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# "A REVIEW ON: COLORECTAL CARCINOMA AND ITS HERBAL APPROACHES"

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## ABSTRACT

The third most prevalent cancer in the world is colorectal carcinoma. Adenomatous polyps, which occur in areas of epithelial cell hyperproliferation and crypt dysplasia, are precursor lesions to colorectal cancer. Chemotherapeutic medicines are used alone or in combination to treat the majority of CRC patients. However, up to 90% of patients with metastatic cancer encounter treatment failure, primarily due to medication resistance that has developed over time and can result in multidrug resistance (MDR). In this study, we looked into recent research on prospective herbal medicine CRC MDR reversal therapies (HMs). **Panax Quinquefolius, Gymnaster Koraiensis, Zingiber Officinale, Punica Grantum, Vitis Vinifera, Camellia Sinensis, Zingiberaceae,** etc. In recent years, emphasis has been drawn to the use of conventional therapies. The purpose of this study was to provide an overview of plants that have been shown to be useful in treating colon cancer, with a focus on their bioactive components and underlying mechanisms of action. The findings suggest that the best plants for preventing colon cancer are grape, soybean, green tea, and pomegranate. These medicinal herbs have a number of cancer-fighting strategies, including upregulating superoxide dismutase, lowering DNA oxidation, triggering apoptosis by stopping the cell cycle in the s phase, and downregulating PI3K and P-Akt expression.

**KEYWORDS:**

Colorectal Carcinoma / Colon Cancer, Epidemiology, Pathophysiology Herbal Approaches / Medicines.

## INTRODUCTION

The words "colon" and "rectum" are combined to form the phrase "colorectal." The last six to eight feet of the intestine, also known as the large intestine, are referred to as the colon, and the final few inches of the large intestine, just before it leaves the body through the anus, are referred to as the rectum. When abnormal tissues develop on the interior of the colon or rectum, colorectal cancer develops. Polyps are a common manifestation of these aberrant tissues. Polyps develop as a tissue outgrowth from the colon wall that is still attached to the wall by a thin stalk. They have a mushroom-like appearance. Particularly in older persons, polyps are rather typical. In the vast majority of cases, polyps do not have malignancy. Some polyps, though, will eventually develop cancer. Unchecked, a malignant polyp develops into a tumour that, through the process of metastasis, engulfs surrounding organs and lymph nodes after penetrating the intestinal wall.

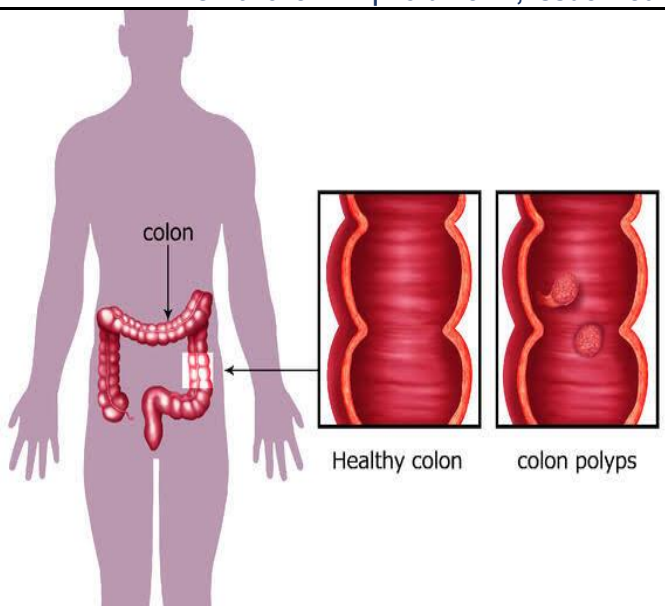


**Fig.1 Showing Healthy colon and Infected colon**

With an incidence of 10.2% and a mortality rate of 9.2%, colorectal cancer (CRC) is the third most prevalent cancer in the world.

