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A Comprehensive Review on Therapeutic Implications of Medicinal Plants in Ovarian Cancer

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Abstract

Although ovarian cancer is the fifth most common cancer in women, it is the eleventh most common cancer. Ovarian cancer accounts for about 2.5% of all cancers in women. The most lethal type of gynecologic cancer is ovarian cancer. Ovarian cancer treatment has become more challenging as a result of inadequate early diagnosis and chemoresistance. Every microanatomic subtype had a specific molecular and epigenetic fingerprint associated with it. According to its histology, OC was divided into four subtypes, of which epithelial ovarian cancer (EOC) is more common than the others. Medicinal plants have contributed a vital role in the therapy of cancer. since ancient times, and the advantage of herbs is that they are less toxic to the human system compared to commercially available drugs. The majority of medicinal plants increase the effectiveness of chemotherapy, allowing us to reduce patient chemoresistance, which caught the attention of researchers. In this overview, we emphasize the role of Scutellaria barbata, Camellia sinensis, curcumin, ashwagandha, Leea indica, Garcinia, Asparagus and Cnidium monnier in ovarian cancer cells' molecular mechanisms.

Keywords: *Chemoresistance, Health and well-being, Medicinal plants, Ovarian Cancer, Therapeutics.*

Introduction

One of the most common gynecological tumors in women is Ovarian Cancer. Women are more frequently diagnosed at an advanced stage because there is a dearth of efficient early-stage screening this is linked to poor patient outcomes. 90% of Ovarian Cancer start out as epithelial tumor. Generally speaking, Ovarian Cancer is referred to as epithelial Ovarian Cancer, it also has number of subtypes, including mixed, clear cell, mucinous, endometrioid and serous [1]. Significant therapeutic potential of β-sitosterol as an anticancer drug against oral cancer KB cells by apoptosis induction and apoptotic pathway targeting [2]. The abdominal cavity, which

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transforms into a special microenvironment characterized by ascites, hypoxia and low glucose levels, is the same location where Ovarian Cancer starts, metastasizes and recurs. Because mitochondrial respiration becomes essential to cancer cells survival in these circumstances, it makes for an excellent metabolic target for chemotherapy-resistance Ovarian Cancer. The Ovarian Cancer tumor microenvironment and the inherent capacity to withstand chemotherapy are both responsible for chemoresistance in Ovarian Cancer. When EOC cells go to the peritoneal cavity, ascites builds up and creates an immunosuppressive microenvironment that exacerbates tumor growth. As the proverb goes, 'let thy food be thy medicine and thy medicine be thy food,' so do we researchers, who also look for drugs in naturally occurring plants. Fascinatingly, the majority of the food we eat has anticancer properties, so for this review, we'd like to look at look at some of the most popular foods and their effects on cancer. Different genetic alterations in different grades of OSCC are revealed by NGS analysis, which helps with minimal intervention techniques and individualized treatment planning [3].

Molecular Markers

Mutated genes determine cancer progression and recurrence in most patients. Mutation performs a crucial function in tumorigenesis and an accurate understanding of genetic mutation can be used to improve cancer diagnosis, prognosis, treatment and resistance mechanisms. Some of the most often observed abnormalities found in breast, brain, colon, lung and Ovarian Cancer include chromosome 17 rearrangements or deletions. According to molecular studies, in addition to the TP53 gene at 17p13.1 and the BRCA1 gene at 17q21, at least one tumour suppressor gene on chromosome 17 contributes in the pathophysiology of this disease region of rare heterozygosity (LOH) at 17p13.3 [4]. The results of the Chisholm et al. experiment, which was validated by other investigations, demonstrated that 12 of the 14 ovarian malignancies in women with BRCA1 mutations disoriented all of chromosome 17, whilst just a piece of 17q arm was lost by the other tumors. The LOH boundary between two tumors with partial heterozygosity for chromosome 17 was enriched in Alu sequences compared to DNA regions with same GC abundance [5]. A priori, each histological subtype was linked to the particular molecular and epigenetic fingerprint, with low-grade ovarian serous carcinoma typically containing mutations in proto-oncogene such as ERBB2, NRAS, BRAF and KRAS; high-grade ovarian serous carcinoma typically mutated at BRCA1/2, RB1, TP53, NF1, and PI3KCA, cyclin depend PTEN, ARID1A, PPP2R1, and mismatch repair deficiency are among the EC subtypes that are affected [6.7]. And mutations in the genes encoding mucin as well as CTNNB1, PPP2R1, PTEN, and PI3KCA; mucinous ovarian carcinoma includes tumor cells mutated KRAS gene and the amplification of ERBB2 occurs frequently [8,9]. The presence of H. pylori in severe carious lesions is associated with higher caries severity, higher DMFT scores, and a plaque ecology that favors Streptococcus mutans [10].

