Journal of Medicines Development Sciences, 2026, Volume 11, Issue 3
https://bbwpublisher.com/journal/JMDS
ISSN Online:2382-6371
ISSN Print:2382-6363

Flavonoids Targeting the PI3K/Akt/mTOR Pathway in Breast Cancer: A Systematic Review

Yin Huang*

University of Nottingham, NG7 2RD, United Kingdom

Author to whom correspondence should be addressed.

Copyright: © 2026 Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY 4.0), permitting distribution and reproduction in any medium, provided the original work is cited.

Abstract: *Background:* The PI3K/Akt/mTOR pathway supports breast-cancer cell growth, survival and motility, and its persistent activation is associated with treatment resistance. Flavonoids are often described as multi-target natural compounds, but their activity against this pathway has not been synthesised consistently. *Methods:* PubMed, Scopus and Web of Science were searched to 23 March 2026 for peer-reviewed preclinical studies of single, naturally occurring flavonoids in human breast-cancer models. Ten studies published from 2021 to 2026 met the eligibility criteria. In vivo risk of bias was assessed with SYRCLE's tool, and in vitro reliability with an adapted ToxRTool. *Results:* The ten compounds were luteolin, sophoraflavanone G, eriodictyol, karanjin, calycosin, kushenol A, ampelopsin, ononin, galangin and tangeretin. Every study reported suppression of at least part of the PI3K/Akt/mTOR cascade, although only five measured all three core nodes. Reduced signalling generally coincided with lower proliferation, cell-cycle arrest or apoptosis; five studies also reported reduced migration or invasion, and seven included an animal experiment. *Conclusions:* The direction of effect was broadly consistent, but the evidence remains preclinical, heavily weighted towards MDA-MB-231 cells, and incomplete for pharmacokinetics, toxicity and pathway-wide target engagement.

Keywords: Breast cancer; Flavonoids; PI3K/Akt/mTOR; Apoptosis; Metastasis; Systematic review

Online publication: September 16, 2026

1. Introduction

Breast cancer is not a single biological entity. Luminal, HER2-positive and triple-negative tumours differ in receptor status, signalling dependencies and treatment response. Even when an initial response is achieved, pathway reactivation and clonal selection can sustain residual disease. The phosphoinositide-3-kinase (PI3K)/Akt/mechanistic target of rapamycin (mTOR) network is relevant across these subtypes because it links receptor signals to protein synthesis, metabolism, survival and motility. Growth factor receptors activate PI3K, which generates PIP_3 and recruits Akt to the membrane; Akt then regulates mTOR-containing complexes and downstream effectors. PTEN provides an important brake by converting PIP_3 back to PIP_2. The network is not linear: feedback and crosstalk mean that inhibition at one point may be followed by

signalling recovery elsewhere.

Flavonoids are structurally varied plant polyphenols found in foods and medicinal plants. Their appeal in cancer research lies less in a single high-affinity target than in the possibility of affecting several connected processes at once. Recent breast-cancer experiments have linked individual flavonoids to lower PI3K/Akt/mTOR activity, apoptosis, cell-cycle arrest and reversal of epithelial-mesenchymal transition (EMT) [1-10]. Those reports use different compounds, cell lines, doses and readouts, which makes the apparent consistency difficult to judge from isolated papers. This review therefore asks a narrow question: how do single flavonoids modulate PI3K/Akt/mTOR signalling in preclinical breast-cancer models, and which cellular or animal outcomes accompany that modulation?

2. Methods

2.1. Search and eligibility

The review was reported in accordance with PRISMA 2020 [11]. A systematic search was conducted in PubMed, Scopus and Web of Science. Search terms combined flavonoid names or subclasses with breast-cancer terms and PI3K, Akt or mTOR terms. The core string was: (flavonoid* OR flavone* OR flavonol* OR flavanone* OR isoflavone* OR anthocyanin*) AND ("breast cancer" OR "breast carcinoma" OR "breast neoplasm*") AND (PI3K OR "phosphoinositide 3-kinase" OR AKT OR "protein kinase B" OR mTOR OR "mechanistic target of rapamycin"). Searches were limited to English-language publications from 2021 to the final search date. Records were deduplicated in EndNote.

