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A Comprehensive Review on Anti-Cancer Properties of Oxalis *corniculata*

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ABSTRACT

Oxalis corniculata L. belongs to the Family (Oxalidaceae), commonly known as "creeping woodsorrel, procumbent yellow sorrel, or sleeping beauty, " a common name. Oxalis corniculate contains several compounds like *β*-sitosterol, betulin, 4-hydroxybenzoic acid, ethyl gallate, methoxyflavones, apigenin, and 7-O-β-D-glucopyranoside were previously isolated from the whole plant of *Oxalis corniculata*. linn. The review reveals that wide ranges of phytochemical constituents have been isolated from the plant like flavonoids, tannins, phytosterols, phenol, glycosides, fatty acids, galactose-glycerolipid, and volatile oil. The leaves contain flavonoids, iso vitexin, and vitexin-2"- O- beta – D- glucopyranose. It is a rich source of essential fatty acids like palmitic, oleic, linoleic, linolenic, and stearic acids. It has been reported that the plant contains anti-inflammatory, anxiolytic, anticonvulsant, antifungal, antiulcer, antinociceptive, anticancer, antidiabetic, hepatoprotective, hypolipidemic, abortifacient, antimicrobial, and wound healing properties.

Keywords- anti-oxidant, anti-cancer, anti-inflammatory, and neuroprotective effect, Flavonoid, glycoside, phenolic compound.

I. INTRODUCTION

Oxalis corniculata Linn. (Family: Oxalidaceae) is well-known in India and is one of the most versatile medicinal plants having a wide spectrum of biological activity. It is commonly known as creeping wood sorrel, an excellent plant in nature with the composition of all the essential constituents required for the normal and good health of humans [1]. Based on the literature, the plant Oxalis corniculata Linn contains several phyto-components such as tannins, palmitic acid, a combination of stearic-8-oleic and linolenic acids. This plant also contains carbohydrates, glycosides, phytosterols, phenolic compounds, flavonoids, proteins, amino acids, and volatile oil [2]. Palmitic acid, 8-oleic acid, Linoleic acid, linolenic, stearic acids, tartaric acid, citric acid, acacetin, 7,4′- diOMe apigenin, P-hydroxybenzoic acid, Vanillic acid, syringic acid, isoorientin, isovitexin, swertisin, β-sitosterol, betulin, ethyl gallate, 5-hydroxy-7,8-dimethoxyflavone, 5-hydroxy-3′, 4′, 6, 7, 8-pentamethoxyflavone, 5-hydroxy-3, 6, 7, 4′-tetra methoxy flavone, apigenin these are the responsible for anticancer activity. Herb is a good appetizer, and removes kapha, vata, and piles; astringent cures dysentery and diarrhea, skin diseases, and quartan fevers. An infusion of the small leaves is externally used to remove warts and opacities of the cornea. The leaves are anti-inflammatory, refrigerant, and antiscorbutic[3]. The oxalis corniculate plant extract showed good anticancer (Hepatocarcinoma) activity based on in-vitro and in-silico analysis [4].

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II. SYNONYMS

- Various more names for **Oxalis corniculata Linn** include[5]
  - *Oxalis corniculata* L. subsp. *Corniculate*
  - *Oxalis corniculata* L. var. *atropurpurea* Planch.
  - *Oxalis corniculata* L. var. *corniculate*
  - *Oxalis corniculata* L. var. *microphylla* Hook.f.
  - *Oxalis corniculata* L. var. *repens* (Thunb.) Zucc.

III. COMMON NAMES [6]

Creeping lady's sorrel, creeping oxalis, creeping sorrel, creeping wood sorrel, creeping wood-sorrel, creeping wood sorrel, Indian sorrel, lady's sorrel, oxalis, procumbent yellow sorrel, procumbent yellow-sorrel, sheep sorrel, sorrel, sour grass, wood sorrel, yellow oxalis, yellow sorrel, yellow wood sorrel, yellow wood-sorrel, yellow wood sorrel.

IV. TAXONOMICAL CLASSIFICATION [7]

- Kingdom : Plantae
- Division : Magnoliophyta
- Class : Magnoliopsida
- Order : Oxalidales
- Family : Oxalidaceae
- Genus : Oxalis
- Species : corniculata
- Botanical Name : Oxalis corniculata Linn.

V. MORPHOLOGICAL CHARACTERS

Foliage: The leaves of the plants (Figure 1) are trifoliate, thin, and heart-shaped. The leaves have a distinct apical depression. The alternate leaves and leaflets along the length of the stem are reticulated[7]
Stem: The stem of the plant (Figure 2) is slender in shape and covered with soft short hairs. Internodes are 5-9 cm long. It is sour and smells sour.[8]
Root: The root (Figure 2) is dark brown, thin, branched, and soft; no smell or taste[6]
Flowers: Flowers (Figure 3) are 6-12 mm wide and have 5 yellow petals[28]
Fruits: Fruits (Figure 4) are capsules, 1-1.5 cm long, cylindrical, pointed and grooved [9]


Fig 1: Leaves


Fig 2: stem & root


Fig 3: flower


Fig 4: Fruit

VI. CHEMICAL CONSTITUENTS OF OXALIS CORNICULATA LINN

Oxalis corniculata Linn contains so many chemical constituents such as. [3]

- *β*-sitosterol

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- Betulin
- 4-hydroxybenzoic acid
- Ethyl gallate
- Apigenin

Were previously isolated from the whole plant of *Oxalis corniculata* Linn. The review reveals that wide ranges of phytochemical constituents have been isolated from the plant

