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Review Article

AN OVERVIEW OF POTENTIAL BENEFITS OF *ASHWAGANDHA (WITHANIA SOMNIFERA)* IN PREVENTION AND TREATMENT OF CERVICAL CANCER

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ABSTRACT

Cervical cancer is the fourth most common cancer affecting women worldwide and the second most prevalent among women in India, following breast carcinoma, with an estimated 127,526 new cases annually. Despite advancements in screening and treatment, recurrent and persistent forms of cervical cancer pose a significant therapeutic challenge. Conventional therapies often come with considerable toxicity, limited efficacy in advanced stages, and poor accessibility in low-resource settings. Therefore, there is an urgent need for safer, multi-targeted therapeutic alternatives. In recent years, *Ashwagandha* is one among the herbal drugs of Ayurveda found useful in different cancers. *Ashwagandha* leaf is rich in an active principle called withaferin A. Withaferin A (WFA), a steroidal lactone has shown significant anti-neoplastic activity across various cancer cell lines, including cervical cancer, in both in vitro and in vivo models. This paper reviews the anticancer efficacy of Withaferin A in cervical cancer and, for the first time, explores the broader therapeutic potential of *Ashwagandha* through an integrative perspective. Emphasis is placed on its anti-proliferative, anti-inflammatory, pro-apoptotic, and adaptogenic properties. The potential mechanism of action are also examined through the lens of *Panchmahabhautika Sanghatana*- the Ayurvedic five-element theory- providing a holistic perspective on *Ashwagandha* impact on cellular pathology. This integrative approach not only bridges traditional Ayurvedic concepts with contemporary biomedical research but also opens a promising pathway for the development of low-toxicity, plant-based therapies for cervical cancer.

INTRODUCTION

Globally, cervical cancer ranks fourth among cancers that affect women and the second most common cancer among women in India after breast carcinoma with an estimated 127,526 new cases diagnosed annually.[1] Cervical carcinoma is also the second leading cause of death, with an estimated 79,906 deaths annually and it is particularly prevalent in underdeveloped nations.[2] Cervical cancer is primarily caused by persistent infections with high-risk HPV, particularly HPV-16 and HPV-18 accounts for about 95% cases.[3] Surgeries, radiation therapy, and chemotherapy are now available treatment options; however, in advanced stages, these can have severe side effects and limited efficacy. Alternative and complementary medicines, especially herbal drugs, have been gaining acceptance because of their possible anticancer effects and less toxicity. *Withania somnifera*, commonly known as *Ashwagandha*, is one such herb and due to its bioactive ingredient, Withaferin A (WA), a*shwagandha* has been utilized for centuries in traditional Ayurvedic medicine and has shown promising anticancer effects.

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In ancient Ayurvedic literature, the therapeutic attributes of *Ashwagandha* are explained in detail. According to the Bhavaprakasha Nighantu, it is classified as a *Rasayana* (rejuvenative), *Balya* (strength-enhancing), *Vrishya* (aphrodisiac), and *Kaphavatahara* (pacifying *Kapha* and *Vata* doshas).

अश्वगन्धाऽनिलश्लेष्मश्वित्रशोथक्षयापहा।
बल्या रसायनी तिक्ता कषायोष्णाऽतिशुक्रला ॥

Reflecting its broad-spectrum efficacy in promoting strength, vitality, anti - inflammatory action and equilibrium of body functions.[4] Similarly, Sushruta Samhita, classifies *Ashwagandha* as a potent

herb for promoting vigor and disease resistance emphasizing its adaptogenic and anti-aging properties.[5] Given these traditional claims, modern research is now validating *Ashwagandha's* role in cancer prevention and treatment.

AIMS

1. To explore the potential of *Ashwagandha* in cervical cancer by targeting oncogenic pathways and restoring normal cell regulation.
2. To explore the potential of *Ashwagandha* in the prevention and management of cervical cancer by understanding its *Panchmahabhotik Sanghathan* and its mechanism of action on cancer cells.

