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Letter to the editor:

AN UP-TO-DATE REVIEW OF DECURSIN AND ITS ANTI-CANCER ACTIVITIES

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Decursin (a pyranocoumarin compound) is a coumarin derivative found in *Angelica gigas*, particularly in its roots. The biosynthesis of decursin is primarily routed through the phenylpropanoid pathway, which is also responsible for the generation of several aromatic amino acids. Phenylalanine undergoes deamination by phenylalanine ammonia-lyase to produce cinnamic acid, which is subsequently hydroxylated at the para-position by cinnamate 4-hydroxylase to yield *p*-coumaric acid. Additional hydroxylation steps convert this intermediate into 2,4-dihydroxycinnamic acid, a key precursor for the formation of the coumarin skeleton and, ultimately, umbelliferone. The attachment of a 2-methylbut-2-ene side chain results in the formation of 7-demethylsuberosin, which then undergoes cyclization to generate decursinol. Subsequent modifications of decursinol give rise to its isomeric derivatives, decursin and decursinol angelate (Sestito et al., 2024).

Decursin has gained significant attention as a potent antitumor agent owing to its substantial inhibitory effects on cancer progression and low toxicity in normal tissues (Hayashi et al., 2023). Moreover, decursin exhibits anti-cancer effects across a wide spectrum of malignancies, including pancreatic, cervical, gastric, colorectal, prostate, liver, esophageal, head and neck, brain (glioblastoma), ovarian, non-small-cell lung, and inflammation-associated colorectal and skin cancers, B-cell lymphoma, FLT3-ITD acute myeloid leukemia, and hypoxia-driven tumors, underscoring its broad potential as a multi-targeted therapeutic agent. This review synthesizes recent advances in decursin research, with a particular focus on its anti-cancer mechanisms and therapeutic properties (Table 1).

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Table 1: Overview of recent studies on the biological and pharmacological activities of decursin

Key message	Reference
Decursin exhibits diverse anticancer activities across multiple malignancies through the modulation of cell cycle regulators, apoptotic pathways, and oncogenic signaling networks. In pancreatic cancer, decursin induces G0/G1 cell cycle arrest and caspase-3–dependent apoptosis, while suppressing migration and invasion via the downregulation of MMP-2/-9 through p38 signaling. In PANC-1 and MIA PaCa-2 cells, it reduces viability and colony formation, underscoring its therapeutic potential in pancreatic malignancies.	Kweon et al., 2020
Decursin promotes apoptosis and inhibits proliferation and migration in HeLa cells through the modulation of Akt signaling. In vitro and in vivo models consistently demonstrate reductions in tumor growth, supporting its potential as a targeted therapeutic agent in cervical cancer.	Zhu et al., 2021
In gastric cancer, decursin disrupts autophagic flux by CTSC expression and activity, resulting in the accumulation of LC3 and SQSTM1. It impairs proliferation and cell cycle progression by targeting CTSC and E2F3, key regulators of tumor aggressiveness. Validation in spheroid and patient-derived organoid models confirmed its ability to reduce tumor growth and modulate autophagy-related proteins.	Kim et al., 2021
Decursin regulates colorectal cancer progression. It modulates the PI3K/Akt pathway and epithelial–mesenchymal transition, leading to the downregulation of Bcl-2, N-cadherin, vimentin, PI3K, and Akt, while upregulating Bax, E-cadherin, and p53, thereby promoting apoptosis and impairing migration.	Yang et al., 2023
Decursin induces apoptosis through ROS generation and subsequent ER stress activation; ROS inhibition with *N*-acetyl-l-cysteine abrogated this effect. Preclinical studies confirmed tumor suppression in xenograft models without systemic toxicity, supporting its therapeutic relevance through the ROS–ER stress axis.	Kim et al., 2024
In prostate carcinoma, decursin inhibits EGFR phosphorylation at Tyr1068 and suppresses downstream ERK1/2 activation, leading to G1 arrest, apoptosis, and the inhibition of proliferation. Both DU145 and 22Rv1 cell lines exhibited reduced colony formation and modulation of Rb, E2F, and CDK inhibitors. Notably, combinatorial inhibition with erlotinib enhanced growth suppression, suggesting potential in EGFR–ERK1/2–driven prostate cancer therapy.	Bhat et al., 2023
In acute myeloid leukemia harboring FLT3-ITD mutations, decursin facilitates proteasome-mediated degradation of FLT3-ITD by upregulating the E2-conjugating enzyme UBE2L6. This mechanism impairs cell cycle progression and survival. Decursin and venetoclax exert synergistic effects, enhancing apoptosis in patient-derived xenograft mouse models, supporting its value as a targeted therapy for FLT3-ITD–driven leukemia.	Zhang et al., 2025
Decursin exerts anti-angiogenic and immunomodulatory effects by promoting proteasomal degradation of HIF-1α, thereby suppressing hypoxia-inducible gene expression and tumor progression. In vitro and allograft mouse studies revealed the inhibition of proliferation, invasion, and angiogenesis, alongside the enhancement of T-cell activation and reduction of immunosuppressive cell populations within the tumor microenvironment.	Ge et al., 2020
In esophageal squamous cell carcinoma, decursin induces G0/G1 arrest and apoptosis via proteasomal degradation of the oncogenic drivers TP63 and SOX2. In vitro and xenograft studies confirmed selective cytotoxicity, reduced migration and invasion, and significant tumor growth suppression without systemic toxicity.	Fang et al., 2025
In head and neck squamous cell carcinoma, decursin targets CXCR7, inducing G0/G1 arrest and suppressing proliferation, migration, and invasion by downregulating cyclin A, cyclin E, and CDK2. Mechanistically, it inhibits the CXCR7–STAT3–c-Myc signaling cascade, thereby impairing tumor progression.	Joo et al., 2022
Decursin suppresses inflammation-driven tumorigenesis by inhibiting LPS-induced PRP4 activation via the TLR4/JNK pathway. In colorectal and skin cancer models, it reduces IL-6, IL-1β, TLR4, and NF-κB expression, while reversing LPS/PRP4-driven cytoskeletal changes and enhancing sensitivity to apoptosis. These results indicate potential utility against PRP4-mediated metastasis and drug resistance.	Ahmed et al., 2020

