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Review article

Apoptosis-Inducing Potential of Gloriosa superba *Root Constituents via Mitochondrial and Death Receptor Pathways in Human Cancers: A Systematic Review*

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Abstract

Gloriosa superba, known as Glory Lily, has been appreciated for many years for its active compounds, colchicine and gloriosine and is being considered for possible roles in cancer treatment. This paper reviews how G. superba root extracts cause apoptosis and explores the mechanisms behind this in several forms of human cancer. Recent research done in the laboratory and on animals suggests that these plant chemicals can regulate both the inner (mitochondrial) and outer (receptor-linked) processes that lead to apoptosis. Numerous studies have found that cancer cells are induced to die when pro-apoptotic proteins such as Bax, p53, caspase-3 and -9 are increased and anti-apoptotic proteins such as Bcl-2 are reduced. In the case of breast, colon and cervical cancer cell lines, the root extracts of G. superba have caused DNA breakdown, the collapse of mitochondrial membranes and the release of cytochrome c. Some other studies reveal that modulating death receptors such as Fas and TRAIL-R also leads to apoptosis in multiple ways. Even with the exciting results, challenges related to the standardisation of extracts, dose-related adverse effects and conducting clinical tests have not been solved. Priority should now be given to extracting individual compounds, developing targeted delivery methods and conducting detailed preclinical testing. The investigation demonstrates that G. superba root-derived chemicals may be useful in apoptosis-targeted cancer therapy.

Keywords: *Apoptosis-Inducing Potential ,* Gloriosa superba *Root Constituents , Mitochondrial*

Introduction

Across the world, cancer is a main reason for sickness and death, leading to more than ten million deaths annually [1]. While chemotherapy, radiation therapy and immunotherapy work well in some cases, they can cause serious side effects, making treatment very costly and leading to the drugs' stopping being effective over time. So, doctors and researchers are turning more often to natural products and phytochemicals from traditional medicinal plants in the hope of finding better, safer therapies. Gloriosa superba L., or Glory Lily, has a long history in Ayurveda and other traditional systems because it is considered to have powerful medicinal effects.

The Colchicaceae family includes Gloriosa superba, a native flower found in Asia and Africa near the tropics and subtropics. For years, many have used the root tubers of this plant to aid in treating ailments such as inflammation, ulcers and infections caused by parasites. Researchers have discovered that part of G. superba, especially its root, contains alkaloids that show strong anticancer properties [2]. Because they affect the cell cycle and induce programmed death known as apoptosis, they are interesting choices for cancer treatment.

Apoptosis serves to control how cells die and to get rid of those that are damaged or unneeded in an organ. When this pathway is disturbed in cancer, cells do not die as they should and instead reproduce rapidly [3]. The critical methods by which apoptosis occurs include the intrinsic or mitochondrial way, as well as the extrinsic or death receptor method. Most anticancer agents help cells use these pathways to cause apoptosis in cancer cells instead of healthy cells.

It appears that compounds from G. superba's roots may start both natural and forced death signals in or trigger the destruction of breast, colon and cervical cancer cells [4, 5]. These results are caused by disturbing the mitochondrial membrane, sending cytochrome c into the cytoplasm, triggering sequences of caspase cascade activation and controlling proteins such as Bcl-2, Bax and p53. Moreover, when Fas and TRAIL receptors are upregulated, they activate the death receptor pathway by turning on caspase-8.

While there are many promising results from G. superba research, it cannot be widely used clinically because of worries about the narrow safety margin of colchicine. A growing number of phytochemical extraction techniques, better formulation approaches and improved delivery techniques are handling these issues over time. This study aims to summarise the effects of G. superba root extracts on apoptosis, focusing on their influence on cancer cells, the experiments performed, the targets they harm, their safety features, and their potential future role in combating cancer.

