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Review Article

PLANT-DERIVED PHYTOCONSTITUENTS WITH ANTI-BREAST CANCER POTENTIAL: MECHANISTIC INSIGHTS, EVALUATION, AND THERAPEUTIC IMPLICATIONS

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ABSTRACT

This study reviews medicinal plants and their phytochemicals that show potential against breast cancer. Relevant research published up to August 2025 was collected from PubMed, Scopus, Web of Science, and Google Scholar. The selected studies reported anticancer activity, mechanisms, or IC_{50} values of plant-derived compounds *in vitro* and *in vivo*. These compounds were grouped as terpenoids, polyphenols, alkaloids, flavonoids, lignans, organosulfur compounds, and other bioactive molecules. Polyphenols (e.g., curcumin, resveratrol), terpenoids (e.g., withaferin A), and ginsenosides showed strong effects with low micromolar IC_{50} values, while alkaloids, such as vinblastine and vincristine demonstrated very high (nanomolar) potency. Some compounds, such as S-allyl cysteine and berberine, were less effective. Flavonoids, including Epigallocatechin gallate and Quercetin, showed notable anticancer activity. The main mechanisms involved are antioxidant action, immune modulation, hormone regulation, inhibition of metastasis and angiogenesis, and induction of cell cycle arrest and apoptosis. Overall, plant-based phytoconstituents show promising potential for breast cancer treatment and prevention, but issues, such as limited clinical evidence, poor bioavailability, and lack of standardization remain challenges. Advances in personalized medicine, synergistic therapies, standardized formulations, and nanotechnology may enhance their clinical use in the future.

Keywords: Breast cancer activity, Medicinal plants, Phytoconstituents, Natural products.

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INTRODUCTION

The most diagnosed disease among women is breast cancer, which has a significant impact on global health. With more than 2.3 million new cases reported each year, it continues to be the leading cause of cancer-related deaths among women, according to the World Health Organization (WHO) [1]. While survival rates have improved due to advancements in hormone therapy, chemotherapy, radiation, and targeted treatments, such as Human Epidermal Growth Factor Receptor 2 inhibitors, several challenges remain [2]. These challenges include the development of non-specific cytotoxicity, cancer recurrence, multidrug resistance, and severe side effects from treatments, which can hinder long-term effectiveness and patient adherence. It is crucial to explore innovative, safe, and successful treatment methods [3].

Medicinal plants have played a crucial role in the drug development process, contributing to nearly 60% of the anticancer medications available on the market today [4]. *Catharanthus roseus* contains various compounds, including vinca alkaloids and paclitaxel from *Taxus brevifolia* illustrate how phytoconstituents can be effectively transformed into established chemotherapeutics [5]. In recent decades, plant-derived bioactive compounds have garnered increased attention due to their potential cytotoxic effects and their ability to influence various signaling pathways associated with cancer development, including hormone signaling, angiogenesis, metastasis, and apoptosis [6].

Numerous phytochemicals derived from natural products, such as lignans, terpenoids, alkaloids, flavonoids, polyphenols, and organosulfur compounds, have demonstrated significant anti-breast cancer effects in pre-clinical models [7]. These substances operate through various mechanisms, such as preventing metastases, inhibiting estrogen receptor (ER) signaling, and inducing apoptosis mediated by mitochondria [8]. In addition, certain phytoconstituents have exhibited synergistic effects when combined with conventional chemotherapeutics, presenting opportunities to reduce toxicity and address resistance [9].

This study aims to provide a thorough analysis of medicinal plants with anti-breast cancer properties, categorized by their main phytoconstituents [10]. This research highlights the importance of phytochemicals in potential future treatments for breast cancer by detailing their mechanisms of action, possible therapeutic applications, and existing limitations [11]. By focusing on phytochemical categorization, modes of action, IC_{50} potency values, current limitations, and potential future clinical applications, this study presents an in-depth examination of medicinal plants that have documented efficacy against breast cancer as shown in the Fig. 1 [12].

METHODS

This study utilized a thorough and systematic method to identify and assess relevant research on medicinal plants and their phytoconstituents that could contribute to the prevention of breast cancer [13]. PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar were among the databases searched for materials published up until August 2025 [14]. Various combinations of the terms "breast cancer," "phytochemicals," "phytoconstituents," "medicinal plants," "anticancer activity," "IC_{50}," and "mechanism of action" were utilized with Boolean operators [15]. Additional pertinent studies were identified by examining the reference lists of retrieved papers and related reviews [16].

The inclusion criteria were strict, focusing only on peer-reviewed English-language articles that presented experimental evidence, whether *in vitro*, *in vivo*, or clinical demonstrating the anticancer potential of plant-derived chemicals [17]. Special emphasis was placed on studies providing IC_{50} values and insights into mechanisms of action [18]. Exclusion criteria encompassed conference abstracts, non-English articles, general

antioxidant studies unrelated to breast cancer, research yielding insufficient anticancer results, and substances with uncertain identification and these were illustrated in the Fig. 2 [19].

Key areas for systematic data extraction and synthesis from the selected research included phytochemical class, plant source, representative compounds, experimental model, IC_{50} range, and mechanisms of action, along with author citations [20]. The study identified trends and differences among various phytoconstituent categories, including alkaloids, flavonoids, lignans, terpenoids, polyphenols, and organosulfur compounds. These findings were presented in comparative tables and graphical representations [21]. The research selection method followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. It started with database searches and progressed through title and abstract screening to a full-text review, ensuring methodological rigor and scientific significance and shown in the Fig. 3 [22].

PHYTOCONSTITUENT-BASED CLASSIFICATION OF MEDICINAL PLANTS WITH ANTI-BREAST CANCER ACTIVITY

Alkaloids

Annona muricata

A therapeutic plant rich in bioactive phytoconstituents, particularly acetogenins, such as annonacin, squamocin, and bullatacin, is *A. muricata*, commonly known as soursop or graviola. The chemical structures of these compounds are illustrated in Fig. 4. The anticancer properties of these substances, especially concerning breast cancer, have been well established. They caused mitochondrial dysfunction and reduced ATP levels, which effectively promoted apoptosis in Michigan Cancer Foundation-7 (MCF-7) breast cancer cells and shortened the lifespan of these cancer cells [23]. The activation of caspase-3 is an essential mediator of programmed cell death; it is the primary mechanism through which ethanolic extracts of *A. muricata* leaves exhibited significant cytotoxicity against the triple-negative breast cancer cells, MDA-MB-231 [24]. The IC_{50} values of annonacin were found to be 8.5 μM in MDA-MB-468 cells and 15 μM in MDA-MB-231 cells, both of which are triple-negative breast cancer cells. These compounds were primarily tested on MCF-7 and MDA-MB-231 cells for a duration of 24–48 h using the MTT assay as show in the Table 1. In contrast, the IC_{50} value for MCF-7 cells was 21.10 μg/mL. Additional findings indicate that annonacin exhibits a broad cytotoxic potential, with IC_{50} values ranging from 32 to 40 μg/mL across various tumor lines [25].

C. roseus

The well-known medicinal plant *C. roseus*, commonly referred to as Madagascar periwinkle, contains significant phytoconstituents, including vincristine, vinblastine, and vinorelbine, which are classified