Key message	Reference
In glioblastoma, decursin activates JNK and p38 signaling, downregulates Bcl-2, CDK4, and cyclin D1, and triggers caspase-dependent apoptosis. Studies in U87 and C6 glioma cells confirmed dose-dependent cytotoxicity, without affecting normal glial cells, suggesting tumor-selective activity.	Oh et al., 2019
In hepatocellular carcinoma, decursin promotes YAP degradation by upregulating phosphorylated LATS1 and βTRCP, thereby activating the Hippo pathway. This leads to cell cycle arrest and apoptosis, which are reversed upon the pharmacological inhibition of MST1/2. In vitro and in vivo models confirmed growth suppression, positioning decursin as a candidate for Hippo/YAP-driven HCC therapy.	Li et al., 2018
In B-cell lymphoma, decursin suppresses Myc expression and induces apoptosis through the inhibition of AKT/mTOR and MAPK signaling downstream of BCR activation. In vitro and in vivo studies demonstrated selective cytotoxicity against lymphoma cells without affecting normal immune cells. It exerts synergistic effects in combination with Myc inhibitors, providing a rationale for therapeutic use in Myc-driven lymphomas.	Kim et al., 2018
In doxorubicin-resistant ovarian cancer, decursin reverses multidrug resistance by inhibiting P-glycoprotein expression, thereby reducing drug efflux and sensitizing tumor cells to chemotherapy. In vitro studies demonstrated that a combination of decursin or *Angelica gigas* Nakai extract with doxorubicin suppresses cell proliferation, induces apoptosis via the activation of caspase-9, caspase-8, caspase-3, and PARP cleavage, and increases sub-G1 populations. These findings highlight the potential of decursin as an adjuvant agent to overcome chemoresistance in ovarian cancer therapy.	Choi et al., 2016
In non-small-cell lung cancer, decursin enhances TRAIL sensitivity by inducing apoptosis through ROS-mediated activation of the PERK/ATF4 branch of the ER stress pathway, leading to the upregulation of DR5 and suppression of survivin and Bcl-xL. In vitro studies demonstrated selective cytotoxicity in TRAIL-resistant NSCLC cells without affecting normal lung cells. Synergistic effects were observed in combination with TRAIL, providing a rationale for therapeutic use in TRAIL-resistant lung cancers.	Kim et al., 2016

Abbreviations: MMP-2, Matrix metalloproteinase-2; Akt, protein kinase B; CTSC, suppressing cathepsin C; LC3, Microtubule-Associated Proteins 1A/1B Light Chain 3; SQSTM1, Sequestosome-1; E2F Transcription Factor; Bcl-2, B-cell lymphoma 2; PI3K, phosphatidylinositol 3-kinase; ROS, reactive oxygen species; ER, Endoplasmic Reticulum; EGFR, epidermal growth factor receptor; ERK, extracellular signal-regulated kinase; Rb, retinoblastoma protein; CDK, cyclin-dependent kinase; Tyr1068, tyrosine 1068 residue; FLT3-ITD, FLT3 gene; UBE2L6, E2 ubiquitin-conjugating enzyme; HIF-1α, hypoxia-inducible factor 1; TP63, Tumor protein p63; SOX2, Sex-determining region Y-box transcription factor 2; CXCR7, CXC chemokine receptor 7; STAT3, signal transducer and activator of transcription 3; LPS, Lipopolysaccharide; PRP4, Pre-mRNA Processing Factor 4 Kinase (PRP4K; gene PRPF4B); TLR4, Toll-Like Receptor 4; JNK, c-Jun N-Terminal Kinase; IL-6, Interleukin-6; IL-1β, Interleukin-1 beta; YAP, Yes-associated protein; LATS1, Large Tumor Suppressor kinase 1; βTRCP, Beta-transducin repeat-containing protein; MST1/2, Mammalian STE20-like protein kinase 1/2; HCC, Hepatocellular carcinoma; mTOR, mechanistic target of rapamycin; MAPK, mitogen-activated protein kinase; BCR, B-cell receptor; Myc, MYC proto-oncogene; PARP, Poly(ADP-ribose polymerase; TRAIL, TNF-related apoptosis-inducing ligand; PERK, PKR-like ER kinase; ATF4, activating transcription factor 4; DR5, death receptor 5.

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Conflicts of interest

The author declares no conflict of interest.

Artificial Intelligence (AI) – Assisted Technology

The author did not use artificial intelligence-based technologies.

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