicin, thiocarbamate, benzyl isothiocyanate, glucosinolates, isothiocyanates, glycosides, zeatin (anti-aging compound), saponins, tannins.	In vitro [crude methanolic leaf extract & dichloromethane] i.MCF-7 ii.MDA-MB-231	2. It reduced NF-κB expression, decreased cell growth in both cells by inducing apoptosis. 3. MO contains zeatin, which is an anti-aging compound. It also has anti-cancer properties and is a good anti-oxidant.	Antioxidant, anti-cancer, anti-inflammatory activity.	66
27	Ferula assa-foetida	1.Nano emulsion containing ferula assa-foetida seed essential oil	1. MCF-7 cell	Increased BAX and decreased BCL2 expression, decreased VEGF and VEGFR.	Antiproliferative, anti-tumor, anti-inflammatory activity, anticancer, antimutagenic, antineoplastic, antioxidant, cytotoxic activity	67
		2.Silver nanoparticles (AgNPs) with aqueous extract of ferula assa-foetida extract	2. MCF-7 cell			
		3. Zinc nanoparticles containing ferula assa-foetida extract	3. MCF-7, MDA-MB-231			
		4.Essential oil of assa-foetida	4.MCF-7	Decreased the viability of MCF-7 cells.		
		5.Ferulic acid	5. MDA-MB-231 6. Mouse xenograft model 7.4T1 cell	Increased caspase 3 and reduced the proliferation of cancer cells about 40% at 100 μM. 100mg/kg significantly reduced tumor volume, weight and growth in mice. Reduced the growth of cancer cell at 500μg/ml.		
		6. Galbanic acid	8. MCF-7, MDA-MB-231	Up-regulation of Bax and caspase-3 and down-regulation of bcl-2.		
		7.Sequiterpene coumarins	MCF-7	Cytotoxic activity against MCF-7 cell		
		8.Farnesiferol C	MCF-7	Decrease cell viability and stopped the cell cycle in G0/G1 phase and induced apoptosis		
28	Mangifera indica	Mangiferin, norathyriol, gallotannin, pyrogallol, gallic acid, quercetin, methyl gallate.	In vitro [cell lines] 1.**Bark:** MDA-MB-231, **Kernel:** MCF-7, MDA-MB-231, **Leaf:** BT-474, MCF-7, MDA-MB-231, **Peel**: MCF-7,	1. Inhibition of NF-KB &nRac1/WAVE2 pathways, suppression of PI3K/AKT pathway, activation of AMPK pathway, inactivation of β-catenin pathway.	Anti-cancer, cytotoxic, anti-inflammatory, antioxidant, immunomodulatory, anti-	68

			Pulp: BT-474, MCF-7, **Peel and plup:** MCF-7, MDA-MB-231 2.**Polyphenols:** MDA-MB-231, MCF10DCIS, **Mangiferin**: MCF-7, MDA-MB-231, T47D, BT-549, **Norathyriol:** MCF-7, **Gallotannin: MCF-7, Gallic acid:** MCF-7, MDA-MB-231, MCF10DCIS. **Pyrogallol:** MCF10DCIS**, Methyl gallate:** MCF10DCIS**, Quercetin:** MCF-7, mice bearing **In vivo [xenograft]** 1.Pulp: mice bearing BT-474 xenografts 2. Polyphenols & quercetin: mice bearing MCF10DCIS 3.Mangiferin & quercetin: mice bearing MDA-MB-231.	2. Modulation of PPARS, intercellular Ca^{2+} signaling, cell cycle regulators, ER activity. .	proliferate and reduce tumor volume in ductal carcinoma in situ breast cancer.	
29	Bacopa monnieri	1.The ethanolic extract of DCM [Dicholromethane] of Bacopa monnieri contains: Bacoside A and B, brahmin, cucurbitacins, and betulinic acid.	1) In vitro i. MCF-7 ii. MDA-MB-231	Inducing cell cycle arrest at G2/M phase	Anti proliferate, antitumorigenic activity, cytotoxic activity, inducing cell cycle arrest.	69
		2. Bacopaside I & II [synergistic action]	In vitro i. MDA-MB-231 & BT-474 (most sensitive) ii. MCF-7, T47D, BT-474 cell lines	1.Inducing cell cycle arrest at G2/M phase. 2. Altered morphology of breast cancer cell lines, 3.Reduced transcript expression of AQP1 in MDA-MB-231 cells.	Anti-proliferative, induce cell cycle arrest, induce apoptosis and reduce migration, reduced invasion in mda-mb-231.	70
30	Camellia sinensis	Epigallocatechin-3-gallate	1**. ER +:** MCF-7, T47D, ZR-75-1 cells 2. **HER2 +:** BT-474, SK-BR-3	1.blocks cell cycle arrest at G0/G1 or G2/M phase.	Anti-proliferative, proapoptotic activity, anti-	71

			3. **TNBC:** MDA-MB-231, MDA-MB-468, Hs578T 4. **Animal models:** **(**Xenograft models) MCF-7 xenografts (ER+), MDA-MB-231 xenografts (TNBC)**,** BT-474 xenografts (HER2+) **Syngeneic models**: 4T1 cells in BALB/c mice to test.	2.Increases pro-apoptotic proteins- p53, Bax, caspase. 3.decreases anti-apoptotic proteins- Bcl-2, survivin. 4.Inhibits telomerase (hTERT). 5.Reduces MMP-2 &9, inhibits β-catenin/Wnt and FAK pathways. 6.Prevents EMT [epithelial to mesenchymal transition] 7. Decreases VEGF 8. Inhibits NF-κB & STAT3 signaling, lowers inflammatory cytokines (IL-6, TNF-α).	metastatic & anti-migratory effects, anti-angiogenic activity, anti-inflammatory, immune modulation.	