Scutellaria barbata

Traditional Chinese medicine says *Scutellaria barbata* has a role in inflammation, increasing blood circulation, diuresis, eliminating heat, blood stasis and removing toxins. A total of 84 products, mainly flavonoids and diterpenoids, polysaccharides, essential oils and steroids, were isolated from *Scutellaria barbata* [11].

Scutellaria barbata, (Figure 1) the main modes of action include mitochondria-mediated apoptosis and inducing cell cycle arrest by regulating cyclin/cyclin-dependent kinase and the cytotoxicity and detoxifying effects helped to raise cancer patients' quality of life [12]. Matrix metalloproteinase is

involved in the migration and infiltration of cancerous cells by fostering link with cancerous cells and its milieu, *Scutellaria barbata D.Don* inhibits infiltration of OC by downregulating matrix metalloproteinase 2 and 9 protein expression [13]. Combining *Scutellaria barbata* and *Hedyotis diffusa willd*, the experiment revealed that EGFR, MAPK1, VEGFA and PIK3CG were viable targets for inhibiting the development and migratory of OC cells through the cell-matrix adhesion pathway. Phytochemicals baicalein, luteolin and quercetin may play significant roles in the suppression of OC by *Scutellaria barbata* and *Hedyotis diffusa willd*, according to molecular docking and polypharmacology studies, but couldn't overlook their use in combination [14]. Data from Lin et al., revealed the positions of flavonoids such as methoxy group and hydroxyl group may be responsible for the interdependent effect of flavonoids with cisplatin on OC cell lines [13,15]. Calotropin inhibits the proliferation, migration, invasion, and aerobic glycolysis of HSC-3 oral squamous carcinoma cells, hence exhibiting anti-cancer effects [16].

Scutellaria baicalensis

Scutellaria baicalensis is an important folk medicinal plant, and its root play a crucial in Chinese medicine (Figure 2). *Scutellaria baicalensis* plant extract and its main compounds shown to have antimicrobial, antitumor, anti-inflammatory, antioxidant, hepatoprotective and neuroprotective effect [17]. The wogonin is a dihydroxy- and monomethoxy-flavone [18], phytochemical derived from *Scutellaria baicalensis* has the potent antiproliferative activity [19]. According to research of Jiang et al., wogonin treatment of OC cells resulted in a decrease in ERα pathway with that MPP which dramatically boosted the suppression of migratory and invasive activity in A2780 cells. Jiang et al. study also showed that nuclear modifications in chromatin condensation and fragmentation in cells, as well as elevated p53 and Bax protein expression and decreased VEGF and caspase 3 cleavage, each resulted in a concentration-dependent suppression of growth, migration and invasion (Figure 3 & Table 1) [19].