Phytochemical Profile of Gloriosa superba

Traditional systems such as Ayurveda and Siddha often value Glory Lily or Kalihari, scientific name Gloriosa superba L., due to its potent pharmacological features, found mostly in these systems. Many of the phytochemicals in root tubers are alkaloids and steroidal saponins, and some have been linked to fighting cancer [6]. All, Fig. 1 showsthe most attention to colchicine and gloriosine since they can stop fast-dividing cells.

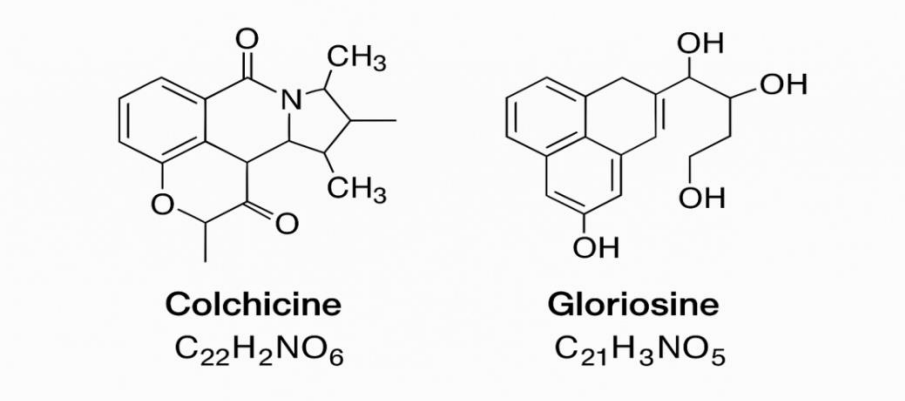

Figure 1 Colchicineand Gloriosine

Extraction and Standardisation

The way G. superba is extracted and its principles are concentrated greatly determine its effects. Traditionally, Soxhlet extraction with either ethanol or methanol is typical and newer approaches, like extracting with microwave and supercritical fluids, result in a better quality of the extract [7]. Colchicine and gloriosine are reliably measured using HPLC and GC-MS in preclinical studies to make sure they are used safely and correctly.

Major Bioactive Constituents

Colchicine, which is well recognised and discovered in Colchicum autumnale, is abundant in G. superba tubers. Attaching to tubulin, it blocks cells from progressing in metaphase and leads to programmed cell death [8]. The way it works resembles what mitotic inhibitors in chemotherapy do, such as paclitaxel and vinblastine. Another tropolone

alkaloid called gloriosine has shown possible anticancer effects by affecting apoptosis, but further investigation of it is needed.

Among its other ingredients are superbine, β-sitosterol, colchicoside and flavonoids. Because of their anti-inflammatory, antioxidant and cytotoxicity, these compounds help the extract treat many conditions [9]. Steroidal saponins in the roots help other cytotoxic agents by making the cell membranes in cancer cells more permeable in the table.1 shows the major bioactive compounds present in the glory lily root.

Table: Phytochemicals in Gloriosa superba Root

Phytochemical	Chemical Class	Molecular Formula	Known Biological Activity
Colchicine	Tropolone alkaloid	$C_{22}H_{25}NO_6$	Antimitotic, induces apoptosis via tubulin binding
Gloriosine	Alkaloid	$C_{22}H_{25}NO_6$ (isomer)	Apoptotic, similar to colchicine, is less studied
Superbine	Alkaloid	$C_{17}H_{24}N_2O_3$	Cytotoxic, a potential apoptosis inducer
β-Sitosterol	Phytosterol	$C_{29}H_{50}O$	Antioxidant, anticancer, and membrane stabilisation
Colchicoside	Glycoside of colchicine	$C_{27}H_{41}NO_{11}$	Less toxic than colchicine, anti-inflammatory
Flavonoids	Polyphenolic compounds	Various	Antioxidant modulates apoptosis-related signalling
Saponins	Steroidal saponins	Various	Permeabilizes cell membranes, supports drug delivery

Traditional Versus Modern Use

Usually, people use the raw root to make remedies for gout, arthritis, snake bites and tumours. Even so, you should take care in processing and measuring this drug because it is highly toxic. At present, emphasis in pharmacology is placed on separating pure chemicals and creating semi-synthetic variants that are safer and have better benefits.In general, the many chemical substances in G. superba tubers help the plant in various ways. The fact that they can prevent cell growth and survival explains why the plant has potential as an anticancer medicine [10].