- flavonoids, tannins, phytosterols, phenol, glycosides, fatty acids, galacto-glycerolipid and volatile oil. The leaves contain flavonoids
- Iso vitexin
- Vitexin-2"- O- beta – D- glucopyrunoside.
- It is a rich source of essential fatty acids like palmitic acid, oleic, linoleic, linolenic and stearic acids

These are all the chemical constituents of Oxalis corniculata Linn which have the following pharmacological activities

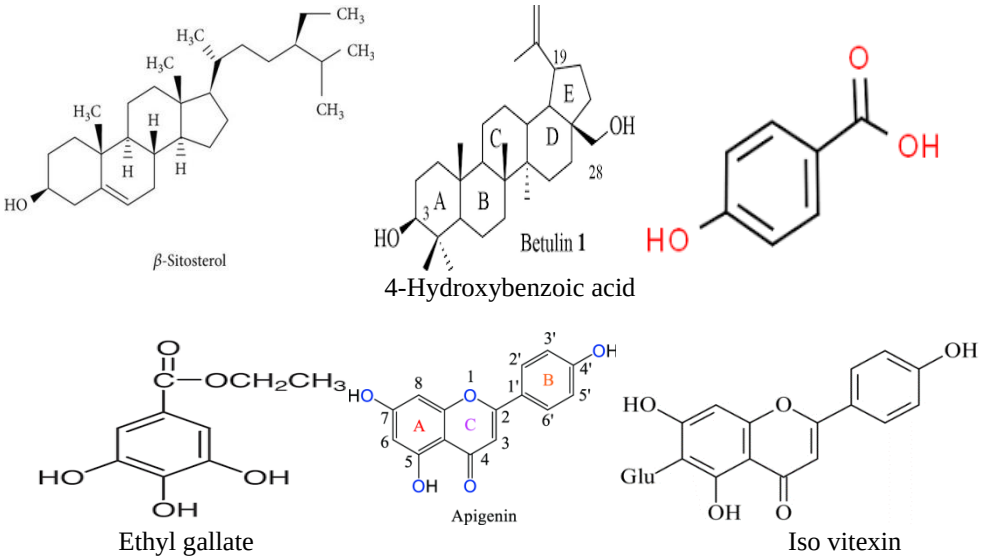

Figure 5 Chemical structure.

VII. *β*-SITOSTEROL

Beta-sitosterol is a sterol which found in plant (cholesterol is the main animal sterol). Sources include Oxalis corniculata Linn , rice bran, wheat germ, corn oil, soybeans, peanuts and their products, *Serenoa repens*, avocados, pumpkin seed, *Pygeum africanum*, and cashew fruit [10].

Among phytosterols, β-sitosterol is usually used for heart disease, hypercholesterolemia, modulating the immune system, and **prevention of cancer**, rheumatoid arthritis, tuberculosis, **cervical cancer**, hair loss, and benign **prostatic hyperplasia**. Furthermore, diverse biological activities whereby natural compounds or extracts were considered including trypanocidal and mosquito larvicidal, even neutralization of viper and cobra venom characteristics were recorded[11].

Mechanism of Action:

Many studies have shown that the anticancer effects of BS were related to the induction of apoptosis by blocking several cell signaling mechanisms [12]. For example, BS activates apoptosis in leukemic cancer cell lines by inducing G2/M arrest. Molecular studies have shown that BS induces endoreduplication in U937 and HL60 cells by promoting spindle microtubule dynamics through the Bcl-2 and PI3K/Akt signaling pathways[13]. BS is also effective against breast, prostate, stomach, and colon tumors by targeting different signaling pathways that induce apoptosis[14,15,16,17,18].

However, the effect of BS on NSCLC remains largely unknown, and the mechanism by which BS induces apoptosis requires further investigation. In this study, we showed for the first time that BS is effective against human NSCLC cells, and an investigation of the molecular mechanism showed that BS promotes apoptotic cell death in A549 and NCI-H460 cells through the accumulation and loss of ROS. ΔΨ. m to p53 activation. In addition, our data showed that BS inhibits Trx/TrxR1 protein expression, which in turn triggers ROS accumulation in A549 and NCI-H460 cells and activation of apoptotic cell death.

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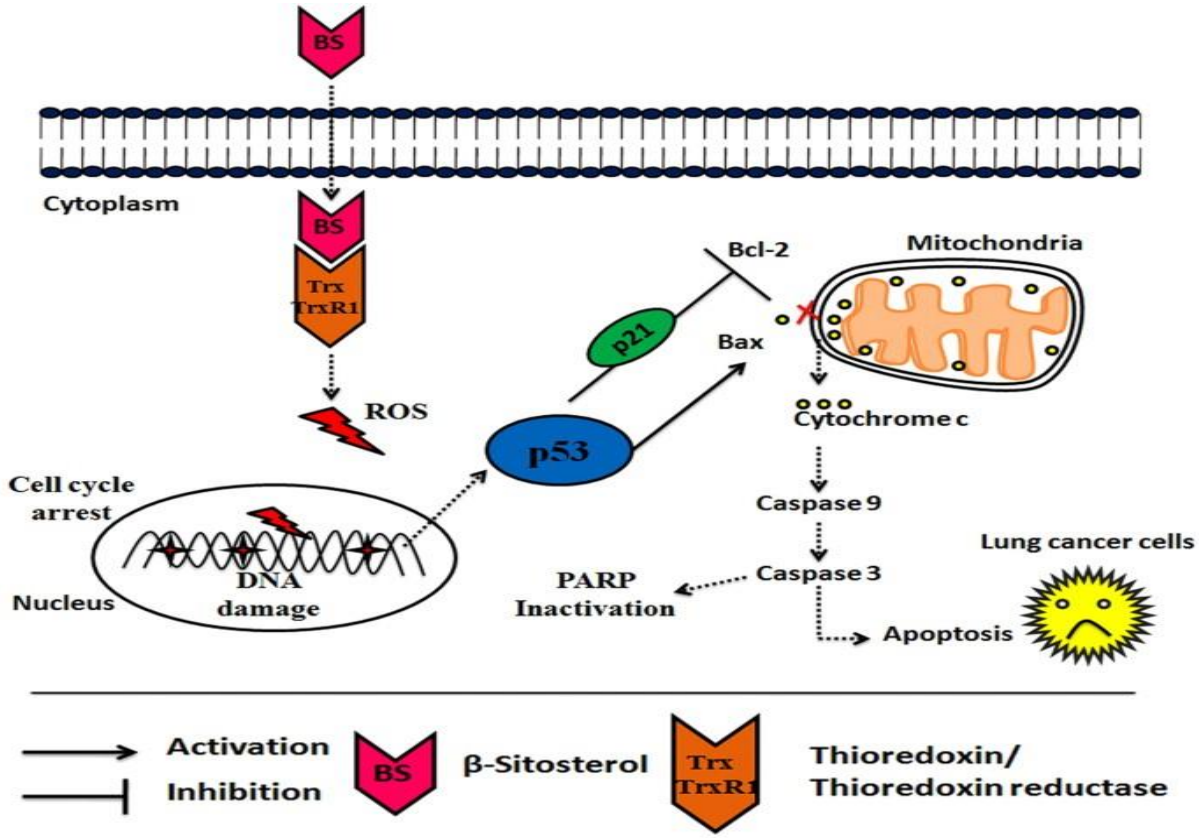