Figure 1. *Scutellaria Barbata* and

Figure 2. *Scutellaria baicalensis*

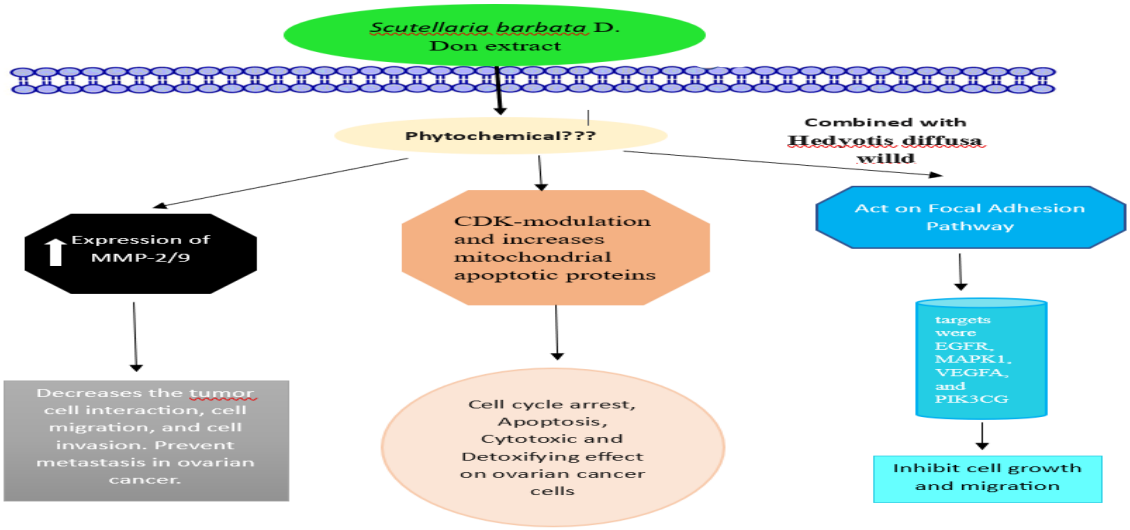

Figure 3. Schematic Representation of Scutellaria Barbata in Ovarian Cancer

In cells treated with wogonin and MPP, the proteins like cyclin D, CDK4, and CDK6 expression were reduced, which in turn resulted in a decrease in the G0/G1 subpopulation [19]. The cells (SKOV3/DDP and C13*) cotreated with cisplatin and wogonin showed significantly increased the antiproliferative activity in the cells and subsequent chromatin condensation and DNA fragmentation. In PI3k/ Akt signaling pathway, wogonin increased the cisplatin sensitivity by downregulating the Akt phosphorylation [20].

Camellia sinensis

Although green tea has anticancer effects, the exact mode of action is still unknown. The polyphenols theogallin, gallocatechin, epicatechin and epigallocatechin, including gallic acid, catechin and its derivatives, are the most prevalent substances in green tea. Theobromine (0.02-0.04%), methylxanthines, caffeine (3-4%) and Theobromine (0.15-0.2%) are all present in small amounts in the fresh leaves range depending on the development. With fermentation, catechins partially transform into non-water-soluble flavonoids like thearubigin, theaflavin and theaflavin acid. Ascorbic acid and vitamin B are present in green tea, but are lost in the production of black tea [21].

When employing a high dose of green tea extract, granulosa cell's p53 and caspase-3 levels increased [22]. Green tea and epigallocatechin gallate (EGCG) reduced cyclooxygenase 1 and cyclooxygenase 2 expression, which in turn reduced PGE2

production, which was stimulated by ET-1 [23]. The antiapoptotic protein Bcl-XL was downregulated and caspase-3 was activated by the EGCG in green tea, which quickly triggered cell death in human OC cells. In addition, the ability of EGCG to reduce the onset and ET-1 induced inhibition of the Endothelin-1/ Endothelin Type A receptor autocrine cycle may be accountable for the anti-angiogenic and anti-inflammatory effects of these drugs on OC. The EGCG treatment of the OVCAR-3 cell line had no inhibitory impact [24]. The circulating exosomal miRNAs miRNA 21, miRNA 184, and miRNA 145 are possible biomarkers to evaluate the risk of malignant transformation in patients with OSMF, OSCC, and leukoplakia [25].

In SMF and Ba/F3 2+4 cells, EGCG decreased basal receptor phosphorylation, suggesting a possible role for EGCG in adjuvant therapy for tumor overexpressing Her-2/neu. This explains how EGCG has a pivotal role in the anticancer effect of camellia sinensis extract [26].

The proliferation of tumor cells may be inhibited by catechin just as effectively as by epicatechin. In addition to apoptosis, necrosis and the autophagy process exhibited by the other phytochemicals, catechin can also cause cell death. Catechin treatment of SKOV-3 cells has demonstrated a considerable reduction in the LRP protein, which has linked has been linked to an increase in intracellular drug concentration and cytotoxicity [27] (Figure 4).

The ability of ethyl gallate and propyl gallate to prevent TGF-mediated Smad-3 phosphorylation despite having no effect on TGF-R1 kinase activity [28]. Black tea's theaflavin has the ability to fight cancer, which is greatly boosted when combined with nanogold. The gold nanoparticle with theaflavin treated cells, showed increased Bax, Bad, Bid, Bim and caspase-3 which are the pro-apoptotic markers whereas antiapoptotic markers like Bcl-2 and Bcl-w got suppressed simultaneously leads to the increased apoptosis [29].

According to the study, people who consume more green tea after receiving an ovarian cancer diagnosis had a higher probability of surviving the disease. As a result, the study came to the conclusion that frequent green tea drinking may offer protection against EOC [30] (Table 1). As a possible marker for early diagnosis and prognosis, elevated salivary MMP-9 levels are correlated with the severity of OSCC and malignant transformation [31].