The normal course of colorectal cancer as it develops from premalignant adenomatous polyps was identified by the National Polyp Study (NPS). The presence of one or more colorectal adenomas but no invasive malignancy was a requirement for eligibility. All polyps were removed from study participants in the beginning, and they were thereafter monitored. 6 years via questionnaires, faecal occult blood tests, barium enema, and colonoscopy. Two thirds of the polyps that were surgically removed at the beginning were adenomatous (neoplastic), having the potential to develop into cancer, and one third were nonadenomatous. The two types of nonadenomatous polyps that don't turn into cancer are principally hyperplastic and regular mucosal tags.

The study demonstrated that polyp growth is extremely prevalent in humans. Following colonoscopic removal, adenomatous polyps are frequently discovered, but only a small proportion—3.3% in the NPS—are pathologically progressed, as indicated by a diameter of.1 cm, high-grade dysplasia, or invasive malignancy, at follow-up exams. Reducing the prevalence of pathologically advanced polyps is increasingly utilised as the major outcome for primary and secondary intervention studies because practically everyone develops adenomas.

## CAUSES AND RISK FACTORS

**Older age:** Although colon cancer can be diagnosed at any age, most cases occur in those over the age of 50. Doctors are unsure of the reason why colon cancer rates in those under 50 have been rising.

**A personal history of colorectal cancer or polyps:** If you've already had colon cancer or noncancerous colon polyps, you have a greater risk of colon cancer in the future.

**Inflammatory intestinal conditions:** Colon cancer risk can be increased by chronic inflammatory illnesses of the colon such Crohn's disease and ulcerative colitis.

**Inherited syndromes that increase colon cancer risk:** Your risk of colon cancer can greatly increase if you have certain gene mutations that have been passed down through your family. The majority of colon cancers are not caused by inherited genes. Familial adenomatous polyposis (FAP) and Lynch syndrome, often known as hereditary nonpolyposis colorectal cancer, are the two most prevalent genetic diseases that increase the chance of developing colon cancer (HNPCC).

**Family history of colon cancer:** If you have a blood family who has had colon cancer, you are more likely to get it yourself. Your risk is increased if multiple family members have colon or rectal cancer.

**Low-fiber, high-fat diet:** A normal Western diet that is high in fat and calories and poor in fibre may be linked to colon and rectal cancer. The findings of this research have been conflicting. People who consume diets heavy in processed and red meat are at an elevated risk of developing colon cancer, according to several research.

**A sedentary lifestyle:** Colon cancer is more prone to occur in those who are inactive. Regular exercise may lower your risk of developing colon cancer.

**Diabetes:** Colon cancer risk is higher in people who have diabetes or insulin resistance.

**Obesity:** When compared to persons who are regarded to be of normal weight, those who are obese have a higher risk of developing colon cancer and a higher risk of dying from the disease.
**Smoking:** Smokers may be more likely to develop colon cancer.
**Alcohol:** Colorectal cancer risk is increased by heavy alcohol usage.
**Radiation therapy for cancer:** The risk of colon cancer is increased by radiation therapy administered to the abdomen to treat prior malignancies.

## DIAGNOSIS

The cancer's stage must be determined as a crucial component of the diagnosis. The various stages of colon cancer indicate how far along the disease is and help choose the best course of action. Here are the stages:
**a. Stage 0:** The colon cancer has not yet reached the inner layer of the colon and has only just begun to grow.
**b. Stage I:** No adjacent lymph nodes have been affected and the colon's inner layers have been invaded by the cancer.
**c. Stage II:** The colon cancer has reached the outer layers or beyond, but it has not progressed to the lymph nodes or other organs.
**d. Stage III:** Lymph nodes nearby have been affected by colon cancer that has migrated from the colon.

