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Anticancer effects of African medicinal plants on breast cancer cell lines: a systematic review

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ABSTRACT

Despite the availability of conventional treatments, breast cancer remains a major global health burden. Breast cancer development is attributed to alterations in molecular signaling pathways that govern cell growth and differentiation. The current therapeutic strategies are often limited by systemic adverse toxicity, drug resistance and high-cost challenges, particularly in low- and middle-income countries. Therefore, there is a need for discovery and development of effective alternative therapeutic agents for breast cancer. Traditional medicinal continue to serve as valuable reservoirs for anticancer agents. This study systematically evaluated the antiproliferative effects and underlying pathomechanisms of African medicinal plants on breast cancer cell lines. A literature search was conducted in Scopus and PubMed focusing on studies that evaluated the growth inhibition properties of plant extracts in *in vitro* and *in vivo* breast cancer models. Thirty studies met the inclusion criteria, majorly utilizing the HCC 1395, MCF-7 and MDA-MB-231 cell lines. Most plant extracts exhibited potent cytotoxicity ($IC_{50} > 20 \mu\text{g/ml}$) and selectivity ($SI < 3$), indicating preferential effects against breast cancer cell lines. The involved molecular pathways included activation of pro-apoptotic markers and suppression of anti-apoptotic metastatic markers. Phytocompounds including terpenoids, polyphenols and phytosterol were majorly implicated. The findings validate the therapeutic effects of African medicinal plants. Furthermore, they support further research into plant-breast cancer treatments.

Keywords: Medicinal plants, Breast cancer, Anticancer activity, Cell lines, Phytochemicals, Systematic review.

INTRODUCTION

The development of breast cancer, a heterogenous disease that is characterized by uncontrolled proliferation of mammary epithelial cells, is attributed to dysregulation of molecular signaling pathways that govern cell apoptosis, growth and differentiation [1]. Whereas internal factors including oxidative stress, hypoxia and genetic mutations contribute to carcinogenesis, external risk factors such as smoking, sedentary lifestyles, ultraviolet radiation and chronic psychological stress also play a role in development of breast carcinoma [2].

Globally, breast cancer is among the leading causes of cancer-related morbidity and mortality, representing a substantial public health challenge [3]. Despite significant advances in early detection and therapeutic interventions, the clinical outcomes for most patients remain poor, as evidenced by persistent mortality, high recurrence rates and substantial treatment-related morbidity [4]. Conventional therapeutic approaches, such as radiation, chemotherapy, surgery and hormonal therapy are often associated with adverse effects, emergence of multidrug resistance and high-cost challenges that are particularly pronounced in developing countries [5].

Consequently, there is a growing interest in natural products, particularly those from medicinal plants as alternative chemotherapeutic agents [6]. Medical plants have attracted considerable scientific attention as complementary or alternative reservoirs of antineoplastic agents [7]. Their affordability, accessibility and cultural acceptance enhance their appeal, particularly in African settings [8]. Notably, plant-derived compounds such as vinblastine and vincristine from *Catharanthus roseus*, paclitaxel from *Taxus brevifolia* are now standard chemotherapeutic agents [5,9]. These findings have invigorated research into African medicinal plants, which have been traditionally used in folk medicine for cancer-related ailments. Due to their rich repertoire of bioactive phytocompounds, various African medicinal plants have shown antiproliferative, anti-metastatic and pro-apoptotic activities *in vitro* and *in vivo* [8,10].

This review consolidates the current evidence on the antiproliferative potential and pathomechanisms of African medicinal plant extracts against breast cancer cell lines, thereby offering a foundation for integrative therapeutic strategies and drug development in oncology.

METHODOLOGY

Literature search

The search strategy was as follows “((Plant extracts)" OR "phytochemical" AND "breast cancer" AND "Africa)" in PubMed and Scopus databases. The search period was unlimited. For eligibility, two authors independently screened the abstracts and whole articles. The third author was involved to settle any disagreement.

