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Review Article

Anticancer for Natural Remedies

Natural Remedies: A Potential Cancer Treatment

Dr. Saripilli Rajeswari

Associate Professor, Centurion University of Technology and Management, School of Pharmacy, Balangir Campus, Balangir - 767001, Odisha, India.

Abstract: The discovery and development of multiple medications for the medical management of multiple kinds of cancer depend heavily on organic components. By modifying multiple pathways, including cellular proliferation, differentiation, apoptosis, angiogenesis, and metastasis, these phytochemicals have shown anticarcinogenic capabilities by preventing cancer onset, expansion, and spread. Treating complex disorders like cancer using drugs focusing on one aspect may have minimal efficacy and frequently result in poor results. Many epidemiological investigations have demonstrated a strong correlation between a regular intake of fruits and vegetables and a lower risk of cancer. Plants, herbs, and other natural items have been employed as therapeutic agents since ages ago. Similarly, most of the components used in medicines nowadays originate from natural sources. Despite successes, creating bioactive substances and pharmaceuticals using organic substances continue to be difficult, largely due to the issue of large-scale sequestration and mechanistic understanding. Researchers are possibly reaching a turning point when they should reassess strategies to discover promising natural products and explore their effectiveness as therapies due to substantial advancements in cancer therapy and the increased usage of modern technology. With an emphasis on solid cancer's root causes and molecular pathways, we review current natural product-based oncology research advancements, what they do, and how to generate innovative systemic therapies.

Keywords: Anticarcinogenic, Metastasis, Natural Remedies for Cancer, Cancer Therapies, and Apoptosis.

***Corresponding Author**

Dr. Saripilli Rajeswari , Associate Professor, Centurion University of Technology and Management, School of Pharmacy, Balangir Campus, Balangir - 767001, Odisha, India.

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11. INTRODUCTION

From prehistoric days, natural ingredients extracted from medicinal plants have been utilized to cure several illnesses. Mesopotamia used herbal remedies with a curative reason for the initial time around 2600 BC[1]. Well, enough conserved are the "Ebers Papyrus" records from 1550 BC, which include over 700 different medications. The medication made from natural ingredients development has several obstacles, including limited stock, difficulties identifying bioactive chemicals, collecting untamed specimens, and difficulty with high throughput screening (HTS). To overcome these obstacles, researchers have begun to utilize synthetic biology techniques to produce natural product-based drugs. This strategy is often more cost-effective and efficient than traditional drug development techniques. However, it is challenging for the assessment precise molecular mechanism of action of natural compounds. Due to these challenges, pharmaceutical businesses have immediately focused on chemical additives to discover novel drugs. Using synthetic biology techniques, researchers can more accurately control the production of natural product-based drugs and better assess their molecular mechanism of action. This approach is often more cost-effective and efficient than traditional drug development techniques, which has shifted focus away from organic sources and towards chemical additives for locating new drugs. Regardless of the type of tumor, recent research indicates that most, by a wide margin, cancer occurrences are brought along by variations in the synthesis of proteins and mutations in the genes that encode proteins. Development hormones and related interactions, anti-apoptotic proteins, tumor suppressors, and transposable elements are a few cell signaling proteins that are changed in cancer and may serve as therapeutic targets. A tumor is brought on by cells growing improperly that can invade or distribute to several bodily regions. This abnormal growth results from genetic material modifications of the cells, which can be inherited or acquired through exposure to a mutagenic substance or radiation. Cancer cell expansion and growth are naturally constrained by controlled cell death, including autophagy and apoptosis. However, when these control mechanisms are impaired, the tumors can keep expanding and developing, leading to a potentially life-threatening condition.

2. QUERCETIN

A polyphenolic phytonutrient flavonoid called quercetin, which originates from cereals, fruits, and vegetables, increases E-cadherin while suppressing vimentin and N-cadherin reversing the epithelial to mesenchymal transition[2]. In kidney and liver malignancies, quercetin lowers TNF-, IL-[1], and iNOS expressions and protects DOX-induced nephrotoxicity[3]. In the case of prostate cancer, it increases TGF—induced downstream gene levels while decreasing VEGF and Ki67 expression[4]. Moreover, quercetin prevents lung cancer by inhibiting A549 cell migration and boosting Bcl2/Bax-mediated apoptosis[5]. Moreover, several studies have demonstrated that controlled distribution using nanotechnology improved the anticancer efficacy of quercetin[3]. The structure of quercetin has been depicted in Figure 1.