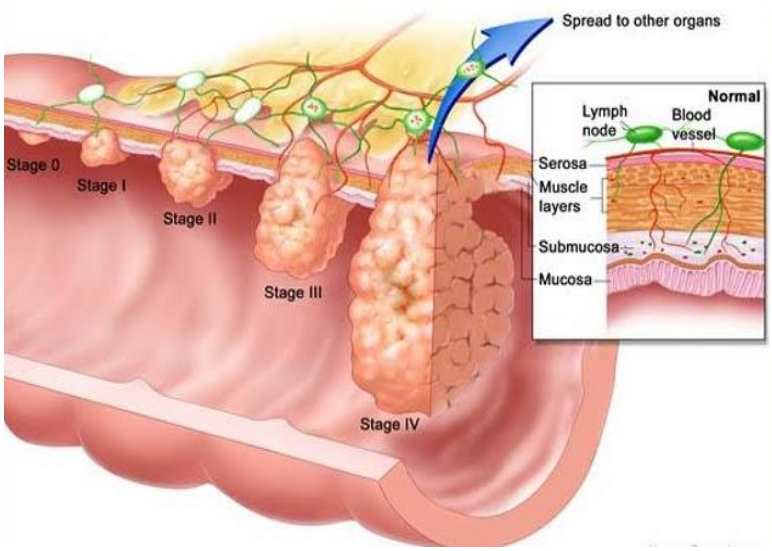


**Fig.2 Stages of colorectal cancer.**

**Diagnostic Colonoscopy:** Similar to a screening colonoscopy, a diagnostic one is performed when symptoms are present or when an abnormality is discovered during another screening test. During this examination, the doctor uses a colonoscope, a short, flexible, lighted tube with a tiny camera at the end, to view the full length of the colon and rectum. It is injected into the rectum and colon through the anus. If necessary, the colonoscope can be used to insert specialised devices to take a sample of any suspicious-looking tissue or to remove polyps. Colonoscopy procedures can be carried out in a doctor's office, a clinic, or an outpatient hospital department.
**Proctoscopy:** If rectal cancer is suspected, this test might be performed. With a proctoscope, a small, stiff, illuminated tube with a camera on the end, the doctor examines the rectum during this test. The anus is used for insertion. Through the scope, the doctor can get a close-up view of the rectum's interior lining. It is possible to visualise the tumour, measure it, and pinpoint its precise location. A tumor's proximity to the muscles that govern sphincter control, for example, can be seen by the doctor.
**Biopsy:** Usually, if a suspected case of colorectal cancer is discovered by any screening or diagnostic test, a colonoscopy is performed to take a biopsy. During a biopsy, the doctor uses a specific device inserted via the scope to extract a small sample of tissue. Less frequently, the diagnosis can require surgically removing a portion of the colon. For more information on the many types of biopsies, how the tissue is used in the lab to diagnose cancer, and what the results may show, see Testing Biopsy and Cytology Specimens for Cancer.
**Computed tomography (CT or CAT) Scan:** A CT scan creates fine-grained cross-sectional images of your body using x-rays. This test can help determine whether colorectal cancer has progressed to your liver, lungs, or other internal organs.
**Magnetic Resonance Imaging (MRI) Scan:** MRI scans produce detailed pictures of the body's soft tissues, just like CT scans do. However, MRI scans substitute radio waves and powerful magnets for x-rays. Before the scan, a contrast agent called gadolinium may be injected into a vein to provide clear images.
Using MRI, abnormal regions in the liver, brain, and spinal cord that could be cancer spread can be examined.

**Endorectal MRI:** Rectal cancer patients might use an MRI scan of the pelvis to determine whether the tumour has spread to adjacent structures. Using this, surgery and other treatments can be planned. Some medical professionals employ an endorectal MRI to increase the test's accuracy. In order to do this test, the physician inserts an endorectal coil, a probe, inside the rectum. During the test, this remains in place for 30 to 45 minutes and may be uncomfortable.

## EPIDEMIOLOGY

Around 10% of all cancers diagnosed each year and cancer-related deaths globally are caused by colorectal cancer. With between one and two million new cases being identified each year, colorectal cancer (CRC) is one of the most prevalent diseases in the world. With 700,000 cancer-related fatalities each year, only lung, liver, and stomach cancers are more common causes of cancer-related mortality. CRC is the second most prevalent cancer among women (9.2%) and males (10%), respectively, according to gender. On the other hand, a concerning increase in patients with colorectal cancer who are under 50 years old has been noted, particularly with rectal cancer and left-sided colon cancer. Although there may be some correlation between genetic, lifestyle, weight, and environmental variables, the precise causes of this increase are not fully understood.