Role in Resistance

As everyone is aware, cancer resistance always makes therapy more challenging. By inhibiting the Akt phosphorylation and Bcl-2 expression and upregulating the expression of cleaved caspase-3, cleaved caspase-9, cytochrome-C and Bax, Cyt-C, green tea and paclitaxel combination triggered apoptosis in OC cells. When combined with green tea, PTX is more effective at decreasing the growth of cancerous cells. Instigating cytotoxicity and cell death in Ovarian Cancer cells may be more effectively accomplished by the mixture of PTX with green tea than by either medication alone [32]. miRNAs have potential as therapeutic targets and biomarkers for OPMD early detection and treatment [33].

In combination of SFN and EGCG inhibited both multidrug resistant and paclitaxel OC cells, but the combination showed maximum effect on paclitaxel resistant ovarian cancer cell by arresting cell cycle at G2/M checkpoint and induces cell death. The process-based study showed that EGCG and SFN combined therapy can reduce mutated DNA of OC cell, decrease hTERT expression and downregulate Bcl-2 expression [34].

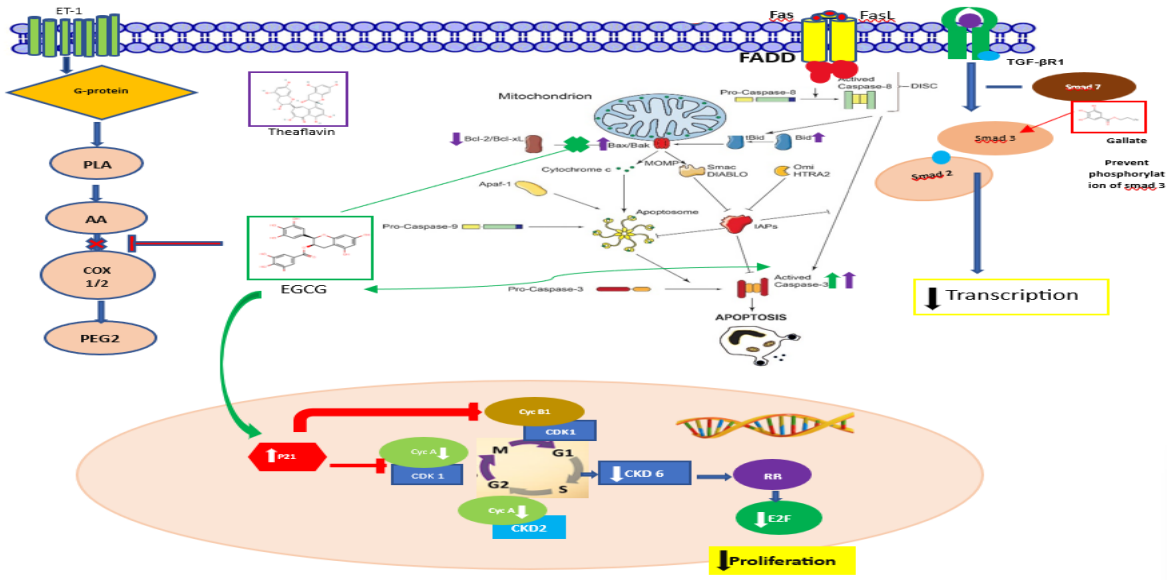

Figure 4. Mechanism Underlying the Potential Impact of Green Tea on Ovarian Cancer.

Turmeric (*Curcuma longa* l)

One of the primary components of the rhizome of *curcuma longa* (L) and another curcuma spp. is curcumin. These phytochemicals are categorized as diarylheptanoids. About 77% of the diarylheptanoids in turmeric, including demethoxycurcumin and bis-demethoxycurcumin are present. Typically, curcumin is employed as a food coloring agent [35]. TNF, Nrf-2, STAT, cyclin D1, cyclooxygenase-2, AP-1, NF-kB, EGFR and mitogen-activated protein kinase are among the carcinogenic molecules that curcumin has been shown to target [36]. Other chemo preventive and antitumor effects of curcumin include suppression of EGFR and protein kinase-C as well as by stimulating the isoenzyme cyclooxygenase-2 expression and by increasing the STAT3 inhibitors expression such as Pias-1 and Pias-3, daily dietary curcumin ingestion reduces STAT3 expression on OC cells [37]. The proliferation of both chemo-resistant and sensitive Ovarian Cancer has been demonstrated to be inhibited by curcumin.

