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Anticancer Activity of Diosgenin Steroidal Saponin

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ABOUT THE STUDY

Diosgenin is a steroidal sapogenin that has been studied extensively for its intriguing bioactivity. As a result, a significant number of researches have been conducted to investigate its possible use in a wide range of medical disorders. Indeed, this compound is known to have anti-inflammatory and antioxidant properties and can be beneficial in a variety of conditions, including blood and cerebral disorders, allergic diseases, diabetes and obesity, menopausal symptoms, and skin ageing; it can also protect against cardiovascular diseases (such as thrombosis and atherosclerosis) and, most importantly, cancer. For medicinal chemists, developing cancer treatments from steroidal compounds has been an appealing option, and numerous potent molecules have emerged.

Several preclinical studies have looked at the effects of diosgenin as a chemopreventive/therapeutic agent against tumours of various organs, demonstrating the molecule's significant interest as a possible antitumor drug. In reality, diosgenin's anticancer action has been researched in a variety of tumour cell lines, and it has been proven that its bioactivity is dependent on both cell type and concentration. Diosgenin, for example, has antiproliferative properties in prostate cancer (PC-3 and DU-145 cells), colon carcinoma (HCT-116 and HT-29 cells), erythroleukemia (HEL cells), squamous carcinoma (A431, Hep2, and RPMI 2650 cells), hepatocellular carcinoma (HepG2 and HCC cells), gastric cancer (BGC-823 cells), lung cancer (A549 cells), and breast cancer (MC (K562 cells). Furthermore, various studies have linked diosgenin's anticancer strategies to a modulation of multiple cell signalling events involved in cell growth/proliferation, differentiation, epithelial-mesenchymal transition migration, and apoptosis, as well as oncogenesis and angiogenesis.

Diosgenin appears to be important in causing apoptotic cell death and preventing malignant transformation throughout various stages of carcinogenesis. Diosgenin's anticancer effects have been shown to be mediated through p53 activation, immunological modulation, cell cycle arrest, modulation of caspase-3 activity, and activation of the transcription STAT3 signalling pathway, among other mechanisms. In this context, studies have shown that diosgenin inhibits osteosarcoma cell proliferation by inducing apoptosis and cell cycle arrest in the G1 phase, as well as inhibiting the proliferation of breast cancer cells (MCF-7 cells) by inducing the proapoptotic p53 protein and increasing caspase-3 levels. Furthermore, diosgenin inhibits the proliferation of PC-3 human prostate cancer cells in a dose-dependent manner, limiting cell migration and invasion by decreasing matrix metalloproteinase production, indicating that this chemical has antimetastatic potential. Diosgenin inhibits the growth of the A549 lung cancer cell line and downregulates hTERT gene expression in these cells in a time-dependent way due to its antioxidant effect. As a result, this sapogenin could be a promising treatment option for lung cancer. COX-2 overexpression was linked to diosgenin-induced apoptosis in HEL cells (human erythroleukemia cell line). Additionally, a rise in the Bax/Bcl-2 ratio, PARP cleavage, and DNA fragmentation accompanied the activation of apoptosis.

The suppression of NF-kappa B nuclear binding and p38 MAPK activation are implicated in the diosgenin-mediated signal cascades for inducing/regulating DNA fragmentation in COX-2 deficient K562 cells. Furthermore, diosgenin stimulates autophagy as well as reactive oxygen species (ROS) formation and suppression of the mammalian target of rapamycin (mTOR) signalling pathway in human chronic myeloid leukaemia cells (K562 and BaF3-WT). Further research revealed that inhibiting autophagy increased the apoptosis mediated by diosgenin. Diosgenin suppresses the STAT3 signalling pathway in human hepatocellular carcinoma (HCC) cells, resulting in cell growth inhibition and chemosensitization, as well as cell cycle arrest at the G1 phase and apoptosis via caspase-3 activation and PARP cleavage. This steroid promotes apoptosis in HepG2 hepatic cells via the Bcl-2 protein family (Bcl-2, Bax, and bid), which is mediated by the mitochondrial/caspase 3-dependent pathway.

Diosgenin also possesses antimetastatic properties; for example, it has been shown that by reducing Vav2 protein activity, it can block the migration of human breast cancer MDA-MB-231 cells, at least partially. Angiogenesis, which is dependent on the action of angiogenic factors such as integrin and VEGF, is also a critical mechanism for the development, invasiveness, and metastasis of solid tumours. In this regard, diosgenin has been shown to limit angiogenesis by inhibiting VEGF expression in PC-3 cells in a dose-dependent way, implying that this steroid can restrict angiogenesis by interfering with this factor. All of these findings point to the compound's potential as a new treatment agent for a variety of cancers. As a result, much work has gone into determining the role of diosgenin and some of its chemical analogues,

as well as combinations of diosgenin and other bioactive compounds, in modulating the growth and proliferation of various types of human tumours, as well as determining its potential mechanism of action. As an example, the combination of diosgenin and thymoquinone shows synergistic antiproliferative and apoptotic effects on squamous cell carcinoma (SCC), and so could be a unique technique for the development of possible antineoplastic therapeutics against SCC.

CONCLUSION

Diosgenin, a steroid saponin present in a variety of plant species, has been identified as a promising bioactive biomolecule with a variety of essential medical qualities, including hypoglycaemic, antioxidant, anti-inflammatory, and anticancer capabilities. Indeed, diosgenin-functionalized iron oxide nanoparticles and hollow manganese ferrite nanocarriers containing tamoxifen and diosgenin have been produced as possible breast cancer therapeutics.