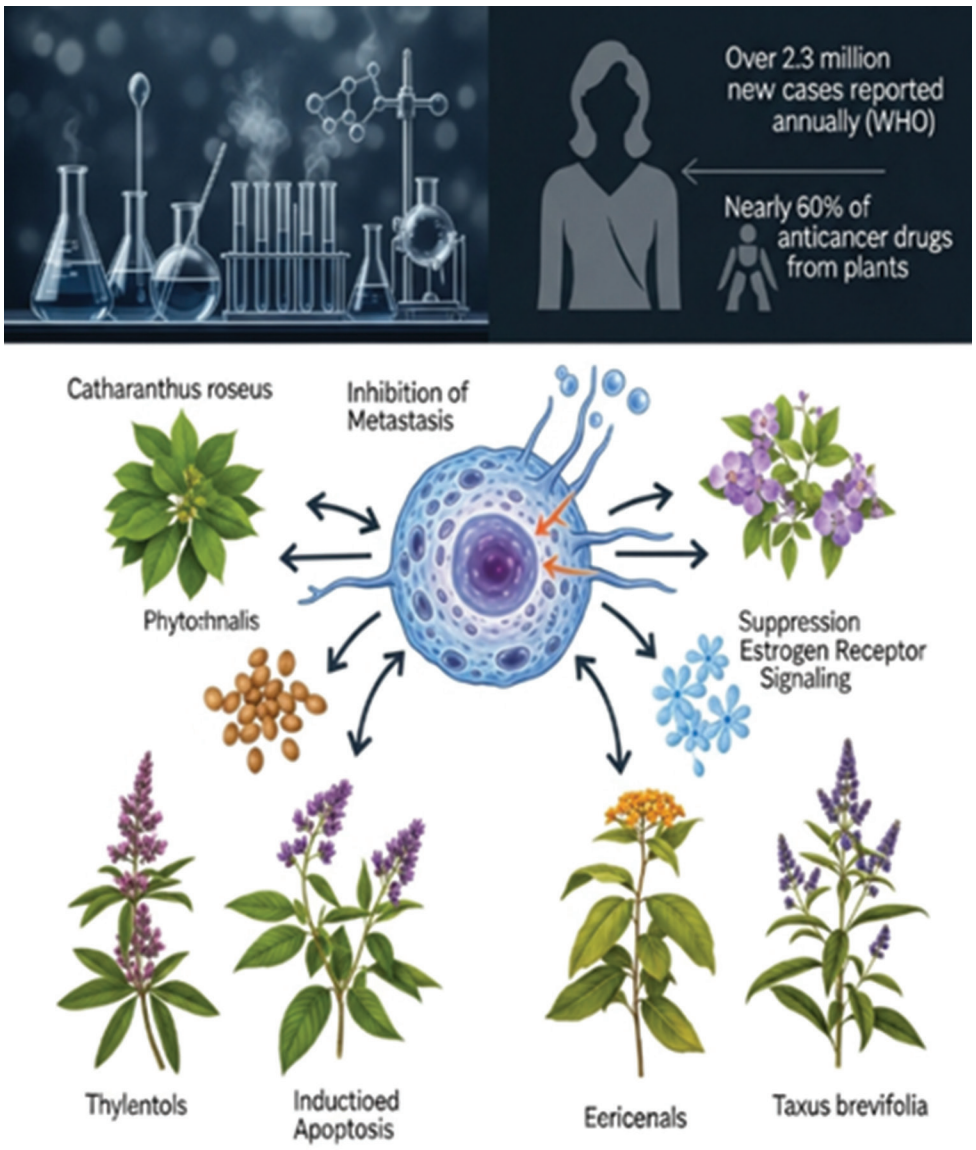

Fig. 1: This illustrates the application of plant derived anticancer mechanisms in treating breast cancer

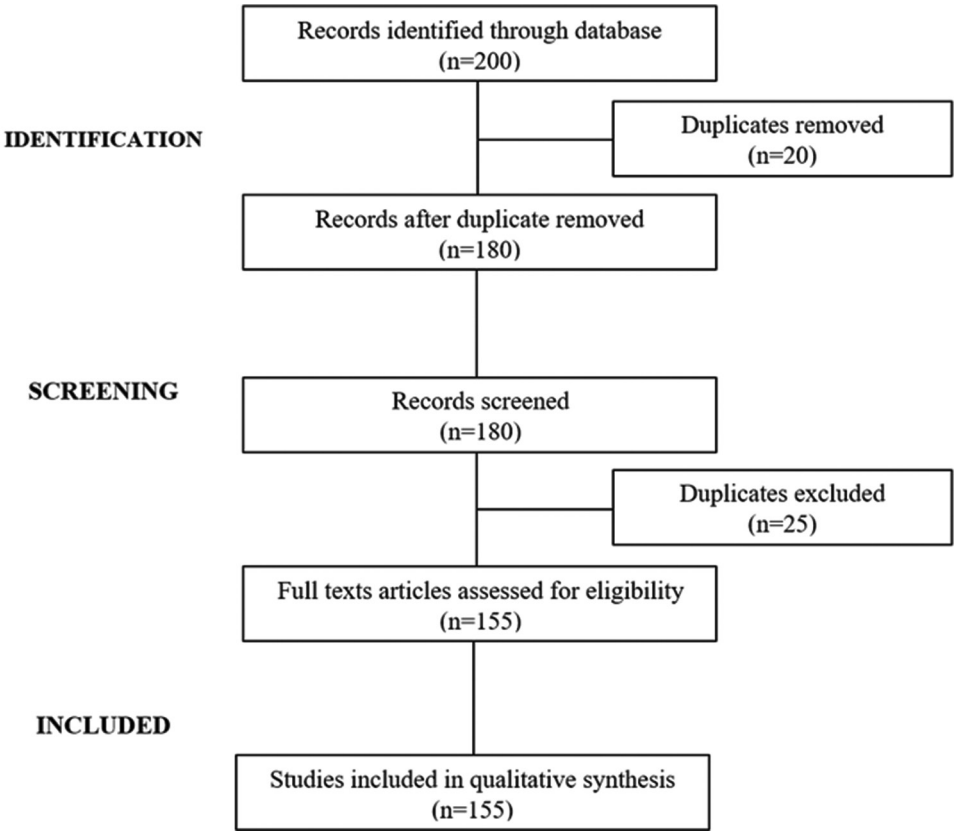

Fig. 2: A Preferred Reporting Items for Systematic Reviews and Meta-Analyses flow diagram summarizing the study selection procedure indicates that 155 studies were selected from 200 records identified and 180 records assessed

as vinca alkaloids. The chemical structures of these compounds are illustrated in Fig. 4 [26]. Vinca alkaloids' anticancer properties were showed that they work by interfering with the synthesis of microtubules, which causes mitotic arrest and inhibits cancer cell growth [27]. Their mechanism of action has enabled the development of these alkaloids into clinically approved chemotherapeutic agents that are now widely used to treat various cancers, including breast cancer [28]. Vinblastine is significantly more potent; tests have shown that its IC_{50} values in MCF-7 cells are around 0.68 nM. At 1-10 nM, vinorelbine exhibits action and causes cell-cycle abnormalities that are consistent with microtubule disruption [29]. The phytoconstituents were assessed on MCF-7, MDA-MB-231, and T47D cells using the MTT test over a period of 24–48 h, as detailed in Table 1. According to these findings, vinca alkaloids are compelling and serve as the gold standard for anticancer medications [30].

Berberis vulgaris

The isoquinoline alkaloid berberine has been extensively studied for its potential to prevent cancer. The chemical structure of berberine is shown in Fig. 4. It may inhibit the growth of MCF-7 breast cancer cells and reduce tumor cell proliferation by inducing apoptosis and causing G1 phase cell cycle arrest [31]. By generating reactive oxygen species (ROS), it leads to mitochondrial dysfunction, resulting in cancer cell death in breast cancer models [32]. For instance, in MCF-7 cells, one study reported an IC_{50} of approximately 52 μM after 48 h, while another study employing different methodologies found an IC_{50} of 272 ± 11 μM for the same cell line [33]. The evaluation was conducted over a period of 48–72 h using MTT and SRB assays on the MCF-7 and MDA-MB-231 cell lines, as detailed in Table 1. In addition, it has been investigated as a chemosensitizer capable of reversing multidrug resistance [34].

Flavonoids

Passiflora spp.

The flavonoid chrysin is one of the primary bioactive phytoconstituents found in *Passiflora species*, commonly known as passionflowers, whose chemical structure is illustrated in Fig. 5 [35]. The development of ER-positive breast cancer cells is inhibited by chrysin, which effectively inhibits aromatase activity and reduces estrogen production [36]. The research indicates that chrysin induces apoptosis in MCF-7 breast cancer cells by modifying the PI3K/Akt signaling pathway, which is a key regulator of cell survival and proliferation [37]. The compound exhibits an IC_{50} range of 20–50 μM for the specific breast cancer cell line, indicating a moderate to high level of cytotoxicity. It is evaluated in MCF-7 cells using the MTT assay for a duration of 24–48 h, as indicated in Table 1.Its primary mechanisms of action include cell cycle arrest, inhibition of the PI3K/Akt and Mitogen-Activated Protein Kinase (MAPK) signaling pathways, and the induction of apoptosis [38].

Glycine max

Soybeans are a significant source of genistein, an isoflavone that has been extensively studied for its potential anticancer properties. The chemical structure of genistein is illustrated in Fig. 5 [39]. By inhibiting tyrosine kinase signaling, which disrupts essential processes required for the survival and proliferation of tumor cells, it induced apoptosis in MCF-7 breast cancer cells [40]. The typical IC_{50} values ranged from 20 to 50 μM, indicating the compound's moderate to high cytotoxic potential. It was administered to T47D and MCF-7 cell lines using the MTT assay for a duration of 48 h, as indicated in Table 1. Its primary mechanisms of action include the induction of apoptosis, the inhibition of the PI3K/Akt and MAPK signaling pathways, and the arrest of the cell cycle [41].

Silybum marianum

It is a plant whose seeds are rich in silymarin, a flavonolignan complex recognized for its potent hepatoprotective and antioxidant properties. The chemical structure of silymarin is depicted in Fig. 5 [42]. By modifying the p53 tumor suppressor and activating caspase-dependent pathways, it inhibited the growth of breast cancer cells and induced cell death [43]. It exhibits a strong anti-breast cancer effect against MCF-7 cells, with an IC_{50} range of 25–50 μM. It was assessed in MCF-7 cells over a duration of 48–72 h using the SRB test, as indicated in Table 1.