Figure 6. Schematic representation of the proposed signaling target of BS on NSCLC cells. BS induces excessive ROS production through inhibition of Trx/TrxR1. ROS accumulation causes DNA damage, p53/p21 activation and triggers the mitochondrial-mediated apoptotic cell death in A549 and NCI-H460 cells.

VIII. BETULIN [(BETULINIC ACID (BA)]

Betulinic acid was proven to induce apoptosis via the mitochondrial pathway by upregulation of the concentration of intracellular ROS level[20]. Interestingly, in cells preincubated with an antioxidative solution, the reaction was not observed[21]

Additionally, betulinic acid has not been reported to have negative effects on normal cells, which is often desirable in anti-cancer treatments. Its selective cytotoxicity was tested on various human tumor cell lines compared to doxorubicin, a cytostatic agent commonly used in cancer treatment. Betulinic acid exhibited up to 2–5 times lower cytotoxicity than doxorubicin.

Betulin itself is inactive when applied to specific cancer cell lines, such as melanoma, neuroblastoma, leukemia or epidermoid carcinoma. However, it can be easily converted to betulinic acid which exhibits anticancer properties. According to this research, the cytotoxic properties of betulinic acid increase with the decrease in intracellular pH[21]

Mechanism of Action:

Current studies showed that BA can mediate the anticancer effect via directly binding or interacting with target molecules, such as cannabinoid receptors and GRP78 [[23, 25, 26]. The anticancer mechanism of BA mainly includes the induction of mitochondrial oxidative stress, the regulation of cell cycle and the inhibition of angiogenesis[24, 27,28]. The direct targets of BA in cancer cells include nuclear factor-κB (NF-κB), specificity protein (Sp) transcription, VEGF and the ZBTB family[25, 29, 30, 31]. This section focuses on the mechanism of BA in the treatment of malignant tumour diseases through various targets.

Inhibition of the NF-κB signalling pathways

The NF-κB pathway is common in cancer and inflammation, which plays an important role in cell proliferation and apoptosis[32, 33]. NF-κB is composed of homo- and heterodimers of five members of the Rel family including p50, p52, p65, RelB, and c-Rel (Rel) [33].

Activation of NF-κB mainly triggers lipopolysaccharide and some inflammatory cytokines such as tumor necrosis factor and interleukin-1. Abnormal activation of IKK leads to increased phosphorylation and destabilization of IKB proteins, and **disruption** of the IKK complex promotes the release and transport of free NF-κB dimers. P65 activates the

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NF-κB complex to translocate into the nucleus, binds to specific DNA, and activates the corresponding promoter, inducing the overexpression of target genes, such as pro-proliferation and anti-apoptotic genes[30]. The activation of NF-κB occurs in the tumor microenvironment of most solid cancers and hematological cancers, which can promote epigenetic changes, epithelial-mesenchymal transformation, angiogenesis, metastasis, drug resistance, and immunosuppression[35,36] . (**Figure 8**).