Eligible reports were original, peer-reviewed experiments evaluating a single naturally occurring flavonoid in a human breast-cancer cell line, an animal breast-cancer model, or both. Experimental evidence had to show modulation of PI3K/Akt/mTOR signalling together with a biological outcome. Extracts and mixtures, synthetic or biotransformed derivatives, murine-only cell-line studies, purely computational analyses and papers in which this pathway was not a primary mechanistic axis were excluded. Titles and abstracts were screened before full-text assessment. Data extraction covered compound class, model, dose, pathway readouts, phenotypic outcomes and in vivo findings. Because the studies were heterogeneous, results were synthesised narratively rather than pooled.

2.2. Quality assessment

Animal experiments were appraised with SYRCLE's ten signalling questions across six bias domains [12]; each judgement was coded as low, high or unclear risk of bias. In vitro reliability was assessed with a review-specific 17-item adaptation of ToxRTool [13], created by merging the two overlapping statistical-reporting items in the original 18-item tool. Items were scored 1 (met) or 0 (not met); totals of 15-17, 11-14 and 0-10 indicated Categories 1, 2 and 3, respectively, and failure on any of five critical items precluded Category 1. One reviewer completed both assessments. The ratings informed interpretation but were not used to exclude studies or weight the narrative synthesis.

3. Results

3.1. Study selection and characteristics

The search yielded 518 records: 111 from PubMed, 342 from Scopus, 62 from Web of Science and three from

other sources. Removing 149 duplicates left 369 titles and abstracts (Figure 1). Twenty-five articles entered full-text review, and 15 were excluded, most often because the intervention was an extract or synthetic derivative, the mechanistic evidence was insufficient, or PI3K/Akt/mTOR was not the main axis. Ten studies remained [1-10]. Their designs ranged from focused signalling experiments in one cell line to combined cell and animal studies. Eight used MDA-MB-231 cells, which immediately limits how confidently the findings can be extended to other TNBC models or receptor-defined subtypes (Table 1). Table 1 reports the principal dose range and treatment period for each study. These exposure details are descriptive only because units, assay windows and in vivo schedules were not comparable across experiments.

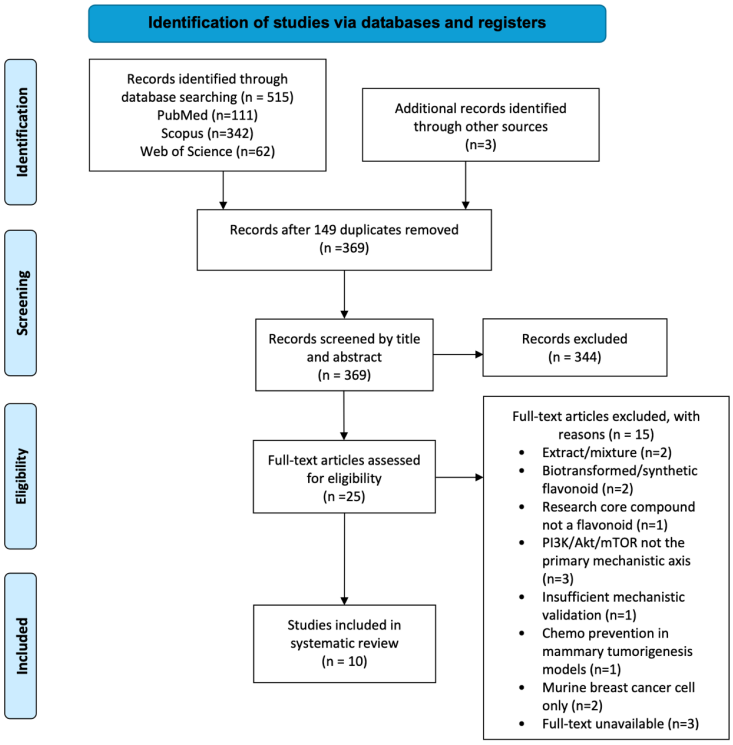


Figure 1. PRISMA flow diagram for study selection.

The counts reproduce the review screening log: 518 identified records, 369 screened, 25 full texts assessed and 10 studies included.

