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PHARMACOLOGICAL ANALYSIS OF THE ANTICANCER EFFECTS OF TAXIFOLIN

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Abstract

The article provides information about taxifolin, which plays an important role in the treatment and prevention of various diseases, including cancer. Many studies have been conducted on taxifolin, and numerous pharmacological properties have been discovered, including anticancer activity. Taxifolin is a potent flavonoid with therapeutic potential in the treatment of diseases, especially inflammation associated with cancer. This article talks about various studies that prove the anticarcinogenic effect of taxifolin: in breast cancer, colon cancer, prostate cancer, skin cancer, etc. Taxifolin treatment prevents cancer progression, metastasis, relapse, and stimulates apoptosis.

Key words: taxifolin, flavonoid, anticarcinogenic effect, treatment

In recent years, new bioactive compounds derived from plants have been identified that play an important role in the treatment and/or prevention of various diseases, including cancer [1]. Flavonoids, which are among the biologically active compounds, have been found to be beneficial to human health. Flavonoids are hydroxylated phenolic compounds containing benzo- γ -pyrone ring in their structure [2]. Natural bioactive compounds, especially flavonoids, have significant effects in the treatment of carcinoma.

They exhibit strong antioxidant activity and neutralize the action of free radicals and chelate metal ions due to the presence of hydroxyl groups [3]. Taxifolin, a flavonoid, has attracted attention for its wide pharmacological effects as well as its anticancer activities such as preventing angiogenesis, reactive oxidative species (ROS) and cell cycle regulators and inducing apoptosis [4,5].

In one study, the effect of taxifolin against breast cancer was assessed by interrupting the lipid and carbohydrate metabolism induced by

carcinogens. The study found that taxifolin corrected the altered metabolism and prevented uncontrolled cell proliferation by interacting with LXR (Liver X receptors) and HMG-CoAR (hydroxymethylglutaryl-coenzyme A reductase) [10]. Taxifolin plays a vital role in reducing the proliferation, migration and invasion of cancer cells in aggressive breast carcinoma, which was assessed by *in vivo* and *in vitro* assessment. According to the results obtained from the studies, taxifolin potently inhibited the proliferation and migration of cancer cells in a dose-dependent manner. In addition, it prevented the epithelial-mesenchymal transition process, reduced the expression of mesenchymal markers.

It also reduced the expression of β -catenin protein and mRNA. Inhibits the growth of primary tumors and prevents breast cancer metastasis to the lungs as assessed in a 4T1 xenograft mouse model [11].

Taxifolin has been reported to directly and indirectly inhibit stemness and epithelial-mesenchymal transition in various tumor-related studies. Taxifolin induces modification of various bioactive substances and promotes differentiation of human umbilical cord-derived mesenchymal stem cells into osteoblasts [12]. Taxifolin has also been documented to enhance inhibition of NF- κ B signaling pathway associated with osteogenic differentiation. There are various signaling pathways involved in stemness regulation such as Janus family tyrosine kinase (JAK)/signal transducer and activator of transcription (STAT), Notch, serine/threonine kinase PI3K/AKT (“AK” of the mouse name, and the “T” was for the word “thymoma” describing the cellular source of the retrovirus), SHH(sonic hedgehog activated tumors) and Wnt/ β -catenin pathways [13]. The inhibitory effect of taxifolin was investigated in

colon cancer cell lines and HCT116 (cell line was isolated from the colon of an adult male with colon cancer) xenograft model. Taxifolin appears to have cytotoxicity against colorectal cancer cell lines (HCT-116 and HT-29) in a dose- and time-dependent manner. Administration of taxifolin to colorectal cancer cell lines causes cell growth arrest, changes in G2 phase regulatory molecules, and induction of apoptosis in a dose-dependent manner. Taxifolin reduced β -catenin, AKT, and survivin gene expression and protein expression both *in vitro* and *in vivo* [14]. These data suggest that targeting the β -catenin gene may contribute to the cell cycle changes and cell cycle regulation resulting from taxifolin treatment, thereby reducing the risk of colorectal cancer.

