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Phytochemicals of the Malvaceae Family and Their Mechanistic Role in Cancer Prevention and Therapy: A Comprehensive Review

Ms. Rutuja Chavan*[1], Dr. Sunil Shingade[1], Ms. Namra Shaikh[1]
[1.]*V.P. College of Pharmacy MadkholSawantwadi*

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ABSTRACT
Globally, cancer continues to be a major cause of illness and death, distinguished by uncontrolled cellular proliferation, resistance to apoptosis, genomic instability, angiogenesis, inflammation, and metastatic progression. Although significant advancements have been achieved in conventional cancer therapies such as surgery, chemotherapy, radiotherapy, hormone therapy, and targeted molecular treatments, their clinical effectiveness is often compromised by adverse effects, toxicity, high cost, and the development of drug resistance. Consequently, there is growing interest in plant-derived bioactive compounds as alternative or complementary anticancer agents. The plant family Malvaceae, widely distributed across tropical and subtropical regions, is recognized for its rich reservoir of pharmacologically active phytochemicals, including flavonoids, phenolic acids, anthocyanins, triterpenoids, sterols, alkaloids, and saponins. This review systematically compiles and analyzes phytochemicals isolated from selected Malvaceae plants and highlights their mechanistic roles in cancer prevention and therapy. Emphasis is placed on molecular mechanisms such as modulation of apoptotic pathways, cell-cycle arrest, oxidative stress regulation, inhibition of angiogenesis and metastasis, suppression of inflammation, and interference with key oncogenic signaling pathways including PI3K/Akt, MAPK, NF-κB, STAT3, Wnt/β-catenin, and p53. The accumulated evidence supports the therapeutic potential of Malvaceae phytochemicals as multi-target anticancer agents and underscores their relevance in future cancer drug discovery and development.

Keywords: Malvaceae; Phytochemicals; Cancer; Anticancer activity; Apoptosis; Cell cycle arrest; Molecular mechanisms; Natural products

I. INTRODUCTION

Cancer represents a multifactorial group of diseases arising from genetic mutations, epigenetic alterations, environmental exposure, lifestyle factors, and chronic inflammation. It is characterized by hallmark features such as sustained proliferative signaling, evasion of programmed cell death, limitless replicative potential, induction of angiogenesis, metabolic reprogramming, immune evasion, and metastatic dissemination. Despite continuous progress in understanding cancer biology, effective and safe management of cancer remains a major challenge in modern medicine. [1]

Conventional cancer treatment modalities, including chemotherapy, radiotherapy, surgery, and targeted therapies, have significantly improved survival rates in several cancer types. However, their use is often associated with severe systemic toxicity, off-target effects, and the emergence of multidrug resistance, which limit therapeutic success. Additionally, many targeted therapies act on single molecular pathways, making them less effective against heterogeneous and adaptive tumor populations. These limitations have driven the search for novel therapeutic agents capable of modulating multiple cancer-related pathways simultaneously with reduced toxicity.[2]

Natural products have been crucial in the search for anticancer drugs, helping to create a number of medicines that are now in clinical use. Medicinal plant-derived phytochemicals possess diverse chemical structures and biological activities, enabling them to interact with multiple molecular targets involved in carcinogenesis. Their ability to regulate oxidative stress, inflammation, apoptosis, angiogenesis, and metastasis makes them attractive candidates for cancer prevention and therapy.[3]

The Malvaceae family comprises more than 4,000 species distributed worldwide and includes medicinally important genera such as *Hibiscus*, *Sida*, *Abutilon*, *Gossypium*, *Malva*, *Urena*, *Thespesia*, and *Grewia*. These plants have been traditionally used in various systems of medicine for the treatment of inflammatory disorders, infections,

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metabolic diseases, and tumors. Phytochemical investigations of Malvaceae plants have revealed a rich composition of bioactive compounds, including flavonoids, phenolic acids, anthocyanins, triterpenoids, phytosterols, alkaloids, and glycosides.[4]

Experimental studies have demonstrated that Malvaceae-derived phytochemicals exert significant anticancer effects through multiple mechanisms, such as induction of apoptosis via intrinsic and extrinsic pathways, cell-cycle arrest at various phases, inhibition of angiogenesis, suppression of inflammatory mediators, modulation of oxidative stress, and inhibition of cancer cell invasion and metastasis. Furthermore, these compounds have been shown to interfere with key oncogenic signaling pathways, including PI3K/Akt, MAPK, NF-κB, STAT3, Wnt/β-catenin, and p53, which are commonly dysregulated in various cancers.[5]

This review aims to comprehensively summarize the phytochemical constituents of selected Malvaceae plants and critically evaluate their molecular mechanisms of action in cancer prevention and treatment, thereby highlighting their potential role as promising multi-target anticancer agents.