Discussion
All the 30 selected medicinal plants demonstrated in-vitro activity against breast cancer cell lines, although the depth of evidence varies considerably. Certain plants, such as Curcuma longa, Nigella sativa, Piper nigrum, Glycine max, and Withania somnifera, have been extensively studied, whereas others show preliminary but promising results.
Several phytochemicals from medicinal plants have demonstrated significant anti-breast cancer activity in in vivo models. Among them, curcumin (Curcuma longa), thymoquinone (Nigella sativa), piperine (Piper nigrum), and soy isoflavones such as genistein and daidzein (Glycine max) provide the strongest and most consistent evidence, showing inhibition of tumor growth, induction of apoptosis, and suppression of metastasis in various breast cancer models. In addition, Foeniculum vulgare and Zingiber officinale have exhibited activity against 4T1 and BaP-induced breast tumors, while Phyllanthus emblica and Aegle marmelos significantly reduced tumor incidence in DMBA-induced rat mammary carcinogenesis. Glycyrrhiza glabra and Ferula assa-foetida have also shown promising effects in 4T1 and xenograft models. Together, these findings suggest that phytochemicals exert their anti-cancer effects through multiple mechanisms including apoptosis induction, anti-mutagenic activity, inhibition of angiogenesis, and chemo-preventive potential, highlighting their value as complementary agents in breast cancer management
The following medicinal plants have demonstrated strong evidence from both in vitro and in vivo studies against breast cancer: Curcuma longa (curcumin), Nigella sativa (thymoquinone), Piper nigrum (piperine), Glycine max (soy isoflavones), Phyllanthus emblica (amla), Zingiber officinale (gingerol), Foeniculum vulgare (fennel), Aegle marmelos, Glycyrrhiza glabra (licorice), and Ferula assa-foetida, Mangifera indica.
Only a few medicinal plants have reached clinical evaluation for anticancer properties: Curcuma longa (curcumin) shows safety and modest efficacy; Glycine max (soy isoflavones) has been studied for breast cancer prevention and recurrence with mixed results; Withania somnifera (ashwagandha) provides supportive therapy by reducing fatigue and boosting immunity; Vitis vinifera (resveratrol and grape extracts) is under limited clinical trials for chemoprevention; Brassica oleracea (sulforaphane from broccoli sprouts) has been explored for anticancer potential; and Phyllanthus emblica (amla) shows early evidence for antioxidant and supportive anticancer roles, though efficacy is not yet fully established and camellia sinensis has been clinically tested for its chemo-preventive effects in cancers like breast, prostate, skin.
Plants like flaxseed and soy isoflavones are primarily active in hormone-dependent (ER+) cells, making them relevant for estrogen-receptor-positive tumors. Most triple-negative studies employed the MDA-MB-231 cell line, which is considered the prototypical model for aggressive, invasive breast cancer. A few studies also extended findings to other TNBC lines such as MDA-MB-468 and BT-549, and to the murine 4T1 model. Most others have been tested in both hormone-dependent and triple-negative models, showing broader anticancer potential.

Several medicinal plants have demonstrated promising anticancer activity against breast cancer cells through diverse mechanisms.

Out of the 30 medicinal plants reviewed, roughly 20–22 exhibit **apoptosis or anti-proliferative activity** in breast cancer cells. Exceptions include Hemidesmus indicus, Piper nigrum, and Ferula assa-foetida, which primarily act through anti-proliferative mechanisms without directly inducing apoptosis, as well as Flax seeds (Linum usitatissimum), Vitis vinifera, Foeniculum vulgare, Smilax zeylanica, and Thespesia populnea, whose anticancer effects are mainly mediated via other mechanisms.

Anti-angiogenic effects have been reported for flax seeds (Linum usitatissimum), Tinospora cordifolia, Centella asiatica, Zingiber officinale, Aegle marmelos, Glycine max, Mimosa pudica, Hemidesmus indicus, Piper nigrum, Aloe barbadensis, and Glycyrrhiza glabra, primarily by suppressing VEGF expression, inhibiting endothelial cell proliferation, and modulating matrix metalloproteinase activity.

Several phytochemicals, including curcumin (Curcuma longa), thymoquinone (Nigella sativa), piperine (Piper nigrum), gingerol (Zingiber officinale), withaferin A (Withania somnifera), resveratrol (vitis vinifera) and bacopa monnieri have shown strong **anti-metastatic** effects by suppressing epithelial–mesenchymal transition (EMT), downregulating MMPs, and inhibiting invasion pathways.

Several medicinal plants have demonstrated **chemo-preventive properties** against breast cancer, including Curcuma longa (curcumin), Vitex vinefera, Zingiber officinale (ginger), Piper nigrum (black pepper), Moringa oleifera, and camelia sinensis. Additionally**, anti-mutagenic activity** has been reported for thymoquinone (Nigella sativa) and allicin (Allium sativum), which protect DNA from mutagen-induced damage, while Ferula assa-foetida shows general anti-mutagenic potential that may contribute to chemoprevention. Anti-mutagenic activity has been reported for thymoquinone (Nigella sativa) and allicin (Allium sativum), which protect DNA from mutagen-induced damage, while Ferula assa-foetida demonstrates general anti-mutagenic potential that may contribute to chemoprevention.

Conclusion

The medicinal plants reviewed in this article exhibit significant anticancer activity against breast cancer cells. Most of these plants have been investigated in *in vitro* studies, demonstrating promising therapeutic potential. While modern medicine provides effective treatment options, the incorporation of herbal medicines may further enhance clinical outcomes. Integrative approaches that combine conventional therapies with plant-based remedies offer considerable promise for the future management of life-threatening diseases such as cancer.

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