The effect of taxifolin on the expression of major human miRNAs was assessed in two cell lines, Hep G2 and primary human hepatocytes, using the Affymetrix GeneChip™ miRNA 3.0 Array. In this study, the expression of four miRNAs, miR-153, miR-204, miR-211, and miR-377-3p, was downregulated by taxifolin treatment. Interestingly, all of these miRNAs are directly associated with ZEB2 protein expression in various cancer model systems. Moreover, taxifolin treatment causes a dose-dependent increase in ZEB 2 protein expression. Taxifolin also reduces ZEB 2 signaling by inhibiting Akt phosphorylation, which triggers carcinogenesis. This result suggests that taxifolin produces confusing and even contradictory results due to its non-specific effects on the cell [15].

The effects of taxifolin plus andrographolide (Andro), a diterpenoid lactone isolated from the functional plant *Andrographis paniculata*, on prostate cancer were evaluated using DU145 prostate cancer cells. The study demonstrated the effects of Andro on inhibiting prostate cancer cell proliferation via mitotic arrest and inducing the intrinsic apoptotic pathway. Co-

administration of taxifolin and Andro, by cleaving poly (ADP-ribose) polymerase and caspase-7 and -9, significantly enhanced the antiproliferative effect by enhancing mitotic arrest and apoptosis. Taxifolin plus andro also enhanced microtubule polymerization in vitro to form twisted and elongated spindles in cancer cells, ultimately leading to mitotic arrest. Depletion of MAD2, a component of the spindle assembly checkpoint (SAC), ameliorates mitotic block, leading to SAC activation, resulting in mitotic arrest. Overall, it has been suggested that combination therapy with taxifolin and andrographolide may activate SAC and thus disrupt microtubule dynamics [16].

Taxifolin was found to be a potential androgen biosynthesis blocking agent. Taxifolin significantly reduced basal, LH-stimulated, 8BR-stimulated, pregnenolone-mediated and progesterone-mediated androgen production by Leydig cells. Taxifolin also inhibited the activities of 3β -hydroxysteroid dehydrogenase and 17α -hydroxylase/ $17, 20$ -lyase enzymes in rat and human testes. These results indicate that taxifolin is a potential competitive inhibitor of these two enzymes, which may play an important role in the treatment of prostate cancer [17]. The anticancer effect of taxifolin against osteosarcoma was evaluated in human osteosarcoma cancer cell lines U2OS and Saos-2. Taxifolin treatment caused a dose-dependent antiproliferative effect by reducing the colony-forming efficiency. Tumor growth was significantly inhibited by taxifolin administration to U2OS xenograft-bearing nude mice. Taxifolin also promoted G1 cell cycle arrest and apoptosis induction in U2OS Saos-2 cell lines. Taxifolin treatment also increased the levels of serine/threonine kinase 1 (AKT), phosphorylated (p-Ser473) AKT, avian myelocytomatosis viral oncogene homolog v-myc (c-myc) and S-phase kinase-associated protein 2 (SKP-2) and significantly decreased

their expression levels. These results demonstrated that taxifolin impairs cell migration and invasion, which is largely associated with the reduction of SKP-2 overexpression, representing a potential competitor in the treatment of osteosarcoma [18].

It has also been reported that one of the anticancer effects of taxifolin is the prevention of skin cancer. In an *in silico* study, EGFR, phosphatidylinositol 3-kinase (PI3K), and Src were reported to be highly potential targets for taxifolin. In the study, taxifolin was shown to bind to EGFR and PI3-K through the ATP binding pocket and inhibit their kinase activity. Taxifolin was also associated with the suppression of UVB-induced EGFR and Akt activation and inhibited the downstream signaling cascade in JB6 P+ mouse skin epidermal cells. Taxifolin also attenuated UVB-induced COX-2 and prostaglandin E2 (PGE2) expression and inhibited EGFR-induced cellular transformation. Topical application of taxifolin suppressed the incidence, volume, and multiplicity of tumors in a mouse model, demonstrating the chemopreventive activity of taxifolin against UV-induced skin cancer by targeting EGFR and PI3K.[19] The anticancer activity of taxifolin against cicatricial cell carcinoma was evaluated, and taxifolin was shown to effectively inhibit the progression of cicatricial cell carcinoma through apoptosis and cell cycle arrest. Taxifolin also suppressed cicatricial cell carcinoma invasion by downregulating the expression of matrix metalloproteinases (MMPs) MMP-2 and MMP-9. Taxifolin was found to inhibit the growth of cicatricial cell carcinoma by inducing apoptosis and cell cycle arrest, and suppressing cell invasion [20].