Malvaceae Plants, Key Phytochemical & Detailed MOA

1. *Hibiscus rosa-sinensis*

Key Phytochemical: Quercetin

Molecular Formula: $C_{15}H_{10}O_7$

Chemical Structure: 2-(3,4-dihydroxyphenyl)-3,5,7-trihydroxychromen-4-one

Mechanism of Action (MOA):

Quercetin, a prominent flavonoid present in *Hibiscus rosa-sinensis*, exhibits potent anticancer activity through multiple molecular pathways. It primarily inhibits the PI3K/Aktsignaling pathway, which plays a crucial role in promoting cancer cell survival and proliferation. By suppressing this pathway, quercetin reduces survival signals within tumor cells.

In addition, it activates the tumor suppressor protein p53, which leads to an increase in pro-apoptotic protein Bax and a decrease in anti-apoptotic protein Bcl-2. This imbalance promotes mitochondrial dysfunction, resulting in the release of cytochrome-c and subsequent activation of caspase-9 and caspase-3, ultimately inducing apoptosis.

Furthermore, quercetin inhibits the epidermal growth factor receptor (EGFR), thereby preventing uncontrolled cell growth. It also suppresses the NF-κBsignaling pathway, reducing inflammation and tumor progression. Another important action includes arresting the cell cycle at the G2/M phase by inhibiting cyclin-B/CDK1 complexes, thereby halting cancer cell division. **[6]**

2. *Hibiscus sabdariffa*

Key Phytochemical: Delphinidin

Molecular Formula: $C_{15}H_{11}O_7^+$

Chemical Structure: 2-(3,4,5-trihydroxyphenyl)chromenylium-3,5,7-triol

Mechanism of Action (MOA):

Delphinidin, an anthocyanidin found in *Hibiscus sabdariffa*, demonstrates significant anticancer effects through modulation of various signaling pathways. It inhibits IκB kinase (IKK), thereby preventing the activation and nuclear translocation of NF-κB, a key transcription factor involved in inflammation and cancer progression.

This inhibition leads to a reduction in the expression of pro-inflammatory and anti-apoptotic proteins such as COX-2 and survivin. Additionally, delphinidin activates death receptors DR4 and DR5, which in turn trigger the extrinsic apoptotic pathway via activation of caspase-8.

Moreover, it blocks the ERK/MAPK signaling pathway, thereby suppressing cell proliferation. Delphinidin also exhibits anti-angiogenic activity by inhibiting VEGFR2 signaling, which prevents the formation of new blood vessels required for tumor growth and metastasis. **[7]**

3. *Abelmoschus esculentus*

Key Phytochemical: Isoquercitrin

Molecular Formula: $C_{21}H_{20}O_{12}$

Chemical Structure: 3-[(2S,3R,4R,5R)-5-[(1R)-1,2-dihydroxyethyl]-3,4-dihydroxyoxolan-2-yl]oxy-2-(3,4-dihydroxyphenyl)-5,7-dihydroxychromen-4-one

Mechanism of Action (MOA):

Isoquercitrin exerts its anticancer activity primarily through the generation of controlled levels of reactive oxygen species (ROS), which disrupt mitochondrial integrity. This leads to depolarization of the mitochondrial membrane and opening of the mitochondrial permeability transition pore (mPTP), resulting in the release of cytochrome-c into the cytosol.