OBJECTIVES

1. To understand the molecular mechanism of HPV oncogenes (E6 and E7) in cervical carcinogenesis and their suppression by *Ashwagandha*.
2. To evaluate the ability of *Ashwagandha* to induce apoptosis and cell cycle arrest, along with its anti-inflammatory, immunomodulatory, adaptogenic, and over all anticancer potential in cervical cancer.
3. To evaluate the role of *Ashwagandha* as a *Rasayana* and adjuvant therapy in cervical cancer.

MATERIALS AND METHODS

Literature search strategy

A comprehensive review of the literature was conducted using databases such as PubMed, Scopus, Google Scholar, and Science Direct.

Inclusion and Exclusion Criteria

Inclusion criteria

- Studies specifically on *Ashwagandha* or its key bioactive compound Withaferin A in cervical cancer and other gynecological cancers.
- Research evaluating molecular mechanisms of *Ashwagandha* on HPV associated cervical cancer.
- Articles published in peer-reviewed journals.
- In vitro, in vivo, and clinical studies evaluating the effects of *Ashwagandha* and Withaferin A, in cervical cancer.

Exclusion Criteria

- Studies without HPV mechanistic insights.
- Non–peer-reviewed articles, editorials.
- Studies focusing only on general herbal medicine without *Ashwagandha*–specific data.

Phytochemistry of *Ashwagandha*

Ashwagandha is rich in bioactive compounds, including: Withanolides (Withaferin A, Withanolide A, Withanone), steroidal lactones, flavonoids and alkaloids, saponins and tannins. Among these, Withaferin A($C_{28}H_{38}O_6$) has been extensively studied for its anticancer properties. It has an ergostane-type steroidal skeleton with an epoxide moiety, which contributes to its bioactivity.[6]

Table 1: *Panchmahabhautika Sanghatana* of *Ashwagandha* [7]

Property	Details
Rasa (taste)	*Madhura* (sweet), *Kashaya (*astringent*), Tikta* (bitter)
Guna (qualities)	*Laghu* (light) *Snigdha* (unctuous)
Virya (patency)	*Ushna* (hot)
Vipaka (post-digestive effect)	*Madhura* (sweet)
Karma (actions)	*Balya* (strength-promoting), *Rasayana* (rejuvenating), *Vajikarana* (aphrodisiac), *Medhya* (cognitive), *Nidrajanan* (sleep-inducing), *Shothahara* (anti-inflammatory)

Effect of HPV Oncogenes on the Cell Cycle

The cell cycle is a highly regulated sequence of events that governs cell growth, DNA replication, and division. It consists of four major phases. In the G_1 phase, the cell experiences marked growth, synthesizes essential proteins, and prepares for DNA replication. The S phase is characterized by the duplication of the cell's DNA to ensure that each daughter cell receives a complete and accurate set of genetic material. This is followed by the G_2 phase, where the cell undergoes further growth and performs critical checks to detect and repair any DNA damage before division. The final stage is the mitotic (M) phase, which involves nuclear division (mitosis) followed by cytokinesis, resulting in the formation of two genetically identical daughter cells.

To maintain genomic integrity and prevent aberrant cell division, the cell cycle is controlled by key checkpoints- the G_1 checkpoint, which assesses cell size, nutrients, and DNA integrity; the G_2 checkpoint, which verifies proper DNA replication and checks for damage; and the M checkpoint, which ensures correct chromosome alignment and attachment to the spindle apparatus during mitosis.