Ho-8910 (Human OC cell line) cells can be significantly induced to undergo apoptosis and have their growth inhibited by curcumin. In the curcumin treated cells, the expression of procaspase-3, Bcl-2 and Bcl-XL was reduced whereas Bax and P53 levels was uplifted. Increased p53 modulates the proportion of Bcl-XL/Bax and Bcl-2/ Bax, which in turn it activates the release of mitochondrial cytochrome C into cytosol. In Ho-8910, it's speculated that Bcl-2 family function as the secondary signal carrier molecule in the curcumin induced cell death [35]. A2780CP cells that had been treated with curcumin displayed decreased β-catenin expression [38]. IL-17A and salivary 1-25dihydroxycholecalciferol levels correlate with orthodontic treatment phases, indicating that vitamin D administration may hasten tooth movement with little harm to surrounding tissue [39].

The curcumin treated cells show that AMPK and its upstream signal p38 are involved in the cytotoxic action on cancer cells. Curcumins cytotoxic effect is mostly caused by AMPKs control of p53 phosphorylation [40]. Calcium homeostasis is regulated by SERCA (Sarco-endoplasmic reticulum calcium ATPase) which has been found to express more frequently in ovarian malignancies. Curcumin induces cell death only in OC cells the healthy cells such as ovarian epithelial cells and peripheral blood mononuclear cells were found unaffected, through increasing concentration of calcium

present in cytosol by suppressing the SERCA activity [41].

In vitro, the DTX-Cur/M nano micelles dramatically reduced the viability and mobility of A2780 cells and increased cell death. In vivo drug revealed a significantly improved antitumor activity in terms of reducing tumor angiogenesis, increasing tumor cell apoptosis and decreasing tumor cell proliferation [42] (Figure 5 & Table 1).

Role in Resistance

Cells treated with curcumin showed a reduction in Bcl-XL and Mcl-1 proteins, thereby causing apoptosis in response to cisplatin. The study showed that curcumin treated cells before cisplatin enhanced the proportion of PARP, cleaved caspase-9 and annexin V infused cells compared with cells treated curcumin or cisplatin exclusively [38]. Muhanmode et al., study suggests that cisplatin treatment followed by accelerate cell death, increasing chemosensitivity in OC cells by considerably reducing the PI3K/AKT/mTOR pathway, resveratrol and curcumin notably increases the epithelial ovarian cancer cells sensitivity to cisplatin therapy [43].

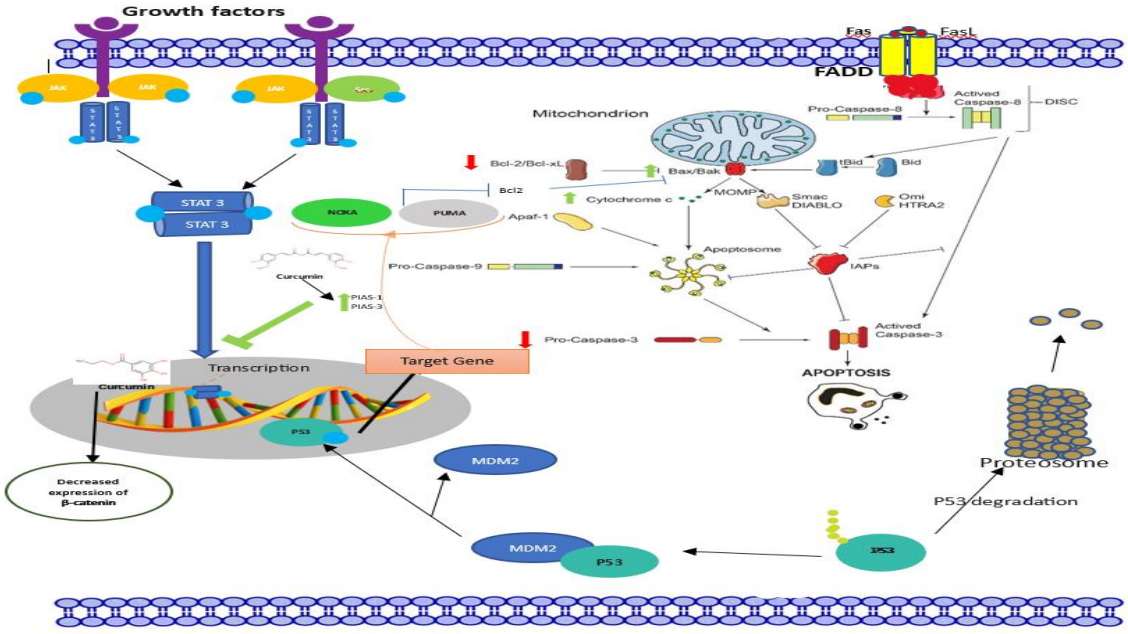

Figure 5. The Multifaceted Mechanism of Action of Turmeric (Curcuma longa) in the Context of Ovarian Cancer