Table 1. Core characteristics and findings of the ten included studies

Compound / study	Subclass	Models and exposure	Pathway evidence	Biological effects	In vivo
Luteolin Wu et al., 2021 [1]	Flavone	BT-20; MDA-MB-231 Exposure: 10–30 μM; 24–96 h.	↓ p-Akt and p-mTOR in BT-20; PI3K not assessed; weak or opposite response at high dose in MDA-MB-231	↓ proliferation, migration and invasion; EMT reversal; ↓ MMP-9/2	No
Sophoraflavanone GCheng et al., 2022 [2]	Flavanone	BT-549; MDA-MB-231 Exposure: 2.5–160 μM (viability); 20–30 μM for 24 h (other assays). Animal dose/schedule: NR.	↓ p-EGFR, p-PI3K and p-Akt; EGFR overexpression attenuated effects	↓ proliferation/ motility; ↑ apoptosis and oxidative stress	Xenograft: ↓ tumour growth

Compound / study	Subclass	Models and exposure	Pathway evidence	Biological effects	In vivo
Eriodictyol He et al., 2022 [3]	Flavanone	MCF-7; MDA-MB-231; MCF-10A Exposure: 10–40 μM; time NR. In vivo: 20 mg/kg; schedule NR.	↓ circ_0007503, p-PI3K and p-Akt; circRNA rescue weakened effects	↓ viability and transformation; ↑ apoptosis	MNU-rat: incidence 83.3% → ~50%
Karanjin Yu et al., 2022 [4]	Furanoflavone	MDA-MB-231; MDA-MB-468 Exposure: 5–160 μg/mL; 48 h.	↓ p-PI3K/PI3K and p-Akt/Akt; 740Y-P reversed effects	↓ proliferation; ↑ apoptosis	No
Calycosin Li et al., 2023 [5]	Isoflavone	MDA-MB-231; BT-549 Exposure: dose/time NR. In vivo: i.p.; dose/schedule NR.	↓ ER-α30, p-PI3K and p-Akt; ER-α30 knockdown strengthened inhibition	↓ proliferation/motility; ↑ apoptosis; EMT reversal	Xenograft: ↓ tumour growth
Kushenol A Liu et al., 2023 [6]	Flavanone	MDA-MB-231; MCF-7; BT-474 Exposure: 0.5–32 μM; 24–72 h (mechanistic assays, 4–16 μM for 48 h). In vivo: gavage daily for 2 weeks; dose NR.	↓ p-Akt and p-mTOR; synergy with PI3K-IN-6; total Akt/mTOR unchanged	↓ proliferation; G0/G1 arrest; ↑ apoptosis	Xenograft: ↓ tumour growth
Ampelopsin Meng et al., 2023 [7]	Flavanonol	MDA-MB-231 Exposure: 10–50 μM; 6–48 h (colonies, 14 d). In vivo: 20/50/100 mg/kg twice weekly for 4 weeks; route reported inconsistently.	↓ PI3K, Akt and mTOR; cyclin B1 overexpression attenuated effects	↓ proliferation; G2/M arrest; ↑ apoptosis	Xenograft: ↓ tumour growth
Ononin Ganesan et al., 2024 [8]	Isoflavone glycoside	MDA-MB-231; 4T1 Exposure: 24 h in EGF-rescue assays; ononin dose NR. In vivo: dose/schedule NR.	↓ p-EGFR, PI3K, Akt and mTOR; EGF stimulation restored signalling	↓ proliferation and motility; ↑ apoptosis; EMT reversal	Mouse model: ↓ tumour and lung metastasis
Galangin Nimal et al., 2025 [9]	Flavonol	MDA-MB-231 Exposure: 50 μM for 48 h (range to 125 μM; combination, 1 μM galangin + 0.08 μM SAHA). In vivo: 25 mg/kg i.p.; 20 d.	↓ PI3K, p-Akt and mTOR; ↑ PTEN; synergy with SAHA	↓ proliferation/motility; sub-G0/G1 arrest; ↑ apoptosis	Xenograft: ↓ tumour growth
Tangeretin Reddy & Shankaraiah, 2026 [10]	Polymethoxylated flavone	SKBR3; MCF-7 Exposure: 1–50 μM; 48 h (MTT) and 24 h (mechanistic assays).	↓ p-HER2, p-PI3K, p-Akt, p-mTOR and Akt kinase activity	↓ clonogenic survival; G2/M arrest; ↑ apoptosis	No

Arrows indicate reported change after flavonoid treatment. "No" means that the study did not include an animal experiment; it does not imply absence of in vivo activity. Exposure entries summarise the principal tested conditions rather than every assay-specific protocol. NR, not reported.