Eligibility criteria

Studies were considered eligible if they reported on the *in vitro* and *in vivo* effects of African medicinal plants against breast cancer cell

lines. Studies that failed to report the outcomes of interest were excluded from the review.

Data extraction

Data were extracted into tables. The information gathered includes the plant names, part used, country studied, type of study, mode of action, cell line and author names.

RESULTS

A total of 107 studies were identified, 30 met the inclusion criteria. Results are summarized in the Preferred Reporting Items for Systemic Reviews and Meta-Analyses (PRISMA) flowchart (Figure 1).

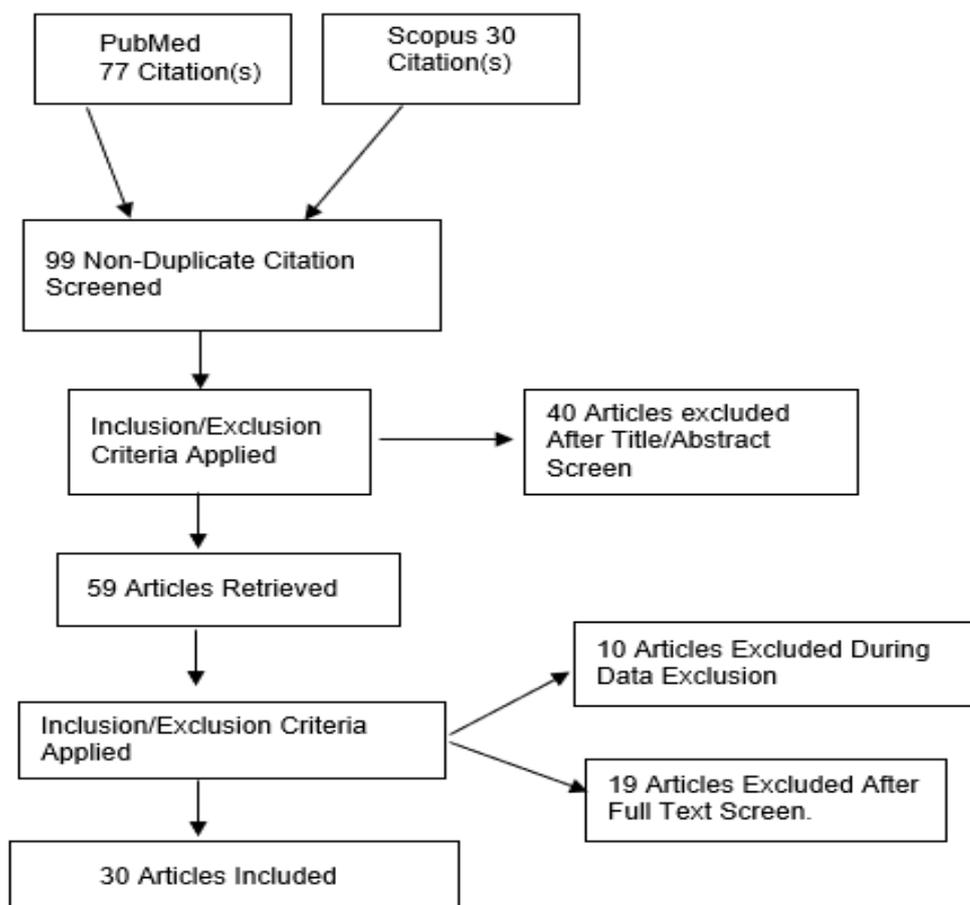


Figure 1: PRISMA flowchart of literature search and study selection.