Disruption of these checkpoints is a hallmark of cancer, and such disruption can occur during infection with high-risk Human Papillomavirus (HPV) types, such as HPV-16 and HPV-18. These viruses produce viral oncoproteins E6 and E7, which interfere with the normal regulatory proteins of the cell cycle. The E6 oncoprotein binds to and promotes the degradation of p53, a crucial tumor suppressor protein often called

the "guardian of the genome." Under normal conditions, p53 halts the cell cycle in response to DNA damage and initiates DNA repair or apoptosis if the damage is beyond repair. Loss of p53 function results in unchecked DNA replication, even when errors are present. Meanwhile, the E7 oncoprotein targets the retinoblastoma protein (pRb), another key regulator of the G_1-S checkpoint. Under normal conditions, pRb binds and inhibits E2F transcription factors, preventing premature progression to the S phase. When E7 inactivates pRb, E2F is released, driving the cell into DNA synthesis regardless of its readiness.

Thus, HPV infection leads to loss of cell cycle control, bypasses critical checkpoints, and promotes uncontrolled cellular proliferation and genomic instability, ultimately leads to the development of cervical and other HPV-associated cancers.[8]

Mechanism of Anticancer Activity

Ashwagandha exerts its anticancer effects by inhibition of HPV oncoproteins, induction of apoptosis, cell cycle arrest, suppression of inflammation, inhibition of angiogenesis, and modulation of key signalling pathways.

- **Modulation of HPV Oncogenes**

  The oncoproteins E6 and E7 of HPV impair tumor suppressor function by degrading p53 and pRb, which results in unregulated cell proliferation. Withaferin A restores the functions of p53 and pRB. The restored p53 then upregulates p21, a cyclin-dependent kinase inhibitor that plays a crucial role in halting cell cycle progression. Elevated p21 levels interfere with the activity of proteins essential for the G2/M transition-such as cyclin B1 and the p34^cdc2 kinase-resulting in a G2/M phase arrest. This cell cycle blockade not only prevents cancer cell proliferation but also predisposes cells to undergo apoptosis, effectively eliminating the malignant cells.[9]

- **Induction of Apoptosis**

  Withaferin A induces apoptosis through both the intrinsic and extrinsic pathways:

  Apoptosis, or programmed cell death, can occur through two primary pathways: the intrinsic and extrinsic pathways. The intrinsic pathway is triggered by mitochondrial dysfunction, which leads to the release of cytochrome c into the cytosol and subsequent activation of caspase-9. In contrast, the extrinsic pathway is initiated by the activation of death receptors on the cell surface, leading to the activation of caspase-8. Both pathways converge at the activation of caspase-3, a key executioner caspase responsible for the cleavage of vital cellular components, ultimately resulting in cell death. Withaferin A, promotes apoptosis by modulating the balance between pro- and anti-apoptotic proteins. It upregulates pro-apoptotic proteins such as Bax and Bak while downregulating anti-apoptotic proteins like Bcl-2 and Bcl-xL. This shift favors mitochondrial outer membrane permeabilization, thereby enhancing the intrinsic apoptotic pathway and facilitating the activation of downstream caspases.[10,11,12]

- **Anti–inflammatory effect by Inhibition of STAT3 and NF-κB Signaling**

  Withaferin A exerts notable anticancer and anti-inflammatory effects by modulating critical intracellular signaling pathways. In particular, it attenuates the STAT3 pathway by inhibiting phosphorylation events—primarily through the suppression of upstream kinases such as JAK2—thereby reducing STAT3 dimerization and its subsequent nuclear translocation. Concurrently, Withaferin A disrupts the NF-κB pathway by preventing the phosphorylation and degradation of IκBα, which impedes NF-κB's nuclear accumulation and transcriptional activity. This dual inhibition culminates in the downregulation of pro-inflammatory cytokines, including IL-6 and TNF-α, and is associated with diminished tumor progression, inflammatory response, and immune evasion.[13]

- **Inhibition of Angiogenesis**

  Tumor angiogenesis is essential for sustained cancer growth because it provides tumors with oxygen and nutrients through the formation of new blood vessels. Withaferin A has been shown to inhibit angiogenesis through multiple mechanisms. It downregulates the expression of vascular endothelial growth factor (VEGF)-a key mediator of angiogenesis-by interfering with transcription factors (such as Sp1) that promote VEGF expression.[14] In addition, Withaferin A suppresses endothelial cell proliferation and directly inhibits the formation of new blood vessels, thereby disrupting the vascular support required for tumor progression and metastasis.