Table 1: The table includes phytoconstituents from medicinal plants that exhibit anticancer potential, along with details about their classifications, modes of action, estimated IC_{50} ranges, types of cell lines, assay type, assay duration and references. To account for variations in cell lines, exposure durations, and experimental methods, IC_{50} values are presented as ranges

S. No.	Plant/Source	Phytoconstituents	Phytochemical Class	IC_{50} values (μM)	Cell line type	Assay type	Assay duration	Mechanism of action	References
1	*Annona muricata*	Annonacin, Squamocin, and Bullatacin (Acetogenins)	Acetogenins	32–40	MCF-7, MDA-MB-231, T47D	MTT	24–48 h	Complex I inhibition, apoptosis	[25]
2	*Catharanthus roseus*	Vincristine, Vinblastine, Vindesine	Alkaloids	1–10	MCF-7	MTT	24–48 h	Microtubule disruption, apoptosis	[29,30]
3	*Berberis vulgaris/ aristata*	Berberine	Alkaloids	52–272	MCF-7, MDA-MB-231	SRB/ MTT	48–72 h	ROS generation, G1 arrest	[33,34]
4	*Passiflora spp.*	Chrysin	Flavonoids	20–50	MCF-7	MTT	24–48 h	Aromatase inhibition, apoptosis	[38]
5	*Glycine max*	Genistein	Isoflavonoids	20–50	MCF-7, T47D	MTT	24–48 h	Phytoestrogen, kinase inhibition	[41]
6	*Silybum marianum*	Silymarin	Flavonolignans	25–50	MCF-7	SRB	48–72 h	Antioxidant, apoptosis sensitizer	[44]
7	*Scutellaria baicalensis*	Baicalein	Flavonoids	20–40	MDA-MB-231	MTT	24–48 h	EMT inhibition, apoptosis	[47]
8	*Petroselinum crispum*	Apigenin	Flavonoids	25–35	MCF-7	MTT	48 h	p53 upregulation, VEGF inhibition	[51]
9	*Kaempferol (various)*	Kaempferol	Flavonoids	20–30	MCF-7	MTT	24–72 h	PI3K/Akt inhibition, apoptosis	[54]
10	*Chrysanthemum spp.*	Luteolin	Flavonoids	30–40	MDA-MB-231	MTT	48–72 h	ERK/Akt inhibition, apoptosis	[57]
11	Quercetin (various)	Quercetin	Flavonoid	20–50	MCF-7	MTT	24–72 h	PI3K/Akt inhibition, apoptosis	[61]
12	*Linum usitatissimum*	SDG (Secoisolariciresinol diglucoside)	Lignans	20–60	MCF-7	SRB	24–48 h	Phytoestrogenic, antioxidant	[63,64]
13	*Arctium lappa*	Matairesinol	Lignans	40–70	MDA-MB-231	MTT	48 h	Apoptosis, autophagy	[67]
14	*Sesamum indicum*	Lariciresinol	Lignans	35–60	MCF-7	MTT	48–72 h	Antioxidant, estrogen-modulatory	[70]
15	*Withania somnifera*	Withaferin A	Terpenoids (steroidal lactone)	0.5–5	MDA-MB-231	MTT	24 h	Caspase activation, STAT3 inhibition	[73]
16	*Azadirachta indica*	Nimbolide	Terpenoids (limonoids)	5–20	MCF-7, MDA-MB-231	MTT	24–48 h	PI3K/Akt inhibition, apoptosis	[76]
17	*Curcuma zedoaria*	Curdione	Terpenoids	20–50	MCF-7	MTT	24–48 h	Mitochondrial apoptosis, ROS	[79]
18	*Taxus brevifolia*	Paclitaxel	Taxane diterpenoid	5–20	BT-549, MCF-7	MTT	24–72 h	Microtubule stabilization, apoptosis	[81]
19	*Tripterygium wilfordii*	Triptolide	Terpenoids	10–100	MDA-MB-231	MTT	24 h	NF-κB inhibition, apoptosis	[85]
20	*Betula species*	Betulinic acid	Terpenoids (triterpenes)	10–50	MCF-7	MTT	24–48 h	Mitochondrial apoptosis	[86,87]
21	*Ocimum sanctum*	Ursolic Acid	Pentacyclic Triterpenoid	20-40	MCF-7	MTT	24–48 h	Mitochondria-mediated apoptosis, PI3K/Akt inhibition, G1 arrest, ROS induction, MMP-2/9 suppression	[90]
22	*Curcuma longa*	Curcumin	Curcuminoids (polyphenols)	5–25	MCF-7, MDA-MB-231	SRB	24–72 h	NF-κB inhibition, apoptosis	[95]
23	*Camellia sinensis*	EGCG	Polyphenols (catechins)	30–100	MDA-MB-231	MTT	24–48 h	Akt/NF-κB inhibition, ROS	[98]
24	*Phyllanthus emblica*	Gallic acid	Polyphenols (tannins)	30–80	MCF-7	SRB	48 h	Antioxidant, apoptosis	[102]
25	*Punica granatum*	Ellagic acid	Polyphenols (ellagitannins)	30–80	MDA-MB-231	MTT	48–72 h	Apoptosis, ER modulation	[106]
26	*Vitis vinifera*	Resveratrol	Polyphenols (stilbenes)	25–60	MDA-MB-231	MTT	48–72 h	ER modulation, apoptosis	[108]
27	S-allylcysteine (SAC)	S-Allylcysteine	Organosulfur amino acid	10–50	MCF-7	MTT	24–48 h	Antioxidant, apoptosis	[112]
28	Diallyl disulfide (DADS)	Diallyl disulfide	Organosulfur compound	10–50	MCF-7	MTT	48 h	Cell cycle arrest, ROS	[117]
29	Diallyl trisulfide (DATS)	Diallyl trisulfide	Organosulfur compound	20–50	MDA-MB-231	MTT	24–48 h	Caspase activation, apoptosis	[120]
30	*Brassica oleracea*	Sulforaphane	Isothiocyanates	3–20	MCF-7, MDA-MB-231	MTT	24–48 h	Nrf2 activation, HDAC inhibition	[123]
31	*Nigella sativa*	Thymoquinone	Quinones	10–40	MCF-7	MTT	24–48 h	NF-κB inhibition, apoptosis	[126]
32	*Panax ginseng*	Ginsenosides (Rg3)	Triterpenoid saponins	10–20	MDA-MB-231	MTT	48 h	Apoptosis, angiogenesis inhibition	[129]
33	*Ocimum sanctum*	Eugenol	Phenylpropanoid	20–35	MCF-7, MDA-MB-231	MTT	24–48 h	Induces apoptosis via Bax/Bcl-2 modulation, caspase-3 activation, mitochondrial dysfunction, ROS generation	[131]

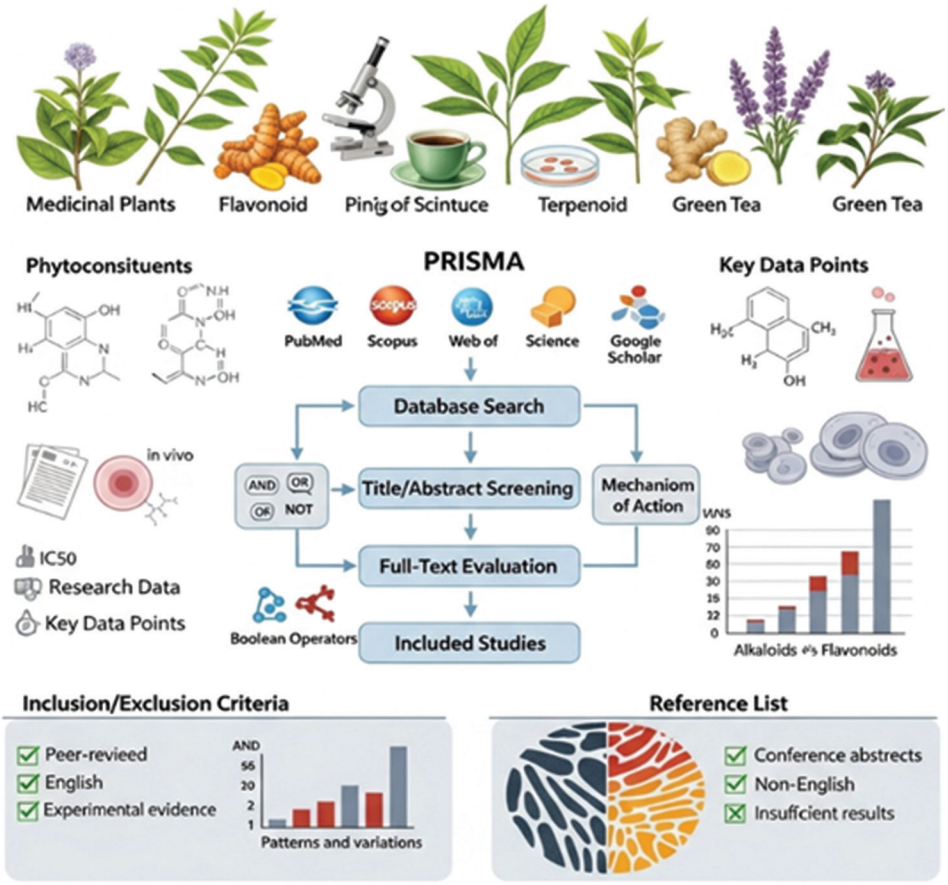

Fig. 3: This illustration demonstrates the application of plant derived anticancer methods in treating breast cancer