Role of Glory Lily Root Extract and Apoptosis Pathways in Cancer

The molecular mechanism by which Gloriosa superba root extract (GLRE) induces apoptosis, particularly through the mitochondrial (intrinsic) pathway. Under carcinogenic stress, the anti-apoptotic protein Bcl-2 is upregulated, preventing the release of cytochrome c (Cyto-C) from mitochondria, thus inhibiting apoptosis. However, GLRE, through the activation of p53, downregulates Bcl-2 and upregulates the pro-apoptotic protein Bax. This Bax protein promotes mitochondrial outer membrane permeabilisation, facilitating the release of Cyto-C into the cytosol.

Once in the cytosol, Cyto-C binds to Apaf-1(apoptotic protease-activating factor-1) in the presence of ATP, forming the apoptosome complex**.** This complex activates procaspase-9, which in turn activates caspase-9. Caspase-9 then cleaves and activates procaspase-3 into caspase-3, a key executioner of apoptosis. Caspase-3 drives DNA fragmentation and cell death.

Figure 2 shows that GLRE favours apoptosis by enhancing the expression of p53, Bax, Apaf-1, and caspases, while inhibiting Bcl-2. This pro-apoptotic action of GLRE suggests its potential role as an anti-cancer agent, especially in inducing apoptosis in cancer cells via mitochondrial signalling pathways. The cascade results in controlled cell death, thereby inhibiting tumour growth.

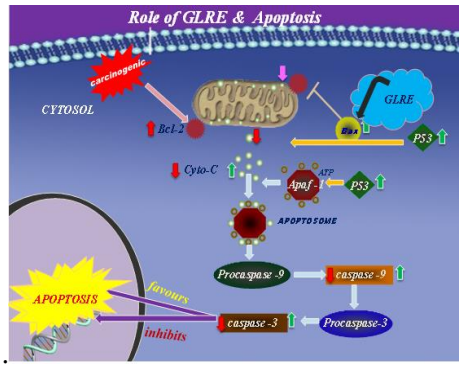

Figure 2 Role of Glory Lily Root Extract andApoptosis Pathways in Cancer

Intrinsic (Mitochondrial) Pathway

The operation of the intrinsic pathway depends on healthy mitochondria, and the Bcl-2 family is mainly responsible for its regulation. Bax and Bak, which promote programmed cell death, trigger the opening of the mitochondrial outer membrane and cytochrome c is released into the cytosol as a result. This release is prevented by anti-apoptotic members, including Bcl-2 and Bcl-xL, which help keep mitochondrial function normal under average conditions. As soon as it enters the cytoplasm, cytochrome c joins with procaspase-9 and Apaf-1, which form the apoptosome that activates caspase-9. As a result, the initiator caspase sets off caspase-3 and other downstream effectors, which damage the DNA, cause the cell membrane to bleb, and ultimately leadto the death of the cell. Apoptosis induced by chemotherapy and natural compounds such as those from Gloriosa superba often works through the mitochondrial pathway.

Extrinsic (Death Receptor) Pathway

The extrinsic pathway is turned on when a death ligand meets its cell surface receptor. Important receptors identified are Fas (CD95), tumour necrosis factor receptor (TNFR) and TRAIL receptors (DR4 and DR5). Ligand attachment to these receptors attracts adaptor proteins such as FADD, which activate procaspase-8. Either way, activated caspase-8 delivers or intensifies the apoptotic signal by either cleaving effector caspases itself or by cleaving Bid, a BH3-only Bcl-2 family member, to stimulate the signal from mitochondria. Immune cells, including cytotoxic T lymphocytes, use the extrinsic pathway to combat cancer, and researchers are studying TRAIL-based treatments to selectively cause tumour cells to die.

