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# Miraculous responses of *Gloriosa superba* L. in the treatment of colon cancer: A brief review

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**Abstract**
We know that there are certain plants that help us to cure ailments; one such crucial plant is *Gloriosa superba* L. which helps to cure various diseases of the human body. One of the main causes of death for the vast majority of people worldwide is colon cancer, a pathologic condition caused by aberrant cell development. Around the world, Colon cancer is a serious health issue. Due to the stage of the malignancy, adverse effects, and altered bio-distribution, there are only a limited number of treatments such surgery, chemotherapy, radiation, and anticancer medications available, this treatment have high risk factor and high cost. Peptides taken from natural sources have come to be thought of as a possible therapeutic. *Gloriosa superba* L. has a long history of usage in traditional medicine, notably for anti-cancer, tumours, kidney issues, haemorrhoids, and wounds. *Gloriosa superba* L. contain colchicine, flavonoid and some other alkaloid which showed anti-cancer property. However, the aim of this study is an investigation of anti-colon cancer activity from some parts of *Gloriosa superba* plant which regulates p53, NF-κB of colon cancer cells (SW620).

**Keywords:** *Gloriosa superba*, p53, Colon Cancer, FR8.2, NF-κB

## Introduction
In 2012, WHO reported that worldwide Colon cancer is one of the top three cancers [1]. Worldwide Colon cancer (CRC) is a serious health problem due to high consumption of Red Meat, alcohol, tobacco, Dietary fibre and Whole Grains. Now a day's various treatments available for colon cancer treatment including surgery, chemotherapy, radiation and anti-cancer drugs [2, 3]. Generally, colon cancer is stage based treatment [1]. Surgery is the only treatment for stage II colorectal cancer (CRC), and adjuvant chemotherapy is not advised on a regular basis. If any of the high-risk characteristics were present, such as poorly differentiated cancer, lymphovascular invasion, perineural invasion, a report of fewer than 12 lymph nodes, bowel obstruction, localised perforation, and positive margins, adjuvant chemotherapy was advised by the European Society of Medical Oncology (ESMO) (last updated in 2013) [4]. I another treatments are chemotherapy, radiation therapy [2]. But this has various bad impressions such as high cost, side effect and no permanent cure [2-4]. Therefore, the world is searching for better alternative cost cost-effective, safe medicine for colon cancer.

A great role has been played by plants and their products since the beginning of human civilization, ethnomedicinal plants. Researchers have studied Ayurvedic medicinal plants extensively in terms of pharmacognosy, chemistry, and pharmacology. A total of 80,000 plants are used in medicine. Traditional medicines are used by 70% of the global population, with most made from plants, and by 80% of developing nations, according to the WHO's Strategy for Traditional Medicine 2014-2023 [3, 4].

*G. superba* is a perennial climber found in tropical and subtropical parts of India, including the foothills of the Himalayas. Plants like *Gloriosa* serve as pot plants in gardens because of their floral beauty [2]. Originally from tropical Africa, it can now be found growing in India, Burma, Malaysia, and Sri Lanka among others. The plant is widely distributed in tropical regions, and it is also used in pots worldwide. In Zambia *G. superba* is national flower emblem. It is widely distributed monocot in India. In India, *G. superba* widely distributed and is the state flower of Tamil Nadu and found Himachal Pradesh, West Bengal, Assam,

Jammu Kashmir and Uttar Pradesh, Maharashtra (Jalgaon, Nandurbar), Assam, Rajasthan (Aravalli hills), Odisha and Punjab. *Gloriosa superba* L. comes from the word "gloriosus", which means handsome, and "*superba*" comes from the word superb, which means splendid or magnificent [1-6]. This studies on anti-colon cancer property of *G. superba* and its effects on apoptosis pathway and the regulatory effect of, p53 and NF-κB that are involved in cellular pathway.

**Taxonomical position** [5, 6]
**Kingdom:** Plantae
**Sub-kingdom:** Trachobionta
**Class:** Liliopsida
**Sub-class:** Lilidae
**Order:** Liliales
**Family:** Colchicaceae
**Genus:** *Gloriosa*
**Species:** *superba*
**Botanical Name:** *Gloriosa superba* L.