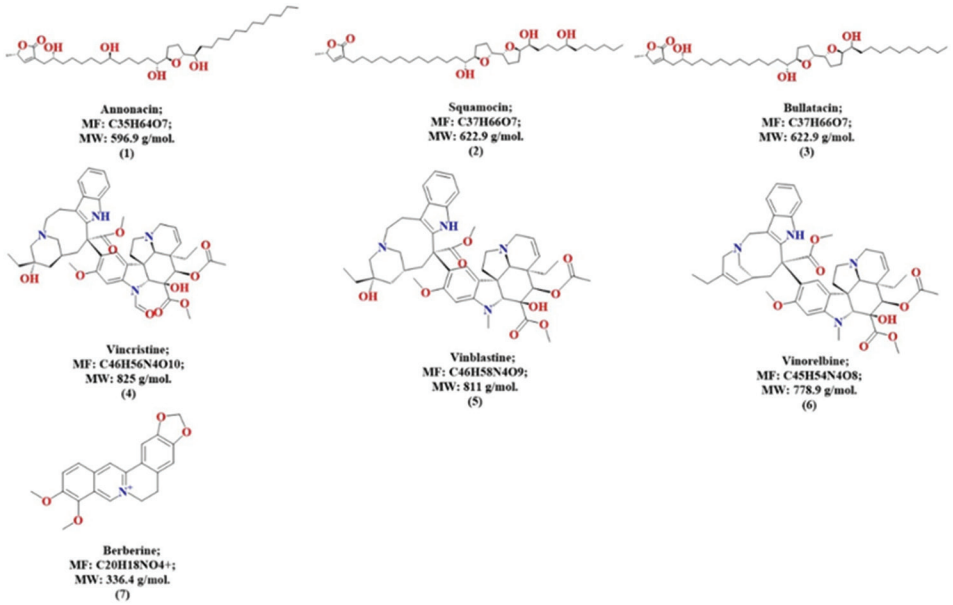

Fig. 4: This figure illustrates the phytoconstituents of alkaloids, such as Annonacin (1), Squamocin (2), Bullatacin (3), Vincritine (4), Vinblastine (5), Vinorelbine (6), and Berberine (7)

It inhibits the STAT3 and ERK signaling pathways, leading to apoptosis and cell cycle arrest. It extracts from milk thistle could serve as a valuable supplement to traditional breast cancer treatments and may act as a direct anticancer agent [44].

Scutellaria baicalensis

A flavonoid known as baicalein, derived from the Chinese skullcap plant *S. baicalensis*, has demonstrated significant anticancer properties against breast cancer. Fig. 5 illustrates the molecular structure of

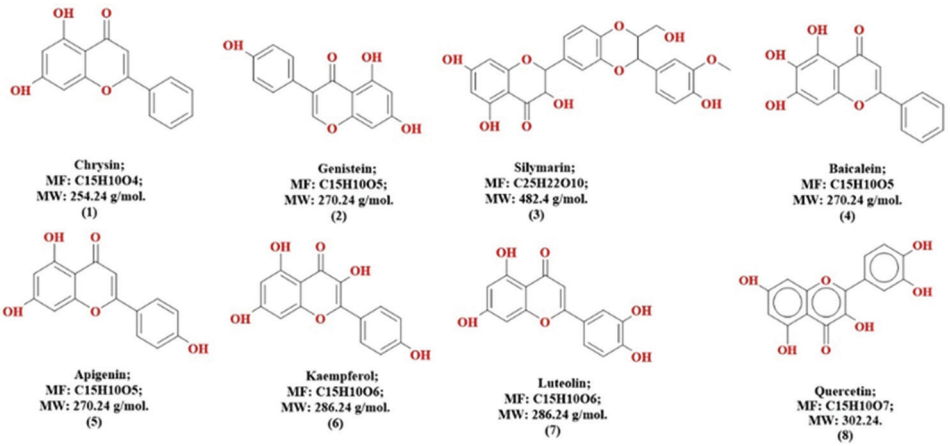

Fig. 5: This figure illustrates the phytoconstituents of flavonoids, such as Chrysin (1), Genistein (2), Silymarin (3), Baicalein (4), Apigenin (5), Kaempferol (6), Luteolin (7), and Quercetin (8)

baicalin [45]. Inducing apoptosis, reducing cancer cell migration, and inhibiting the epithelial-mesenchymal transition (EMT), which is a critical phase in cancer progression, are among its mechanisms of action [46]. Breast cancer cell lines MCF-7 and MDA-MB-231 exhibit an IC_{50} range of 20–40 μM for baicalein. The MTT test was used to measure the effects of baicalein on MDA-MB-231 cells over a period of 24–48 h, as shown in Table 1. It is known to induce caspase-dependent apoptosis and to inhibit the process of EMT [47]. According to breast cancer models, it has been shown to prevent the spread of the disease, suggesting that it could be a valuable natural substance for breast cancer treatment [48].

Petroselinum crispum

Apigenin is a dietary flavonoid known for its significant anticancer properties against breast cancer and is found in foods, such as celery, chamomile, and *P. crispum* (parsley), Fig. 5 illustrates the molecular structure of apigenin [49]. It induces apoptosis by upregulating p53, an essential tumor suppressor protein, and it inhibits the production of vascular endothelial growth factor (VEGF), which contributes to a reduction in angiogenesis [50]. The IC_{50} values for apigenin in MCF-7 breast cancer cells range from 25 to 35 μM. It was evaluated in MCF-7 cells for a duration of 48 h using the MTT assay, as presented in Table 1. By inhibiting VEGF-mediated angiogenesis, apigenin induces cell cycle arrest in the G2/M phase. Its significant cytotoxic effects on MCF-7 breast cancer cells demonstrate the value of apigenin as a natural cancer therapy [51].

Kaempferol

A dietary flavonoid that is often found in tea, broccoli, kale, and spinach, kaempferol has potent anticancer properties against breast cancer. Its chemical structure is illustrated in Fig. 5 [52]. Apoptosis and autophagy are two mechanisms of action that involve the induction of cell cycle arrest through mitochondrial dysfunction and the suppression of the PI3K/Akt signaling pathway [53]. It exhibits significant cytotoxic effects in MCF-7 and BT-549 breast cancer cells, with an IC_{50} range of 20–30 μM. Using the MTT assay, it was tested on MCF-7 cells for durations ranging from 24 to 72 h, as detailed in Table 1. Its primary modes of action include promoting apoptosis and inhibiting the PI3K/Akt signaling pathway. This compound demonstrates a potent antiproliferative impact in breast cancer models [54].

Chrysanthemum

Luteolin, a compound found in celery, parsley, and *Chrysanthemum* flowers, has shown potential as a treatment for breast cancer. The chemical structure of luteolin is illustrated in Fig. 5 [55]. Blocking the Akt and ERK signaling pathways is one of its mechanisms of action, as these pathways are crucial for the proliferation and survival of cancer cells [56]. In breast cancer cells, it has an IC_{50} value ranging from 30 to 40 μM. It was assessed in MDA-MB-231 cells over a period of 48–72 h using the MTT assay, as outlined in Table 1. In addition to reducing MMP-9 expression, it activates caspase-dependent apoptotic pathways. It demonstrates potential as a natural therapy for breast cancer due to its strong anti-proliferative effects against ER-positive breast cancer cells [57].

Quercetin

Apples, onions, *Moringa oleifera*, and *Phyllanthus emblica* are rich sources of quercetin, a flavonoid known for its strong anticancer properties against breast cancer. The chemical structure of quercetin is illustrated in Fig. 5 [58]. The impact arises from the alteration of various signaling pathways, including PI3K/Akt, Mitogen-Activated Protein Kinase MAPK, and NF-κB, which are crucial regulators of cell proliferation, survival, and metastasis [59]. It inhibits tumor growth by inducing apoptosis and promoting cell cycle arrest, along with other multitargeted effects [60]. It has an IC_{50} range of 20–50 μM and exhibits cytotoxic effects on breast cancer cells. Using the MTT test, the effects of quercetin on MCF-7 cells were examined over a period of 24–72 h, as detailed in Table 1. It decreases cell survival by inhibiting the PI3K/Akt and MAPK signaling pathways. In xenograft models, it effectively suppresses the growth of breast cancer tumors, highlighting its potential as a natural therapeutic agent for both the prevention and treatment of breast cancer [61].

Lignans

Linum usitatissimum

It is a valuable source of secoisolariciresinol diglucoside (SDG), which is an anticancer lignan. Fig. 6 illustrates the chemical structure of this compound [62]. It indicates that flaxseed lignans may modify hormone-dependent signaling and suppress tumor growth in mice with estrogen-dependent breast cancer by competing with estradiol for binding to ERs. SDG, a notable lignan present in flaxseed, exhibits potent anti-cancer properties in MCF-7 breast cancer cells. Its IC_{50} values, ranging from 20 to 60 M, demonstrate its efficacy in suppressing cell proliferation [63]. Using the SRB assay, it was evaluated in MCF-7 cells over a duration of 24–48 h, as outlined in Table 1. Clinical pilot trials further corroborated these pre-clinical results, demonstrating that flaxseed intake is associated with a reduction in tumor growth indicators among breast cancer patients [64].