The study evaluated the effect of taxifolin on human P-gp activity. The transporter inhibition activity was assessed in human P-gp stable

expression cells (ABCB1/Flp-InTM-293) together with the MDR reversal activity of taxifolin in human cervical carcinoma HeLa S3 cell line. The result of the study showed that ABCB1 expression was decreased dose-dependently by taxifolin treatment. Taxifolin also inhibited P-gp activity through non-competitive inhibition of rhodamine 123 and doxorubicin efflux, demonstrating significant resensitization of MDR cells to chemotherapeutic agents [21]. These results demonstrated that taxifolin as a P-gp modulator can be used as a synergistic treatment for multidrug-resistant cancers. The primary anticancer activity of taxifolin is related to the regulation of β -catenin and PI3K pathway, which modulates a number of cancer conditions. Taxifolin was found to primarily bind to two specific molecular targets in the cancer microenvironment, PI3K and EGFR, and regulate a number of signaling pathways and the expression of various proteins [19]. After analyzing all the above-mentioned data, taxifolin was found to play the most important role in the regulation and occurrence of EMT-Epithelial-mesenchymal transition (EMT) in various cancer models. Stem cells and EMT are mainly regulated by β -catenin and PI3K pathway, which causes the overexpression of stem cell markers such as SOX2 and OCT 4, as well as mesenchymal cell markers (N-cadherin and vimentin) [13, 22].

Activation of the PI3K/AKT pathway and accumulation of β -catenin in tumors have been shown to be associated with mutational inactivation of the p53 tumor suppressor, which often impairs cancer cell apoptosis [23, 24]. Furthermore, there is a cross-talk between Wnt and EGFR signaling in cancer; it has been suggested that Wnt ligands can activate EGFR signaling, whereas EGFR can activate β -catenin via the receptor tyrosine kinase-PI3K/Akt pathway; EGFR has been shown to form a complex with β -catenin and increase cancer cell invasion and metastasis [25]. Taxifolin

treatment results in inhibition of the expression of these proteins, preventing cancer progression, metastasis, cancer recurrence and promoting apoptosis. Moreover, regulation of these signaling pathways results in alteration of various other protein molecules involved in cancer development and progression in various tissues such as SKP2, LXR, survivin, ZEB2, c-Myc and others. Furthermore, chemotherapeutic activity of taxifolin is also associated with re-sensitization of resistant cancer cells by inactivating P-gp and inhibiting drug efflux from cancer cells. Taxifolin treatment results in inhibition of expression of these proteins, preventing cancer progression, metastasis, cancer recurrence and stimulating apoptosis. Moreover, regulation of these signaling pathways results in alteration of various other protein molecules involved in cancer development and progression in various tissues such as SKP2, LXR, survivin, ZEB2, c-Myc and others. In addition, the chemotherapeutic activity of taxifolin is also associated with re-sensitization of resistant cancer cells by inactivating P-gp and inhibiting drug efflux from cancer cells.

By acting as an EGFR PI3K receptor antagonist, taxifolin exhibits a variety of chemotherapeutic activities including antiproliferative, antiangiogenic, stem cell, and EMT regulation in various cancer model systems. Taxifolin also acts through the ARE formation process to regulate gene expression and exhibit excellent chemopreventive activity. Due to the low solubility of taxifolin (0.1% at room temperature), it is very difficult to absorb and metabolize in the body, which significantly limits its bioavailability and efficacy [26-28].

Taxifolin has also been shown to have limited ability to cross the blood-brain barrier (BBB), which limits its efficacy against intracerebral amyloid- β aggregation [29]. To enhance the bioavailability, solubility, and permeability of



taxifolin, micronization technology should be used to produce microscopic and homogeneous amorphous taxifolin nanoparticles [30]. Taxifolin is a potent flavonoid with therapeutic potential in the treatment of diseases, especially cancer-related inflammation. Flavonoids, plant-derived substances, are highly effective with fewer side effects than synthetic molecules in the treatment of various diseases, including cancer, making these chemicals popular among medicinal agents. Moreover, since they are widely available, acceptable, affordable, and safe for frequent use, these phytochemicals can be used as adjunctive therapies to prevent human cancer and slow down its progression.

Taxifolin has been extensively studied and has been found to have numerous pharmacological properties, including anticancer activity. Taxifolin's widespread biological activity has led to its being used in a variety of markets, from dietary supplements to chemotherapeutic drugs. A series of human studies on taxifolin's activity under various physiological conditions confirms its non-toxicity and excellent biological activity.

Əlavə məlumatlar.