3.2. Pathway modulation and biological outcomes

Across the ten studies, Akt was the most consistently measured node. Ampelopsin, ononin, galangin and tangeretin reduced signals across PI3K, Akt and mTOR [7-10]. Kushenol A lowered phosphorylated Akt and mTOR without changing their total abundance; direct PI3K reduction was not shown, although combining the compound with PI3K-IN-6 strengthened pathway inhibition [6]. Luteolin acted selectively in androgen-receptor-positive BT-20 cells, where Akt/mTOR activity and MMP-9 expression fell, while the response in

MDA-MB-231 cells was weaker and could reverse at higher concentrations [1]. These differences matter: an unmeasured node should not be described as inhibited, and a cell-line-specific response should not be generalised to the entire subtype.

Several experiments connected the core cascade to an upstream regulator. Sophoraflavanone G reduced EGFR phosphorylation; EGFR overexpression weakened its effects on PI3K/Akt signalling and cell behaviour [2]. Ononin also acted through EGFR, with ligand stimulation partially restoring signalling [8]. Tangeretin reduced phosphorylated HER2 in SKBR3 cells [10]. Eriodictyol lowered circ_0007503, and overexpression of this circular RNA attenuated pathway suppression [3]. Calycosin reduced the truncated estrogen-receptor variant ER-α30, while ER-α30 knockdown enhanced inhibition of PI3K/Akt [5]. The studies therefore point to several entry routes rather than one flavonoid-specific binding mechanism.

All ten compounds reduced proliferation or viability, but potency was not directly comparable because doses, exposure times and assays differed. Cell-cycle data were available for four compounds: ampelopsin and tangeretin produced G2/M arrest, kushenol A produced G0/G1 arrest, and galangin increased the sub-G0/G1 fraction [6,7,9,10]. Nine studies reported apoptosis. Recurrent changes included a higher Bax/Bcl-2 balance, caspase-9 and caspase-3 activation, and PARP cleavage, consistent with mitochondrial intrinsic apoptosis. Migration or invasion was examined in five studies; all five reported inhibition together with higher E-cadherin and lower N-cadherin, vimentin or matrix metalloproteinases [1,2,5,8,9]. Ononin provided the strongest metastasis-specific evidence: in a 4T1-luc mouse model it reduced the primary tumour and pulmonary metastatic burden while suppressing EGFR/PI3K/Akt/mTOR and EMT-associated proteins [8].

Seven studies included an animal component [2,3,5-9]. Five used subcutaneous xenografts and reported lower tumour volume or weight. Eriodictyol was tested in an MNU-induced rat model and reduced mammary-tumour incidence from 83.3% to about 50% at 20 mg/kg [3]. Ononin was the only compound evaluated with a dedicated lung-metastasis outcome [8]. Body weight did not decline in the studies that reported it, but this is a weak substitute for a structured toxicity assessment. No study supplied the pharmacokinetic measurements needed to connect an effective cell-culture concentration with achievable systemic exposure.

3.3. Study quality

For the seven animal studies, 51 of 70 SYRCLE judgements were unclear. Random allocation, allocation concealment, random housing and blinding were rarely described; no domain was consistently judged high risk, but absence of reporting is not evidence that safeguards were used (**Figure 2**). In vitro ToxRTool scores ranged from 14 to 16 of 17. Nine studies met the threshold for reliability without restriction and one was reliable with restrictions. Cell-line authentication, positive controls and compound-purity reporting were the most common gaps. Study-level counts, scores and principal unmet criteria are reported in Table 2.

Table 2. Study-level summary of quality and reporting assessments

Study/compound	SYRCLE L/U/H	ToxRTool score/category	Principal unmet ToxRTool item(s)
Luteolin	NR	16/17; Cat. 1	Positive control
Sophoraflavanone G	1/9/0	15/17; Cat. 1	Cell-line characterisation; positive control
Eriodictyol	3/7/0	16/17; Cat. 1	Positive control
Karanjin	NR	15/17; Cat. 1	Cell-line characterisation; positive control
Calycosin	3/7/0	16/17; Cat. 1	Cell-line characterisation