Arctium lappa

Matairesinol is present in species including Forsythia intermedia and *A. lappa* (burdock root), is converted into enterolactone and a bioactive substance that has antioxidant and estrogen-modulating qualities. The chemical structure of this compound is illustrated in Fig. 6 [65]. It is

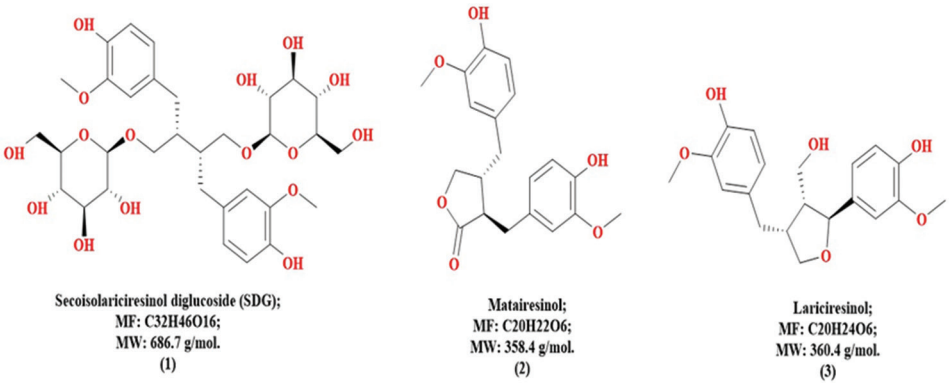

Fig. 6: This figure illustrates the phytoconstituents of lignans, such as Secoisolariciresinol diglucoside (1), Matairesinol (2) and Lariciresinol (3)

a lignan produced from plants that may have anticancer properties, especially in malignancies linked to hormones [66]. The compound exhibits moderate cytotoxicity in MCF-7 and MDA-MB-231 cells, with an IC_{50} ranging from 40 to 70 µM. It was evaluated for 48 h in MDA-MB-231 cells using the MTT assay, as detailed in Table 1. Its mechanism of action involves altering ERs and generating ROS. In addition, this discovery emphasizes how it reduces the risk of breast cancer by modifying ERs, which subsequently affects hormone-dependent pathways that contribute to tumor development [67].

Sesamum indicum

The lignan lariciresinol, found in sesame seeds (*S. indicum*) and flaxseed (*L. usitatissimum*), serves as an essential pre-cursor to mammalian lignans, such as enterolactone and is generated by the metabolism of gut microbes. The chemical structure of this compound is illustrated in Fig. 6 [68]. It may have an impact on ER-mediated pathways, which might prevent hormone-dependent cancer cell growth [69]. The dietary lignan lariciresinol, found in *L. usitatissimum* (flaxseed), *S. indicum*, and *A. lappa*, demonstrates moderate cytotoxicity. This adverse effect is indicated by an IC_{50} value ranging from 35 to 60 µM in MCF-7 and T47D breast cancer cells. It was evaluated in MCF-7 cells for a duration of 48–72 h using the MTT assay, as outlined in Table 1 [70].

Terpenoids and steroidal lactones

Withania somnifera

Withaferin A is a steroidal lactone derived from *W. somnifera* (commonly known as ashwagandha) that has demonstrated significant potential in combating breast cancer through various mechanisms. The chemical structure of this compound is illustrated in Fig. 7. It activates caspases, leading to apoptosis in cancer cells, which is a process of programmed cell death [71]. In addition, Withaferin A effectively suppresses key oncogenic signaling pathways by inhibiting NF-κB and STAT3, two proteins critical for tumor growth, survival, and resistance [72]. In MCF-7 and MDA-MB-231 breast cancer cells, it exhibits potent cytotoxic effects, with an IC_{50} range of 0.5–5 µM. In MDA-MB-231 cells, it was examined over a 24-h period using MTT and Annexin V assays, as detailed in Table 1. Its anti-cancer mechanisms include the inhibition of the STAT3 signaling pathway and the induction of apoptosis through the activation of caspases [73].

Azadirachta indica

Nimbolide, a limonoid terpenoid obtained from *A. indica*, sometimes referred to as neem, has strong anti-breast cancer effects via several pathways. The chemical structure of this compound is illustrated in Fig. 7 [74]. Blocking the PI3K/Akt signaling pathway, which is a crucial regulator of cell survival, proliferation, and resistance, slows the progression of breast cancer [75]. It demonstrates cytotoxic effects on breast cancer cells, with an IC_{50} range of 5–20 µM. It was applied to MCF-7 and MDA-MB-231 cells for 24–48 h, with the MTT test conducted as detailed in Table 1. It triggers apoptosis through the generation of ROS, effectively halting cell division. Furthermore, its anti-cancer effects are linked to the suppression of the PI3K/Akt signaling pathway [76].

Curcuma zedoaria

White turmeric, scientifically known as *C. zedoaria*, is the source of curdione, a sesquiterpene with potent anticancer properties specifically against breast cancer. The chemical structure of this compound is illustrated in Fig. 7 [77]. By inducing mitochondrial dysfunction, this process mainly triggers the intrinsic apoptotic pathway, resulting in the programmed cell death of cancer cells [78]. With IC_{50} values between 20 and 50 µM, it has cytotoxic effects. Using the MTT test, it was evaluated in MCF-7 cells for durations of 24–48 h, as outlined in Table 1. Across mitochondrial mechanisms, it triggers apoptosis and successfully prevents the invasion of cancer cells. It shows curdione's potential as a natural therapeutic agent in the battle against breast cancer by demonstrating its strong cytotoxic effects on MCF-7 breast cancer cells [79].

T. brevifolia

The diterpenoid paclitaxel is one of the most important natural chemotherapeutic agents used in the treatment of breast cancer. It is derived from the Pacific yew, which is scientifically classified as *T. brevifolia*. The chemical structure of this compound is illustrated in Fig. 7 [80]. Its cytotoxic effectiveness is demonstrated by its micromolar IC_{50} values, which typically range from 5 to 20 µM in MDA-MB-231 cells. It was conducted on BT-549 and MCF-7 cell lines using the MTT assay over a period of 24–72 h, as outlined in Table 1 [81]. It induces G2/M cell cycle arrest and apoptosis by stabilizing microtubules, thus preventing their depolymerization. It also activatescaspase-3 and the cleavage of PARP while blocking important survival pathways, such as PI3K/Akt and NF-κB. It also reduces the risk of metastasis by lowering the levels of MMP-2 and MMP-9, which makes it harder for cells to invade and move around.As a result, ongoing research is concentrated on combination therapies and nanoformulations to improve therapeutic outcomes [82].

Tripterygium wilfordii

The Thunder God Vine (*T. wilfordii*) contains a diterpenoid epoxide called triptolide, which has strong anticancer effects against breast cancer. The chemical structure of this compound is illustrated in Fig. 7 [83]. The methods of action include inducing apoptosis, inhibiting NF-κB signaling, and downregulating heat shock protein 70, which is a molecular chaperone associated with cancer cell survival and resistance [84]. It demonstrates strong cytotoxicity, with IC_{50} values ranging from 10 to 100 nM. It was examined for a duration of 24 h in MDA-MB-231 cells using the MTT assay, as detailed in Table 1. It exhibits anti-cancer properties by blocking NF-κB signaling and triggering apoptosis. This natural substance showed strong cytotoxic effects on breast cancer cells, indicating that it is a possible therapy option for breast cancer [85].

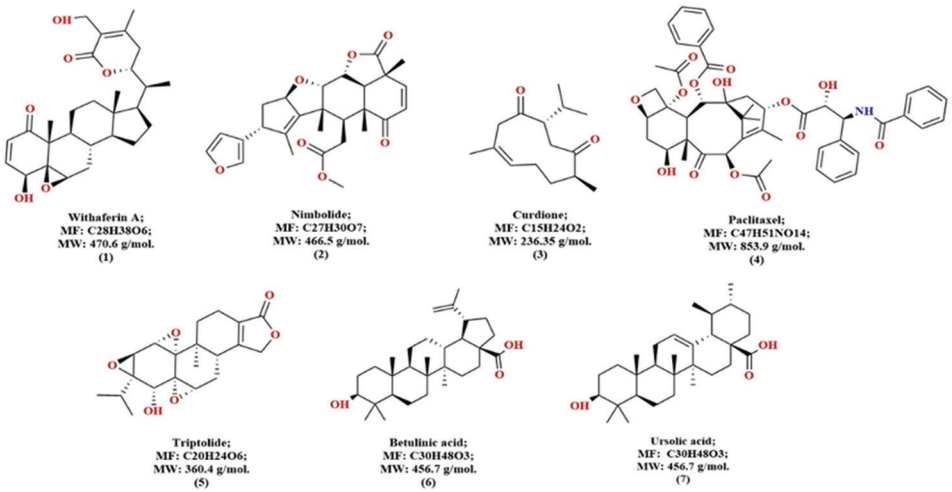

Fig. 7: This figure illustrates the phytoconstituents of terpenoids and steroidal lactones, such as Withaferin A (1), Nimbolide (2), Curdione (3), Paclitaxel (4), Triptolide (5), Betulinic acid (6) and Ursolic acid (7)