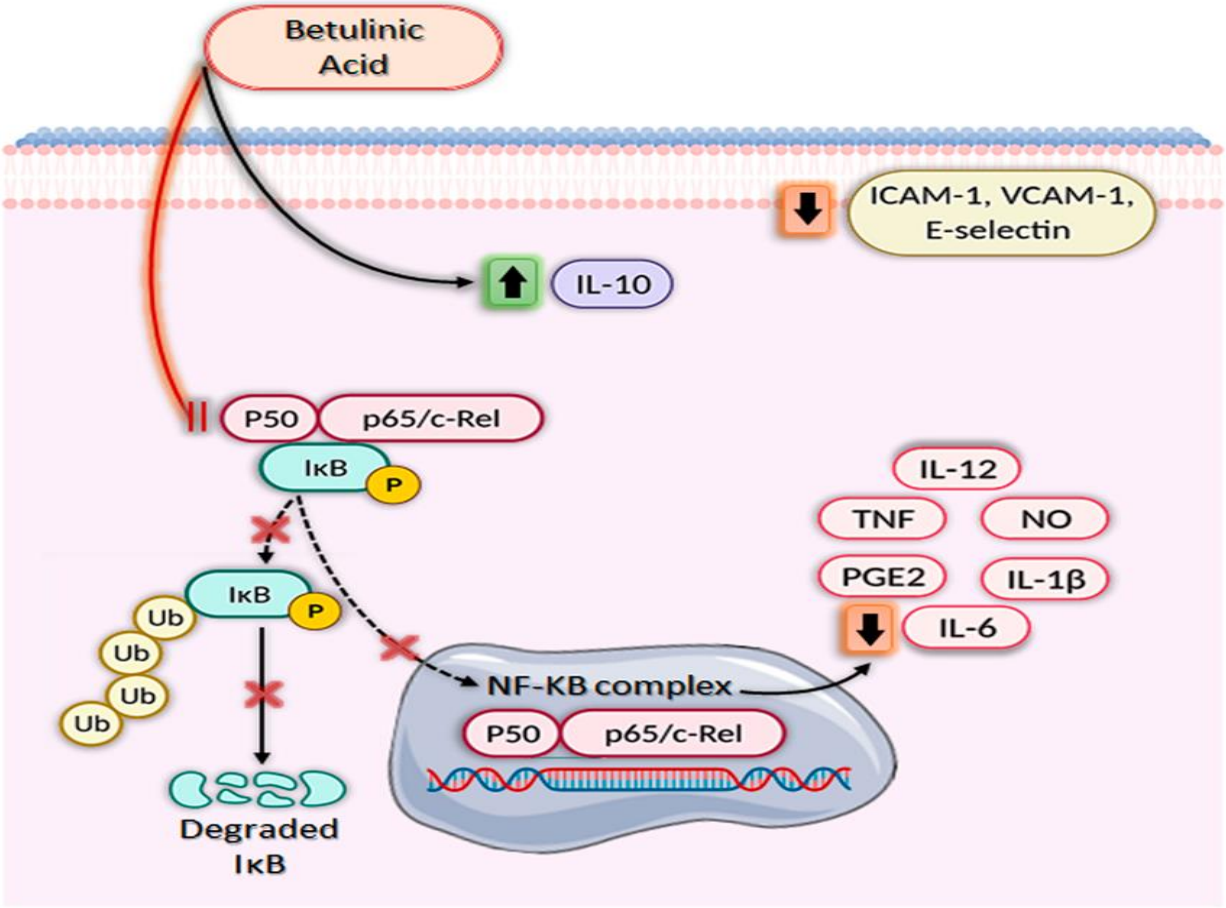

Figure 7. Immunomodulatory activities of betulinic acid in vitro. Betulinic acid (BA) has broad-spectrum anti-inflammatory activity, significantly increasing IL-10 production, decreasing ICAM-1, VCAM-1, and E-selectin expression and inhibiting nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), leading to downregulation of several pro-inflammatory genes. BA inhibits the NF-κB signaling pathway by preventing IκB phosphorylation and degradation by ubiquitination via the proteasomal degradation machinery. As a result, NF-κB is not activated and does not move from the cytoplasm to the nucleus, which prevents the transcription of pro-inflammatory mediators such as IL-1β, IL-6, IL-12, NO. PGE2, and TNF.

BA treatment can induce apoptosis of human prostate cancer cells by reducing the stability of IKK-α and its phosphorylation at serine 32/36, which limits NF-κB/p65 nuclear translocation and prevents DNA binding. The viability of androgen-responsive LNCaP cells and androgen-refractory DU145 cells were reduced by 30% and 50%− 55% after 12 h of treatment with BA (IC_{50} was 40 μM), respectively. The expression of NF-κB/p65 in the nucleus of both cell lines decreased in a dose-dependent and time-dependent manner [37]. In addition, NF-κB binding sites may exist in the vasodilator-stimulated phosphoprotein (VASP) promoter region, and BA can reduce the transcriptional activation of NF-κB by inhibiting the expression of p-p65, thus downregulating the expression of VASP and promoting the apoptosis of gastric cancer cells [38]. This mechanism **indicates** that BA **may act** as a **new** drug **in** the treatment of gastric cancer patients. BA significantly decreased NF-κB/p65 **levels** in multiple myeloma (MM) cells. **Morphological changes** including condensed nuclei and broken **chromatin** were observed in MM **cells after BA treatment.** When the IC_{50} was 40 μM, there was lower expression and lower nuclear binding levels of NF-κB/p65 in MM cells. It affected the expression of cyclin A, cyclin-dependent kinase 2, $P21^{Waf1/Cip1}$ and $p27^{Kip}$ and increased the percentage of S-phase cells and the number of apoptotic cells to exert its antiproliferative effect.