Table 1: African medicinal plants with antiproliferative activities against breast cancer cell lines

Medicinal plant	Part used/ extract	Region	Type of Study	Major Compounds	Model cell line	Effect/mode of action	Author
<i>Zanthoxylum paracanthum</i>	Root	Kenya and Tanzania	<i>In vitro</i>	Stigmasterol, sesamin, 8-oxochelerythrine, canthin-6-one, 8-cetonyldihydrochelerythrine	HCC 1395	SI>3	Kaigongi et al., 2020 [11]
<i>Anonidium mannii</i>	Stem bark	Cameroon	<i>In vitro; in vivo</i>	Flavonoids and alkaloids	MCF-7 DMBA-induced breast tumor rat model	<i>In vitro</i> assay: Moderate toxicity (IC ₅₀ -61.5µg/ml); <i>In vivo</i> assay: in rats; ↓ in tumor burden, breast tumor incidence and volume (95.34-99.14%, 28%, 92% respectively) ↑SOD, catalase, glutathione; ↓MDA, nitrite	Mefegue et al., 2021 [12]
<i>Acridocarpus orientalis</i>	Leaves and stem	Somalia	<i>In vitro</i>	Flavonoids, β-sitosterol, betulinic, morin and betuline.	MCF-7 and MDA-MB-231	Induces apoptosis (↑ <i>Caspase-3</i> , ↓ <i>Bcl-2</i> , ↑ <i>Bax</i> , ROS-mediated mitochondrial dysfunction	Balhamar et al., 2019 [13]
<i>Tulbaghia violacea</i>	Leaves (methanol and aqueous)	South Africa	<i>In vitro</i>	1,2,4-triazine-3,5(2H,4H)-dione, vanillin, schisandrin, taurolidine, and α-pinene	MDA-MB-231	↑cell adhesion of cancer cells to ECM analogue (↑ transcription levels of SNAIL1 and 3; ↑CDON); ↓ ability of cancer cells to invade other tissues (↑transcription level of LRP2BP, ↓Gli2)	Alaouna et al., 2024 [14]
<i>Brucea antidysenterica</i>	Leaves and stem	Cameroon	<i>In vitro</i>	3, (3-(3-methyl-1-oxo-2-butenyl))1H indole, hydnocarpin, (20R)-O-(3)-α-L-arabinopyranosyl-pregn-5-en-3β,20-diol, (20R)-O-(3)- β-D-glucopyranosyl(1→2)-α-L-arabinopyranosyl-pregn-5-en-3β,20-diol, canthin-6-one, cleomiscosin C, bruceolline F or 3-(2',3'-dihydroxy-3'-méthylbutyl)- 1N-β-glucopyranosylindole.	MDA-MB-231	Exhibited IC ₅₀ ≤ 20µg/ml; Altered mitochondrial membrane permeability; ↑caspases activity 3/7, 8 and 9	Youmbi et al., 2023 [5]
Recipe A: <i>Anchomanes difformis</i> , <i>Garcinia kola</i> , <i>Kigelia africana</i> , <i>Xylopia aethiopica</i> Recipe B <i>Allium ascalonicum</i> , <i>Antiaris</i>	-	Nigeria	<i>In vitro</i>	-	MCF-7 and MDA-MB-231	Exhibited IC ₅₀ <20µg/ml; ↑expression levels of PARP and caspase 3; altered cell cycle at the G0/G1 phase	Alabi et al., 2020 [7]