Betula species

Betulinic acid is a naturally occurring pentacyclic triterpenoid that is primarily extracted from the bark of medicinal plants, such as birch trees (*Betula* species). The chemical structure of this compound is illustrated in Fig. 7. It is well-known for its strong anticancer properties. With IC_{50} values ranging from 10 to 50 μM, betulinic acid exhibits significant cytotoxic effects on various breast cancer cell lines, including MCF-7, MDA-MB-231, and T47D [86]. Using the MTT assay, the effects of betulinic acid were evaluated on MCF-7 breast cancer cells over a period of 24–48 h, as detailed in Table 1. The anticancer mechanism of betulinic acid involves mitochondria-mediated apoptosis, leading to the release of cytochrome c, activation of caspase-3, and a shift in the Bax/Bcl-2 ratio [87]. In addition, it inhibits NF-κB signaling pathways, which subsequently reduces the production of anti-apoptotic and metastatic genes. By downregulating VEGF and HIF-1α, it exhibits anti-angiogenic properties that prevent tumor vascularization and proliferation. Furthermore, it enhances the production of ROS and induces G1 phase arrest in the cell cycle, which has been shown to promote oxidative stress-mediated apoptosis [88].

Ursolic acid

Ursolic acid, a bioactive pentacyclic triterpenoid, is present in several therapeutic plants, including *Ocimum sanctum* and *Rosmarinus officinalis*. The chemical structure of this compound is illustrated in Fig. 7 [89]. It exhibits strong anticancer properties against breast cancer cells; in MCF-7 cells, the IC_{50} values range from 20 to 40 μM, while it shows similar activity in MDA-MB-231 cells. It was evaluated over a period of 24–48 h using the MTT assay in MCF-7 and MDA-MB-231 cell lines, as outlined in Table 1 [90]. This compound reduces tumor development and spread by inhibiting key signaling pathways, such as STAT3, PI3K/Akt, and NF-κB, as well as inducing G1 cell cycle arrest and triggering mitochondrial death [91].

Phenolic compounds

Curcuma longa

A diarylheptanoid polyphenol derived from *C. longa* (turmeric), curcumin shows potential as an anticancer agent for breast cancer by targeting various pathways. The chemical structure of this compound is illustrated in Fig. 8 [92]. It effectively reduces tumor cell proliferation, survival, and resistance by inhibiting key oncogenic signaling pathways, including NF-κB, STAT3, and PI3K/Akt [93]. In addition, curcumin suppresses angiogenesis and induces apoptosis, further hindering tumor growth and metastasis [94]. It exhibits cytotoxic effects on MCF-7 and MDA-MB-231 breast cancer cells, with an IC_{50} range of 5–25 μM. MTT/SRB assays were conducted to investigate the effects of curcumin on MCF-7 and MDA-MB-231 cell lines over a period of 24–72 h, as detailed in Table 1. It is believed to possess anticancer properties by inhibiting NF-κB signaling. Studies using breast cancer models indicate pro-apoptotic effects, and a summary of pre-clinical and clinical data supports its potential effectiveness as a treatment [95].

Camellia sinensis

The proliferation of breast cancer cells is inhibited by epigallocatechin gallate (EGCG) through several mechanisms; it suppresses the Akt and NF-κB signaling pathways, prevents angiogenesis, and activates apoptosis. The chemical structure of this compound is illustrated in Fig. 8. These combined effects work to inhibit tumor development and reduce cancer cell proliferation [96]. Its anti-proliferative properties have been demonstrated to enhance the susceptibility of breast cancer cells to chemotherapy, thereby increasing the effectiveness of standard treatments [97]. The effects of EGCG depend on the dose, and the IC_{50} values are usually in the tens of micromolar range. Generally, EGCG demonstrates antiproliferative effects on breast cancer within the 30–100 μM range, suggesting its potential as a natural therapeutic agent. It was assessed over a period of 24–48 h in MDA-MB-231 cells using the MTT assay, as outlined in Table 1 [98].

P. emblica

Gallic acid is a naturally occurring phenolic molecule that exhibits potent anticancer effects, particularly against breast cancer. It can be found in *P. emblica* (Amla), green tea, and grapes. The chemical structure of this compound is illustrated in Fig. 8 [99]. By causing mitochondrial dysfunction and activating intrinsic cell death pathways, ROS mediate apoptosis, which is their main mode of action [100]. It has strong antioxidant qualities in addition to its pro-apoptotic actions, thus playing a dual function in aging [101]. It has cytotoxic properties with IC_{50} values ranging from 30 to 80 μM. It was evaluated for 48 h in MCF-7 cells using the SRB assay, as detailed in Table 1. By causing oxidative stress, gallic acid prevents cancer cells from growing. It may cause mitochondrial-mediated apoptosis in breast cancer cells, making it a viable natural therapy option [102].

Punica granatum

Ellagic acid is a polyphenolic antioxidant found in *P. emblica* and *P. granatum* (pomegranate) that possesses significant chemo preventive and therapeutic properties against breast cancer [103]. The chemical structure of this compound is illustrated in Fig. 8. By inhibiting the formation of DNA adducts and promoting the death of cancer cells, it protects genetic material from damage caused by carcinogens [104]. It exhibits strong anti-proliferative properties against breast cancer cells

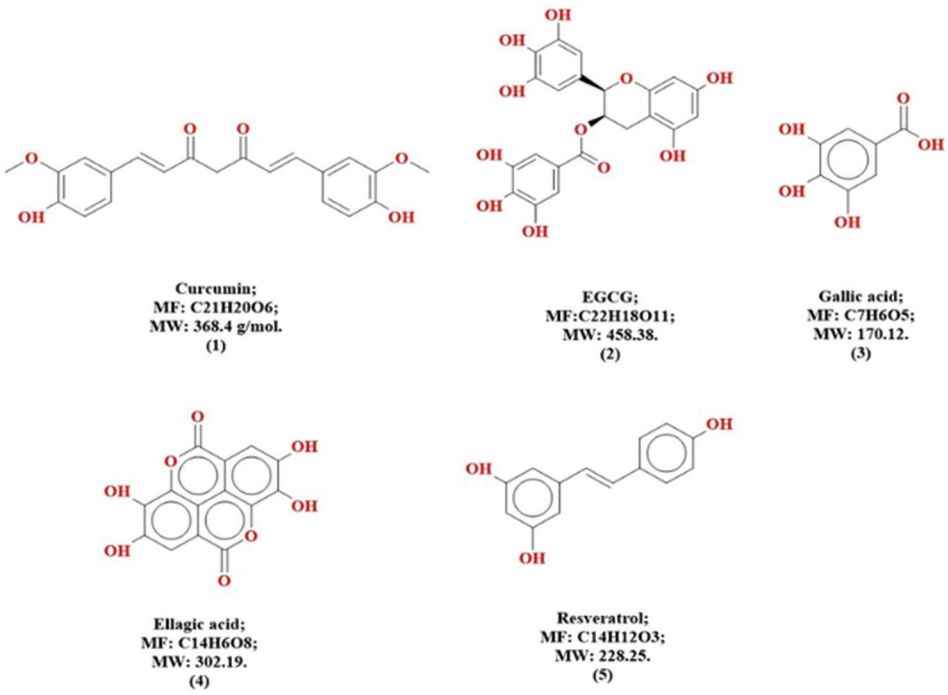

Fig. 8: This figure illustrates the phytoconstituents of phenolics, such as Curcumin (1), epigallocatechin gallate (2), Gallic acid (3), Ellagic acid (4) and Resveratrol (5). The structural representations of these compounds underscore their significant potential in combating breast cancer

and can protect breast tissue from oxidative damage [105]. It inhibits the growth of cancer cells by inducing oxidative stress, which harms effects on breast cancer cells, exhibiting an IC_{50} value of 30–80 μM. The MTT assay was conducted to evaluate the effects of ellagic acid on MDA-MB-231 cells over a period of 48–72 h, as detailed in Table 1 [106].

Vitis vinifera

Resveratrol, a naturally occurring polyphenol found in *V. vinifera* (red wine and grapes), has been extensively studied for its potential role in the prevention and treatment of breast cancer. The chemical structure of this compound is illustrated in Fig. 8. It limits tumor development and spreads by inhibiting angiogenesis, reducing the PI3K/Akt signalling pathway, and blocking ER signalling [107]. It also triggers apoptosis, which encourages cancer cells to die. With an IC_{50} range from 25 to 60 μM, it has cytotoxic effects on breast cancer cells. It was assessed over a period of 48–72 h in MDA-MB-231 cells using the MTT assay, as outlined in Table 1. The activation of the p53 tumor suppressor is the main cause of this growth suppression. Further evidence supporting resveratrol's chemopreventive potential and anticancer activities established resveratrol as a promising natural substance for the prevention and treatment of breast cancer [108].