**Synonym**
**English:** Malabar glory lily [2, 4]
**Hindi:** Kalihari, Languli [2, 4]
**Bengali:** Ulatchandal, Agnisikha [8,7]
**Sanskrit:** Agni-sikha[4]
**Santhali:** Samonom [8]
**Rajasthan:** Kalgari [8]
**Punjabi:** Kariari, Mulim [7, 8]
**Tamil:** Kalappaikilangu, Salem, Kannoru [7, 8]
**Telugu:** Agnisikha [8]
**Oriya:** Phulbani [8]
**Marathi:** Kal-lavi [6]

**Morphological identification**
**Habitat:** *Gloriosa superba* L. is an annual or perennial; belong in monocot [4, 7, 9].

**Height:** Climbing, sometimes erect herb growing 5 to 6 meters, with tuberous roots.

**Stem:** The stem is glabrous, sparsely branching, and climbs up to 4 m (Fig1) [1].

**Tuber:** Fibrous horizontal tuber bent into an L or V shape [4].

**Leaves:** The upper leaves are ovate to lanceolate, 5-14 cm x 1.6-5 cm, with an obtuse base and parallel veins. It appears in whorls of 3-4, opposite or alternate (Fig 2) [4, 6, 8].

**Flower:** Flowers are large bright yellow and red with wavy edges, at branch ends, solitary, every year flower appears from November to March (Fig1) [3,4,7].

**Inflorescence:** Generally Cymose [7, 8].

**Androecium:** It has six stamens, hypogynous, dorsifixed anthers which are dorsifixed, versatile, and shed bright yellow pollen abundantly [8, 4].

**Gynoecium:** It has a superior ovary, tricarpellary, syncarpus, monolocular, numerous ovules on parietal placentas, a short style deflected at right angles to the ovarian axis, and a trifid stigma.
**Fruit:** Oblong capsule shape, 4-7 cm diameter, containing up to 20 seeds [1, 4].

**Seed:** Seeds of *G. Superb* L. are rounded, numerous, between 4 mm and 5 mm in diameter, surrounded by a fleshy, red sarcotesta [1].

**Fig 1:** Whole plant part

**Fig 2:** Leaf

**Colon Cancer**
Colon and rectal cancers are two extremely prevalent and aggressive cancer forms that are included in colorectal cancers (CRCs). Rectal cancer is the eighth most frequent cancer worldwide, whereas colon cancer ranks fourth [10]. Combined, CRCs represent 11% of all cancer cases identified globally, making them the third most often diagnosed cancer type. These days, colonoscopies are more widely available, and more people are receiving a diagnosis of colorectal cancer (CRC), sometimes at an earlier stage and sometimes at an advanced one. The World Health Organisation (WHO) reports that 862,000 people worldwide lost their lives to colorectal cancer in 2018-a total of 1.80 million new cases were identified with the disease [9-11]. About 145,600 cases of CRC are detected in the USA each year. Colon cancer accounts for 1,014,200 of these instances, with rectal cancer making up the remaining portion. Typically, 29% of CRC cases occur in the rectum and 71% in the colon. Each year in the USA, over 50,000 people pass away with CRC. The incidence of colorectal cancer (CRC) declined by 2.2% annually in males between

2008 and 2014, whereas it stayed steady in women. In addition, between 1999 and 2015, the annual death rate from CRC declined by 1.8% for men and 1.4% for women [12, 13].

The American College of Physicians (ACP), the Multi-Society Task Force (MSTF), and the American Cancer Society (ACS) all advocate different screening programmes [11]. Environmental variables and/or genetic factors may raise the risk of getting colorectal cancer (CRC). Age over 50, low socioeconomic status, being overweight or obese, leading a sedentary lifestyle, smoking tobacco, drinking excessive amounts of alcohol, eating a low-fibre, high-fat diet, consuming red meat, processed meat, and burnt or charred meat, diabetes mellitus and insulin resistance, acromegaly, renal transplantation with long-term immunosuppressant, long-term androgen deprivation therapy, familial adenomatous polyposis (FAP), mutated MMR gene syndromes such as hereditary nonpolyposis colorectal cancer (HNPCC) or Lynch syndrome and Muir-Torre syndrome, hamartomatous polyposis syndromes like Peutz-Jeghers syndrome, Cowden syndrome, and Juven syndrome are among the various risk factors for developing colorectal cancer (CRC) [14, 15].