Role of p53 and Mitochondrial Dynamics

When something like DNA damage or increased activity of oncogenes happens to a cell, p53 or the ‘guardian of the genome,’ helps initiate apoptosis. When p53 is activated, it triggers Bax, Puma and Noxa to help push cells toward a death considered mitochondrial apoptosis. Apart from its transcription duties, p53 can engage with Bcl-2 group proteins in the mitochondria to help start MOMP.

How many and how apoptotic cells are, also depends on the mitochondrial processes of fission and fusion. Drp1 and similar proteins help mitochondrial fission, and too much mitochondrial fission increases the chances of apoptosis, while too much fusion protects against cell death. Increasing research is showing that mitochondria become altered in cancer cells, using this change to bypass apoptosis. Interfering with mitochondrial division and fusion offers a new way to make tumour cells more responsive to substances that promote apoptosis.

Colchicine, naturally found in Gloriosa superba, can activate different types of apoptosis either by stimulating the p53 protein or making mitochondria more permeable. Because of these mechanisms, apoptosis pathways offer exciting new targets for herbal drugs used in cancer treatment.

Apoptosis Induction by Gloriosa superba in Various Cancers

Because of its colchicine, Gloriosa superba’s root extract is valuable for stimulating cancer cell apoptosis. In cancer, the regular process of apoptosis is no longer controlled properly. In experiments, G. superba has been confirmed to use intrinsic and extrinsic pathways to restore apoptotic processes in breast, colon, cervical, liver and pancreatic cancers, both in a laboratory and in vivo.

Breast Cancer Models: MCF-7 and MDA-MB-231

Tissues from G. superba produced powerful cytotoxic effects on MCF-7 and MDA-MB-231 breast cancer cell lines. Pro-apoptotic Bax was highly expressed by MCF-7 cells while Bcl-2 was reduced, suggesting that an intrinsic pathway was involved [11]. Cell shrinkage, condensed chromatin and DNA fragmentation were features of apoptosis, along with an increased level of caspase-3, in triple-negative MDA-MB-231 cells. How toxic these effects were depended on how much each drug contained, and colchicine, present in all three compounds, disrupted microtubules and turned on apoptosis signalling [10].

Colon Cancer Models: HT29 and HCT116

Cells from HT29 (mutant p53) and HCT116 (wild-type p53) species were tested with G. superba extracts in colon cancer experiments. The use of colchicine-rich extracts led to a G2/M phase arrest and impaired mitochondrial activity, which resulted in cytochrome c release and the activation of caspase-9 [12]. In particular, the high apoptosis seen in HCT116 cells may be due to the fact that p53 is intact and able to increase the expression of Puma and Bax, both of which encourage death in cells. In HT29 cells, ROS and ER stress caused apoptosis even though Omi cleavage was reduced. It appears that G. superba can work around problems related to p53 inactivation, which often develops in colon cancer treatment.

Cervical Cancer: HeLa Cells

Both the extrinsic pathway and the intrinsic pathway were identified as ways that G. superba extracts trigger apoptosis in HeLa cells. Nuclear breakdown was observed with DAPI, and flow cytometry identified that apoptosis was taking place among annexin V-detected cells. Therapy led to higher Fas and TRAIL receptor expression, and it turned on caspase-8 and caspase-3 [13]. Signals showed that as mitochondrial polarisation dropped, cytochrome c was released, a sign of collaboration between the two signalling pathways. Importantly, treatment raised p53 levels, which may be due to a reduction in the HPV oncogenes E6 and E7. That G. superba can once again cause apoptosis highlights its capacity to identify virally altered cells.