***Withania somnifera* (Ashwagandha)**

Acylsterylglucosides, anahygrine, anaferine, cuseohygrine, isopelletierine, saponins, sitoindosides, withanolides and withaferins are some of the phytochemicals of *Withania somnifera* (WS). Ashwagandha has an extended tradition of using it as an adaptogen for stress relief [44,45]. When ashwagandha is supplemented, there is a correlation between increased NK cells activity and decreased MICA expression in tumor cells. MICA and MICB have a modest level of expression in normal cells, but their expression has been reported to increase in response to cell surface damage, mutagen-induced cell mutation, and infection. Ashwagandha administration boosted the invasion of Natural killer cells and reduced the intensity of OC cells in Hens [46] (Figure 6 & Table 1).

Role in Resistance

The injection of WFA only or in combination with cisplatin caused apoptosis and decreased proliferation in cisplatin-resistant (A2780 and CP70) and cisplatin-sensitive (A2781 and CaOV3). Additionally, an in vivo study showed that CSC makers (Oct4, CD117, CD24, CD34

and CD44) can be used to stop metastasis and remove cells that express these markers [47].

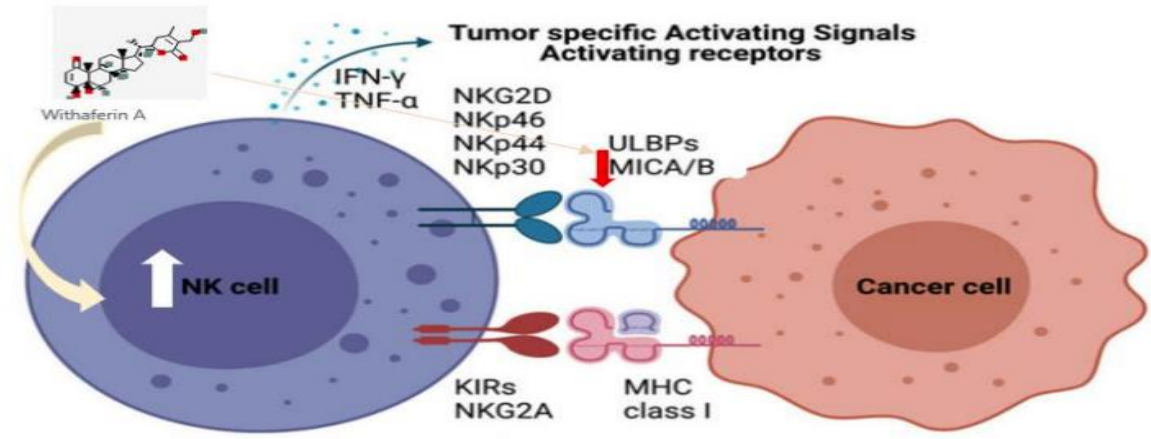

Figure 6. Schematic Representation of Ashwagandha in Ovarian Cancer

Leea indica

Leea indica leaf extract renders ovarian cancerous cells more sensitive to natural killer cell intruded cytotoxicity. The cancerous cells showed the upregulation of stress ligand expression when treated with methyl gallate. In cancerous cells treated with methyl gallate before treated with small concentration of oxaliplatin, stress ligands expression level was increased and vulnerability to natural killer cell intruded cell lysis was elevated. In addition, natural killer cells inhibited the growth of OC previously treated with methyl gallate and reduced the secretion of interleukin-1β and tumor necrosis factor-α in the macrophage of human U937 cells [48].

Asparagus

Asparagus racemosus is a medicinal plant available in Asia, Africa, and Australia. In India, this plant extract is used for indigestion, stomach ulcer and in the problem related to reproduction in female [49], and it shows to have anticancer activity. Kalai et al., researched the activity of *Asparagus racemosus* extract in the OC cell line (SKOV3) showed the anticancer activity in the dose depended manner [50].

