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Exploring anticancer properties of Moringa and its derived by-products against colon cancer in humans and animals

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Abstract

Colon cancer continues to be the main cause of neoplasm-related morbidity and mortality in humans and animals worldwide, necessitating the exploration of new, efficient, and less toxic medicinal approaches. Depending on the adverse effects, including genetic toxicity, myelosuppression, hypertension, neurotoxicity, gastrointestinal toxicity, impaired chemosensitivity, nephrotoxicity, cardiovascular issues, neutropenia, etc., conventional therapies such as chemotherapy and radiotherapy need to be replaced by better therapeutic alternatives. This review aims to explore the potential therapeutic benefits of *Moringa oleifera* and its nano-formulations against colon cancer. Moringa, a medicinal plant rich in active chemicals, and its biosynthesized nanoparticles have the potential to hinder the growth and proliferation of colon cancerous cells through arresting cell division at different phases, promoting cell death by disrupting mitochondrial membrane potential, inducing oxidative stress leading to DNA damage, and hindering cancer-causing signalling pathways like PI3K/Akt, NF-κB, and ERK. Moreover, the review highlights that future research should evaluate the safety and efficacy of Moringa and its NPs through clinical trials, observing the effect of Moringa and its NPs on different molecular pathways, enhancing the production of NPs, assessing the use of combination therapy, and examining whether Moringa plays a role in treating other cancers. Although the findings of laboratory and animal studies are encouraging, further *in vivo* and clinical evaluation is needed to validate these findings in practical applications. In conclusion, Moringa and its NPs demonstrate promising potential for translation into therapeutic agents for CC.

1. Introduction

Cancer occurs when aberrant cells divide uncontrollably and can infiltrate adjacent tissues, travelling through the lymphatic and circulatory systems to other parts of the body (Nabi and Le 2021). Specifically, colon cancer (CC) is one of the deadliest malignant diseases, with significant prevalence and death rates (Li and Lai 2009). In particular, CC develops due to alterations in the normal colonic epithelium, including the development of adenomatous polyps that can multiply and enlarge, resulting in genetic and epigenetic mutations that accumulate over time (Kasi et al. 2020). Most studies report that 5% of CC are caused by genes that a person inherits, mainly through conditions such as Lynch syndrome or familial adenomatous polyposis (FAP). The majority of CCs are random (Xiao et al. 2019).

Clinically, the most frequently encountered symptoms in CC are constipation and abdominal pain. Additionally, bloating and abdominal distension are significant clinical indicators, particularly in distal CC (Hirai et al. 2016). The global burden of CC is expected to rise by 60%, reaching roughly 2.2 million new cases and 1.1 million fatalities annually by 2030 (Wong et al. 2021). Similarly, like humans, animals are also affected, including dogs, and share common illnesses with humans, such as an increase in tumor-related colorectal disorders. In dogs, carcinoma of the colon accounts for around 60% of large intestine tumors (Herstad et al. 2021). Another study found spontaneous colon tumors commonly known as adenocarcinomas in dogs, which exhibit invasive development and potential for progression similar to those in humans (Wang 2019).

Various treatment protocols, including chemotherapy and radiotherapy, have been adopted to treat this lethal disorder. Chemotherapy is a conventional cancer treatment that uses chemicals to either kill or reduce the growth of cancer cells, but it can also cause side effects by affecting rapidly dividing normal cells, as shown in Fig.

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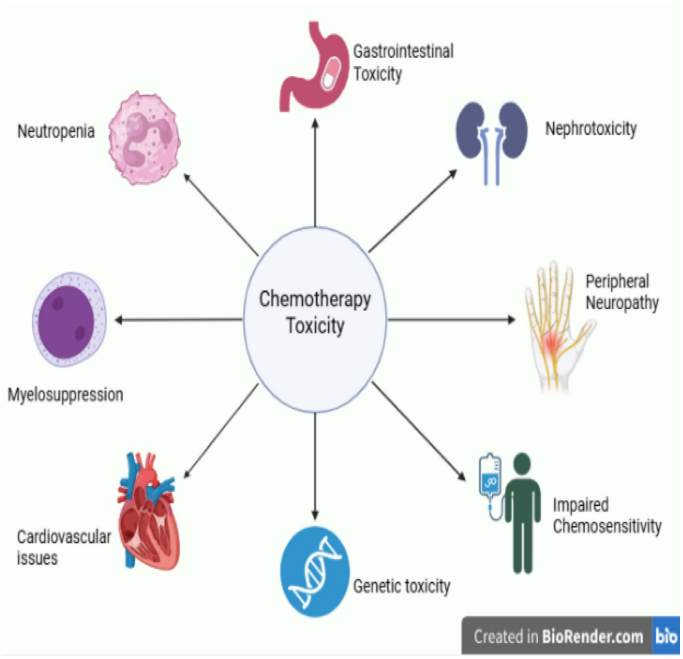