Study/compound	SYRCLE L/U/H	ToxRTool score/category	Principal unmet ToxRTool item(s)
Kushenol A	2/8/0	14/17; Cat. 2	Purity; cell-line characterisation; replicate reporting
Ampelopsin	4/6/0	15/17; Cat. 1	Cell-line characterisation; positive control
Ononin	3/7/0	16/17; Cat. 1	Purity
Galangin	3/7/0	15/17; Cat. 1	Purity; cell-line characterisation
Tangeretin	NR	15/17; Cat. 1	Supplier details; purity

Abbreviations: L/U/H, low/unclear/high risk-of-bias judgements; NR, no animal component; Cat., reliability category. Category 1 denotes reliable without restriction and Category 2 reliable with restrictions. The adapted ToxRTool scores describe reporting reliability rather than an independently validated risk-of-bias scale.

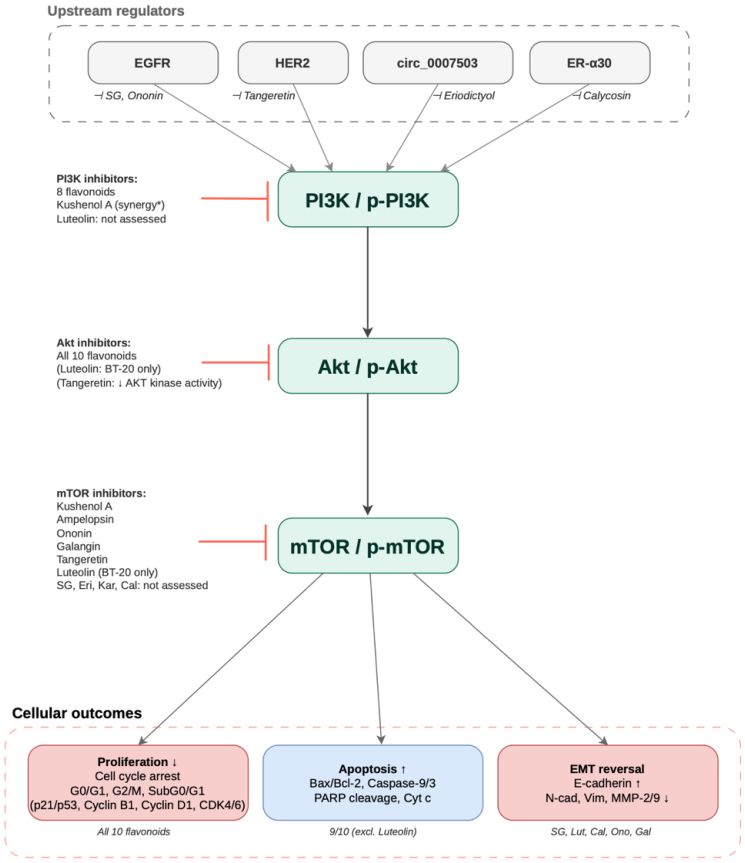


Figure 2. Reported points of flavonoid action within the PI3K/Akt/mTOR network.

The diagram distinguishes reported upstream regulators, core signalling nodes and downstream cellular outcomes. Labels summarise the included studies [1-10]; they do not establish direct binding unless a study performed a target-specific experiment.

4. Discussion

The most defensible conclusion is directional rather than quantitative. Ten structurally different flavonoids

were associated with weaker PI3K/Akt/mTOR signalling and less aggressive behaviour in breast-cancer models, yet the studies do not establish a common potency, dose or direct molecular target. Some measured phosphorylated proteins, others total protein or circulating concentrations, and several stopped at PI3K/Akt without examining mTOR. The apparent breadth of inhibition may therefore reflect both biology and the choice of assays. A future study that measures the same nodes, at matched time points and with pathway rescue experiments, would be more informative than another isolated viability experiment. To avoid conflating evidence sources, citations [1-10] identify the included experiments, whereas [14-18] are used only to contextualise mechanism and translation. This distinction matters because feedback and pathway crosstalk described in the wider cancer literature [14] support caution but do not enlarge the review sample.