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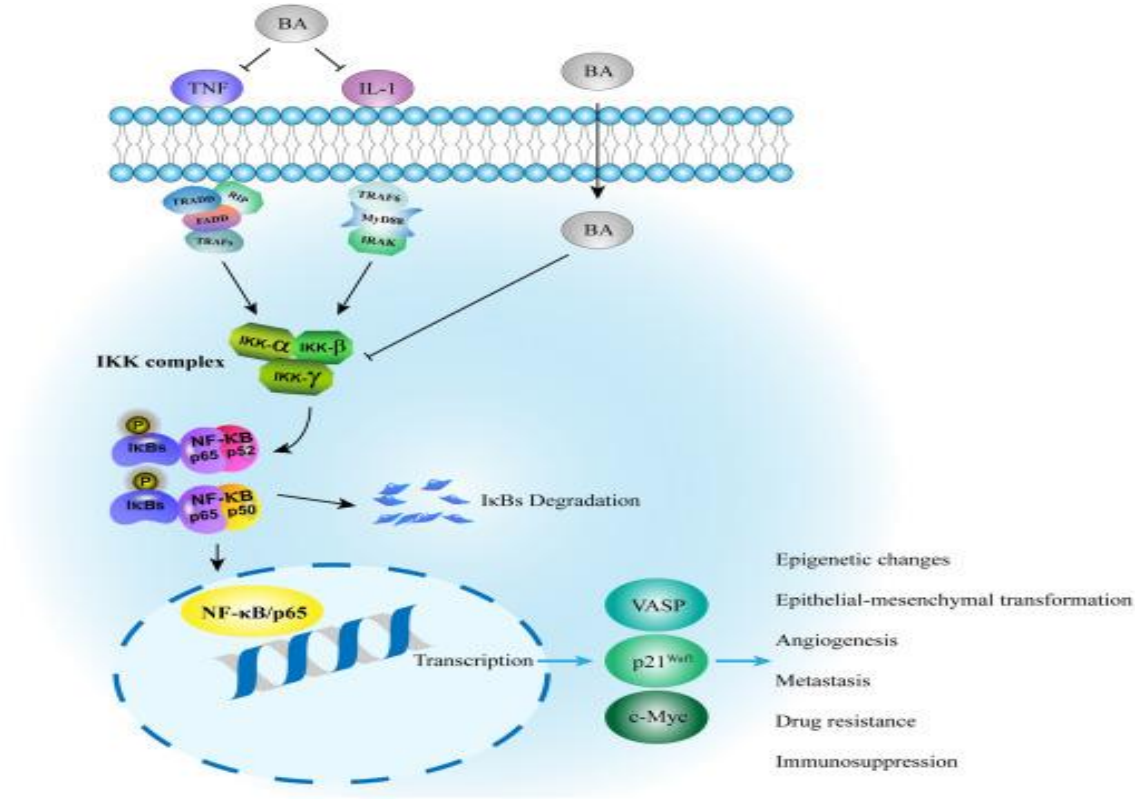

Figure 8:- Inhibition of NF-κB by BA to treat tumour disease. BA can directly affect the IKK complex and affect the activation of the IKK complex and the phosphorylation of IKB protein, which limits the nuclear translocation of NF-κB/p65 and inhibiting its binding to DNA. As a result, BA decreases the protein levels of VASP, $P21^{Waf1}$ and c-Myc, which can promote epigenetic changes, epithelial-mesenchymal transformation, angiogenesis, metastasis, drug resistance, and immunosuppression. BA also interferes with the NF-κB pathway by inhibiting the interaction between tumour necrosis factor and its membrane receptors. Abbreviations: IL-1, interleukin-1; TNF, tumour necrosis factor; VSAP, vasodilator-stimulated phosphoporotein.

IX. APIGENIN

Apigenin is a natural flavonoid commonly found in fruits and vegetables such as Oxalis corniculata Linn, parsley, celery, thyme, and oranges. The chemical structure of apigenin is similar to estrogen, and it may mimic estrogen. Therefore, extensive studies have shown that apigenin has potent antioxidant and anticancer activities in ER-positive and ER-negative breast cancer[39].

X. MODE OF ACTION

The biological and pharmacological activities of apigenin are diverse, and include anti-inflammatory, antioxidant, anticancer, anti-proliferative and anti-spasmodic. The cytotoxic effects of apigenin on various cancer cells, including osteosarcoma cells, prostate cancer, bladder cancer, liver cancer and colon cancer cells, have been well studied. Apigenin has been shown to potently inhibit apoptosis by altering signaling pathways, but the underlying mechanisms need further elucidation. Apigenin exhibited significant chemo-preventive properties by inhibiting the progression and metastasis of choriocarcinoma cells such as JAR and JEG3 by modulating the extracellular signal-regulated kinase 1/2 (ERK1/2) MAPK and PI3K-Akt signaling networks. The ERK1/2 phosphorylation was concentration-dependently enhanced by apigenin therapy and the effect was enhanced by the manifestation of ERK1/2 inhibitor and PI3K-Akt inhibitor[40]. It was reported recently that apigenin at various concentrations (0, 1, 10, 20, and 50 μmol/L) could dose-dependently induce G1 arrest in HepG2. Apigenin (20 μmol/L) incubated in the cells for 24 h could also inhibit cell cycle progression at the G1 phase through upregulation of cyclin D1 and downregulation of CDK4 *via* the p38 MAPK-p21 signaling networks[41]. Apigenin administered orally at 20 and 50 μg/mouse/d, for 20 weeks significantly reduced prostate tumor size and eliminated distant-site metastases to liver, lymph nodes and lungs in transgenic adenocarcinoma of the mouse prostate (TRAMP) mice.

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1. Apigenin: Inhibition and Prevention of Various Types of Cancer

Natural products or active compounds of medicinal plants show a role in cancer management through modulating various biological activities[42,43,44] In this regard, in vivo and in vitro studies have proven that apigenin plays a significant role in cancer management through modulating cell signalling pathways and the process of carcinogenesis (**Figure 9**). The role of apigenin in cancer inhibition and treatment of various cancers are described in Table 2 and **Figure 10**.