Fig. 1. *Toxicity of chemotherapy*

1 (Bahadoer et al. 2021). Neoadjuvant chemotherapy, while beneficial in tumor reduction, carries significant risks such as chemotoxicity, surgical delays, and complications such as intestinal obstruction or perforation (Gosavi et al. 2021). A study reported chemotherapy in dogs and their adverse effects, like vomiting, diarrhea, neutropenia, and inappetence (Cunha et al. 2017). In another study, chemotherapy adverse effects were recorded at least once in 124 canines, with serious consequences reported in fifty dogs. Twenty-three of them suffered gastrointestinal problems, while thirty-one dogs had myelotoxicity consequences. Severe adverse effects resulted in the hospitalization of thirty-seven dogs, the cessation of chemotherapy in twelve dogs, and the euthanasia or mortality of nine dogs (Chavalle et al. 2022). Other research demonstrated that epirubicin treatment in feline patients bearing tumors has been linked to a variety of side effects, the most notable of which include gastrointestinal toxicity, anorexia, and lethargy. In addition, a subgroup of cats demonstrated epiphora, upper respiratory symptoms, and immune system disorders such as hypersensitivity (Elliott et al. 2025). The most prevalent adverse effect of chemotherapy in feline recorded was neutropenia, which limit dose. Human patients and pet animal owners may also experience financial hardship as a result of chemotherapy because of the high expense of supportive care, drugs, and treatment (Nelson et al. 2020). Mode of action and limitations of most common chemotherapeutic agents is given in Table 1.

Radiotherapy is another significant clinical option for CC in addition to surgery and chemotherapy, particularly for patients with intermediate or advanced CC who are not fit for chemotherapy and have no likelihood of a successful outcome from surgery (Tan et al. 2022). However, its efficacy is limited due to the low radio-sensitivity of CC and the high risk of collateral damage to surrounding normal tissues. Radiation can cause elevated levels of pro-inflammatory cytokines, which can lead to tissue damage in non-tumoral areas (Ball et al. 2017). According to research, conventional fractionated definitive radiotherapy (CFDRT) caused moderate to severe acute cutaneous and gastrointestinal toxicity, with late consequences such as colorectal strictures, but hyper-fractionated radiotherapy caused lesser gastrointestinal symptoms such as colitis or diarrhoea in canines (Murakami and Rancilio 2025). The FLASH Radiotherapy for canines with mouth tumors is successful, although it generates substantial severe complications, particularly osteoradionecrosis when bone is in the course of the treatment field. Other concerns comprised necrosis and mucosal ulcers (Børresen et al. 2023). Similarly, another study found that acute radiation-induced negative impacts appeared in the skin and inside the mouth of dogs. Acute ocular adverse reactions included keratoconjunctivitis sicca (Mayer et al. 2019). Another study revealed that cats with sinonasal malignancies treated with radiotherapy applying a cyclical hypo-fractionated strategy obtained satisfactory tumour control but encountered moderate toxicities such as leukotrichia, alopecia, and ocular discharge (Frezoulis et al. 2022). Research demonstrated that in felines with squamous cell carcinoma of the mouth, radiation combined with zoledronate provided therapeutic advantages but also induced widespread acute toxicities such as dermatitis, mucositis, hyper-salivation, lethargy, and anorexia (Lundberg et al. 2022).

On the other hand, nanotechnology advances science and engineering by manipulating materials at the nanoscale from 1-100 nm and has demonstrated significant potential in medical applications, particularly in cancer therapy (Gowda et al. 2024). Nanomaterials are increasingly considered as the most effective therapeutic agents against a broad range of disorders (Mehwish et al. 2024). Nanoparticles (NPs) can improve cancer therapy by accurately delivering anticancer medications to tumour areas (Banazadeh et al. 2023; El-Dawy et al. 2023). Additionally, NPs have demonstrated a wide range of biomedical properties, such as antibacterial, anti-inflammatory (Ibrahim et al. 2024; Waliaveettil and Anila 2024), anticoagulant (Yenurkar et al. 2025), antioxidant (Samrot et al. 2022), and anti-diabetic activities (Loyola-Leyva et al. 2025). NPs can be formulated by using various sources, including artificial synthesis via top-down and bottom-up processes, as well as from the environment via volcanic ash, dust storms, anthropogenic, and other natural processes (Chimbekujwo et al. 2024; Ali et al. 2025). NPs can be synthesized using various techniques, including chemical methods (sol-gel, hydrothermal, solvothermal, vapour synthesis) (Khan 2020; Sati et al. 2025), biological methods (microbial, plant-based synthesis) (Alsaiari et al. 2023; Akhreim et al. 2024), and mechanical methods (milling, mechanical alloying) (Suryanarayana et al. 2022; Zhao et al. 2024).