Role in Resistance

Zhang et al. study showed the combination of *Asparagus officinalis* and Paclitaxel is highly effective against paclitaxel-resistant MES-TP cells and paclitaxel-sensitive MES cells by increasing the cleaved caspase9, cleaved caspase 3, cleaved caspase 8 and MCL-1while decreased expression of Bcl-XL when combined with paclitaxel treatment alone [51].

Garcinia

The polyisoprenylated benzophenone (garcinol, xanthochymol and guttiferone) and xanthone derivates from *Garcinia mangostana* has the antiapoptotic, antioxidant, anti-inflammatory, antimicrobial, antiulcer and histone acetylase transferase (HAT) inhibiting properties [52,53].

The compound Garcinone E (2,3,6,8-tetrahydroxy-1,4,7-tris(3-methylbut-2-enyl) xanthen-9-one) molecular weight of 464.5 g/mol [54] derived from *Garcinia mangostana* showed to have a related properties in both basic susceptible cell line and multidrug resistance cell line, it additionally revealed the promising antiproliferative effects and also decrease the multidrug resistance possibility in OC cells. In A2780 and HEY cells, Garcinone E triggers the decrease in mitochondrial membrane potential, it induces the ER stress by drastically increases the expression of PERK

(Protein kinase R-like ER kinase), such as eIF2, CHOP (C/EBP homologous protein), Bip (Immunoglobulin binding protein), XBP-1 (X-binding protein-1) and IRE-1 (Inositol requiring kinase-1). Cell invasion and migration are important steps in the development of malignant tumor. Garcinone E shows that it downregulates Rac, RhoA, MMP-2 and MMP-9 and upregulates Tissue inhibitors of metalloproteinases 1 and 2 protein levels, indicating that its reduction of HEY cells invasion is carried out by lowering MMP protein levels and by decreasing Rho GTPase and MMP activity, the protein which involved in migration [55].

The key xanthone derivative isolated from *Garcinia mangostana* pericarp α-mangostin (7-o-methyl-4-desprenylcostatin) [56], exhibits numerous pharmacological effects including antioxidant, antiproliferative, anti-inflammatory and antiapoptotic activity [57]. Apigenin and α-mangostin treated SKOV-3 cells, exhibited the decrease in cell density, and increased plasmolysis and detached cells were observed, but the effect of α-mangostin had the most dramatic impacts. In terms of apoptosis, apigenin induced early apoptosis while doxorubicin and α-mangostin induced late apoptosis and necrosis. The compound (α-mangostin) treated SKOV-3 cells showed a transient increase in caspase-9 and caspase-3 activity. Apigenin and α-mangostin halted the cell cycle by acting on G2/M checkpoint after 24 and 48 hours of the treatment, respectively. In SKOV-3 cells injected with α-mangostin and apigenin, there was a notable increase of the transcripts for the apoptosis-associated gene BCL2 and the inflammation-associated gene COX2 [58].

α-mangostin treated OVACAR-3 cells, showed the morphological alterations like increased protrusions of cell membrane, rapturing of plasma membrane, cellular disintegration and karyorrhexis. The increased Caspase-3,8 and 9, proapoptotic Bax protein, ROS and decreased Bcl-2 and MMP were noted which leads to induction early, late apoptotic, necrotic cells and migration of tumor cells [59].

Cnidium Monnieri

Numerous medicinal plants, notably *Angelica pubescens* and *cnidium monnieri*, contain natural phytochemical osthole (7-methoxy-8-(3-methyl-2-butenyl)-2H-1-benzopyran-2-one). In A2708 and OV2008 cell lines treated with osthole showed some of the morphological changes were which include chromatin condensation, karyopyknosis and apoptotic body, elevated the phosphorylation at tyrosine 15 in cdc2 and cleaved caspase 3&9 while downregulated the MMP 2&9, cd2 and cyclin B1 expression which tend to tigger both early and late apoptosis and has inhibitory effect on migratory and invasion in OC cells [60], Gasdermin E (c-GSDME) cleavage could result in the pyroptosis [61,62] (Table 1).