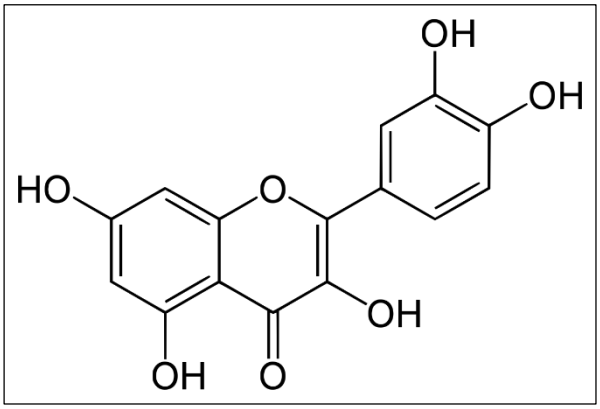

Fig 1: Structure of Quercetin

3. SILYMARIN

Silymarin is an extract of the milk thistle (Silybum marianum) which exhibits anticancer effects on N-Nitrosodiethylamine (NDEA)-induced rat hepato-carcinogenesis by reducing the extent of lipid peroxidation, decreasing MMP-2, MMP-9, and COX-2 levels, restoring GSH levels and various antioxidant enzymes activities[6]. In addition, Silibinin suppresses the spread of MDA-MB-231 cells while inducing death in MCF-7, inhibiting the growth of breast cancer cells by inhibiting replication within the insulin growth factor receptor (IGFR) [7]. In addition, Silymarin has been found to inhibit the growth of several other separate types, including pancreatic and prostate cancer. Furthermore, Silymarin showed anti-inflammatory and anti-angiogenic properties, possibly contributing to its anti-cancer effects. It also inhibits mitomycin C-human melanoma caused by accelerated cell death—a 375- S2 cells by downregulating p53 and Bcl-2 mediated apoptosis. Silibin is also excellent against G1 tract cancers. The structure of Silymarin has been depicted in Figure 2. Moreover, it was shown to have anti-oxidant, anti-mutagenic, and anti-viral properties, suggesting it may be beneficial in opposition to several cancers and many illnesses. For example, inhibiting p53 and Bcl-2-mediated mortality prevents mitomycin C-induced cell death in human melanoma A 375-S2 cells. Additionally, a helpful in treating G1 tract tumors is Silibinin. Silibin is additionally known to possess anti-oxidant, anti-mutagenic, and anti-viral characteristics, indicating it could prove helpful in the fight against various cancers in additional illnesses[9,10].

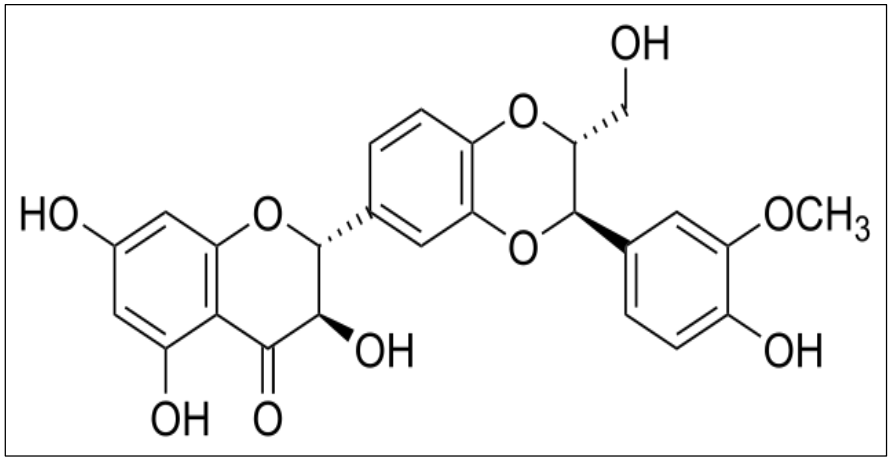

Fig: 2 - Structure of Silymarin

4. INDOL-3-CARBINOL (I3C)

Additionally, indol-3-carbinol, mostly found in cruciferous vegetables like broccoli, cauliflower, and cabbage, has anticancer properties[11]. I3C has reportedly been shown to have anticancer properties by using a new method, which includes reduced cell growth. Diindolylmethane (DIM) and I3C were shown to suppress the G1 cell process stage in breast and prostate cell lines for malignancy, according to previous research[11]. I3C can slow the spread of tumors, which could lead to its potential use as an anticancer agent. However, further research is needed to understand more about the functioning of I3C on cancer cells. This research suggests that I3C could potentially impede the emergence of cancer cells by suppressing the G1 cell process stage. It could lead to better treatments for cancer and a greater understanding of the mechanisms of I3C on cancer cells. More investigation is required to comprehend how I3C can be used in the fight against cancer (Figure [01]). To truly maximize the potential of I3C in cancer treatments, further research is needed to explore the full extent of its mechanisms and effects on cancer cells.