Additionally, isoquercitrin suppresses Akt phosphorylation, thereby inhibiting survival signaling pathways in cancer cells. It also downregulates cyclin-D expression, causing arrest

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of the cell cycle at the G1 phase and preventing further cell proliferation.**[8]**

4. *Gossypiumherbaceum*
Key Phytochemical: Gossypol
Molecular Formula: $C_{30}H_{30}O_8$
Chemical Structure: 2,3,8-trihydroxy-6-methyl-4-propan-2-ylnaphthalene-1-carbaldehyde 7-(8-formyl-1,6,7-trihydroxy-3-methyl-5-propan-2-ylnaphthalen-2-yl)
Mechanism of Action (MOA):
Gossypol functions as a BH3 mimetic, meaning it binds to anti-apoptotic proteins such as Bcl-2, Bcl-XL, and Mcl-1, thereby neutralizing their function and promoting intrinsic apoptosis.
It also inhibits lactate dehydrogenase-A (LDH-A), disrupting cancer cell metabolism and reducing energy production. Furthermore, gossypol enhances the activation of caspase-9 and caspase-3, leading to programmed cell death. It also induces DNA fragmentation, a hallmark of apoptosis in cancer cells. **[9]**

5. *Sida cordifolia*
Key Phytochemical: Ephedrine
Molecular Formula: $C_{10}H_{15}NO$
Chemical Structure: (1R,2S)-2-(methylamino)-1-phenylpropan-1-ol
Mechanism of Action (MOA):
Ephedrine demonstrates anticancer potential by inducing oxidative stress through increased ROS generation, which leads to mitochondrial damage. This process is accompanied by a reduction in anti-apoptotic protein Bcl-xL levels.
Additionally, ephedrine disrupts microtubule polymerization, which interferes with mitotic spindle formation and results in mitotic arrest. These events collectively activate caspase-3-mediated apoptosis, ultimately leading to cancer cell death. **[10]**

6. *Sidarhombifolia*
Key Phytochemical: Cryptolepine
Molecular Formula: $C_{16}H_{12}N_2$
Chemical Structure: 5-methylindolo[3,2-b]quinoline
Mechanism of Action (MOA):
Cryptolepine exhibits strong anticancer activity through direct interaction with DNA. It intercalates into DNA strands, disrupting normal replication and transcription processes.
Moreover, it inhibits topoisomerase-II activity, leading to the accumulation of double-strand DNA breaks. This damage activates tumor suppressor protein p53 as well as DNA damage response pathways involving ATM/ATR kinases.As a result, cancer cells undergo S-phase cell cycle arrest and eventually apoptosis. **[11]**

7. *Urena lobata*
Key Phytochemical: Kaempferol
Molecular Formula: $C_{15}H_{10}O_6$
Chemical Structure: 3,5,7-trihydroxy-2-(4-hydroxyphenyl)chromen-4-one
Mechanism of Action (MOA):
Kaempferol exerts anticancer effects by inhibiting the MAPK/ERK signaling pathway, which is essential for cancer cell growth and proliferation. It also reduces the expression of HER2, a receptor commonly overexpressed in certain cancers.
Additionally, kaempferol induces mitochondrial-mediated apoptosis by increasing the expression of pro-apoptotic protein Bax. It also inhibits angiogenesis by reducing VEGF levels, thereby limiting tumor blood supply and growth. **[12]**

8. *Abutilon indicum*
Key Phytochemical: β-Sitosterol
Molecular Formula: $C_{29}H_{50}O$
Chemical Structure: (3S,8S,9S,10R,13R,14S,17R)-17-[(2R,5R)-5-ethyl-6-methylheptan-2-yl]-10,13-dimethyl-2,3,4,7,8,9,11,12,14,15,16,17-dodecahydro-1H-cyclopenta[a]phenanthren-3-ol
Mechanism of Action (MOA):
β-Sitosterol promotes apoptosis by increasing the expression of pro-apoptotic protein Bax and decreasing anti-apoptotic protein Bcl-2. This shift in balance activates both extrinsic and intrinsic apoptotic pathways through caspase-8 and caspase-9 activation.
It also inhibits NF-κBsignaling, thereby reducing tumor-associated inflammation. Furthermore, β-sitosterol helps in limiting cancer metastasis and progression. **[14]**

9. *Malva sylvestris*
Key Phytochemical: Malvin
Molecular Formula: $C_{29}H_{35}O_{17}^{+}$
Chemical Structure: (complex anthocyanin glycoside structure as given)
Mechanism of Action (MOA):
Malvin exhibits anticancer activity by inhibiting key enzymes involved in DNA synthesis, thereby preventing replication of cancer cells. It induces cell cycle arrest at the S-phase, halting cell division.Additionally, it suppresses ROS-induced DNA damage, protecting normal cells while