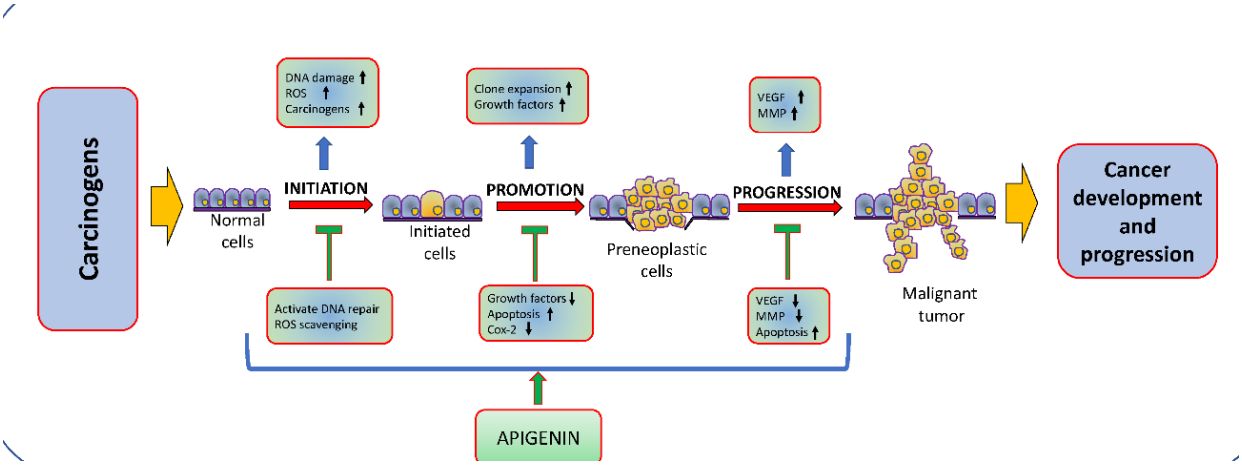

Figure 9. Anti-cancer activity of apigenin: Apigenin plays a significant role in cancer inhibition through inhibiting carcinogenesis by many various molecular interactions and processes, such as the regulation of the apoptotic machinery, aberrant cell signaling and oncogenic protein network.

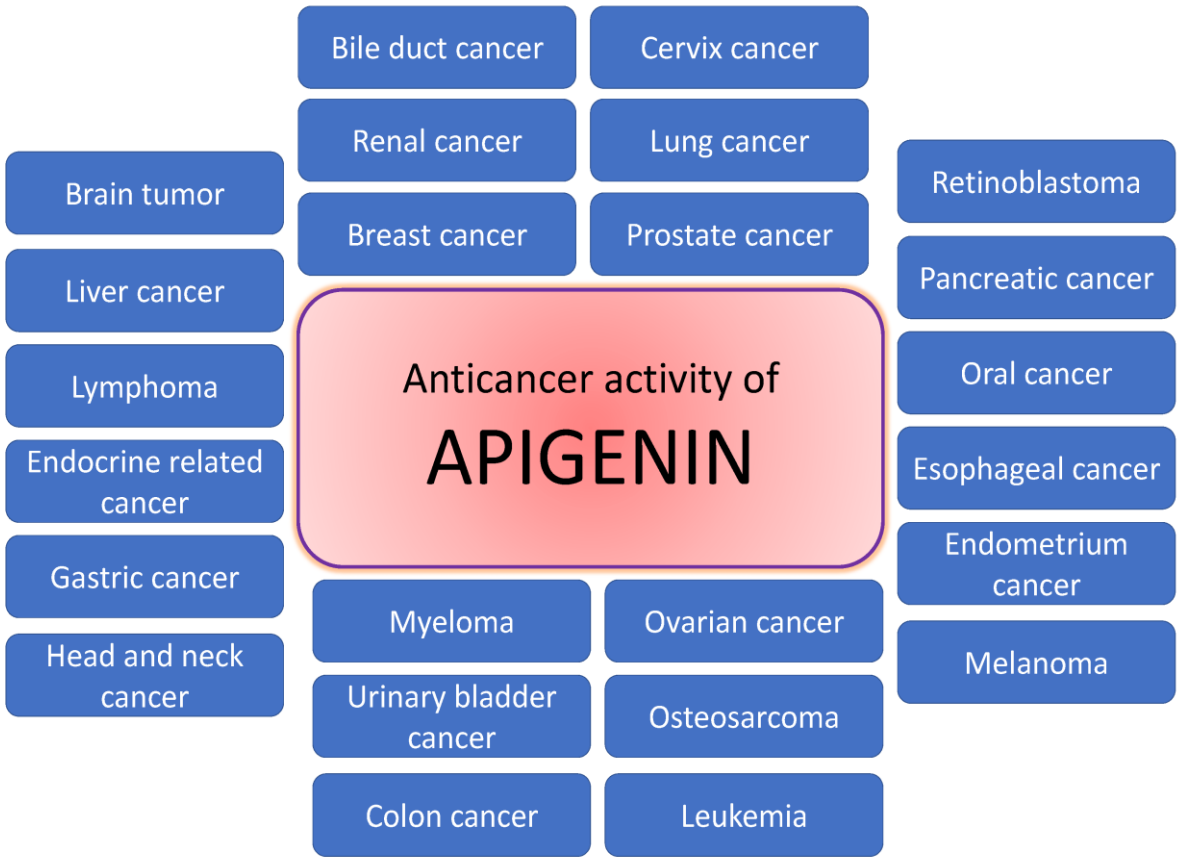

Figure 10. Protective effect of apigenin against different cancer types. Apigenin has been widely investigated for its anti-cancer activities and low toxicity. Apigenin has been shown to inhibit a number of human cancers both in vitro and in vivo through a variety of biological mechanisms.

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1.1 Breast Cancer

The effects of apigenin on estrogen-sensitive, antiestrogen-sensitive MCF7 breast cancer cells and two MCF7 sublines with acquired resistance to either fulvestrant or tamoxifen were investigated. The results showed that apigenin acts as both an estrogen and an antiestrogen in a dose-dependent manner. In addition, growth inhibition of cancer cells and antiestrogen-resistant sublines at high concentrations of apigenin and a combination of apigenin with either tamoxifen or fulvestrant showed synergistic growth inhibition. Taken together, the results indicated that apigenin has potential as a novel therapeutic agent against antiestrogen-resistant breast cancer due to its ability to target both ERα-dependent and -independent pathways[45].