Organosulfur compounds

S-Allylcysteine (SAC)

A bioactive compound derived from aged garlic extract, SAC, has demonstrated significant anticancer properties specifically against breast cancer. The chemical structure of this compound is illustrated in Fig. 9 [109]. Its mechanisms of action include inducing apoptosis, inhibiting cell proliferation, and modulating oxidative stress pathways, all of which contribute to the prevention of tumor growth and development [110]. The chemo preventive potential of SAC was convincingly demonstrated *in vivo*, found that it significantly reduced the incidence of mammary tumors in rats [111]. It exhibits a moderate level of cytotoxicity, with an IC_{50} range of 10–50 μM. It was evaluated over a period of 24–48 h in MCF-7 cells using the MTT assay, as detailed in Table 1. It promotes apoptosis, thereby inhibiting the growth of cancer cells [112]. It's effectiveness as a natural agent for the prevention and treatment of breast cancer, reporting that it successfully diminished the proliferation of ER-positive breast cancer cells [113].

Diallyl disulfide (DADS)

A sulfur-containing compound derived from garlic oil, known as DADS, exhibits significant anticancer effects against breast cancer. The chemical structure of this compound is illustrated in Fig. 9 [114]. Its mechanism of action includes reducing the potential for metastasis, promoting apoptosis through ROS, and inducing cell cycle arrest in the G2/M phase [115]. Its role in triggering apoptosis by modulating histone acetylation, its cytotoxic effects on MCF-7 breast cancer cells [116]. The compound demonstrates a moderate level of cytotoxicity, with an IC_{50} range between 10 and 50 μM. MCF-7 cells were utilized to investigate the effects of DADS over a period of 48 h, using the MTT assay, as outlined in Table 1. It induces apoptosis, which inhibits the growth of cancer cells [117].

Diallyl trisulfide (DATS)

A sulfur-rich organosulfur molecule found in onions and garlic, DATS, has demonstrated significant anticancer effects against breast cancer. The chemical structure of this compound is illustrated in Fig. 9 [118]. It disrupts cancer cell survival pathways through various mechanisms, including inhibiting Akt signaling, downregulating the anti-apoptotic protein Bcl-2, and inducing caspase-dependent apoptosis [119]. The IC_{50} value for DATS varies between 20 and 50 μM, indicating its cytotoxic effects. In the MDA-MB-231 cell line, the MTT assay was conducted over a duration of 24–48 h, as detailed in Table 1. It promotes apoptosis by activating caspase cascades within cancer cells. It exhibited strong therapeutic potential *in vivo* by significantly inhibiting breast tumor growth in xenograft models [120].

Brassica oleracea

Sulforaphane, an isothiocyanate found in *B. oleracea* crops, such as kale, broccoli, and Brussels sprouts, possesses significant anti-breast cancer

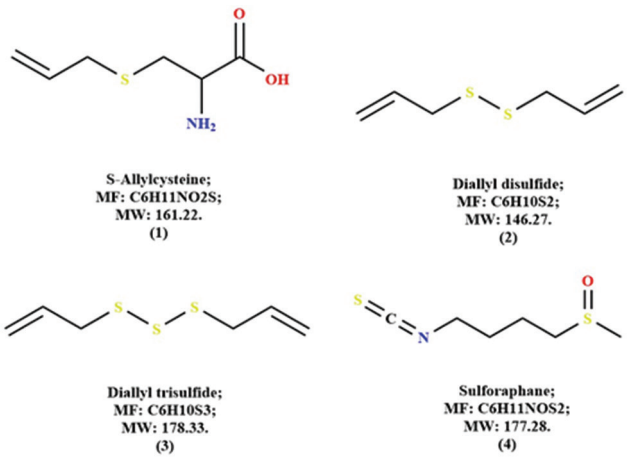

Fig. 9: This figure illustrates the phytoconstituents of organosulfur, such as S-allylcysteine (1), Diallyl disulfide (2), Diallyl trisulfide (3), and Sulforaphane (4)

chemo preventive and therapeutic properties. The chemical structure of this compound is illustrated in Fig. 9 [121]. It activates the Nrf2 pathway and inhibits histone deacetylases (HDACs), thereby inducing apoptosis and autophagy two critical processes for cellular survival and epigenetic regulation [122]. It exhibits strong cytotoxicity in breast cancer cells, with an IC_{50} ranging from 3 to 20 μM. Using the MTT assay, it was evaluated in MCF-7 and MDA-MB-231 cell lines over a duration of 24–48 h, as outlined in Table 1. It works by inhibiting HDAC and activating the Nrf2 pathway, which contributes to its anti-cancer properties. The chemo preventive potential of sulforaphane in mammary carcinogenesis, while its ability to suppress breast cancer stem cells [123].

Other bioactive compounds

Nigella sativa

Thymoquinone, which is derived from black cumin (*N. sativa*), has shown notable anticancer properties against breast cancer through several mechanisms. The chemical structure of this compound is illustrated in Fig. 10 [124]. It inhibits the NF-κB and Akt signaling pathways, generates ROS, and induces apoptosis to prevent the survival and proliferation of cancer cells [125]. It exhibits cytotoxic effects on MCF-7 breast cancer cells, with an IC_{50} range of 10–40 μM. It was assessed in MCF-7 cells over a period of 24–48 h using the MTT assay, as detailed in Table 1. It inhibits the progression of cancer cells by blocking NF-κB signaling. In addition, it has been demonstrated to induce apoptosis in MCF-7 cells and significantly reduce tumor size in animal models, highlighting its potential as both a therapeutic and adjuvant treatment for breast cancer [126].

Panax ginseng

Ginsenoside Rg3 is an important bioactive saponin known for its strong anticancer effects against breast cancer. The chemical structure of this compound is illustrated in Fig. 10 [127]. By inhibiting the production of matrix metalloproteinase (MMP) and VEGF, two critical substances for tumor vascularization and invasion, it reduces angiogenesis and metastasis [128]. In breast cancer cells, it exhibits cytotoxic effects, with IC_{50} values ranging from 10 to 20 μM. In MDA-MB-231 cells, it was tested for a duration of 48 h using the MTT assay, as outlined in Table 1. It induces apoptosis and effectively inhibits angiogenesis. In addition, it has demonstrated the ability to prevent the metastasis of breast cancer cells and induces apoptosis, while significantly reducing tumor vascularization [129].

Eugenol

Eugenol, a naturally occurring phenylpropanoid, is primarily found in fragrant plants, such as *O. sanctum*, commonly known as holy basil. The chemical structure of this compound is illustrated in Fig. 10 [130]. The reported IC_{50}values for MCF-7 and MDA-MB-231 breast cancer cells are between 20 and 35 μM, respectively. It was examined using the MTT test over a duration of 24–48 h in MCF-7 and MDA-MB-231 cell lines, as detailed in Table 1 [131]. This suggests that the drug has a strong effect against breast cancer. The mechanism of action involves the activation of caspase-3, cleavage of PARP, alteration of the Bax/Bcl-2 ratio, and substantial suppression of the NF-κB and cyclin D1 pathways, leading to apoptosis and inhibition of cell proliferation [132].

MECHANISM OF ACTION

Plant-derived phytoconstituents exhibit anti-cancer properties by targeting specific characteristics of breast cancer through various cellular and molecular mechanisms. One well-recognized mechanism is the induction of apoptosis, a regulated form of cell death [133]. Certain compounds, such as thymoquinone (from *N. sativa*), curcumin (from *C. longa*), DATS (found in garlic), and withaferin A (from *W. somnifera*), can induce mitochondrial dysfunction, alter the Bax/Bcl-2 ratio, and activate the caspase cascade, ultimately leading to programmed cell death [134]. Substances, such as genistein (derived from *G. max*), paclitaxel (extracted from *T. brevifolia*), and berberine (from *Berberis* spp.) work by inhibiting cyclin-dependent kinases or stabilizing microtubules. This action prevents breast cancer cells from progressing through the G1, G2, or M phases of the cell cycle [135].

Inhibiting metastasis and angiogenesis represents a critical therapeutic strategy. Various phytochemicals, such as baicalein (derived from *S. baicalensis*), nimbolide (from *A. indica*), and ginsenoside Rg3 (extracted from *P. ginseng*), contribute to reducing tumor invasiveness and vascularization [136]. They achieve these effects by decreasing VEGF signaling, preventing EMT, and inhibiting MMPs [137].

Hormonal control is crucial in breast cancers that are dependent on estrogen. Research shows that phytoestrogens, such as genistein, punicalagin (found in pomegranates), and SDG (found in flaxseed) inhibit the growth of estrogen-induced tumors [138]. They accomplish their goal through two primary mechanisms: By inhibiting the enzyme aromatase or by competing with natural estrogens for binding to ERs [139]. The mechanisms outlined suggest that phytoconstituents function via multiple pathways, demonstrating diverse effects such as immunological modulation, oxidative stress reduction, cell cycle arrest, angiogenesis suppression, hormonal regulation, and apoptosis induction. These diverse actions make them promising candidates as additional or alternative therapeutic agents for the treatment of breast cancer [140].