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targeting cancerous cells. Malvin also reduces the expression of matrix metalloproteinase-9 (MMP-9), which plays a role in tumor invasion and metastasis, thereby exerting anti-metastatic effects. **[15]**

10. *Malva neglecta*
Key Phytochemical: Apigenin-7-O-glucoside
Molecular Formula: $C_{21}H_{20}O_{10}$
Chemical Structure: 5-hydroxy-2-(4-hydroxyphenyl)-7-[(3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)oxan-2-yl]oxychromen-4-one
Mechanism of Action (MOA):
Apigenin-7-O-glucoside exerts anticancer effects by inhibiting the PI3K/Aktsignaling pathway, thereby suppressing cancer cell survival and proliferation. It induces G1 phase cell cycle arrest by downregulating cyclin-D1 and CDK4 expression.
Furthermore, it increases the Bax/Bcl-2 ratio, promoting mitochondrial dysfunction and cytochrome-c release, which subsequently activates caspase-9 and caspase-3, leading to apoptosis.In addition, it suppresses MMP-9 expression, thereby reducing cancer cell invasion and metastasis, particularly in breast cancer models. **[16]**

11. *Althaea officinalis*
Key Phytochemical: Rutin
Molecular Formula: $C_{27}H_{30}O_{16}$
Chemical Structure: 2-(3,4-dihydroxyphenyl)-5,7-dihydroxy-3-[…]oxychromen-4-one
Mechanism of Action (MOA):
Rutin exhibits significant anticancer potential by activating the tumor suppressor protein p53, which plays a crucial role in maintaining genomic stability and inducing apoptosis in damaged cells. It also enhances DNA repair mechanisms, thereby preventing mutation accumulation.
Additionally, rutin suppresses the PI3K/Aktsignaling pathway, reducing cancer cell survival and proliferation. It further inhibits angiogenesis by downregulating vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF), thereby limiting tumor blood supply and growth. **[17]**
12. *Bombax ceiba*
Key Phytochemical: Lupeol
Molecular Formula: $C_{30}H_{50}O$
Chemical Structure: (complex pentacyclic triterpenoid structure as given)
Mechanism of Action (MOA):
Lupeol demonstrates potent anticancer effects by inhibiting the PI3K/Akt and mTORsignaling pathways, both of which are critical for cancer cell growth and metabolism. It also suppresses NF-κB activation, thereby reducing inflammation and tumor progression.
Moreover, lupeol inhibits matrix metalloproteinases (MMP-2 and MMP-9), preventing cancer cell invasion and metastasis. It also induces mitochondrial-mediated apoptosis, leading to programmed cell death in cancer cells. **[18]**

13. *Hibiscus cannabinus*
Key Phytochemical: Hibiscetin
Molecular Formula: $C_{15}H_{10}O_9$
Chemical Structure: 3,5,7,8-tetrahydroxy-2-(3,4,5-trihydroxyphenyl)chromen-4-one
Mechanism of Action (MOA):\
Hibiscetin exhibits strong anti-metastatic activity by acting as a potent inhibitor of MMP-9, an enzyme involved in extracellular matrix degradation and tumor spread.
It also promotes apoptosis by activating caspase-3, a key executioner enzyme in programmed cell death. Additionally, hibiscetin suppresses ERK1/2 phosphorylation, thereby inhibiting cancer cell proliferation and survival. **[19]**

14. *Hibiscus mutabilis*
Key Phytochemical: Scopoletin
Molecular Formula: $C_{10}H_8O_4$
Chemical Structure: 7-hydroxy-6-methoxychromen-2-one
Mechanism of Action (MOA):
Scopoletin exerts anticancer effects primarily through anti-inflammatory and pro-apoptotic mechanisms. It inhibits NF-κB and COX-2 pathways, thereby reducing inflammation-associated cancer progression.
Furthermore, it induces the generation of mitochondrial ROS, which leads to oxidative stress and triggers apoptosis in cancer cells. **[20]**

15. *Pavoniaodorata*
Key Phytochemical: Gallic Acid
Molecular Formula: $C_7H_6O_5$
Chemical Structure: 3,4,5-trihydroxybenzoic acid
Mechanism of Action (MOA):
Gallic acid induces apoptosis through the generation of reactive oxygen species (ROS), which disrupt mitochondrial membrane integrity and lead to its collapse.This process activates caspase-9 and caspase-3, promoting programmed cell death. Additionally, gallic acid suppresses epigenetic regulatory enzymes such as histone deacetylases (HDAC), thereby influencing gene expression and inhibiting cancer progression. **[21]**