## PATHOPHYSIOLOGY

The molecular pathways for both sporadic and CRC linked to colitis are involved in the aetiology of colorectal cancer (CRC).

**Sporadic Colorectal Carcinoma:**

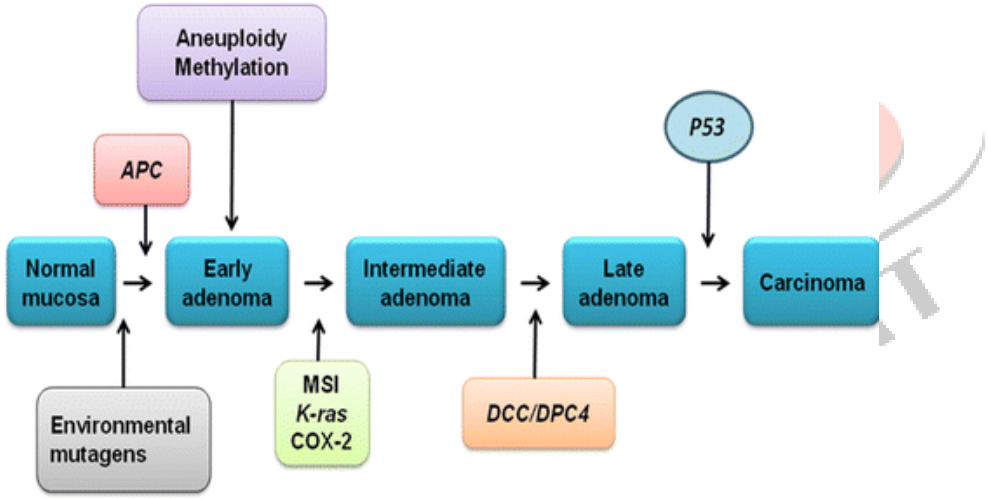


**Fig.3 Pathway of Sporadic Colorectal Carcinoma**

**APC gene & Wnt Signalling pathway:** Produces the APC protein, which stops the -catenin protein (involved in stem cell renewal) from building up. A mutation in the APC protein causes the -catenin protein to build up and unnecessarily high levels of stem cell renewal.A big 312-kDa protein that is encoded by the APC gene has been found to have many cellular activities and work through a variety of molecular pathways. APC's role in the negative regulation of the canonical Wnt signalling pathway, which it accomplishes by downregulating the transcriptional activator -catenin, is one of the first and best-known actions of the protein.

A vast protein complex is formed in order to achieve this, and its main players include APC, the scaffold protein Axin, GSK3, casein kinase 1, -catenin, and the E3-ubiquitin ligase TrCP. This complex has the ability to phosphorylate -catenin at particular locations, resulting in the protein's destruction by the proteasome. Because it interacts with the T cell factor (TCF) family of transcription factors and mediates the transcriptional activation of target genes crucial for carcinogenesis, -catenin plays a crucial function in the canonical Wnt pathway. It has been demonstrated that APC mutations that activate the canonical Wnt pathway alter the cell cycle and promote cell proliferation, survival, and differentiation.

Additionally, APC may negatively regulate the canonical Wnt pathway using alternative methods. For instance, APC can be transported into the nucleus where it can assist the export of -catenin back to the cytoplasm and remove it from particular genomic loci.

Additionally, APC might perform other nuclear tasks unrelated to the Wnt pathway, like DNA repair and cell-cycle regulation. APC, for instance, has been discovered to be attached to DNA, where it may prevent the

advancement of the cell cycle. Oncogenic APC mutations' precise effects on these nuclear processes are still being fully understood.

**TP53 Pathway:** The TP53 gene, also referred to as the "guardian of the genome," is found on the short arm of chromosome 17. It encodes proteins that control the cell cycle, DNA repair, senescence, and apoptosis. 50–75% of CRC cases had TP53 mutations or loss of function; the loss of p53-mediated apoptotic pathways is a significant prognostic factor. from an adenoma to a cancerous tumour. A crucial step in the development of colorectal cancer, excessive cell proliferation activities and uncontrolled cell cycle are enhanced by the loss of function of the p53 gene. The most frequent TP53 mutations in CRC, according to research, are missense mutations (48%) that change GC to AT, followed by point mutations (37.5%) with transitions at CpG sites.