The upstream data offer a plausible explanation for the varied responses. EGFR, HER2, circ_0007503 and ER-α30 can all feed into PI3K/Akt signalling, so compounds acting at different entry points may converge on the same central cascade. That convergence does not prove that a flavonoid is a selective pathway inhibitor. Rescue experiments in the sophoraflavanone G, eriodictyol, karanjin, calycosin, ampelopsin and ononin studies strengthen causality [2-5,7,8], but off-target effects remain likely. The selective response of BT-20 cells to luteolin is a useful warning that receptor context can change the direction or size of the effect [1]. The circRNA finding is biologically plausible in light of broader circRNA-PI3K/Akt crosstalk [15], but that background literature does not independently validate circ_0007503 as a breast-cancer target.

Downstream phenotypes were more consistent than the upstream mechanisms. Reduced Akt activity can release pro-apoptotic Bcl-2-family signalling and favour mitochondrial permeabilisation, which fits the repeated caspase-9/caspase-3/PARP pattern. PI3K/Akt suppression can also reduce EMT-associated transcription and matrix remodelling, consistent with the E-cadherin, vimentin and MMP changes. Even so, most studies measured signalling and phenotype in parallel; they did not always show that the phenotype disappeared when the pathway was restored. The ononin metastasis model and the rescue experiments are therefore more persuasive than correlations alone. The apoptotic interpretation is supported by experimental evidence that Akt phosphorylation of BAD couples growth-factor signalling to the intrinsic death machinery [16]. Likewise, independent TNBC models show hybrid epithelial/mesenchymal states and a functional requirement for vimentin during metastasis [17]; this supports the relevance of the marker changes without reducing EMT to a simple binary switch.

Translation is the central weakness. MDA-MB-231 cells dominate the evidence base, while luminal and HER2-positive models are sparse and patient-derived systems are absent. Subcutaneous xenografts simplify the tumour microenvironment and do not reproduce immune interactions in an intact host. In addition, flavonoids often have limited aqueous solubility, extensive metabolism and low systemic exposure. Without plasma and tumour concentrations, it is impossible to know whether the active in vitro doses are pharmacologically realistic. Claims of safety are also premature because body weight and routine histology cannot replace dose-ranging toxicology. The bioavailability challenge is well documented for dietary flavonoids and has prompted multiple delivery-system strategies [18]; it cannot, however, substitute for compound-specific exposure data in the included studies.

The next step should be comparative rather than merely confirmatory. The same panel of subtype-representative cell lines should be tested with authenticated cells, verified compound purity, positive pathway controls and predefined readouts for p-PI3K, p-Akt, p-mTOR and feedback activation. Orthotopic, immunocompetent or patient-derived models would improve biological relevance. Pharmacokinetic sampling

should be built into animal experiments, and combination studies should distinguish additivity from true synergy. Existing hints of enhanced activity with luteolin plus MK-2206 or rapamycin, kushenol A plus PI3K-IN-6, and galangin plus SAHA [1,6,9] justify such work, but they do not yet support clinical use.

This review also has methodological limitations. The search was restricted to English-language articles published from 2021 onward and to three bibliographic databases; grey literature was not searched. A single reviewer conducted screening, data extraction and quality appraisal, increasing the possibility of missed records or subjective judgements. Heterogeneity in models, doses, exposure times and outcome definitions precluded meta-analysis, and the adapted ToxRTool was used as a reporting-reliability aid rather than a validated risk-of-bias instrument for these experiments. Publication and selective-reporting bias cannot be excluded; the findings should therefore be read as a structured narrative synthesis rather than a quantitative estimate of effect.

5. Conclusion

The available preclinical literature presents a coherent but incomplete signal: ten flavonoids reduced at least part of the PI3K/Akt/mTOR cascade and were accompanied by anti-proliferative, pro-apoptotic or anti-metastatic effects in breast-cancer models. Evidence was strongest where pathway rescue, receptor perturbation or in vivo confirmation linked signalling changes to phenotype. It was weakest where a single cell line, one protein readout or body weight alone carried the interpretation. The findings support further mechanistic and pharmacokinetic study, particularly across receptor-defined subtypes and in clinically relevant models; they do not yet establish efficacy, safety or a therapeutic dose in patients.

Disclosure statement

The author declares no conflict of interest.

References

[1] Wu HT, Lin J, Liu YE, et al., 2021, Luteolin Suppresses Androgen Receptor-Positive Triple-Negative Breast Cancer Cell Proliferation and Metastasis by Epigenetic Regulation of MMP9 Expression via the AKT/mTOR Signaling Pathway. Phytomedicine, 81: 153437.