Nanotechnology is also utilized for the treatment of pets. For instance, the nanorods of gold were utilized in photothermal treatment to treat breast cancer in cats. Hyaluronic acid nanomaterials coated with cisplatin and paclitaxel were used against dogs with oral sarcoma and glandular cancer (Al-obaidi et al. 2024). Similarly, another study reported biomedical applications of AgNPs that displayed significant antibiofilm activity against *Staphylococcus pseudintermedius*, which was isolated from dogs with otitis externa. The antifungal action of AgNPs was demonstrated against the fungal dermatophyte *Microsporum canis*, which caused ringworm in both dogs and cats (Frippiat et al. 2025).

In recent years, plant-derived nanoagents have emerged as promising candidates for colon cancer treatment due to their biocompatibility, targeted delivery, and ability to enhance the therapeutic potential of phytochemicals (López-Cabanillas Lomelí et al. 2024). For example, *Curcuma longa* (turmeric) NPs have shown enhanced cytotoxicity against CC cells through ROS generation and

Table 1. Modes of action and limitations of conventional chemical drugs in the treatment of colon cancer

Chemical drug	Chemical formula	Brand	Composition	Mode of action	Limitations	References
5-fluorouracil	$C_4H_3FN_2O_2$	Adrucil	5-FU,a pyrimidine analogue, forms by fluorinating uracil.	Disrupts deoxynucleotide pool. Impairing DNA fragmentation and RNA synthesis.	Myelosuppression, chemo-resistance, genetic and epigenetic variation, gastrointestinal toxicity.	(Vodenkova et al. 2020)
Capecitabine	$C_{15}H_{22}FN_3O_6$	Xeloda	Transformed by the body into its active form, 5-fluorouracil.	Converting capecitabine into 5FU, thymidine phosphorylase inhibits DNA synthesis.	Adverse toxicity in clinical settings, bioavailability issues, and limited efficacy.	(Masuda et al. 2017; Adebayo et al. 2023; Pouya et al. 2023)
Oxaliplatin	$C_8H_{14}N_2O_4Pt$.	Eloxatin (by Sanofi)	Platinum-based drug coordinated to two amine groups.	DNA cross-links, prevent transcription and DNA replication and cancer cells die.	Peripheral neuropathy, limited efficacy.	(Ma et al. 2021; Żok et al. 2021)
Irinotecan	$C_{20}H_{24}N_2O_5$	Camptosar	Camptothecin, lactone ring, piperidine ring, active metabolite SN38.	DNA to form a ternary complex with topoisomerase I, DNA strands leading to apoptosis.	Neutropenia, diarrhoea, tumor relapse.	(Liu et al. 2022; Saurav et al. 2024)
Cisplatin	$PtCl_2(NH_3)_2$	Platinol	Cis-diamminedichloroplatinum (II) square planner geometry, yellow–orange crystalline.	DNA cross-links, cisplatin prevents replication and induces apoptosis.	Dose-limiting toxicity, nephrotoxicity, cardiovascular issues, chemoresistance	(Casanova et al. 2021; Rottenberg et al. 2021; Ranasinghe et al. 2022)
Bevacizumab	$C_{6638}H_{10160}N_{1720}O_{2108}S_{44}$	Avastin	4 polypeptide chains, a chimeric monoclonal antibody, molecular weight of almost 149 KDa.	Preventing angiogenesis, Block VEGF, especially in metastatic colon cancer.	Impaired chemosensitivity, survival disparities, biological resistance, and limited effectiveness.	(You et al. 2020; Filippo et al. 2021; Taïeb et al. 2021)
Cetuximab	$C_{6484}H_{10042}N_{1732}O_{2023536}$	Erbitux	Two N-linked glycosylation Asn 88, 229, glycosylation features α 1-6 fucosylated structures.	Inhibits the attachment of Epidermal Growth Factor (EGF) by binding to the cell's EGF Receptor.	Induces mutations in HRAS, KRAS, and NRAS genes, change signalling pathways	(Váradi et al. 2020; Wu et al. 2020)
Fruquintinib	$C_2H_{19}N_3O_5$	FRUZAQLA	C-O coupling of 4-chloro-6,7-dimethoxyquinazoline , 6-hydroxy-N, 2-dimethylbenzofuran-3 -carbonyl amide	Obstruct the proliferation of colon cancer, modulation of epithelial-mesenchymal transition (EMT).	Gastrointestinal diseases, hypertension, and tiredness.	(Syaj and Saeed 2024; Song et al. 2025)