**Colitis-associated colorectal carcinoma:**

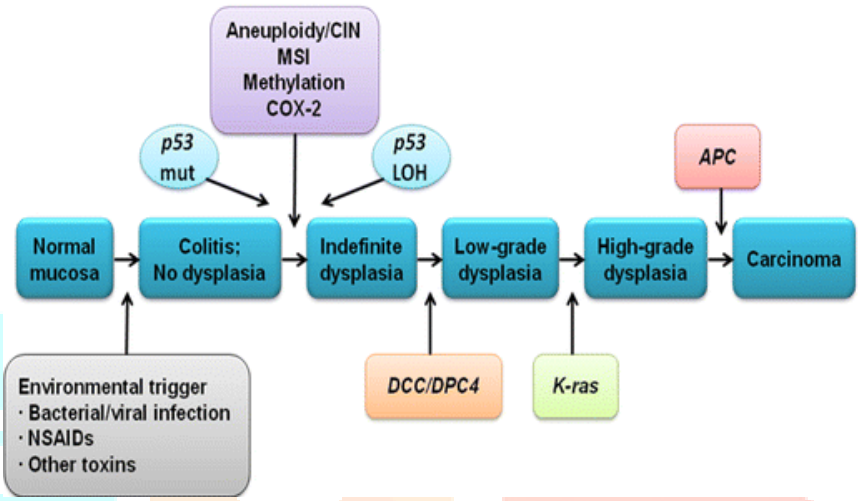


**Fig.4 Pathway of colitis-associated colorectal carcinoma**

**Chromosomal Instability (CIN) Pathway:** The most frequent genetic instability in CRC is chromosomal instability, which is defined as a significant increase in the gain or loss of either the full or significant sections of chromosomes. Around 85% of adenocarcinoma transitions are found to have CIN, which is characterised by oncogene activation (KRAS and BRAF), TSG inactivation (APC and TP53), and a loss of heterozygosity for the long arm of chromosome 18 (18q LOH), which encourages CRC growth.

**Microsatellite (MSI) Pathway:** Microsatellite instability (MSI), a defining trait of malignant cells, is another type of genomic instability in CRC. The hallmark of HNPCC or Lynch syndrome, MSI is present in >95% of cases of HNPCC. However, the underlying mechanism for CIN is still developing in the majority of sporadic CRCs, while MSI accounts for just 15-20% of all CRC cases. Except in Lynch syndrome, MSI is infrequently seen in polyps. Additionally, individuals with Lynch syndrome frequently develop MSI CRCs as a result of germline mutations in one of the MMR genes (MLH1, MSH2, MSH6, and PMS2); mutations in the MLH1 or MSH2 gene increase the risk of cancer development (70-80%), whereas mutations in the MSH6 or PMS2 gene have a relatively lower risk of cancer development (25-60%). On the other hand, sporadic MSI CRCs commonly exhibit lack of MMR activity as a result of aberrant DNA methylation's silencing of MLH1.