Another experiment was performed to examine human cancer cell lines with different expression levels of neu/HER2 and found that apigenin has a strong anti-growth effect in breast cancer cells overexpressing HER2/neu. In addition, induction of apoptosis was also observed in neu/HER2-overexpressing breast cancer cells in a dose- and time-dependent manner.

In addition, apigenin complex inhibits Akt function in tumor cells. Apigenin directly inhibited PI3K activity while indirectly inhibiting Akt kinase activity and HER2/neu activity autophosphorylation and transphosphorylation resulting from depleting HER2/neu protein in vivo was also noticed[46].Mechanistic role of the caspase cascade in apigenin-induced extrinsic and intrinsic apoptosis, exploring the development of noncytotoxic anticancer drugs. Treatment with apigenin (1-100 µM) significantly inhibited the proliferation of human breast cancer cells in a dose- and time-dependent manner. In cells exposed to apigenin, this inhibition resulted in activation of apoptosis and secretion of cytochrome c. Apigenin **is** then cleaved **by** caspase-9, which is involved in mitochondria-mediated apoptosis. Furthermore, apigenin activated caspase-3, which works downstream of caspase-9, and caspase-3 activation was followed by dissociation of capases-6,7,8 [47]. The effects of 5-fluorouracil and apigenin combination therapy on proliferation and apoptosis were investigated in human breast cancer cells. Breast cancer cells exposed to ErbB2 overexpression were resistant to 5-fluorouracil. Surprisingly, co-administration of apigenin significantly reduced resistance. 5-fluorouracil plus apigenin significantly reduced cell proliferation compared to 5-fluorouracil alone. The findings suggest that 5-fluorouracil and apigenin work synergistically to limit cell growth and induce apoptosis via Akt activation and ErbB2 expression [48].

1.2 Cervical Cancer

Apigenin inhibited the growth of human cervical carcinoma cells through the apoptotic pathway. In addition, apigenin significantly reduced cancer cell viability, and apigenin-induced apoptosis in cervical cancer cells and induction of sub-G1 phase were confirmed. Apigenin-treated cancer cells were arrested in the G1 phase, which was associated with a significant increase in p21/WAF1 protein expression of the p21/WAF1 protein. WAF1/p21 induction appears to be transcriptionally accelerated and p53 dependent[49]

1.3. Ovarian Cancer

Apigenin, as a dietary flavonoid, has been found to have antitumor properties. Apigenin inhibits the proliferation and tumorigenesis of human ovarian cancer cells by inhibiting differentiation or DNA-binding protein 1 (Id1). Apigenin inhibited the expression of Id1 by promoting the transcription factor [50]. Another study in ovarian cancer found that apigenin induced early apoptosis at 24 h, while α-mangostin and doxorubicin triggered late apoptosis and necrosis at 72 h. Moreover, in cancer cells treated with apigenin, caspase-9 activity increased dramatically after 24 h, and both α-mangostin and apigenin arrested the cell cycle at the G2/M phase [51].

XI. ISO VITEXIN (ISOVITEX)

Various constituents and bioactive compounds are found in various plant materials such as leaves, stems, roots, flowers, fruits and seeds. The development of advanced and modern instrumental techniques facilitates the identification and characterization of particles and bioactive compounds. Phytocompounds are useful components (phenol, vitamin, amino acids, minerals) in various plant materials that have potential therapeutic effects on various biological activities, such as the treatment and prevention of cancer, cardiovascular disease and other chronic diseases.

Mode of action

Isovitex is a flavonoid that has antitumor effects in various types of cancer. However, it is unclear whether its mechanism of action in osteosarcoma (OS) involves epigenetic regulation or microRNAs, DNA methyltransferase 1 (DNMT1) or their targets. It was shown that isovitexin significantly inhibited survival, increased apoptosis, and decreased CD44, ALDH1, CD133, and ABCG2 mRNA levels in spheres derived from MG63 (MG63-SC) and U2OS cells (U2OS-SC). Isovitex has also shown in vivo anticancer activity against osteosarcoma. Isovitex inhibited tumor growth and reduced tumor size of U2OS-SC xenografts in nude mice, complemented by decreased CD133 protein levels, increased apoptotic rate, decreased proliferative cell nuclear antigen expression (PCNA), decreased DNMT1 expression and activity increased miR-34a and decreased Bcl-2 levels. Researchers identified Bcl-2 as a direct target of miR-34a. In addition, isovitexin showed synergistic effects with 5-aza-2'-deoxycytidine, miR-34a mimic, and ABT-263 in inducing apoptosis, inhibiting cell survival, downregulating CD44, ABCG2, CD133, and ALDH1 expression levels. mRNA and reduces the rate of

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spheroid formation in MG63-SC and U2OS-SC cell lines. These results suggested that isovitexin-mediated epigenetic regulation involved the DNMT1/miR-34a/Bcl-2 axis, which contributed to stemness inhibition and increased apoptosis in OS cell-derived spheres[52]

Molecular Mechanisms of Apigenin and Isovitexin Involved in Cancer Treatment

The natural compound's role in the prevention of cancer has been rationalized mostly via modulation of the cell signaling pathways[53,54]. Apigenin and isovitexin regulate different molecular pathways such as apoptosis, autophagy, cell cycle arrest and angiogenesis and regulate the expression of different genes**.** (**Figure 11**)