apoptosis induction (Venkatadri et al. 2020). Similarly, *Camellia sinensis* (green tea) based NPs have demonstrated significant tumor suppression via modulation of Wnt/β-catenin and NF-κB signaling (Letchumanan et al. 2025). In comparison, *M. oleifera* is also renowned for its diverse range of phytochemicals, which demonstrate unique biological activities (Hegazy et al. 2023; Bagheri et al. 2024; Camilleri and Blundell 2024). It is a medicinal plant from the family Moringaceae and known by common names, such as drumstick tree and horseradish tree (Klimek-Szczykutowicz et al. 2024). Moringa has been used as an efficient and well-known therapeutic agent in traditional medicine for centuries (Bebas et al. 2023). Moreover, among the 13 species of the Moringa genus, *M. oleifera* is the most researched and utilised species due to its pharmacological and phytochemical profile that is relevant to human health (Ma et al. 2020; Hamada et al. 2024). Among the bioactive substances of Moringa are the potent antioxidant vitamins A and C (Khan et al. 2023; Arshad et al. 2025) and polyphenolic compounds, such as kaempferol, quercetin, glycosides, terpenoids, tannins, and Saponins (Xie et al. 2024). The large levels of these antioxidants in Moringa determine its antioxidant activities (Garofalo et al. 2024), resulting in significant anticancer capabilities (Szlachetka et al. 2020) as well as hypotensive (Menichetti et al. 2025), anti-inflammatory (Shahbaz et al. 2024; Imran et al. 2023), antibacterial (El-Sherbiny et al. 2024), anti-ulcerous (Ibrahim and Al-Qadhi 2025), hypoglycaemic (Sahoo et al. 2024), and hypocholesterolaemic properties (Munir et al. 2025). For instance, the aqueous and methanol extracts of *M. oleifera* leaves have demonstrated a strong cytotoxic effect against various cancer cell lines, such as human HCT-116 colon cancer, murine melanoma B-16, and human colon carcinoma (Pappas et al. 2021).

In rats with DMBA-induced mammary cancer, the aqueous extract of *M. oleifera* leaves and benzyl isothiocynate dramatically suppressed tumour and decreased serum levels of IL-1β (Rojas-Armas et al. 2024). Similarly, research reported that Wistar rats with oral cancer were used

to examine the effects of *M. oleifera* leaf extract. *M. oleifera* limited oral cancer by lowering vascular endothelial growth factor (VEGF) and preventing the growth of blood vessels (Hartono et al. 2019). Another study evaluated that extract from *M. oleifera* leaves and pods improved blood counts and dramatically decreased chemically caused skin cancer in mice (Saradha et al. 2024). Moreover, another research found that the ethanol-based extract of *M. oleifera* has anti-leukemic properties that reversed the severe anaemia and significant leucocytosis induced by benzene (Akanni et al. 2014).

M. oleifera has been widely used in the synthesis of various NPs, such as gold (Au), silver (Ag), iron (Fe), copper oxide (CuO), zinc oxide (ZnO), magnesium oxide (MgO), nickel oxide (NiO), bismuth, and cerium oxide (CeO) (Aslam et al. 2023; Perumalsamy et al. 2024). Studies revealed that *M. oleifera*-synthesized silver NPs have strong, broad-spectrum antimicrobial properties (Mohammed and Hawar 2022), while AuNPs derived from *M. oleifera* exhibited substantial antidiabetic, antioxidant, and anticancer effects (Kiran et al. 2021). ZnONPs demonstrated moderate to good antibacterial efficacy (Kalaiyarasi et al. 2023; Maqsood et al. 2023), and CuONPs displayed selective cytotoxicity against cancer cells like MCF-7 breast cancer cells, while sparing normal cells such as NIH/3T3 (Suardana et al. 2024; Barani et al. 2024). This selectivity makes CuO NPs a promising therapeutic option for minimising side effects during chemotherapy for cancer (Sarani et al. 2024). Similarly, MgO NPs demonstrated powerful antioxidant properties, which effectively scavenge oxidant radicals and reactive oxygen species (ROS). One of their potential therapeutic applications is treating diseases associated with oxidative stress, such as cancer and cardiovascular diseases. MgONPs have displayed dose-dependent toxicity against PA-1 cancer cells (Vijayakumar et al. 2023). Thus, the inclusion of both Moringa extracts and Moringa-derived nanoparticles provides a comprehensive view of its therapeutic potential. While extracts offer natural bioactive compounds, nanoparticles enhance bioavailability, targeting efficiency, and anticancer activity, making them promising candidates for colon cancer therapy. This review critically explores the anticancer mechanisms and potential applications of *Moringa oleifera* and its biosynthesized nanoparticles in the treatment of CC, with particular attention to their molecular targets, pharmacodynamics, and future research directions in humans and animals.