## HERBAL APPRAOCHES

**Table 1. Herbal approaches for CRC.**

| Sr. No. | SCIENTIFIC NAME | PARTS USED | IMPORTANT CHEMICAL COMPOUNDS | MECHANISM/CELL-ULAR EFFECT |
|---|---|---|---|---|
| 1 | Vitis vinifera | Seed | Procyanidins, Catechin, Epicatechin | Decreased VEGF, TNF, MMP-1, MMP-3, MMP-7, MMP-8, MMP-9, and MMP-13 protein expression.<br><br>Decreased MPO (myeloperoxidase) activity.<br><br>Elevated p53Bax/Bcl-2 ratio, cleaved PARP, and mitochondrial-mediated apoptosis.<br><br>Suppressed proliferation, sphere formation, nuclear translocation of β -catenin, and Wnt/ β –catenin signalling. |
| 2 | Camellia sinensis | Leaf | Catechin, epigallocatechin gallate, Theaflavins (TF-2, TF-3, TF-1), Phenolic compounds (p-hydroxyphenyl ethanol, pinoresinol & Dihydroxyphenyl ethanol). | Stopped VEGF expression, ERK-1 and ERK-2 activation, and VEGF promoter activity.<br><br>MMP-9 and VEGF secretion inhibition.<br><br>The reduction of COX-2 expression was associated with the inhibition of edoema development, and promoter analysis showed that NF-B AP-1, CREB, and/or NF-Il-6 (C/EBP) were modulated.<br>Inhibition of a range of α5 and β1 by lower expression. |
| 3 | Purple-fleshed potatoes | Fruit | Anthocyanin, β-catenin, cytochrome | Regardless of the p53 status, PA therapy also increased cytochrome c levels, suggesting that the mitochondria may be the source of the apoptotic pathway.<br><br>Reduced amounts of nuclear and cytoplasmic β-catenin. |
| 4 | Zingiberaceae | Rhizomes | Turmerone | LDH release; ROS production.<br><br>Decrease in the potential of the mitochondrial membrane.<br><br>Leakage of cytochrome c.<br><br>Caspases 3 and 9, the initiators, were activated in a dose-dependent manner.<br><br>Bax expression was upregulated while Bcl-2 and Bcl-xL mRNA expression was downregulated. |
| 5 | Panax quinquefolius | Root | Ginsenosides (protopanaxadiol or protopanaxatriol). | Reduced experimental colitis. |

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| | | | | Attenuated colon cancer development caused by AOM/DSS.<br><br>Activation of proinflammatory cytokines.<br><br>DSS was suppressed.<br><br>Inflammatory cytokine gene expression was downregulated. |
| 6 | Myrtaceae | Leaf | Phenolics, flavonoids, betulinic acid | Inhibition of HUVECS migration and angiogenesis of tube formation on Matrigel matrix (in vitro).<br><br>Reduced nutrient and oxygen delivery, which causes a growth and enlargement of the tumour (in vivo).<br><br>An expanded area of tumour necrosis. |
| 7 | Gymnaster koraiensis | Aerial part | Gymnasterkoreaynes B,C, E, 2,9,16-heptadecatrien-4,6-dyne-8-ol | A notable rise in serum IL-6; A significant decrease in COX-2 expression. |
| 8 | Allium fistulosum | Edible portions | p-Coumaric acid, ferulic acid, sinapic acid, quercitrin, isoquercitrin, quercetol, kaempferol. | Downregulated expression of MMP-9 and ICAM.<br><br>Metabolite profiling and potential active phytochemical components.<br><br>Decreased expression of inflammatory molecular markers. |
| 9 | Zingiber officinale | Rhizome | 6-Paradol, 6- and 10-dehydrogingerdione, 6- and 10-gingerdione, 4-, 6-, 8-, and 10-gingerdiol, 6-methylgingerdiol, zingerone, 6-hydroxyshogaol, 6-,8-,10- dehydroshogaol, diarylheptanoids. | Reduced DNA synthesis, G0/G1phase arrest, and apoptotic induction. |
| 10 | Cydonia Oblonga Miller | Leaf & Fruit | Phenolic compound (flavonol and flavone heterosides, 5-O-caffeoylquinic acid. | Repression of pathways activated by the NFkB activator (AP-1), mitogen-activated protein kinases, namely PKC, and the growth factor receptor (GFR).<br><br>Arresting the cell cycle and angiogenesis.<br><br>Apoptosis, antioxidant, and anti-inflammatory actions are induced. |
| 11 | Sedum Kamtschaticum | Aerial Part | Buddlejasaponin IV | Induced apoptosis through a mitochondrial-dependent process that was set off by a decrease in Bcl-2 protein levels, the activation of caspase |