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16. *Kosteletzkyavirginica*
Key Phytochemical: Luteolin
Molecular Formula: $C_{15}H_{10}O_6$
Chemical Structure: 2-(3,4-dihydroxyphenyl)-5,7-dihydroxychromen-4-one
Mechanism of Action (MOA):
Luteolin demonstrates anticancer activity by inhibiting insulin-like growth factor-1 receptor (IGF-1R) and the PI3K/Aktsignaling pathway, thereby reducing cancer cell proliferation and survival.
It also blocks epithelial–mesenchymal transition (EMT), a key step in metastasis, by increasing E-cadherin expression and decreasing N-cadherin levels. Additionally, luteolin suppresses angiogenesis by inhibiting VEGF signaling. **[22]**

17. *Hibiscus acetosella*
Key Phytochemical: Cyanidin
Molecular Formula: $C_{15}H_{11}O_6^+$
Chemical Structure: 2-(3,4-dihydroxyphenyl)chromenylium-3,5,7-triol
Mechanism of Action (MOA):
Cyanidin induces apoptosis through activation of death receptors via the TRAIL pathway, leading to activation of caspase-8 and caspase-3.It also suppresses Aktsignaling, thereby inhibiting cancer cell survival. Furthermore, cyanidin prevents metastasis by downregulating MMP-2 and MMP-9 expression, which are essential for tumor invasion. **[23]**

18. *Hibiscus taylori*
Key Phytochemical: Myricetin
Molecular Formula: $C_{15}H_{10}O_8$
Chemical Structure: 3,5,7-trihydroxy-2-(3,4,5-trihydroxyphenyl)chromen-4-one
Mechanism of Action (MOA):
Myricetin exerts anticancer effects by inhibiting PI3K and Aktsignaling pathways, thereby suppressing cancer cell growth and survival.
It also stabilizes the tumor suppressor protein p53, enhancing apoptosis. Additionally, myricetin promotes mitochondrial-mediated apoptosis and inhibits STAT3 signaling, which is involved in cancer progression and immune evasion. **[24]**

19. *Gossypium arboreum*
Key Phytochemical: Tannic Acid
Molecular Formula: $C_{76}H_{52}O_{46}$
Chemical Structure: (complex polyphenolic structure as given)
Mechanism of Action (MOA):
Tannic acid exhibits anticancer activity through direct interaction with DNA. It binds to DNA and inhibits topoisomerase I and II enzymes, thereby preventing DNA replication and transcription.This leads to activation of DNA damage response pathways and induces cleavage of poly (ADP-ribose) polymerase (PARP), ultimately resulting in apoptosis. **[25]**

20. *Hibiscus asper*
Key Phytochemical: Protocatechuic Acid
Molecular Formula: $C_7H_6O_4$
Chemical Structure: 3,4-dihydroxybenzoic acid
Mechanism of Action (MOA):
Protocatechuic acid demonstrates anticancer potential by inhibiting MMP-2 and MMP-9, thereby reducing tumor invasion and metastasis.
It also exerts anti-inflammatory effects by suppressing tumor necrosis factor-alpha (TNF-α), which contributes to cancer progression. Furthermore, it activates caspase-3, leading to apoptosis in cancer cells. **[26]**

21. *Hibiscus macranthus*
Key Phytochemical: Ellagic Acid
Molecular Formula: $C_{14}H_6O_8$
Chemical Structure: 6,7,13,14-tetrahydroxy-2,9-dioxatetracyclo[...]hexadeca-3,10-dione
Mechanism of Action (MOA):
Ellagic acid exhibits potent anticancer activity primarily through epigenetic regulation and induction of mitochondrial apoptosis. It inhibits DNA methyltransferases (DNMT1, DNMT3A, DNMT3B), leading to hypomethylation and reactivation of tumor suppressor genes such as *p53* and *p21*. This restoration enhances cell cycle control and promotes apoptosis.
Furthermore, ellagic acid increases the Bax/Bcl-2 ratio, resulting in mitochondrial outer membrane permeabilization and release of cytochrome-c. This activates the apoptosome complex (Apaf-1 + caspase-9), followed by executioner caspase-3 activation, leading to programmed cell death.
In addition, it suppresses tumor angiogenesis by downregulating VEGF expression and inhibiting HIF-1α-mediated signaling pathways. **[27]**