Table 1. Summary of Medicinal Plants Employed in Ovarian Cancer Treatment

S.No	Medicinal Plant	Phytochemical	Study model	Target and mechanism	Reference
1.	*Scutellaria barbata*	Unknown	*Invitro*	Mitochondria-mediated apoptosis, cell cycle arrest by regulating cyclin/cyclin dependent kinase. Decreased expression of matrix metalloproteinase 2 and 9 protein.	[12, 13]

	Scutellaria barbata and *Hedyotis diffusa willd*	Unknown	*Invitro*	EGFR, MAPK1, VEGFA and PIK3CG inhibited the cell migration by cell matrix adhesion pathway.	[14]
2.	*Scutellaria baicalensis*	Unknown	*Invitro*	Elevated p53 and Bax protein expression and decreased VEGF and caspase 3 cleavage and downregulating of Akt phosphorylation.	[19, 20]
3.	*Camellia sinensis*	Epigallocatechin gallate (EGCG)	*Invitro*	Reduced COX-1, COX-2 and Bcl-XL, expression, decreased PGE2, increased ET-1 and caspase-3.	[23]
		Theaflavin	*Invitro*	Increased Bax, Bad, Bid, Bim and caspase-3, decreased Bcl-2 and Bcl-w.	[29]
4.	*Curcuma longa*	Curcumin	*Invitro*	Expression of procaspase-3, Bcl-2 and Bcl-XL was reduced, Bax and P53 levels was uplifted and decreased β-catenin expression.	[35, 38]
5.	*Withania somnifera*	Withaferin A	*Invitro*	Increased NK cells activity and decreased MICA expression.	[46]
6.	*Leea indica*	Methyl gallate	*Invitro*	Reduced the secretion of IL-1β and TNF-α.	[48]
7.	Asparagus	*Asparagus officinalis* and Paclitaxel	*Invitro*	Increasing the cleaved caspase9, cleaved caspase 3, cleaved caspase 8 and MCL-1while decreased expression of Bcl-XL.	[51]
8.	Garcinia mangostana	Garcinone E	*Invitro*	Decrease in mitochondrial membrane potential, it induces the ER stress by drastically increases the expression of PERK, such as eIF2, CHOP, Bip, XBP-1 and IRE-1. Downregulates Rac, RhoA, MMP-2 and MMP-9 and upregulates Tissue inhibitors of MMP 1 and 2 protein levels, Decreasing Rho GTPase and MMP activity, the protein which involved in migration.	[55]
		α-mangostin	*Invitro*	Increased Caspase-3,8 and 9, Bax protein, ROS and decreased Bcl-2 and MMP.	[59]

		α-mangostin and Apigenin	*Invitro*	Halted the cell cycle by acting on G2/M checkpoint. Increase BCL2 and COX2.	[58]
9.	*Cnidium monnieri*	Osthole	*Invitro*	Elevated the phosphorylation at tyrosine 15 in cdc2 and cleaved caspase 3&9, downregulated the MMP 2&9, cd2 and cyclin B1 expression.	[60]

ABBREVIATIONS: BAD-Bcl-2-associated agonist of cell death, BAX-Bcl-2-associated X protein, BCL- B-cell lymphoma, BID- BH3 interacting domain death agonist, BIM- Bcl-2 interacting mediator of cell death, CIS-Cisplatin, CTNNB1- Catenin Beta 1, DXT-Deep X-ray therapy, EGCG- Epigallocatechin Gallate, ER- Endoplasmic Reticulum, ERBB2-Erb-B2 receptor tyrosine kinase 2, ET-Endothelin, HB-SB- Hedyotis diffusa willd and Scutellaria barbata, HER 2- Human epidermal growth factor receptor 2, HTERT- Telomerase reverse transcriptase (human), LRP- low density lipoprotein receptor- related protein 1, MMP- Matrix metalloproteins, NEU-Neuroglioblastoma cell line, NF1-Neurofibromin 1, OC- Ovarian cancer, P53-Tumor protein 53, PGE2- Prostaglandin E2, PI3KCA- Phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha, PPP2R1- Protein phosphatase 2 scaffold subunit A alpha, PTEN- Phosphatase and homolog, PTX- Paclitaxel, RB1- RB transcriptional corepressor 1, SNF-Sulforaphane, SERCA- Sarco-endoplasmic reticulum calcium ATPase, TGF-β-Transforming growth factor beta, TGF-βR-TGF-β receptor.

Conclusion

In terms of patient fatalities, OC is the most prevalent gynecological cancer. OC, formerly described as ‘silent killer’, has a variety of nonspecific symptoms that. When acknowledged, may enable early discovery and improved survival. The majority of herbs, fruits and vegetables have demonstrated anticancer potential, which also helps with chemosensitivity during therapy. Prevention is crucial for all illnesses, but it is particularly important for cancer. The presence of natural component is essential to the growth of the tumor.

Conflict of Interest

The author hereby declares that there is no conflict of interest.

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