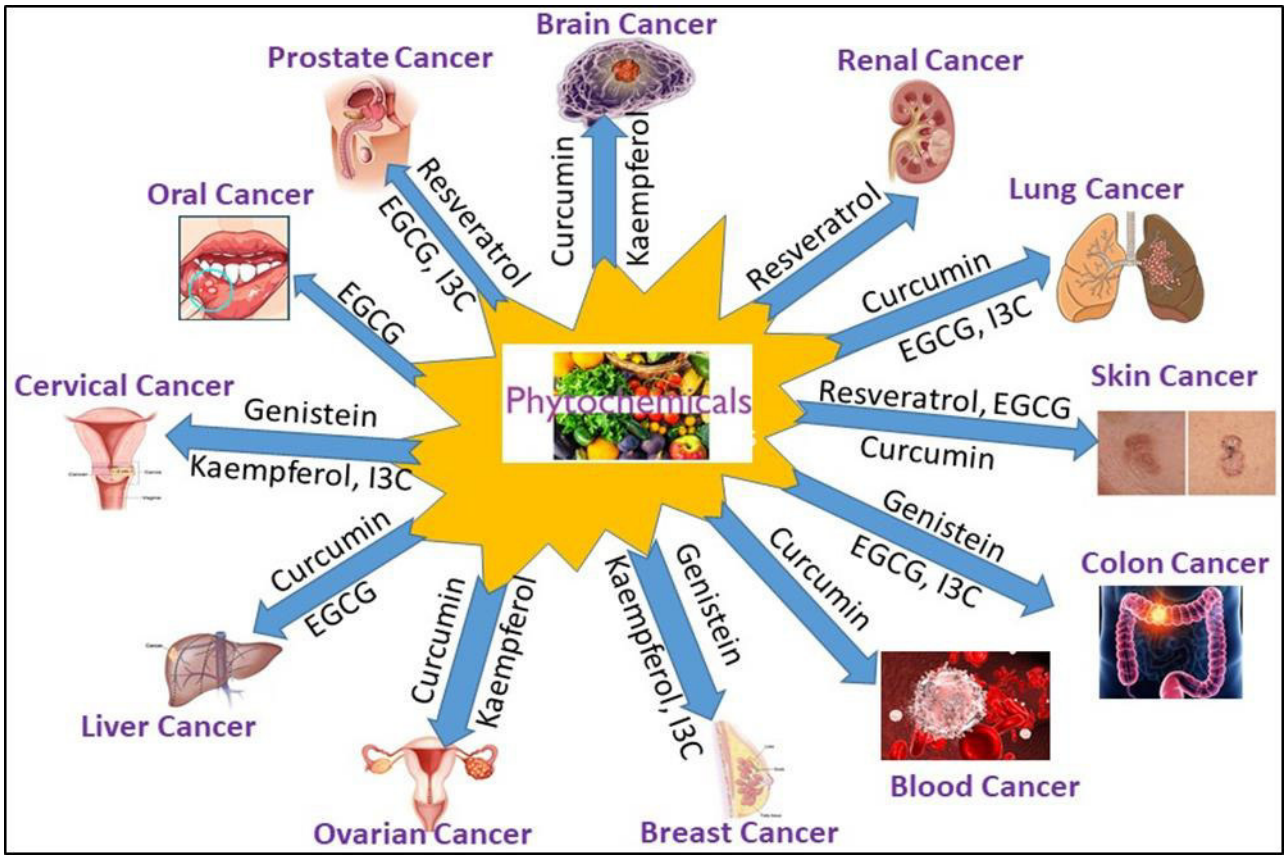

Fig 3 Pictorial representation of cancer types that could be prevented/managed by natural products (phytochemicals). IC3, Indol-3-carbinol.[12]