22. *Hibiscus surattensis*
Key Phytochemical: Fisetin
Molecular Formula: $C_{15}H_{10}O_6$
Chemical Structure: 2-(3,4-dihydroxyphenyl)-3,7-dihydroxychromen-4-one
Mechanism of Action (MOA):

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Fisetin exerts anticancer effects by targeting the Wnt/β-catenin signaling pathway. It promotes degradation of β-catenin via activation of the destruction complex (APC, Axin, GSK-3β), preventing its nuclear translocation and transcription of oncogenic genes such as *c-Myc* and *cyclin-D1*.
This leads to downregulation of cyclin-D1 and inhibition of CDK4/6 activity, resulting in G1 phase cell cycle arrest. Fisetin also activates both intrinsic and extrinsic apoptotic pathways by triggering caspase-8, caspase-9, and caspase-3, thereby inducing apoptosis.
Additionally, it suppresses PI3K/Aktsignaling, further reducing cancer cell survival and proliferation. **[28]**

23. *Hibiscus furcatus*
Key Phytochemical: Chlorogenic Acid
Molecular Formula: $C_{16}H_{18}O_9$
Chemical Structure: (1S,3R,4R,5R)-3-[(E)-3-(3,4-dihydroxyphenyl)prop-2-enoyl]oxy-1,4,5-trihydroxycyclohexane-1-carboxylic acid
Mechanism of Action (MOA):
Chlorogenic acid inhibits STAT3 phosphorylation, thereby preventing its dimerization and nuclear translocation. This suppresses transcription of genes involved in proliferation (*cyclin-D1*), survival (*Bcl-XL*), and angiogenesis (*VEGF*).
It also interferes with IL-6/JAK/STAT signaling, reducing inflammation-driven tumor growth. Additionally, chlorogenic acid inhibits endothelial cell migration and tube formation, thereby suppressing tumor angiogenesis.
Moreover, it enhances DNA repair mechanisms through modulation of p53 and PARP pathways, contributing to genomic stability. **[29]**

24. *Sida acuta*
Key Phytochemical: Vasicine
Molecular Formula: $C_{11}H_{12}N_2O$
Chemical Structure: (3S)-1,2,3,9-tetrahydropyrrolo[2,1-b]quinazolin-3-ol
Mechanism of Action (MOA):
Vasicine inhibits cancer cell proliferation by interfering with DNA synthesis and replication processes, leading to S-phase cell cycle arrest.
It also induces mitochondrial oxidative stress by increasing ROS generation, which damages mitochondrial components and leads to loss of membrane potential (ΔΨm). This triggers the release of cytochrome-c and activation of the intrinsic apoptotic pathway via caspase-9 and caspase-3. **[30]**

25. *Sidatiagii*
Key Phytochemical: Palmitic Acid
Molecular Formula: $C_{16}H_{32}O_2$
Chemical Structure: Hexadecanoic acid
Mechanism of Action (MOA):
Palmitic acid induces apoptosis by integrating into mitochondrial membranes and altering their structural integrity, resulting in mitochondrial dysfunction and depolarization.
This disruption leads to release of cytochrome-c and activation of the apoptotic cascade through caspase-9 and caspase-3. Additionally, palmitic acid inhibits survival signaling pathways such as PI3K/Akt, thereby enhancing apoptotic sensitivity in cancer cells. **[31]**

26. *Malachracapitata*
Key Phytochemical: Apigenin
Molecular Formula: $C_{15}H_{10}O_5$
Chemical Structure: 5,7-dihydroxy-2-(4-hydroxyphenyl)chromen-4-one
Mechanism of Action (MOA):
Apigenin inhibits the Akt/mTORsignaling pathway, leading to reduced phosphorylation of downstream targets such as p70S6K and 4E-BP1, thereby suppressing protein synthesis and tumor growth.
It induces mitochondrial apoptosis by increasing Bax expression and decreasing Bcl-2, resulting in cytochrome-c release and activation of caspase-9 and caspase-3.
Furthermore, apigenin inhibits epithelial–mesenchymal transition (EMT) by downregulating transcription factors such as *Snail*, *Slug*, and *Twist*, thereby reducing metastasis. **[32]**