<i>africana, Bridellia scadens, Calliandra haematocephala, Citrus medica, Mangifera indica, Nauclear latifolia, Tricalysia macrophylla, Trichilia monadelpha</i>							
<i>Moringa oleifera</i>	Seeds	Nigeria	<i>In vitro</i>	-	MCF-7	Moderate -low IC ₅₀ : 26-280 µg/ml	Adebayo et al., 2017 [15]
<i>Bulbine frutescens</i>	Bulb	South Africa	<i>In vitro</i> <i>In silico</i>	Anthraquinones, flavonoids and terpenoids	MDA-MB-231; T47D	IC ₅₀ <20 µg/ml; ↑ <i>p21</i> , caspase 3; down expression of cyclin <i>D1</i> , <i>CDK4</i> , <i>Bcl-2</i> and <i>survivin</i> ; down regulation of notch signaling pathway by inhibiting γ -secretase ↑ ROS production	Kushwaha et al., 2019 [16]
<i>Kigelia Africana</i> (Lam.)	Stem bark	Kenya	<i>In vitro</i>	Terpenoids, phenols, steroids and flavonoids	HCC 1937	IC ₅₀ : 20-100 µg/ml (Moderate toxicity)	Mukavi et al., 2020 [17]
<i>Fagara leprieuri, Fagara xanthoxyloides, Mondia whitei</i> and <i>Xylopi aethiopica</i>	Fruits, fruits, roots and pods, respectively	Tunisia	<i>In vitro</i>	Phenols and flavonoids	MDA-Mb-231; MCF-7	IC ₅₀ <20 µg/ml; ↑transcription levels of caspase 3	Choumessi et al., 2012 [18]
<i>Rubus fairholmianus</i>	Roots-Zinc oxide-nanoparticles	South Africa	<i>In vitro</i>		MCF-7	↑markers of apoptosis cytoplasmic cytochrome c and caspase 3/7; ↑ levels of pro-apoptotic proteins (p53 and Bax); ↑ ROS production; ↓ anti-apoptotic proteins (Bcl-2); reduction in intracellular ATP	George et al., 2020 [19]
<i>Zanthoxylum capense</i>	Stem barks, knobs and leaves	South Africa	<i>In vitro</i>	Chelerythrine, 6-hydroxydihydrochelerythrine, rutaecarpine, dodecyl-trans-p-coumarate, sesamin, catechin, lupeol, sitosterol, pheophytin and lutein	MCF-7	Decreased the viability of MCF-7 tumor cells by at least 23% at concentration of 1 µg mL ⁻¹	Bodede et al., 2017 [20]
<i>Tulbaghia violacea</i>	Leaves (hexane, methanol and butanol extracts)	South Africa	<i>In vitro</i>	Phenols, glycosides, flavonoids (Oyerinde & Risenga, 2025)	MCF-7; MDA-Mb-231	Increase in expression levels of caspase 3/7, p21, Bax, CDK2 proteins, A reduction in Rb expression	Motadi et al., 2020 [21]
<i>Eucalyptus camaldulensis</i>	Leaves- Cerium oxide nanoparticles	South Africa	<i>In vitro</i>	-	MCF-7	Reduced the viability of MCF-7 cells to 78%, 74%, and 69% after 24, 48, and 72 h, respectively.	Tameh et al., 2024 [22]
<i>Uvari dendron anisatum, Fagaropsis angolensis, Hydnora abyssinica, Prunus Africana</i>	Root, bark, rhizome, bark, respectively	Kenya	<i>In vitro</i>	-	HCC 1395	IC ₅₀ : 20-100 µg/ml	Onyancha et al., 2018 [23]
<i>Annona senegalensis</i> Pers, <i>Allophylus africanus</i> P. Beauv	Stem bark	Tanzania	<i>In vitro</i>	Flavonoids, alkaloids, saponins and terpenoids	HCC 1396	IC ₅₀ -20-100 µg/ml; SI:3-8	Biseko et al., 2019 [24]