Minnətdarlıq.

Bu məqalə milli səviyyədə aparılmış araşdırma və təhlil nəticəsində ərsəyə gəlmişdir; hazırlanmasında iştirak edən bütün həmkarlarımı təşəkkür edirəm.

Müəlliflərin töhfələri.

Konsepsiya və dizayn, Məlumatların əldə edilməsi, təhlili və ya təfsir, Əlyazmanın tərtibi, Əlyazmanın mühüm intellektual məzmun üçün tənqidi təftişi, Statistik təhlil, Məlumatların idarəedilməsi, Araşdırma, Əldə edilmiş dəstək, maliyyə və nəzarət: bütün müəlliflər bərabər qaydada. Müəlliflər yekun əlyazmanı oxuyub və təsdiq edib.

Maliyyələşdirmə.

Məqalənin hazırlanması məqsədilə aparılan təhlil və araşdırmalar üçün heç bir kənar maliyyə əldə edilməmişdir. Heç bir digər qurum və ya sponsor təşkilatlararasıdırmanın və ya tədqiqatın və ya təhlilin dizaynı və aparılmasında; məlumatların toplanması, idarə edilməsi, təhlili, məlumatların təfsirində, habelə əlyazmanın hazırlanması, nəzərdən keçirilməsi və ya təsdiqində heç bir rola malik olmayıb; əlyazmanın nəşrə təqdim edilməsi haqqında qərarların verilməsində iştirak etməmişdir.

Məlumat və materialların əlçatanlığı.

Təhlil zamanı istifadə olunan və/yaxud təhlil edilən məlumatlar (datalar) müəlliflərə və ya jurnalın redaksiyasına müraciət etməklə əldə edilə bilər.

Bəyannamələr.

Etik Komitənin icazəsi və məlumatlı razılıq.

Hər bir iştirakçıdan yazılı və ya uyğun olduqda şifahi məlumatlı razılıq alınıb. Etik Komitə (ATUJ, Azərbaycan) bu təhlili təsdiq edib.

Nəşr üçün razılıq.

Nəzərdə tutulmur.

Maraqların toqquşması.

Müəllif(lər) hər hansı maraqların toqquşmasını bəyan etməyiblər.

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TAXIFOLININ XƏRÇƏNG ƏLEYHİNƏ TƏSİRİNİN FARMAKOLOJİ ASPEKTDƏN ANALIZI

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Xülasə

Məqalədə müxtəlif xəstəliklərin, o cümlədən xərçəngin müalicəsində və qarşısının alınmasında mühüm rol oynayan taksifolin haqqında məlumat verilir. Taksifolin üzərində çoxlu tədqiqatlar aparılmış və xərçəng əleyhinə fəaliyyət də daxil olmaqla çoxsaylı farmakoloji xüsusiyyətlər aşkar edilmişdir. Taksifolin xəstəliklərin, xüsusən də xərçənglə əlaqəli iltihabın müalicəsində terapevtik potensiala malik güclü təsir göstərən flavonoiddir. Bu məqalədə taksifolinin antikanserogen təsirini sübut edən müxtəlif tədqiqatlardan bəhs edilir: döş xərçəngi, bağırsaq xərçəngi, prostat xərçəngi, dəri xərçəngi və s. Taksifolinlə müalicə xərçəngin inkişafının, metastazın, residivlərin qarşısını alır və apoptozu stimullaşdırır.

Açar sözlər: taksifolin, flavonoid, xərçəng əleyhinə təsir

АНАЛИЗ ПРОТИВОРАКОВОГО ДЕЙСТВИЯ ТАКСИФОЛИНА С ФАРМАКОЛОГИЧЕСКОГО АСПЕКТА

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Резюме

В статье дана информация о таксифолине который играет важную роль в лечении и предотвращении различных заболеваний, в том числе и рака. Было проведено множество исследований таксифолина, и были обнаружены многочисленные фармакологические свойства, в том числе и противораковая активность. Таксифолин является мощным флавоноидом с терапевтическим потенциалом при лечении заболеваний, особенно воспалений, связанных с раком. В данной статье говорится о различных исследованиях, которые доказывают антиканцерогенный эффект таксифолина: при раке молочной железы, толстой кишки, простаты, раке кожи и др. Лечение таксифолином предотвращает прогрессирование рака, метастазирование, рецидив и стимулирует апоптоз.

Ключевые слова: таксифолин, флавоноид, противораковый эффект