**Development of Colorectal Cancer (CRC)**

The genesis of colorectal cancer involves many successive mutational events. Adenoma-carcinoma sequence, which is present in sporadic adenoma and FAP, accounts for 70% of CRC cases; the remaining 30% are caused by other pathways, such as Lynch syndrome's MMR gene defect, sessile serrated polyps' BRAF mutation, and MYH-associated polyposis syndrome's base-excision repair (BER) gene defect [16,17]. CRC is formed in the adenoma-carcinoma sequence when tumour suppressor genes, such as the p53 gene on chromosome 17p, the activation of the oncogene KRAS on chromosome 12p, the deletion of the colon cancer (DCC) gene on chromosome 18q, and the adenomatous polyposis coli (APC) gene on chromosome 5q, cease to function. In the adenoma-carcinoma sequence, loss of function of the APC gene is thought to be the crucial first stage. While deletion of the p53 gene happens at the end of the adenoma-carcinoma sequence, loss of the DCC gene causes adenoma to develop into a later stage. In 35-45% of CRC cases, KRAS oncogene activation takes place. It is linked to poor survival rates, increased aggressiveness of colorectal cancer, and lower reactivity to some chemotherapeutic drugs, especially those that target the epidermal growth factor receptor (EGFR) in metastatic colon cancer [18]. MMR genes have a role in fixing errors that arise during DNA replication.

Multiple DNA mutations found in MMR-deficient cells have the potential to cause cancer and MSI. In the United States, 12-15% of cases of CRC are sporadic, with somatic MMR gene mutations and MSI. They are virtually invariably in the right colon, primarily affect middle-aged to older Caucasians without a family history of colorectal cancer, and have a generally favourable prognosis. A common feature of CRC is aberrant DNA methylation of CpG islands, or CpG island methylator phenotype (CIMP). MMR insufficiency occurs occasionally as a result of CIMP-associated methylation of MLH1.

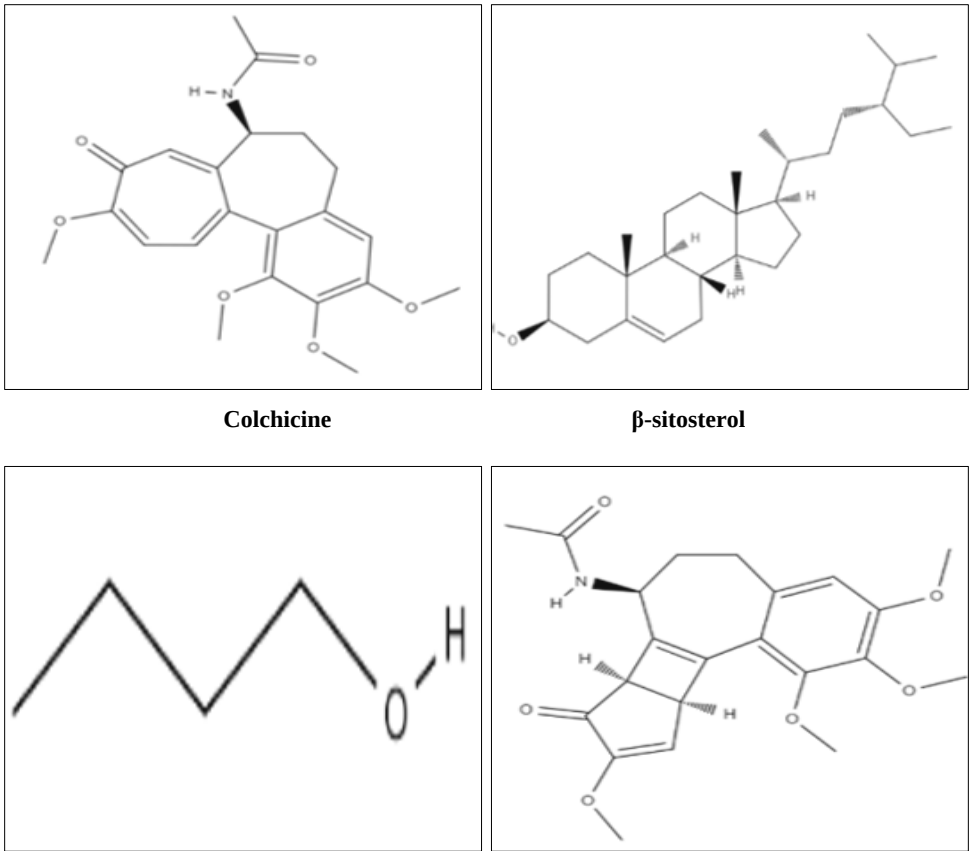
In the USA, germ line mutations in the MMR genes (hMSH2, hMLH1, hPMS1, and hPMS2) are found in HNPCC and account for 3-6% of all CRC cases [11].10% of cases of colorectal cancer had a mutation in the BRAF (B-Raf proto-oncogene serine/threonine kinase) gene (valine-to-glutamate alteration at residue 600-V600E). BRAF mutation causes the development of both classic serrated adenoma (TSA) and sessile serrated adenoma (SSA). BRAF mutation-related colorectal cancers are typically right-sided, develop in old age, are more common in women, and are linked to multiple sclerosis. Proximal right-sided CRC has a poor prognosis, while BRAF-mutated CRC with MSI is typically linked to a better prognosis [19].