<i>Antiaris africana</i> Engler	Leaves	Senegal	<i>In vitro</i>	Hydroxycinnamates, rutin isomer, and cardiac glycosides	MCF-7	EC ₅₀ : 64.6 ± 13.7 µg/mL	Thiam et al., 2022 [25]
<i>Ocimum basilicum</i> Mentha longifolia leaves, <i>Linum usitatissimum</i> seeds, <i>Allium sativum</i> Nigella sativa, <i>Piper nigrum</i> L., <i>Zingiber officinale</i> , and <i>Foeniculum vulgare</i>	Leaves, leaves, seeds, bulb, fruit, rhizome, seeds, respectively	Sudan	<i>In vitro</i>	Phytosterol, terpenoids, Vitamin E, fatty acids	MCF-7; MDA-MB-231	IC ₅₀ <20	Hago et al., 2023 [26]
<i>Detarium microcarpum</i> , <i>Guiera senegalensis</i> , and <i>Cassia siamea</i>	Stem bark	Nigeria	<i>In vitro</i>	Isorhamnetin, eupatorin, alpinumisoflavone, procyanidin B3, syringin, and gallic acid	MCF-7	IC ₅₀ : High to moderate toxicity (8-200; Antioxidant effect; Antimigration; induced cycle arrest at the S phase	Adebayo et al., 2019 [27]
<i>Limoniastrum monopetalum</i> and <i>Bauhinia variegata</i>	Leaves, branches, fruits	Egypt	<i>In vitro</i>	-	MCF-7	IC ₅₀ of 100 µM; caspase-3 activation and cleavage of cytokeratin-18, ↑p53, ↓ Bcl-2 gene, Decreased expression levels of VEGF, MMP-2 and MMP -9 proteins	Mahmoud et al., 2024 [28]
<i>Aristolochia baetica</i> and <i>Origanum compactum</i>	Root and aerial	Moroccan	<i>In vitro</i>	Aristolochic acid and betulinic acid	MCF-7	IC ₅₀ 200-250µg/ml	Chaouki et al., 2010 [29]
<i>Carissa edulis</i> and <i>Pappea capensis</i>	Leaves and stem bark, respectively	Kenya	<i>In vitro</i>	Flavonoid, phenolic, tocopherols and terpenoids	HCC 1395	IC ₅₀ <20µg/ml; ↑p53 and Bax	Muruthi et al., 2024 [30]
<i>Albizia gummifera</i> , <i>Rhamnus staddo</i> ,	Leaf, stem bark and root bark	Kenya	<i>In vitro</i>	Flavonoids, glycosides, terpenoids, phenols	HCC 1395	IC ₅₀ < 20 µg/ml; SI >3; ↑ p53; ↓ VEGF	Mbugua et al., 2022 [31]
<i>Jatropha curcas</i> , <i>Pyrenacantha staudtii</i> , <i>Jatropha gossypifolia</i>	Root bark	Nigeria	<i>In vitro</i>		MCF-7	IC ₅₀ 23-38µg/ml; ↓β1-integrin expression; induced a block in G2/M phase; activation of caspases 7 and 9; ↓PCNA expression	Engel et al., 2014 [32]
<i>Erica glabella</i> , <i>Erica racemosa</i> , <i>Hippia frutescens</i> , <i>Slavia Africana-lutea</i>	Leaves, stem,	South Africa	<i>In vitro</i>	Terpenoids, flavonoids, oxepane, chlorogenic acid, fatty acids	MDA-MB-231	IC ₅₀ <30µg/ml; induction of apoptosis	Adu-Amankwaah et al., 2022 [33]
<i>Euclea crispa</i>	Leaves and stem	South Africa	<i>In vitro</i>		MCF-7	IC ₅₀ <45.7µg/ml	Rademan et al., 2019 [34]
<i>Euphorbia tirucalli</i>	Stem	South Africa	<i>In vitro</i>	Terpenoids and flavonoids	MCF-7 and MDA-MB-231	Overexpression of p21; Bax, caspase 8	Choeni & Motadi, 2016 [35]
<i>Albizia zygia</i>	Roots	Ghana	<i>In vitro</i>	Flavonoids, saponins and alkaloids	MCF-7	SI-2.4; induce apoptosis	Appiah-Opong et al., 2016 [36]
<i>Ximenia americana</i>		Tanzania	<i>In vitro</i>	Tannins and glycosides	MCF-7; MDA-MB-231	IC ₅₀ -1.7; 33µg/ml	Voss et al., 2006 [37]
<i>Solanum schimperianum</i>	Leaves	Sudan	<i>In vitro</i>	N-caffeoyl putrescine, N-feruloyl putrescine, N-coumaroyl, N-caffeoyl agmatine, N-sinapoyl putrescine	MCF-7; MDA-MB-231	IC ₅₀ -19.83±3.83; 7.01±0.25µg/ml	Almoulah et al., 2017 [38]