2. Synthesis of Moringa nanoparticles

The biofabrication of metal NPs using Moringa plant extract consists of three important steps, as illustrated in Fig. 2 (Barman et al. 2023). The process begins with the reduction phase, during which the metal ions are reduced to form small clusters of metal atoms, which are called the primary particles (Virk et al. 2023). This is followed by the growth phase, where these initially unstable particles undergo heterogeneous nucleation and subsequently aggregate into larger, more thermodynamically stable structures (Perumalsamy et al. 2024; Sun et al. 2024). The final step is termination, which marks the point where these NPs attain their definitive morphology and stability. This structural stabilization is largely attributed to the Moringa plant extract, which acts as both a reducing and capping agent, effectively preventing further agglomeration and maintaining nanoparticle integrity (Vidaarth et al. 2024).

3. Mechanism of action of Moringa and its NPs

Moringa and its biosynthesized NPs exhibit significant anticancer properties through the induction of cell cycle arrest, apoptosis, oxidative stress, and modulation of oncogenic signalling pathways in colon cancerous cells, as illustrated in Fig. 3.

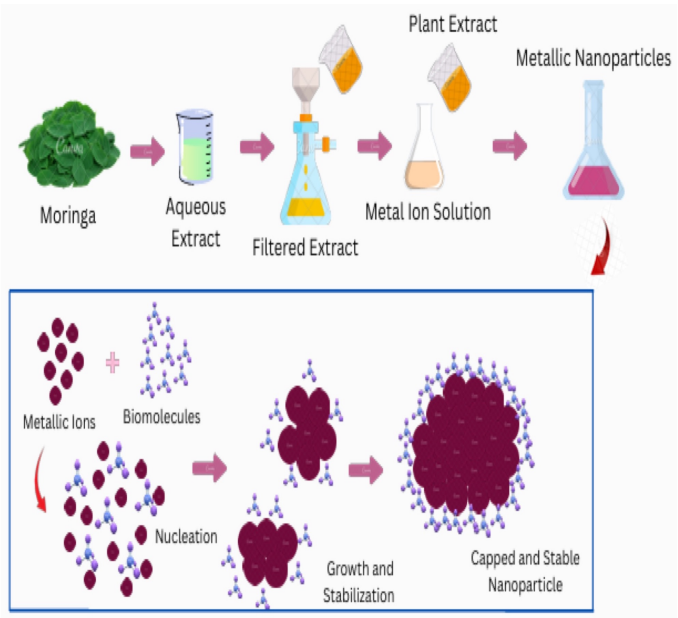

Fig. 2. *Biosynthesis of nanoparticles from Moringa oleifera*

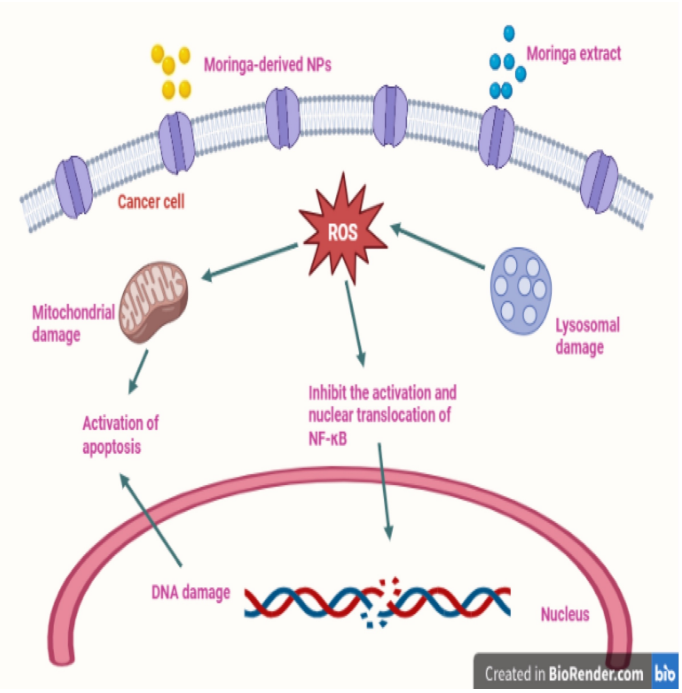

Fig. 3. *Mechanisms of action of nanoparticles synthesized from Moringa against colon cancer*

3.1. Induction of cell cycle arrest

Cell cycle arrest occurs when oxidative damage and redox imbalance exceed the cellular tolerance threshold. This stress causes DNA damage and eventually stops the cell cycle, which prevents the multiplication of cancerous cells (Hernandes et al. 2023). *M. oleifera* extracts have antiproliferative properties due to isothiocyanates produced from glucosinolate hydrolysis. These chemicals triggered apoptosis and

disrupted the cell cycle by affecting signalling pathways like NF-κB (Cuellar-Núñez et al. 2020). Moringa peptides promoted Caco-2 cell cycle arrest, mostly through membrane rupture and internal interaction with cancer-related proteins and DNA (Avilés-Gaxiola et al. 2024). Silver (Ag) NPs derived from *M. oleifera* leaves showed strong cytotoxic activity against SW480 and HCT116 colon cancer cells by supporting their ability to effectively limit cancer cell proliferation (Althomali et al. 2022). The study found that *M. oleifera* leaf extract supplemented with Ag NPs demonstrated potent anti-cancer properties against azoxymethane-induced CC in rats, leading to improved biological efficiency, potentially causing cell cycle arrest in cancer cells (Aboulthana et al. 2021).