The majority of BRAF-mutated colorectal cancer patients are CIMP-positive [20]. Tumours resulting from serrated adenoma are therefore known to be CIMP-positive. Microsatellite instability affects almost half of CIMP-positive tumours. CIMP-positive cancer accounts for 20-30% of all CRC cases, while CIMP-positive and microsatellite unstable cancer accounts for 10-12% of cases [11].

**Bioactive component of *Gloriosa superba***

Whole part of *G. superba* L. contains various important bioactive components due to presence of alkaloids. Its amino alkaloid obtained from the tyrosine and amino acids phenylalanine Extracts of this plant contain monoethyl ester of 2, 6-dihydroxybenzoic acids [4, 21]. *G. superba* L. contain alkaloid including Colchicine and Gloriosine. Chemically Colchicine is known as N-[(7S)-1, 2, 3,10-tetramethoxy-9-oxo-5,6,7,9-tetrahydrobenzo [a] heptalen-7-yl] acetamide [4]. Colchicine is active anti-cancer property including Colon cancer [22,23]. The many amount of colchicine detected using by 9.0 mg with 30 μM tyrosine medium fed [4].

However, colchicine can regulate growth of various cancer cells cycle such as cyclins and Cyclin Dependent Kinases (CDKs), DNA damage, G1-S transcriptional regulation,HT-29, mitotic checkpoint and the ubiquitin ligase regulatory pathways [4, 24-28]. In addition, contain sitosterol, glucoside, β-and gamma lumicolichicines, β-sitosterol, flucoside and 2-H-6-MeO benzoic acid. Its leaves flower and tubers contain glycosides, 3-O-demethylcolchicine-3-O-α-D-glucopyranoside, flavonoids, N-formyldeMe-Colchicine, steroids, phenols and saponins. Carbohydrates, Viatmin C, Vitamin E and minerals presence of tuber of *G. superba* L.[28-34]. Tubers, leaves, and seeds of *G. superba* L. contain Chloroform and n-butanol which have anticancer property [33, 34].