Key: SI- Selective Index, IC₅₀-Half maximal inhibitory concentration, SOD-Superoxide dismutase, MDA-Malonaldehyde, VEGF-Vascular endothelial growth factor, MMP-2- Matrix metalloproteinase, PCNA-Proliferating cell nuclear antigen, ECM-Extracellular matrix, CDON-Cell adhesion associated, oncogene regulated, PARP-Poly (ADP-ribose) polymerase

DISCUSSION

Despite significant advances in diagnosis of breast carcinoma, its treatment remains a formidable challenge. The persistent limitations associated with current therapeutic strategies, including drug resistance, non-specific targeting and toxicity are associated with poor disease outcomes. These limitations have intensified the quest for new therapeutic agents that offer improved selectivity, with less adverse effects. Medicinal plants, long utilized in folk medicine, play a significant role in the development of new anticancer agents. Different plant species have been scientifically validated for their antiproliferative effects, with both *in vitro* and *in vivo* studies confirming their therapeutic potential across various types of cancer, including breast cancer [7].

In this systematic review, various studies were found to have reported on the *in vitro* antiproliferative effects of African medicinal plant extracts on various cell lines, including MDA-MB-231, HCC 1395 and MCF-7. Breast tumors are highly heterogeneous, comprising diverse cell sub-populations with varying carcinogenesis potential. The intratumoral complexity poses a therapeutic challenge. Nevertheless, utilization of *in vitro* culture models permits researchers to circumvent the heterogeneity aspect by offering controlled and reproducible environments for investigating specific cellular and molecular features of breast cancer [39]. The model involves cultivation and maintenance of isolated cells, either as monolayers, 2D cultures, or in more complex configurations such as 3D [40]. The models have, therefore, become important tools for analyzing the biological activities of medicinal plants.

Studies have adopted traditional cell culture methods, the 2D, for evaluating the efficacy of medicinal plants on breast cancer [41-43]. The most common breast cancer lines derived from different patients include MCF-7, HCC 1395, MDA-MB-231 and T47D, each representing distinct molecular subtypes [44-46]. The MCF-7, an estrogen receptor-positive (ER+), carries mutations that are commonly found in luminal breast cancers, including GATA3 and PIK3CA [47, 48]. The MDA-MB-231, acquired from a 51-year-old woman with metastatic breast cancer, is an example of a triple-negative breast cancer (TNBC) and expresses mutated *p53* that contributes to aggressive tumor behaviors [49]. The T47D, derived from an infiltrating ductal carcinoma, expresses high levels of progesterone receptors (PR). Therefore, it is an ideal experimental model for PR-responsive luminal A subtype breast cancer [50].

Advances in cell culture technologies have enhanced the use of *in vitro* models. For example, microfabrication techniques in 2D models, which alter the monolayer surface using nanostructure topographies, have influenced cell proliferation, morphology and signaling pathways [43]. Nevertheless, 2D cultures have limitations, particularly in replicating cell phenotypes that are associated with disease progression, such as cell function, invasive behavior and expressions of pathological markers. Consequently, 3D models have gained attention due to their ability to mimic *in vivo*-like tumor behaviors and architecture [43].