3.2. Induction of apoptosis

Apoptosis is a key mechanism through which tumor cells undergo cell death. Oncological treatment is greatly dependent on apoptosis since many pharmacological agents target the induction of apoptosis in cancer cells to decrease tumor volume and inhibit metastasis (Xie et al. 2020). The extract of *M. oleifera* induces apoptosis in HT29 CC cells by disrupting mitochondrial membrane potential, a pivotal event in the apoptosis process (Reda et al. 2020). Furthermore, the anti-proliferative effect of *M. oleifera* Lam. leaf extract was attributed to the inhibition of ERK1/2 phosphorylation, a key signaling pathway involved in cellular proliferation and survival, which enhances apoptosis in cancer cells (Tragulpakseerojn et al. 2017). Moreover, Cuellar-Núñez et al. (2020) observed that *M. oleifera* extract increases apoptosis by restricting the AKT Signaling pathway in HCT-116. A similar trend was demonstrated in another study by Kraiphet et al. (2018), where boiled *M. oleifera* pods induced apoptosis in colon cancerous cells, mediated by mitochondria in the AOM/DSS mouse model. Increased BAX and decreased BCL-2 values triggered tumor cell death and reduced metastasis.

Similarly, Ag NPs Moringa leaf powder downregulated oncogenes that manage parts of the cell cycle, cell growth, and invasiveness of cancer cells such as Ki-67, Wnt, β-catenin, Cyclin D1, TGF-β, and Snail, mainly through ROS production pathways in CC cells (Susanto et al. 2024). NiO NPs synthesized using *M. oleifera* caused overproduction of ROS and oxidative stress in the body, leading to a significant apoptosis in CC cells (Ezhilarasi et al. 2016). *M. oleifera* Ag NPs exhibited very high toxicity to HCT-116 cells with an IC50 of 6.51 μg/ml and it causes cell death by triggering the cascade of apoptosis (Abdel-Rahman et al. 2022). Moreover, Ibrahim et al. (2022) observed that *M. oleifera* extract and *M. oleifera* extract-Ag NPs, separately and together, significantly increased the number of apoptotic nuclei and upregulated the tumor-suppressor p53 protein in HT-29 human CC cells (Ibrahim et al. 2022).

3.3. Induction of Oxidative stress

According to Hayes et al. (2020), a high amount of reactive oxygen species (ROS), such as hydroxyl radicals, hydrogen peroxide (H_2O_2), and superoxide, compared to antioxidant defense capacity, results in various diseases, including cancer. Furthermore, *M. oleifera* triggered increased oxidative stress in HT29 CC cells by increasing ROS production and, therefore, resulted in impaired mitochondrial activity, reduced ATP production, and subsequent apoptotic cell death (Reda et al. 2020). These effects were further enhanced by the hexane fraction of *M. oleifera* extract (Jinghua et al. 2018). The exposure to glucosinolate-rich hydrolysed extract (GEH) of *M. oleifera* induced oxidative stress and subsequent apoptosis in CC cells (Shafiq et al. 2024; Cuellar-Núñez et al. 2020). Ag NPs synthesized using *M. oleifera* modified the key genes related to pathways linked to cell proliferation, DNA damage, and oxidative stress (Avilés-Gaxiola et al. 2024). Furthermore, the Moringa Ag nano-extracts restored biochemical parameters to their normal values, thereby reducing the side effects of oxidative stress in azoxymethane (AOM)- induced colorectal cancer models (Aboulthana et al. 2021).