Hepatic and Pancreatic Models

The roots of G. superba led to colchicine exposure, which prompted oxidative stress, lowered the mitochondrial potential and activated caspase-3 in HepG2 hepatocellular carcinoma cells. A decrease in Survivin made cells more sensitive to apoptosis [14]. When resistance to apoptosis is common in pancreatic cancer models, G. superba extracts led to higher DR4 and DR5 expression, which may activate extrinsic apoptosis pathways. Additional p53 expression and damage to the mitochondria were found as well. They confirm that G. superba can cause apoptosis in a wide range of tumours, even those that are resistant.

Both in Vitro and In Vivo Studies

In vitro data have been confirmed in mouse xenograft models of both breast and colon cancer. Giving G. superba extract as mouth drops reduced tumour growth and increased the markers of apoptosis in the tumour tissue. It was found that cleaved caspase-3 increased and Ki-67 expression decreased on immunohistochemistry, meaning that cell death was increased in these tumours [15]. There was little evidence of toxicity to the system, suggesting a suitable therapeutic range. These investigations confirm that G. superba could act as the main treatment for some cancers and also complement chemotherapy drugs.

Mechanistic Insights and Molecular Targets

Studies of how mechanically these root extracts work have provided insight into their uses in cancer therapy. To learn about the pathways of apoptosis induction, several techniques such as studying gene and protein expression, looking at mitochondria, measuring DNA fragmentation, plus using Western blotting, flow cytometry and qPCR were employed**.**

Gene and Protein Expression Profiling.

Expression studies done repeatedly have demonstrated that using G. superba extract changes important apoptotic genes and proteins. It has been shown by qPCR and transcriptomics that higher levels of Bax, Puma and p53 can block tumour growth in combination with lower amounts of anti-apoptotic Bcl-2 and Survivin [16]. This triggers the natural (mitochondrial) pathway that leads to apoptosis. Cleaved caspase-3, caspase-9 and cytochrome c were found to be expressed in greater amounts by Western blot, revealing that apoptosis had started and finished as expected.

Investigating Mitochondrial Membrane Potential

An early sign of apoptosis is when the mitochondrial membrane loses its potential (ΔΨm). JC-1 and rhodamine 123 staining, together with flow cytometry, have proven that cancer cells exposed to G. superba extracts have lost much of their ΔΨm [14]. Because of the disruption, cytochrome c is released from the mitochondria into the cell's cytoplasm, which triggers caspase-9 and caspase-3 to activate. These results support the idea that oxidative stress in mitochondria may cause the apoptosis seen in G. superba treatment.

DNA Fragmentation and TUNEL Assays

The main feature of apoptosis is that DNA is broken into small fragments by endonucleases. A strong positive signal has appeared in the TUNEL assays of cancer cells treated with radiotherapy, proving the presence of internucleosomal DNA breakage [17]. It was confirmed by agarose gel electrophoresis that apoptosis had occurred, since the classical DNA banding pattern was present in the treated groups. By using microscopy, researchers were able to validate that apoptosis indeed takes place rather than the other types of death seen in cells.

Western Blot, Flow Cytometry, and qPCR Data Interpretation

By using western blotting, we found both caspase activation and PARP cleavage, which signal the end of apoptosis. Growing levels of cleaved PARP with declining levels of full-length PARP are related to DNA damage and collapse of the nucleus [18]. With flow cytometry, we have shown that annexin V binds to cells whose membranes are damaged, and propidium iodide cannot enter. Consistently, qPCR confirms that the extract affects gene transcription, especially regarding apoptosis.

The group analysis of these results outlines a clear flow: Mitochondria lose their function → caspases trigger cell death → DNA breaks into fragments → the cell dies by apoptosis.

Apoptosis induced by G. superba is characterised by early mitochondrial events, the regulation of apoptotic genes by transcription, the involvement of effector caspases and nuclear DNA changes. Using strong molecular biology methods, we found that G. superba could usefully support cancer therapy as a natural apoptotic agent.