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| | | | | 3, and the subsequent cleavage of PARP. |
| 12 | Ginkgo biloba | Fruit & leaf | Terpene lactones and flavonoid glycosides. | An increase in caspase 3 activity, a decrease in the expression of the Bcl-2 mRNA, and an increase in the expression of the p53 mRNA. |
| 13 | Rubus occidentalis | Fruit | β-Carotene, α-carotene, ellagic acid, ferulic acid, coumar | Impaired routes for signal transduction that activate AP-1 and the NFB RU-ME fraction. |
| 14 | Oryza sativa | Seed | Enolic compound (tricin, ferulic acid, caffeic acid, and methoxycinnamic acid). | Reduced the quantity of SW480 and HCEC cells that were still viable as well as their capacity to form colonies. This was accomplished by increasing the activation of caspases 8 and 3. |
| 15 | Olea europaea | Leaf | Olea europaea Oleuropein and hydroxytyrosol | Caspases 3, 7, and 9 are activated.<br><br>Mitochondrial membrane potential and cytochrome c release are decreased.<br><br>An increase in the Ca2+ content inside cells. |
| 16 | Cannabis sativa | Dry Flowers & Leaf | Cannabidiol, phytocannabinoids | Decreased cell proliferation in preneoplastic polyps and lesions that are CB1-sensitive and caused by AOM.<br><br>CB1 and CB2 receptor activation inhibits the growth of colorectal cancer cells. |
| 17 | Smallanthus Sonchifolius | Root | Fructan | A decrease in the incidence of colon cancers that exhibit altered -catenin. |
| 18 | Punica Granatum | Peel | Gallic acid, protocatechuic acid, cateachin, rutin, ellagic acid, Punicalagin. | Decreased levels of TGF-, Bcl-2, EGF, CEA, CCSA-4, MMP-7, COX-2, cyclin D1, and survivin.<br><br>Downregulated expression of the genes c-Myc, K-ras, and β-catenin. |
| 19 | Aloe vera | Gel | Polysaccharides | Cellular factors like extracellular signal-regulated kinases 1/2, cyclin-dependent kinase 4, and cyclin D1 are induced, while PAG increases the expression of caudal-related homeobox transcription factor 2.<br><br>Cell cycle progression is inhibited. |
| 20 | Glycine max | Seed | Anthoxanthin , Saponin. | An increase in the protein levels of Rab6, a key small GTP-binding protein.<br><br>Alkaline phosphatase activity was increased while PKC activation was suppressed.<br><br>Repression of IκBα oxidative phosphorylation in PMA-stimulated cells. |

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| | | | | Reduction of PKC and COX-2 expression. |
| 21 | Opuntia ficus-indica. | Fruit | Betalain pigment indicaxanthin | Reactivation of the suppressed mRNA expression and accumulation of p16INK4a; Demethylation of the tumor suppressor p16INK4a gene promoter. |
| 22 | Piper betle | Leaf | Hydroxychavico | electrophilic metabolite formation, scavenging action, increased apoptosis, and antioxidant capacity. |
| 23 | Mentha spicata | Leaf | Acetic acid 3-methylthio propyl ester (AMTP), methyl thio propionic acid ethyl ester (MTPE). | Displayed antimutagenic properties.<br><br>The tetraterpene -cryptoxanthin and the monoterpene auraptene (7-geranyloxycoumarin) stimulated the synthesis of antibodies. |
| 24 | Scutellaria barbata | Leaf | Scutellarein, scutellarin, carthamidin, isocarthamidin, wogonin | Increase in the sub-G1 phase and suppression of cell proliferation in the human colon cancer cell line. |
| 25 | Morus alba | Leaf | Epicatechin, myricetin, quercetin hydrate, luteolin, kaempferol, ascorbic acid, gallic acid, pelargonidine, p-coumaric acid. | Cytotoxic impact on colon cancer cells in humans (HCT15).<br><br>Additionally, iNOS was downregulated by apoptosis induction.<br><br>DNA fragmentation.<br><br>An increase in caspase 3 activity. |
| 26 | Podocarpus elatus | Fruit | Phenolic and anthocyanin | Lessening of colon cancer cell growth. Cell cycle stalling in the S phase.<br><br>Telomerase activity was downregulated by 93%, and telomere length fell. Brought about morphological changes in HT-29 cells. |
| 27 | Annona squamosa Linn. | Leaf | Acetogenins (annoreticuin & isoannoreticuin) and alkaloids dopamine, salsolinol, and coclaurine. | Reactive oxygen species (ROS) generation, lactate dehydrogenase (LDH) release, and inhibition of tumour cell growth and proliferation are some of the side effects.<br><br>Caspases 3/7, 8 and 9 activation. |
| 28 | Hibiscus Cannabinus | Seed | Gallic acid, p-hydroxybenzoic acid, caffeic acid, vanillic acid, syringic acid, and p-coumaric and ferulic acids. | Cytotoxic action against human colon cancer HCT116 cells; induction of apoptosis through blocking the mid G1-late G1-S transition, resulting in arrest of the G1 phase of the cell cycle. |
| 29 | Orostachys Japonicus. | Leaf & Stem | Flavonoids, triterpenoids, 4-hydroxybenzoic acid, 3,4- dihydroxybenzoic acid, polysaccharide. | Apoptosis and proliferation in HT-29 colon cancer cells were both inhibited by the tumour suppressor protein p53 at the G2 stage of the cell cycle. |