3.4. Oncogenic signalling pathway suppression

Oncogenic signalling pathways, such as PI3K/Akt, NF-κB, and ERK, are upregulated in cancer, promoting tumorigenesis, metastasis, and resistance to therapy (Sanchez-Vega et al. 2018). The *M. oleifera* extract was shown to interfere with the NF-κB and ERK pathways and simultaneously activate Nrf2 antioxidant defence system, thereby hindering the growth and maturation of CC cells (Sodvadiya et al. 2020). According to Tragulpakseerojn et al. (2017), the *M. oleifera* extract was found to restrain the growth of HCT116 cells by downregulating the ERK1/2 phosphorylation and AKT expression. The, *M. oleifera*-AgNPs altered the expression of key genes involved the development of colon carcinogenesis, such as downregulation of LRP6 and upregulation of LRP5 expression (Althomali et al. 2022). Similarly, downregulation of Ki-67 gene expression, associated with cancer cell growth, was observed on treatment with increasing concentrations of *M. oleifera* leaf powder-AgNPs, with effective suppression of the survival of colorectal cancer cells at a concentration of 1414 μg/mL (Susanto et al. 2024). Furthermore, Sayed et al. (2021) revealed that ZnONPs interrupted PI3K/Akt and NF-κB signalling, inhibiting cell growth and causing apoptosis in cases of colorectal cancer. These NPs also disrupted angiogenesis and migration of endothelial cells, which curtailed the blood supply and metastasis of cancer (Liu et al. 2021). The mode of action of different Moringa extracts against colon cancer is given in Table 2.

4. Anticancer effects against colon cancer in animals

Researchers have investigated the anticancer properties of Moringa extracts and their NPs formulations against different kinds of cancers in animals. According to a study, lymphoma has been reported at high prevalence in cats and dogs, weakening their immune systems. However, *M. oleifera* induced the apoptosis of the cancerous cells by expressing the Caspase-3 gene when treating the EL4 murine lymphoma cell line at 40 μg/ml (Shekarabi et al. 2025). Similarly, adenocarcinomas are also among the most frequent neoplasms reported in cats and dogs (Negoescu et al. 2025). Nevertheless, *M. oleifera* inhibited the growth of adenocarcinoma in urethane-induced lung cancer rats by upregulating glutathione, superoxide dismutase, EGFR-mRNA, and downregulating malondialdehyde (Ibrahim et al. 2023). Similarly, multiple studies have elaborated on the effect of *M. oleifera* extract against the CC. For instance, a study conducted by Phannasil et al. (2020) revealed that *M. oleifera* pods showed therapeutic potential against AOM/DSS-induced colon carcinogenesis in mice via regulating tumor-related proteomes. Similarly, Moringa leaf extract modulated AOM/DSS-associated CC in rodents through anti-inflammatory processes in colon tissues (Cuellar-Núñez et al. 2021). Furthermore, *M. oleifera* leaf extract alleviated colonic injury, which is a risk factor for CC, induced by sodium nitrate in a rat model by suppressing oxidative stress, hyperproliferation, and apoptosis, indicating its chemotherapeutic potential (Hassan et al. 2024). According to another study, in DMH-induced Sprague Dawley rats, the neoplastic lesion of CC was inhibited by *M. oleifera* leaf extract through lessened aberrant

Table 2. Modes of action of Moringa extracts against colon cancer

Plant part	Extraction solvent	Dose	Cancer cell line/ Animal model	IC_{50} Value	Mode of action	References
Leaf	Methanol	0.55 mg/mL	HCT-116, HT-29	0.17 mg/mL, 0.19 mg/mL	Induce apoptosis by blocking the AKT Signalling pathway	(Cuellar-Núñez et al. 2020)
Leaf	dd-H_2O	0.3mg/mL	HT-29	-	Induce apoptosis by losing mitochondrial membrane potential	(Reda et al. 2020)
Leaf	100% Methanol	44.02 µg/mL	HCT-116	-	Induce apoptosis by suppressing ERK1/2 phosphorylation and lowering pro-survival signals	(Tragulpakseerojn et al. 2017)
Pod	Water	3.0% (diet)	Mouse model	-	Induce apoptosis via lowering BCL-2 levels and raising BAX expression	(Kraiphet et al. 2018)
Leaf	Methanol	0.55mg/mL	HCT-116, HT-29	0.17 mg/mL, 0.19 mg/mL	Trigger cell cycle arrest by affecting signalling pathways like NF-Kb	(Cuellar-Núñez et al. 2020)
Leaf	Tris-HCL buffer	500µg/mL	Caco-2	-	Promote cell cycle arrest through membrane rupture and internal interaction with cancer-related proteins and DNA	(Avilés-Gaxiola et al. 2024)
Leaf	Hexane	100-200 µg/mL	HT-29	-	Oxidative stress triggers mitochondrial apoptosis	(Jinghua et al. 2018)
Leaf	Methanol	0.55 mg/mL	HCT-116, HT-29	0.17 mg/mL, 0.19 mg/mL	Oxidative stress leads to mitochondrial damage and apoptosis	(Cuellar-Núñez et al. 2020)
Leaf	dd-H_2O	1.8 mg/mL	HT-29	-	Induce oxidative stress	(Reda et al. 2020)
Leaf	Methanol	6% (w/w)	Mice model		Suppressing NF-κB and ERK signalling pathways	(Sodvadiya et al. 2020)
Leaf	100% Methanol	19 µg/mL	HCT-116	-	Downregulation of ERK ½ phosphorylation decreases AKT expression	(Tragulpakseerojn et al. 2017)