5. RESVERATROL

Two phenol rings are connected by an ethylene bridge in the naturally occurring polyphenol (stilbenoid) resveratrol (3, 5, 4-trihydroxystilbene). It exists in two different isomeric types: cis- and trans-resveratrol, with the former being the more common. This polyphenol effectively slows the emergence of individual colon cancers by upregulating pro-

apoptotic Bax and downregulating anti-apoptotic Bcl-2, according to a new research study[13]. Additionally, it inhibits the emergence of human HCT116 cells through sirtuin-mediated NF-B suppression. Polyphenol also reduces inflammation and oxidative stress, which further helps prevent cancer's emergence. It also has anti-aging effects and can help to improve heart health[14]. The most frequent disease related to the skin is skin cancer, and several studies have found that resveratrol may be able to treat it. Resveratrol exhibits its anticancer properties by either inducing apoptosis and autophagy or inhibiting angiogenesis and metastasis. Additionally, polyphenol is a potent antioxidant, scavenging free radicals that can damage DNA and cause cell death. These characteristics have links to the prevention of multiple chronic illnesses, including heart disease, type 2 diabetes, certain types of cancer, and neurodegenerative diseases. By increasing the activity in the dehydrogenase pyruvate complex in colon tumor cells, it may also mitigate the Warburg effect. Resveratrol's antitumor effectiveness is also an outcome of the phase-I antioxidant enzyme cytochromes P450 metabolic process being hindered. The structure of Resveratrol has been depicted in Figure 4. Moreover, resveratrol has been found to modulate signaling pathways and alter gene expression, suggesting it has the potential to be a cancer chemopreventive agent[15-17].

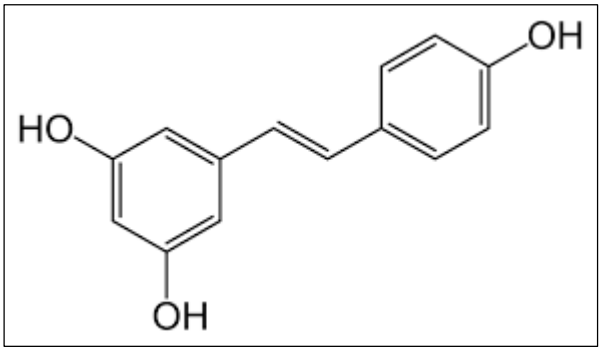

Fig: 4 - Structure of Resveratrol

6. MELATONIN

Animals' pineal glands release the neurohormone melatonin (N-acetyl-5-methoxy tryptamine), which has plenty of physiological impacts, including the control of circadian rhythm and antioxidant capacity. Melatonin stimulates mitochondrial dysfunction-mediated apoptosis, reducing pancreatic ductal adenocarcinoma (PDAC) when combined with sorafenib[18]. Inhibition of PDAC progression was observed in animal models with the process of synthesis of melatonin and sorafenib, suggesting that these two compounds could be a promising therapeutic approach for pancreatic cancer. Furthermore, according to research, melatonin inhibits MMP-9 catalytic function via communication with its online presence in a gastric cancer cellular line[19]. Therefore, it suggests that melatonin can potentially prevent the emergence of pancreatic cancer by blocking the way it acts of MMP-9, which is a major factor in the emergence of this cancer[20]. The structure of Melatonin has been depicted in Figure 5. Furthermore, combining melatonin and sorafenib is more effective than the two compounds alone, making it an attractive option for treating pancreatic cancer.

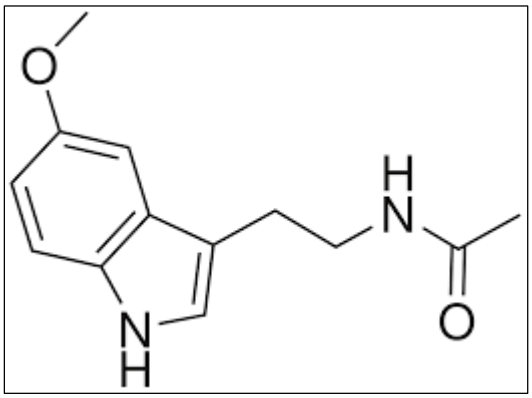

Fig: 5 - Structure of Melatonin

7. TAURINE

Sulfur-containing taurine (2-aminoethanesulfonic acid) is a common amino acid found in animals. By triggering a process influencing cascade of apoptosis, it reduced mammary tumorigenesis[21]. Taurine significantly reduced the widening of Nasopharyngeal Carcinoma (NPC) cells and promoted cell demise by triggering ER and mitochondrial stress-mediated mortality. Moreover, apoptosis-related genes and proteins influence mitochondrial dysfunction and caspase activation[22]. Taurine was revealed to be an effective agent in reducing cancer cells, making it a potential therapeutic target for NPC. Furthermore, taurine was noticed to induce autophagy, an essential process for cancer cells' survival and development, which may contribute to its anti-tumorigenic effects. The structure of Taurine has been depicted in Figure 6.