CURRENT LIMITATIONS AND CHALLENGES

One of the primary challenges is the low bioavailability of certain substances, such as curcumin, resveratrol, and quercetin. These compounds exhibit low water solubility, rapid metabolism, and poor systemic absorption, all of which significantly reduce their therapeutic effectiveness *in vivo* [141]. Another considerable obstacle is the variability in the phytochemical composition of plant extracts, which can differ based on factors, such as species, geographic origin, growth conditions, harvesting time, and extraction methods. In addition, when crude extracts contain a mixture of bioactive components, it becomes challenging to identify unique modes of action or establish clear structure–activity relationships [142].

A significant limitation is the lack of robust clinical trials; although *in vitro* and *in vivo* studies have shown promising results, only a few phytochemicals, such as paclitaxel, vinca alkaloids, and, to a lesser extent, curcumin, have advanced to clinical trials. The safety, toxicity, and efficacy of most other phytochemicals remain poorly understood [143]. Many of these compounds demonstrate cytotoxic effects against cancer cells only at high micromolar (μM) IC_{50} values and exhibit low selectivity toward normal breast epithelial cells. This

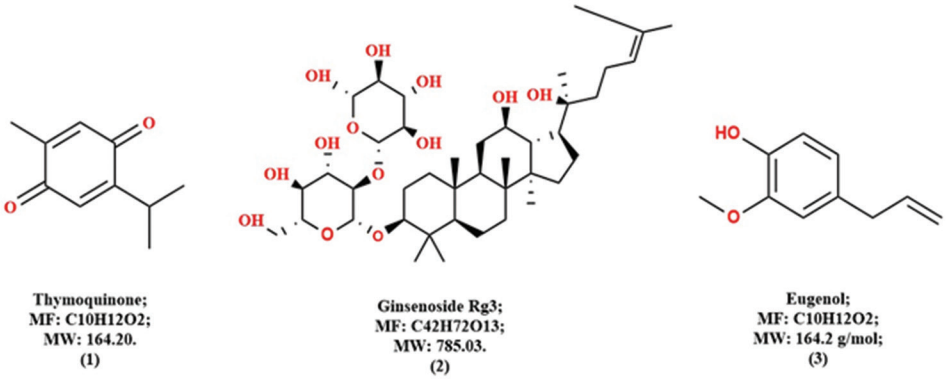

Fig. 10: This figure illustrates the phytoconstituents of bioactive compounds, such as Thymoquinone (1), Ginsenoside Rg3 (2), and Eugenol (3).

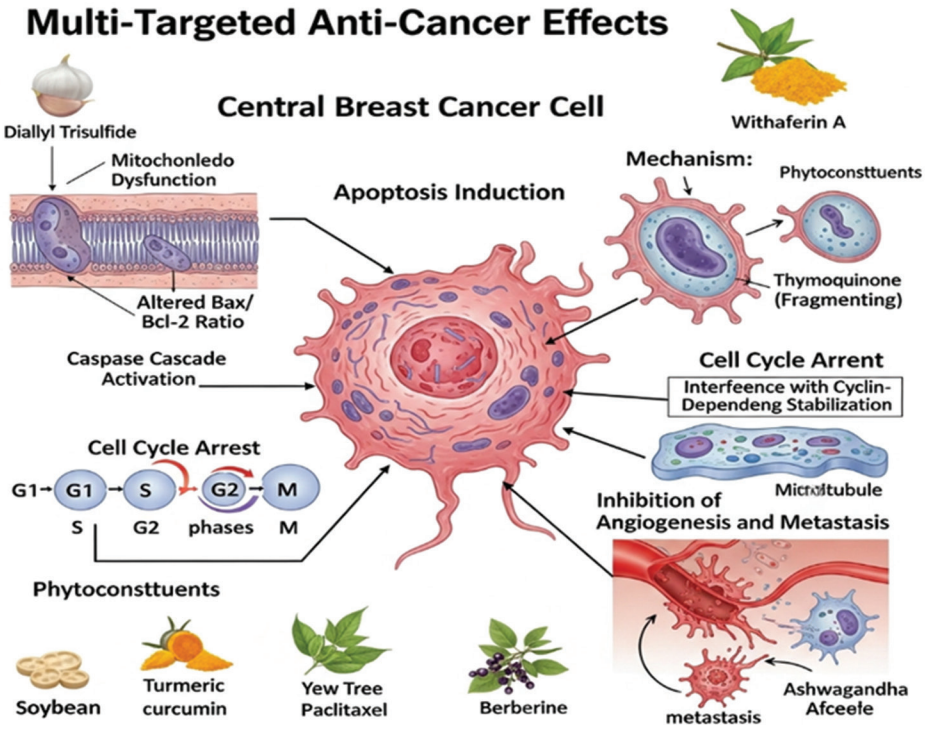

Fig. 11: There are strategies that target multiple pathways of phytoconstituents derived from plants to prevent breast cancer

poses a major hindrance to the clinical application of phytoconstituents due to their poor therapeutic index [144].

For example, flavonoids, such as chrysin (20–50 μM), apigenin (25–35 μM), kaempferol (20–30 μM), luteolin (30–40 μM), and baicalein (20–40 μM) typically require doses that far exceed clinically acceptable levels. Similarly, substantial concentrations of phenolic compounds, such as gallic acid (30–80 μM), ellagic acid (30–80 μM), resveratrol (25–60 μM), curcumin (5–25 μM), EGCG (30–100 μM), and quercetin (20–50 μM) are necessary to achieve significant cytotoxic effects. Organosulfur compounds, including SAC (10–50 μM), DATS (20–50 μM), and DADS (10–50 μM), show even lower efficacy, while lignans, such as matairesinol (40–70 μM), lariciresinol (35–60 μM), and SDG (20–60 μM) exhibit similar limitations. Some terpenoids, such as betulinic acid (10–50 μM) and ursolic acid (20–40 μM), demonstrate modest activity, but only a select few, including paclitaxel (5–20 μM), nimbolide (5–20 μM), withaferin A (0.5–5 μM), and triptolide (10–100 nM), achieve pharmacologically significant potency.

In addition to these challenges, phytochemicals often fail to achieve effective therapeutic plasma concentrations due to serious pharmacokinetic limitations, including poor solubility, rapid metabolism, limited oral bioavailability, short half-lives, and systemic toxicity [145]. Drug-drug interactions may also present clinical issues; for instance, resveratrol and genistein can interfere with cytochrome P450 enzymes, affecting chemotherapy metabolism and potentially leading to adverse interactions [146].

Innovative delivery methods are being explored to overcome these challenges. Curcumin-loaded lipid nanoparticles and solid lipid carriers enhance systemic bioavailability and reduce IC_{50} values [147]. Quercetin-loaded PLGA nanoparticles facilitate increased intracellular uptake and apoptosis [148]. Resveratrol nanoemulsions, micelles, and nanosponges improve metabolic stability and circulation half-life [149]. Moreover, EGCG-gold nanoparticles boost cytotoxicity induced by ROS [150]. Withaferin, triptolide nanocarriers, and its water-soluble prodrug minnelide significantly improve the therapeutic index by

minimizing intrinsic toxicity, while liposomes enhance tumor selectivity and decrease overall toxicity as shown in the Fig. 11 [151].

However, only a limited number of nano-phytoconstituent systems have advanced beyond early-phase studies, indicating that clinical translation remains restricted [152]. Despite the strong mechanistic effects and broad anticancer potential of phytoconstituents, their lack of clinical validation, high μM IC_{50} requirements, and poor pharmacokinetics continue to impede their incorporation into standard treatment regimens [153].

CONCLUSION

The treatment of breast cancer with medicinal plants and their phytoconstituents offers a diverse array of potential therapeutic compounds. These substances, which include alkaloids, flavonoids, lignans, terpenoids, polyphenols, organosulfur derivatives, and other bioactive components, can induce apoptosis, interrupt the cell cycle, inhibit angiogenesis and metastasis, modulate hormone signaling, and regulate oxidative stress and immune responses. Pre-clinical studies consistently illustrate their ability to target various hallmarks of breast cancer, presenting advantages over traditional single-target chemotherapeutics.

Although the positive outcomes are promising, several challenges remain, including limited bioavailability, a lack of standardized formulations, inadequate clinical validation, and potential drug-drug interactions. To address these issues, we need innovative solutions, such as delivery systems utilizing nanotechnology, effective combinations of medicines and phytochemicals, and comprehensive clinical research. The translational potential of these natural compounds will be further enhanced by omics-based approaches and personalized medicine frameworks.

Phytoconstituents show significant promise as adjuvants or supplementary therapies for breast cancer. By addressing current challenges in advanced pharmaceutical technology, future treatments for breast cancer could become safer, more effective, and more patient-centered. If plant-derived compounds can effectively bridge the gap between traditional medical knowledge and modern molecular research, we may achieve this improvement.

AUTHOR'S CONTRIBUTION

Pranavi Juturu designed the study, handled the data collection and conducted the analysis, and prepared the initial draft of the article. Dr. Velmurugan Vadivel supervised the study, contributed to data analysis and interpretation and provided essential revisions. All authors have reviewed and approved the final version of the manuscript.

CONFLICT OF INTEREST

The author declares no conflict of interest, financial or otherwise.

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