**Colchicine** **β-sitosterol**

**N-butanol** **Lumicolchicine**

**Fig 2:** Important bioactive chemical compound of *G. superba* L. [9, 10]

**Anti-Colon Cancer Activity**

Surgery is the only treatment for stage II colorectal cancer (CRC), and adjuvant chemotherapy is not advised on a regular basis. If any of the high-risk characteristics were present, such as poorly differentiated cancer, lymphovascular invasion, perineural invasion, a report of fewer than 12 lymph nodes, bowel obstruction, localised perforation, and positive margins, adjuvant chemotherapy was advised by the European Society of Medical Oncology (ESMO) (last updated in 2013). One of the following chemotherapy regimens should be used for six months as part of the adjuvant chemotherapy: Leucovorin (LV), capecitabine, 5-flurouracil (FU) with LV and oxaliplatin (FOLFOX), or capecitabine and oxaliplatin (Capeox) in combination. Curative surgical tumour excision for Stage III colorectal cancer is followed by adjuvant chemotherapy consisting of six cycles of either FOLFOX or Capex [35, 36]. However, the traditional medicine of colon cancer treatments best way for cost effective, long-lasting therapeutic benefit, and risk free. As a result, they guarantee the delivery of traditional drugs to the CRC, increasing their effectiveness [3, 4, 15, 28].

In the Indian Medicine Systems *G. superba* L. are various used [4]. Reported that rhizome *G. superba* L. have strong anti-colon cancer activity. To study anti-colon cancer property of the extracted of *G. superba* L. protein hydrolysate, SW620 (colon cancer cells) were treated with various concentrations such as 0, 10, 20 to 30 ng/mL of protein hydrolysate and after 5 days they study on the cells in an MTT assay. Protein hydrolysate significantly induced SW620 cells without normal cell. Cell treatment with 30 ng of protein/mL, cell of SW620 was remarkably decreased showing a 40% inhibitory property. NF-κB and p53 serve as strong molecular targets for anti-cancer treatment[1].Some various cellular proteins that regulate the apoptotic response including p53 and NF-κB and being that control the cell line response[37, 38]. NF-κB plays a critical role in the genotoxic and cytotoxic stress response pathways, both of which malfunction frequently in cancer tissues. Partial purified protein hydrolysate fraction 8.2 (FR8.2) strongly regulated p53 at 20 ng of protein/mL. FR8.2 inhibited the cell division and cell death [1]. Colchicine is isolated from *G. superba* L. Naturally occurring Colchicine is a very inexpensive alkaloid that have used pharmacy from earlier time. Colchicine results in the arrest of mitosis and induction of apoptosis of Colon cancer cells [22, 23, 38, 39]. Colchicine inhibits proliferation due to its ability to bind to the ends of microtubules and block microtubule elongation [22, 23, 40, 41]. Colchicine inhibits colon cancer cell growth by inducing caspase-3-promoted cell death [1, 22, 41, 42].

Flavonoids are secondary polyphenolic metabolites of plant and over 4000 broad class such flavones, flavanols and isoflavone [43, 44]. Several flavonoids have been shown to have anti-cancer properties and prohibition of cell proliferation. *G. superba* L. contains flavonoids and fight Colon cancer growth. The flavonoids inhibit estrogen-producing enzymes, thereby reducing colon cancer risk. Checkpoint inhibitors have recently been shown to benefit individuals with MSI-H or dMMR colorectal cancer. When patients receiving nivolumab + ipilimumab for metastatic colorectal cancer (CRC) with dMMR or MSI-H, Overman *et al*. saw a long-lasting therapeutic benefit [9, 41, 46-50].

**Conclusions**

In this study provides evidence that *G. superba* L. have important bioactive component that work against colon

cancer as well as anti-colon-cancer activity. Traditionally *G. superba* L. has been claimed for a huge number of pharmacological activity and therapeutic uses.FR8.2 of rhizome of *G. superba* L. inhibited cell line of colon-cancer cell by inhibiting apoptosis via up-regulation of p53 and down-regulation of NF-κB.

Moreover, this study provided the anti-colon cancer property of *G. superba* L., which can answer of the disease cancer. Natural important bioactive components present in this plant source of new compounds in cancerous drug discovery research. Bioactive component of the said plat including protein hydrolysate, Colchicine and Flavonoids regulate cell cycle of SW620 (Colon Cancer). The study also highlights the huge possibilities of the plant for several other medicinal uses [51-69]. So, this plant has the promising aspects toward healing the colon cancer.

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**Conflict of interest**

The author declares no conflict of interest.

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