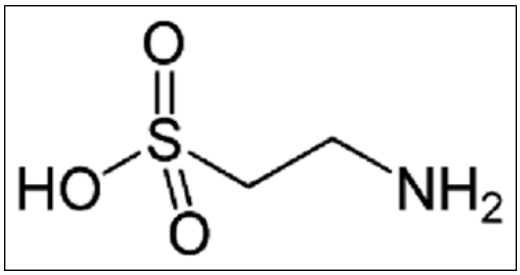

Fig:6 - Structure of Taurine

8. ASTAXANTHIN

Astaxanthin, the king of carotenoids, acts by downregulating the manifestation of Bcl-2, p-Bad, survival (antiapoptotic), and upregulating the manifestation of Bax and Bad (proapoptotic), Smac/Diablo, and cytochrome-c, astaxanthin, influences apoptotic cell death[23]. Apoptotic death was further verified with the turning on of caspase-3, caspase-9, and caspase-8[24]. It suggests that astaxanthin could be utilized as a possible treatment agent for cancer. Treatment with astaxanthin, lutein, and beta-carotene jointly led to apoptosis and slowed the enlargement of MCF-[10]A. By causing cell cycle arrest, astaxanthin inhibited the enlargement of and generated cell apoptosis. Activating caspases indicates apoptosis, which is the programmed death of cells. The study's result suggests that astaxanthin could cause cancer cells to go into demise, which would help impede the spread of malignant cells and possibly even lead to their death. The combination of astaxanthin with other compounds, such as lutein and beta-carotene, was also proven to be efficient in inducing apoptosis and slowing the growth of the cells[25]. The structure of astaxanthin has been depicted in Figure 7. Therefore, astaxanthin is an effective compound for triggering caspase activation, which is crucial for inducing apoptosis and ultimately reducing The proliferation of tissue cancer cells.

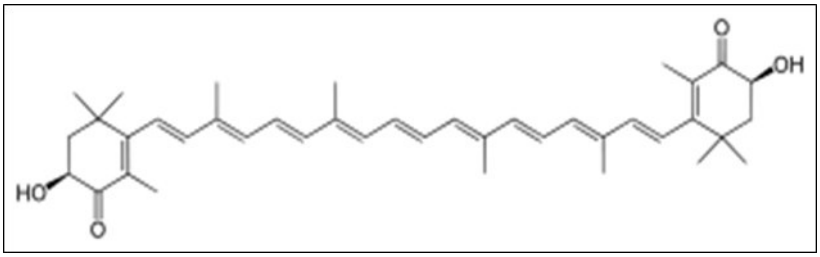

Fig: 7 - Structure of Astaxanthin

9. TANNIC ACID

By lowering the amount of MMP-2 and MMP-9 and increasing E-cadherin activity, tannic acid used to treat cells with prostate cancer inhibited the capacity to invade and migrate[26]. Tannic acid suppresses cell growth in gingival squamous cell carcinoma by arresting the cell phase. Tannic acid also inhibited cell migration and invasion in oral cancer cell lines[27]. These findings suggest that tannic acid can be a potential therapeutic agent for treating oral cancer. A study has demonstrated that acid inhibits the way it acts of MMP-2 and MMP-9, which are enzymes that help cancer cells invade and migrate. It also increases how it acts of E-cadherin, which helps keep cells together[28]. The structure of Tannic Acid has been depicted in Figure 8. By inhibiting the way, it acts of these enzymes and increasing the way it acts of E-cadherin, tannic acid can help to prevent the spread of cancer cells.