crypt foci, regulation of liver/kidney markers, and rebuilding tissue architecture, thus offering chemopreventive and protective action versus carcinogenesis (Kumawat and Une 2024). Moreover, the CC progression was reduced by *M. oleifera* AgNPs in male Wistar rats through inhibiting angiogenesis and cell invasion. MO-AgNPs also inhibited endothelial tube formation, micro-vessel sprouting, and spheroid growth, therefore, reducing vascularization of the tumors, cancerous invasion, and metastasis (Al-Shalabi et al. 2025). According to another study, in an AOM-induced rat model, *M. oleifera* nano-extract inhibited CC by reducing biochemical parameters to normal, increasing the expression of tumor protein 53 and Adenomatous polyposis coli (APC), restoring antioxidant enzyme gene patterns, and preserving the histology of the colonic tissue (Aboulthana et al. 2021). Furthermore, in the rat model, *M. oleifera* leaf silver nano-extract repressed CC progression by complementing antioxidant defence, escalating polyphenolic level, enhancing free radical scavenging action, and was further amplified with the higher cytotoxicity against CC cells via effective bioavailability of phytochemicals (Shousha et al. 2019; Luecha et al. 2024). Hence, *M. oleifera* and its NPs show significant potential for preventing colon cancer in animal studies by boosting antioxidant defences, promoting apoptosis, reducing inflammation, and inhibiting angiogenesis and metastasis, underscoring their potential as natural, multi-targeted agents against cancer. However, there is still a limited amount of research that specifically targets Moringa extract and NP-based treatments for colon cancer in domestic animals.

5. Challenges and limitations

The variability in cytotoxic efficacy between colon cancer cell lines (HCT 116 more sensitive than Caco-2) with Moringa leaf extract-loaded PLGA-CS-PEG nanoparticles was demonstrated by a study showing dose-dependent decreases in viability of HCT 116 but resistance in Caco-2 cells. The bioavailability and delivery challenges are linked to the use of nanoparticles like PLGA-CS-PEG that improve stability and controlled release, but comprehensive in vivo data are still limited, as referenced in studies including those that tested nanocomposites of Moringa extracts (Abd-Rabou et al. 2017). Toxicity and safety profiles show minimal effects on normal cells in some studies; for example, cytotoxicity was selective toward cancer cells with lower impact on normal cells like BHK-21 or CD34+ hematopoietic stem cells. This suggests a potentially favourable safety profile, but more data is needed. Standardization issues arise from the complex phytochemical composition, as seen in studies comparing various Moringa extracts (ethanolic, aqueous, sequential extraction) with differential cytotoxic effects due to varying active metabolites. Mechanistic insights into apoptosis and gene expression modulation are still developing. Still, studies have shown that Moringa leaf extracts and their nanoparticles induce apoptosis and cell cycle arrest through molecular pathways involving caspase and BID proteins in cancer cells (Khor et al. 2020).

6. Future perspectives

Future research on *Moringa oleifera* and its nanoparticle (NP) derivatives should focus on four critical directions. First, preclinical and clinical trials shall be mandatory to evaluate the long-term safety, pharmacokinetics, and therapeutic efficacy of Moringa and its NPs in the treatment of colon cancer. Second, a deeper understanding of their molecular mechanisms is needed through advanced studies targeting key signalling pathways and gene expression profiles. Third, optimising Moringa NP synthesis processes to improve their stability, biocompatibility, and tumor targeting capabilities. Fourth, investigating Moringa-based combination therapies with other anti-cancer medications or treatments to yield synergistic effects, potentially overcoming drug resistance and improving therapeutic outcomes. Finally, investigating the potential of Moringa and its NPs in other cancer types may reveal broader anticancer potentials, supporting their development as versatile, multi-targeted therapeutic agents.

7. Conclusion

Moringa and its nano-formulations exhibited powerful antiproliferative properties in colon cancer by inducing cell cycle arrest and apoptosis through ROS-mediated oxidative stress, while blocking oncogenic pathways, such as Wnt/β-catenin. Moringa-based nanoparticles, particularly AgNPs, enhance these effects by amplifying cytotoxicity, modulating gene expression, and inhibiting proliferation and metastatic indicators. Despite promising *in vitro* and *in vivo* findings, the translation of these outcomes to clinical practice is hampered by the lack of clinical trials. Therefore, extensive *in vivo* research and well-controlled clinical trials in humans and animal models are essential to validate the therapeutic efficacy and safety of Moringa and its NPs. Collectively, the multi-targeted mechanisms of action underscore Moringa's potential as a promising candidate in colon cancer therapy and warrant further investigation and development to fill the treatment gaps in chemotherapy-resistant CC.

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