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| 30 | Long pepper (PLX) | Fruit | Piperidine alkaloids, piperamides, piperlongumine. | Time-dependent induction of apoptosis in HT-29 colon cancer cells after DNA fragmentation.<br><br>Caspase-independent apoptosis that was induced.<br><br>Promoted the formation of whole cell ROS. |
| 31 | Crocus sativus L. | Flower | Carotenoid, pigment, crocin, crocetin | Apoptosis and DNA damage were induced.<br>Induction of a full G2/M halt and p53 pattern-dependent caspase 3 activation.<br><br>Caused a notable delay in the S/G2 phase transit with mitotic entrance. |
| 32 | Phyllanthus emblica L. | Seed, Pulp | Trigonelline, naringin, kaempferol, embinin, catechin, isorhamnetin, Quercetin. | Suppressed Proliferation. Independent of the p53 stemness trait, induced apoptosis (in HCCSCs).<br><br>Antiproliferative properties.<br>Reduced c-Myc and cyclin D1 expression and cell proliferation.<br>Activated the intrinsic apoptotic signalling system in the mitochondria. |
| 33 | Butea monosperma | Flower | n-Butanol | Antiproliferative impact that is notable.<br>Significantly reduced the expression of Wnt signalling proteins such c-Myc, cyclin D1, APC, and GSK-3.<br>A rise in ROS levels inside cells. |

## CONCLUSION

CRC is a serious public health issue that has serious effects on individuals and their families. Rectal cancer affects patients under the age of 55 in about 30% of cases, and recent research indicating an increase in CRC cases at age 50 compared with age 49 clearly shows that patients under 50 are already at an elevated risk of developing precancerous polyps and cancer. According to the review's findings, medicinal plants containing a variety of phytochemicals, including flavonoids, poly-phenol compounds like caffeic acid, catechins, saponins, polysaccharides, triterpenoids, alkaloids, glycosides, and phenols like quercetin and luteolin, as well as kaempferol and luteolin glycosides, can inhibit tumour cell growth and also trigger apoptosis. Different processes govern how plants and the primary components of plants regulate transcription and the cell cycle. The induction of superoxide dismutase, which neutralises free radicals, the reduction of DNA oxidation, the induction of apoptosis by causing a cell cycle arrest in the S phase, the downregulation of PI3K, P-Akt protein, and MMP, the downregulation of antiapoptotic Bcl-2 and Bcl-xL proteins, and the reduction of proliferating cell nuclear antigen (PCNA), cyclin A, Additionally, plant chemicals raise the expression of BAD, Bax, caspase 3, caspase 7, caspase 8, and caspase 9 proteins as well as cell cycle inhibitors such p53, p21, and p27. In general, this study demonstrated that medicinal plants may be able to stop colon cancer cells from growing and multiplying. However, more research on these substances in in vivo models is necessary before they may be used clinically.

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