The use of animal models in *in vivo* studies is significant in preclinical cancer research. The animal models provide a more accurate representation of human tumor biology, when compared with *in vitro* studies [51]. For example, in their study, Zingue et al [52], found that dichloromethane/methanol stem barks extract of *C. adansonii* significantly reduced tumor volume and weight in female Wistar rats. Similarly, Luo et al [53] observed that methanolic root extracts of *Prunella vulgaris* inhibited tumor growth in a 4T1 breast cancer BALB/c mouse model. *In silico* models are also emerging as complementary tools in analysing the effects of plant extracts on breast cancer. The computational model simulates biological systems by utilizing mathematical equations and has been used to predict drug-target interactions, pharmacodynamics and absorption [40].

In this review, the included studies showed that the plant extracts exhibited cytotoxic activities with $IC_{50} < 20 \mu\text{g/ml}$, indicating a high growth inhibitory potency against breast cancer cell lines. Low IC_{50} values suggest that the plant extracts can effectively inhibit the proliferation of cancer cells, even at low concentrations, in tandem with the National Cancer Institute (NCI) criterion, whereby an $IC_{50} \leq 20 \mu\text{g/mL}$ is considered to be highly active, between 21–100 $\mu\text{g/mL}$ moderately active and that of $IC_{50} > 100 \mu\text{g/mL}$, weakly active or inactive [30]. Moreover, most of the plant extracts exhibited selectivity indices (SI) > 3 , indicating preferential cytotoxic effects on cancer cells when compared to normal cells. Notably, a high SI value is important in cancer drug development because it reflects the ability of an agent to differentiate between neoplastic and healthy cells, reducing the likelihood of systemic toxicity [30,31].

The reviewed studies demonstrated that African medicinal plant extracts exert significant growth inhibitory effects through different molecular pathways, largely by modulating critical regulators of the cell cycle and programmed cell death [54]. For example, *C. edulis* and *P. capensis* induced antiproliferative effects on HCC 1395 cells by upregulating *p53* and *Bax* genes [30]. Similarly, *R. afairholmianus* extracts upregulated apoptotic markers in MCF-7 [19] whereas bulb extracts of *B. frutescens* inhibited the proliferation of MDA-MB-231 cells by upregulating *p21* while downregulating *cyclin D1*, *CDK4*, *Bcl-2* and *survivin* [16]. These findings are in tandem with established apoptotic pathways, where *p53*, a tumor suppressor gene, responds to cellular stress and DNA damage by promoting apoptosis and cell death [55,56]. In addition, upregulation of *p21*, a downstream effector of *p53*, inhibits cyclin-dependent kinases (CDKs) leading to cell cycle arrest at the G1 or G2/M checkpoints, thereby suppressing uncontrolled proliferation [27-34].

Downregulation of anti-apoptotic proteins, including *Bcl-2* alter mitochondrial integrity, thereby promoting the release of cytochrome c, and subsequent activation of executioner caspases such as caspase-3, and 9 [57]. The caspases further induce the cleavage of cellular components, resulting in morphological hallmarks of apoptosis, such as chromatin condensation, membrane blebbing and nuclear shrinkage [58].

Moreover, it was noted that some plant extracts exerted anti-metastatic effects by suppressing genes involved in epithelial-mesenchymal transition (EMT) and cell invasion, which are critical processes in cancer progression and metastasis. Some plant extracts inhibited the expressions of pro-metastatic genes, including Cell Adhesion Associated, Oncogene Regulated (CDON) and Snail Family Transcription Repressor 1 (SNAIL) [14]. As a transmembrane cell adhesion receptor, CDON is involved in regulation of cell motility and invasive behaviors whereas SNAIL represses E-cadherin and promotes mesenchymal, migratory phenotypes in cancer cells [14].