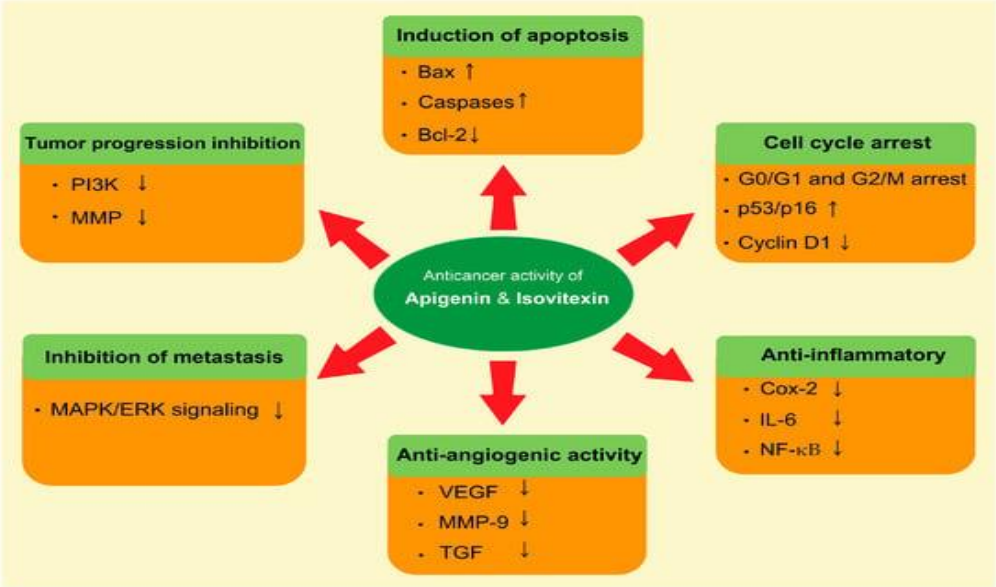

Figure 11. Role of apigenin and isovitexin in cancer management through the modulation of different signaling pathways. The complex cell nature of the tumor is characterized by a number of molecular interactions and mechanisms. Abbreviations: Bax, BCL2-associated x protein; Bcl-2, B-cell lymphoma 2; Cox-2, cyclooxygenase-2; ERK, extracellular signal-regulated kinase; IL-6, interleukin-6; MAPK, mitogen-activated protein kinase; MMP, matrix metallopeptidase; NF-κB, nuclear factor-κB; PI3K, phosphatidylinositol-3 kinase; TGF, transforming growth factor; VEGF, vascular endothelial growth factor. Downward arrow ↓, decrease; Upward arrow ↑, increase.

Apigenin has a noteworthy role in apoptosis induction. It increased apoptosis through the activation of the MAPK signaling pathway, as well as through the reduction in sulfiredoxin expression[55]. Apigenin was demonstrated to increase poly-(ADP-ribose) polymerase (PARP) proteolytic cleavage and to trigger a rapid enhancement of caspase-3 activity. PARP cleavage and DNA fragmentation showed that apoptosis increased after apigenin treatment. These effects were related to a modification in the ratio of Bax/Bcl-2 in favor of apoptosis[56]. Apigenin inhibited lung tumor cell proliferation and vascular endothelial growth factor (VEGF) transcriptional activation in a concentration-dependent manner.. The mechanism of apigenin-suppressed VEGF transcription was suggested to occur via the reduction in HIF-1α[57].

Apigenin has been shown to increase autophagy and cell death in primary effusion lymphoma, in addition to significantly reducing ROS. Moreover, apigenin has been reported to activate p53, which enhances catalase and suppresses STAT3, as evaluated via the silencing of p53[58].

Apigenin administration resulted in G2/M phase cell cycle arrest. The levels of p53 and its protein $p21^{CIP1/WAF1}$ are enhanced when the cells were treated with apigenin[59]. Additionally, apigenin showed a role in the enhancement of autophagy and apoptosis through the inhibition of the PI3K/Akt/mTOR pathway[60]. Apigenin is also a potential chemopreventive agent that inhibits tumor growth and metastasis via the regulation of the ERK1/2 MAPK and PI3K/Akt signaling pathways[61]. Additionally, through inhibiting the Wnt/β-catenin pathway, apigenin considerably decreased tumor cell proliferation, invasion, migration, and organoid growth[62].

Different authors have indicated that isovitexin induces autophagy and apoptotic cell death of different cancer cells via the upregulation of PARP, Bax, and MAPK, and the downregulation of ERK1/2 and Bcl-2, involving decreased phosphorylation of PI3K, Akt, and mTOR in tumor tissues[63,64]. Experimental findings have demonstrated that isovitexin has anti-inflammatory and antioxidant properties, which influence multiple signaling pathways related to tumor progression and metastatic growth. In addition, isovitexin arrested tumor cell growth at the G2/M cell cycle phase and consequently led to apoptosis induction. This induction of apoptosis appears to be mediated by caspases activation[63].

XII. CONCLUSION

The ethanol-botanical, phytochemical, and pharmacological information about Oxalis corniculata (L) is gathered from the previously published information. The plant has a wide range of pharmacological actions that have been reported, making it effective in treating a wide range of diseases. It was noted because it had a small number of phytoconstituents linked to a limited number of biological functions. Therefore, it is necessary to identify the other phytoconstituents that can be employed as lead compounds to create innovative drugs with potent therapeutic effects. It is crucial to isolate and characterize phytoconstituents, to understand the mechanism of action of isolated compounds, and to conduct clinical trials on substances. The importance of medicinal plants in basic healthcare has grown in the current global environment. Consequently, the information supplied may be useful for requires additional investigation to screen the substances accountable for various bioactivities and to clarify the molecular mechanism of action.

ACKNOWLEDGEMENT

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DATA AVAILABILITY STATEMENT

All of the data supporting the findings of the presented study are available for corresponding author on request.

DECLARATIONS

Conflict of interest: The authors declare that they have no conflict of interest.
Author Contributions: All authors Participate Equally.
Ethical approval: The manuscript has not been published or submitted to another journal, nor is it under review.

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