27. *Malva parviflora*
Key Phytochemical: Ferulic Acid
Molecular Formula: $C_{10}H_{10}O_4$
Chemical Structure: (E)-3-(4-hydroxy-3-methoxyphenyl)prop-2-enoic acid
Mechanism of Action (MOA):
Ferulic acid inhibits tumor angiogenesis by suppressing VEGF expression and VEGFR2 signaling, thereby preventing endothelial cell proliferation and new blood vessel formation.
It also reduces ROS-mediated DNA damage, thereby protecting genomic integrity. In cancer cells, it promotes apoptosis by enhancing caspase activation and increasing pro-apoptotic proteins such as Bax. **[33]**

28. *Hibiscus altissima*
Key Phytochemical: Vitexin
Molecular Formula: $C_{21}H_{20}O_{10}$

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Chemical Structure: 5,7-dihydroxy-2-(4-hydroxyphenyl)-8-[…]chromen-4-one
Mechanism of Action (MOA):
Vitexin inhibits both PI3K/Akt and ERK/MAPK signaling pathways, thereby suppressing cancer cell proliferation and survival.
It downregulates cyclin-D1 and CDK4, leading to G1 phase arrest. Additionally, vitexin induces apoptosis by activating caspase-3 and promoting mitochondrial dysfunction. It also reduces inflammatory mediators contributing to tumor progression. **[34]**

29. *Hibiscus coddii*
Key Phytochemical: Catechin
Molecular Formula: $C_{15}H_{14}O_6$
Chemical Structure: (2R,3S)-2-(3,4-dihydroxyphenyl)-3,4-dihydro-2H-chromene-3,5,7-triol
Mechanism of Action (MOA):
Catechin induces oxidative stress in cancer cells by increasing intracellular ROS levels, leading to mitochondrial membrane permeabilization and release of cytochrome-c.
This activates the intrinsic apoptotic pathway via caspase-9 and caspase-3. Additionally, catechin inhibits NF-κBsignaling, thereby reducing inflammation, proliferation, and survival signals. It also decreases anti-apoptotic protein Bcl-2, enhancing apoptosis. **[35]**

30. *Grewia asiatica*
Key Phytochemical: Pelargonidin
Molecular Formula: $C_{15}H_{11}O_5^+$
Chemical Structure: 2-(4-hydroxyphenyl)chromenylium-3,5,7-triol
Mechanism of Action (MOA):
Pelargonidin induces apoptosis through activation of death receptors (DR4/DR5), triggering the extrinsic apoptotic pathway via caspase-8, which subsequently activates caspase-3.
It also inhibits HER2 signaling, reducing proliferation in HER2-positive cancers. Furthermore, pelargonidin suppresses metastasis by decreasing expression of matrix metalloproteinases (MMPs) and other invasion-related proteins. **[36]**

II. Conclusion

The growing global burden of cancer and the limitations associated with existing therapeutic strategies necessitate the exploration of alternative and complementary treatment approaches. Phytochemicals derived from the **Malvaceae** family represent a valuable source of bioactive compounds with significant anticancer potential. The evidence compiled in this review demonstrates that Malvaceae plants contain a wide range of secondary metabolites capable of targeting multiple hallmarks of cancer, including uncontrolled proliferation, resistance to apoptosis, angiogenesis, inflammation, oxidative stress, and metastasis.

These phytochemicals exert their anticancer effects through diverse molecular mechanisms, such as modulation of apoptotic signaling pathways, regulation of cell-cycle checkpoints, inhibition of oncogenic transcription factors, suppression of pro-inflammatory mediators, and interference with key intracellular signaling cascades. The multi-targeted nature of Malvaceae-derived compounds offers a distinct advantage over conventional single-target therapies and may contribute to overcoming drug resistance and reducing treatment-related toxicity.

Although preclinical studies provide substantial support for the anticancer efficacy of Malvaceae phytochemicals, further investigations are required to validate their clinical relevance. Future research should focus on standardized extraction methods, bioavailability enhancement, toxicological evaluation, molecular target validation, and well-designed clinical trials. Overall, Malvaceae plants hold considerable promise as a reservoir of novel anticancer agents and may play a significant role in the development of safer and more effective strategies for cancer prevention and therapy.

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