[2] Cheng W, Liu D, Guo M, et al., 2022, Sophoraflavanone G Suppresses the Progression of Triple-Negative Breast Cancer via the Inactivation of EGFR-PI3K-AKT Signaling. Drug Dev Res, 5: 1138–1151.

[3] He J, Fu H, Li C, et al., 2022, Eriodictyol Inhibits Breast Carcinogenesis by Targeting circ_0007503 and Repressing PI3K/Akt Pathway. Phytomedicine, 102: 154159.

[4] Yu J, Yang H, Lv C, et al., 2022, The Cytotoxicity of Karanjin Toward Breast Cancer Cells Is Involved in the PI3K/Akt Signaling Pathway. Drug Dev Res, 7: 1673–1682.

[5] Li Y, Hu S, Chen Y, et al., 2023, Calycosin Inhibits Triple-Negative Breast Cancer Progression Through Down-Regulation of the Novel Estrogen Receptor-α Splice Variant ER-α30-Mediated PI3K/AKT Signaling Pathway. Phytomedicine, 118: 154924.

[6] Liu T, Gong J, Lai G, et al., 2023, Flavonoid Extract Kushenol A Exhibits Anti-Proliferative Activity in Breast Cancer Cells via Suppression of PI3K/AKT/mTOR Pathway. Cancer Med, 2: 1643–1654.

[7] Meng M, Yang Q, Ouyang Z, et al., 2023, Ampelopsin Induces MDA-MB-231 Cell Cycle Arrest Through Cyclin B1-Mediated PI3K/AKT/mTOR Pathway In Vitro and In Vivo. Acta Pharm, 1: 75–90.

[8] Ganesan K, Xu C, Wu J, et al., 2024, Ononin Inhibits Triple-Negative Breast Cancer Lung Metastasis by Targeting the EGFR-Mediated PI3K/Akt/mTOR Pathway. Sci China Life Sci, 9: 1849–1866.

[9] Nimal S, Kumbhar N, Pote MS, et al., 2025, Reversal of Epithelial to Mesenchymal Transition in Triple Negative Breast Cancer Through Epigenetic Modulations by Dietary Flavonoid Galangin and Its Combination with SAHA. Cell Commun Signal, 1: 163.

[10] Reddy JS, Shankaraiah P, 2026, Tangeretin Suppresses HER2-Driven Breast Cancer via PI3K/AKT/mTOR Inhibition and Caspase-Dependent Apoptosis. Bangladesh J Pharmacol, 1: 1–10.

[11] Page MJ, McKenzie JE, Bossuyt PM, et al., 2021, The PRISMA 2020 Statement: An Updated Guideline for Reporting Systematic Reviews. BMJ, 372: n71.

[12] Hooijmans CR, Rovers MM, de Vries RBM, et al., 2014, SYRCLE's Risk of Bias Tool for Animal Studies. BMC Med Res Methodol, 14: 43.

[13] Schneider K, Schwarz M, Burkholder I, et al., 2009, "ToxRTool", A New Tool to Assess the Reliability of Toxicological Data. Toxicol Lett, 2: 138–144.

[14] Glaviano A, Foo ASC, Lam HY, et al., 2023, PI3K/AKT/mTOR Signaling Transduction Pathway and Targeted Therapies in Cancer. Mol Cancer, 1: 138.

[15] Xue C, Li G, Lu J, et al., 2021, Crosstalk Between circRNAs and the PI3K/AKT Signaling Pathway in Cancer Progression. Signal Transduct Target Ther, 1: 400.

[16] Datta SR, Dudek H, Tao X, et al., 1997, Akt Phosphorylation of BAD Couples Survival Signals to the Cell-Intrinsic Death Machinery. Cell, 2: 231–241.

[17] Grasset EM, Dunworth M, Sharma G, et al., 2022, Triple-Negative Breast Cancer Metastasis Involves Complex Epithelial-Mesenchymal Transition Dynamics and Requires Vimentin. Sci Transl Med, 656: eabn7571.

[18] Yuan D, Guo Y, Pu F, et al., 2024, Opportunities and Challenges in Enhancing the Bioavailability and Bioactivity of Dietary Flavonoids: A Novel Delivery System Perspective. Food Chem, 430: 137115.

Publisher's note

Bio-Byword Scientific Publishing remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.