Toxicity, Limitations, and Safety Concerns

Even though Gloriosa superba is very effective in fighting cancer, the use of colchicine raises concerns about its safety,limiting the use of this herb. Although colchicine is a good anti-mitotic agent, it has a very small range between doses known to be toxic and non-toxic, and it has a reputation for being highly poisonous. Depending on the way colchicine is administered in rodents, the LD_{50} (the dose that kills half of the population) is reported as lying between 1.2 and 2.5 mg/kg [19]. Since there isn't much difference between a useful and dangerous amount, it is very important to keep dosages exact.

Cytotoxicity to Normal Cells

Tests done in a laboratory setting indicate that both colchicine and the G. superba extract could have nonspecific cytotoxicity on normal human cell line fibroblasts and in the renal epithelial cells at elevated concentrations. For this reason, there are concerns about the safety of the system with oral or IV use [20]. While the use of nanoparticles and liposomes is being studied to limit unwanted toxicity, more evidence is required to use these strategies safely in patients.

Dosing, Standardisation, and Safe Formulations

Developing standard dose and treatment formulations for G. superba therapies remains a key problem. How much of each alkaloid a plant has can change a lot, depending on where it is grown, when it is harvested, and the method used to process it [21]. It is also true that having safer and reproducible formulations is difficult, and full medicine and toxicity studies must be completed before new CAR-T drugs can enter clinical trials. It is thought by a few

studies that using G. superba with other herbs might lessen side effects while still working against cancer, though this has yet to be shown by clear clinical trials.

Translational Potential and Future Perspectives

The research results showing G. superba able to induce apoptosis and hinder cancer progression suggest it is a good candidate for medicines. It is, however, a difficult process to turn laboratory findings into treatments. It needs testing through the drug development process, developing better ways to deliver the drug, proving the drug effective in a real clinical setting and examining if new drugs are complementary.

Drug Development in Progress

The main steps of natural product-based drug discovery are often extraction, evaluating their activities, analysing their structures, making them into formulations and testing for toxicity. Colchicine is a recognised drug for the treatment of gout and familial Mediterranean fever in the species G. superba. Even so, chemotherapy treatments for cancer require new versions of the drug to be selective and less harmful. Patented molecules for semi-synthetic colchicine analogues show lower risk of systemic toxicity and better results in treating disease [22].

Nanoparticle Delivery Systems

One way to control the colchicine side effects is by using nanoparticles to deliver it. Using biodegradable nanoparticles made of PLGA or chitosan means G. superba extracts or colchicine can be delivered just to tumours, allowing healthy cells to be spared. With these nanocarriers, drugs are absorbed more easily by the body, fewer unwanted effects occur, and the release can be regulated. In recent work, liposomal and gold nanoparticle drugs have been found to damage cancer cells but barely affect healthy ones [23]. Such developments could make delivering plant-derived cancer agents much easier and more effective.

Potential for Combination Therapies

When used with other chemotherapy agents, G. superba may have beneficial results. In some cases, lower-dose colchicine makes the drugs paclitaxel or doxorubicin more effective and helps patients receive smaller doses, which reduces side effects. The results of docking analyses and synergy tests suggest there could be benefits from combining G. superba products and common cancer drugs in future trials [24].

Conclusion

The medicinal plant Gloriosa superba shows strong anticancer abilities by encouraging cancer cells to die through both intrinsic and extrinsic pathways. Its beneficial components, with colchicine being one, disturb mitochondria, affect genes associated with apoptosis and induce caspase activity. Analysis of breast, colon, cervical, hepatic and pancreatic cells, both in the lab and in living animals, shows it can target tumour cells specifically. Despite their use, normal cell toxicity and a narrow margin of safety continue to be significant problems.With advances in Western blotting, qPCR and mitochondrial techniques, we now recognise its way of working more clearly. Also, using nanoparticles to deliver drugs may improve both safety and effectiveness. Since no major clinical trials are available, the preliminary experiments suggest that G. superba might be effective against cancer in combination treatments.Essentially, G. superba combines elements of traditional medicine with those of modern oncology. More work in biology, with a focus on similar standards, lessening toxicity and clinical research, should be done in order to maximize immunotherapy in cancer treatments.

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