The reported pathomechanisms were associated with the presence of bioactive phytochemicals in the studied medicinal plants. Phytochemicals, including polyphenols, terpenoids, phytosterols and alkaloids have been identified as key components with anticancer effects. Polyphenols such as flavonoids, luteolin, apigenin, quercetin, and kaempferol inhibit tumor-promoting signaling pathways [30]. In HCC 1395 cells, the compounds increased the expressions of *p53* and *Bax* genes. Against the MCF-7 cell line, polyphenol compounds reduced the Bax/Bcl-2 ratio, enhanced cytochrome release as well as the subsequent activation of caspases 3, 7 and 9, thereby reducing apoptosis [59,60]. On T47D cells, polyphenols downregulated *Bcl-2*, Akt, and PI3K while upregulating *p53*, *p21* and apoptotic caspases [56,61,62]. Some phytochemicals also alter mitochondrial metabolism; for example, flavone was found to detach hexokinase from the outer mitochondrial membrane, inhibiting glycolysis in MCF-7 and MDA-MB-23 cells [63].

Certain flavonoids act as pro-oxidants in cancer cells, generating ROS, thereby promoting cell death [64-66]. For instance, Kaushik et al [67] demonstrated that flavonoids induced G2/M cell cycle arrest followed by ROS-mediated apoptosis in MCF-7 and MDA-MB-231 cell lines. Similarly, Palit et al. demonstrated that the flavanone, hesperetin, induced intrinsic mitochondrial apoptosis via ROS in MCF-7 cells [68].

Resveratrol has been shown to modulate the p53-dependent apoptotic pathway, activate ERK1/2 and suppress VEGF, EGFR and COX-2 pathways, ultimately reversing epithelial-mesenchymal transition (EMT) and inhibiting metastasis in TBC models [69]. The anti-metastatic effects of resveratrol have also been reported in preclinical models. Other polyphenols such as quercetin, kaempferol, luteolin and gallic modulate NF-KB, EGFR/P13k/AKT, MAPK and STAT signaling pathways, thereby regulating angiogenesis, apoptosis, migration, histone acetylation and gene methylation [70-75]. Notably, some polyphenols enhance the efficacy of chemotherapy and radiation, suggesting the potential for combination therapy in resistant subtypes, including TNBC [30,76].

Terpenoids, including phytol, menthol, D-limonene, are among the largest naturally occurring group of phytochemicals. Paclitaxel, a diterpenoid, remains a cornerstone in metastatic breast cancer therapy [77]. Terpenoids have been demonstrated to induce mitochondrial apoptosis, modulate ROS and suppress proliferation in breast cancer cell lines. Muruthi et al. [30] attributed the antiproliferative effects of *C. edulis* and *P. capensis* to the presence of phytochemicals, including terpenoids, which upregulated *p53* and *Bax* expressions in HCC 1395 cell lines. Kuete et al. [79] also reported that diterpenoids exert their inhibitory effects on MCF-7 cell lines by inducing apoptosis mediated by MMP loss and increased ROS levels. Similarly, Hou et al. [80] demonstrated the antiproliferative activities of diterpenoids isolated from *Euphorbia kansui* in MDA-MB-435 cells. Phytosterols, including sitosterol and stigmasterol have been reported to have inhibitory effects against breast cancer cell lines [80-84]. Kaigongi et al. [11] demonstrated that stigmasterol isolated from *Z. paracanthum* inhibited HCC 1395 proliferation while enhancing pro-apoptotic signaling by upregulating *bax* genes. Various studies have reported the anticancer activities of alkaloids [85-91].

Additionally, in their studies, Rampogu et al [92]. also reported alkaloids including berbamine, fangchinoline and capsaicin that have shown to induce autophagy in treating breast cancer. Furthermore, Habli et al [86]. reported the anticancer effect of hisrsutin, isolated from plant of the genus *Uncaria* on murine breast cancer.

CONCLUSION

African medicinal plants exert growth inhibitory effects against breast cancer cell lines by modulating key molecular pathways, including programmed cell death. Due to their accessibility, less adverse effects and affordability, medicinal plants are promising candidates for the development of anticancer therapies. Nevertheless, further preclinical and mechanistic studies are needed to support their potential in breast cancer treatment.

Conflict of interest

The authors declared no conflict of interest.

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