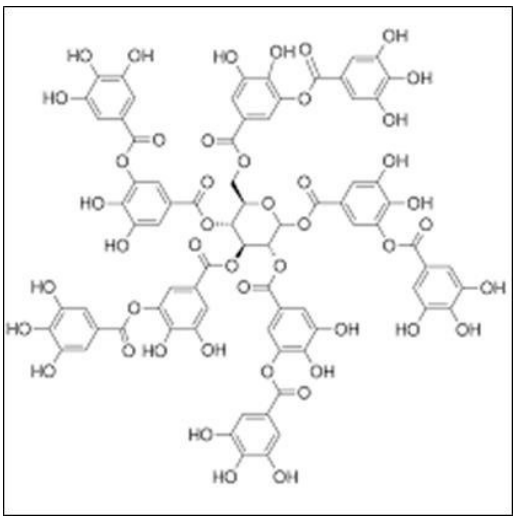

Fig: 8 - Structure of Tannic Acid

10. CURCUMIN

Malignant pleural mesothelioma (MPM), a disease brought on by asbestos exposure, may be handled with curcumin with little to no side effects by inhibiting Bcl-2, curcumin, and gallic acid caused apoptosis in MDA-MB-231 cells[29]. It indicates that curcumin and gallic acid can effectively treat MPM with few side effects, providing hope for those suffering from this deadly disease. In laboratory tests, curcumin and gallic acid reduced the manifestation of Bcl-2. This protein is responsible for inhibiting apoptosis and increasing the manifestation of p53, a protein necessary for initiating apoptosis[30]. It suggests that curcumin and gallic acid can induce apoptosis in MPM cells, thus providing an effective treatment option with minimal side effects. Furthermore, these findings demonstrate the potential of curcumin and gallic acid to be used as natural and safe alternatives to chemotherapy as an apoptosis-inducing treatment for MPM[31]. The structure of Curcumin has been depicted in Figure 9.

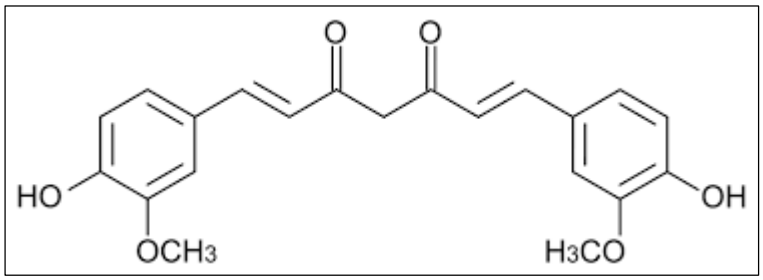

Fig: 9 - Structure of Curcumin

11. FERULIC ACID(FA)

FA prevents the growth, invasion, and spread of cancer cells. It targets and kills cancer cells while leaving healthy cells unharmed[32]. FA can be used alone or in combination with other treatments, such as chemotherapy or radiation therapy, to maximize treatment effectiveness. Treatment with FA greatly reduces the ability of human cervical cancer cells (Hela and Caski) to survive [33]. This research has shown that FA treatment reduces the term of proteins essential for the upsurge and survival of cancer cells, such as NF-kB and Akt. It has also been demonstrated to prevent cancer cells from migrating, thus avoiding spreading throughout the body. The structure of Ferulic Acid has been depicted in Figure 10. Furthermore, FA treatment is shown to cause cytotoxicity (cell death) in both Hela and Caski cancer cells, further highlighting its potential as an ability anti-cancer therapy[34].

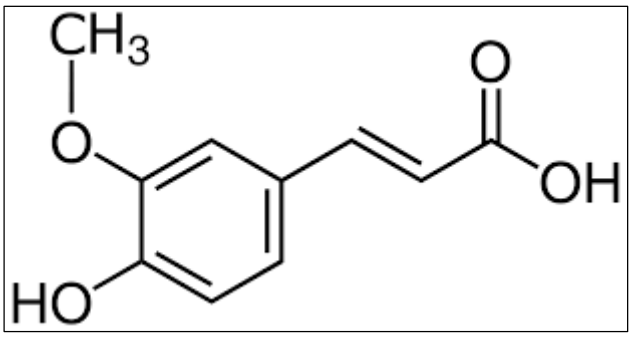

Fig: 10 - Structure of Ferulic Acid(FA)

12. CONCLUSION

This review article offers a thorough understanding of the function of natural products in cancer therapy by modifying the developmental route for programmed cell death and self-renewal. In the realm of anticancer research, natural substances are emerging as a viable therapeutic agent because due to its low cost and simple availability. Although the clinical applications of natural compounds are constrained by their poor intestinal absorption, quick catabolism, low bioavailability, and low aqueous solubility, recent efforts have been made to circumvent these restrictions by using nanoparticles, micelles, lipids, liposomes, and metal complexes.

13. CONFLICT OF INTEREST

Conflict of interest declared none.

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