Synergistic Effects with Conventional Therapies

Withaferin A, a bioactive compound derived from *Withania somnifera* (*Ashwagandha*), has shown promising results when used in combination with standard chemotherapeutic agents. Studies have demonstrated that its co-administration with cisplatin leads to increased cytotoxic effects on cancer cells and helps reduce the development of drug resistance.[15] Similarly, when combined with paclitaxel, Withaferin A enhances apoptosis and contributes to improved tumor regression. In the context of radiotherapy, it has been found to sensitize cervical cancer cells to radiation, thereby amplifying radiation-induced apoptosis.[16] One of the major advantages of using Withaferin A in combination therapy is that it allows

for the use of lower doses of conventional chemotherapeutic drugs, which significantly reduces systemic toxicity. This not only minimizes side effects but also improves overall patient outcomes, making it a valuable adjunct in modern cancer treatment strategies.

Safety and Toxicity of Withaferin A

- **Preclinical Toxicity Studies**

  Withaferin A has been evaluated for toxicity in animal models.

  Acute Toxicity: No lethal effects at therapeutic doses

  Chronic Toxicity: No significant organ damage observed.

  Hematological Parameters: No major alterations in blood counts or liver enzymes.

- **Clinical Safety**

  Although clinical studies specifically evaluating Withaferin A in cervical cancer are currently limited, broader clinical and preclinical investigations on *Ashwagandha (Withania somnifera)* indicate a favourable safety profile. The herb is generally well-tolerated when administered within recommended therapeutic doses. Reported adverse effects are infrequent and typically mild, including occasional gastrointestinal disturbances (such as nausea or abdominal discomfort) and neurological symptoms (such as drowsiness or dizziness) in sensitive individuals.[17]

Potential role of *Panchmahabotik sanghatan* of *Ashwagandha* in cervical cancer

- ***Tikta* (Bitter) *Rasa*:** *Tikta Rasa* is recognized for its *vishagna* (anti-toxic) properties, making it effective in eliminating *Ama* (toxins) from the body. This taste is instrumental in the *(Sodhan)* purification of *Rakta Dhatu* (blood tissue) and *Dushya* (vitiated tissues), which are often implicated in the pathogenesis of tumors. Furthermore, *Tikta Rasa* exhibits significant *(Ropan)* anti-inflammatory activity, anti-proliferative and healing properties, contributing to the inhibition of tumor growth and overall suppression of neoplastic processes.[18]
- ***Kashaya* (Astringent) *Rasa*:** *Kashaya Rasa*- the astringent taste- is known for its drying (*Kledohara*) and constricting (*Stambhana*) effects. These actions help manage excess bodily fluids and secretions, which can be especially useful in uncontrolled tissue growth or fluid accumulation.[19]
- From a modern medical viewpoint, these traditional properties may align with the concept of anti-angiogenesis- the process of blocking new blood vessel formation that tumors need to grow. By helping dry out abnormal fluids and tightening tissues, herbs with *Kashaya Rasa* might reduce the blood supply feeding tumors. In this way, they could support cancer treatment by slowing down tumor growth and spread.[20]
- ***Madhura* (sweet) *Rasa*:** *Madhura rasa* possesses *Brimhana* (nourishing) and *Jivana* (life-sustaining) properties, which support tissue regeneration and cellular nourishment. In cancer patients, these properties can help counterbalance the cytotoxic effects of chemotherapy or radiation by promoting *Ojas* (vital essence) and strengthening *Dhatus* (body tissues). *Madhura Rasa* also pacifies *Vata* and *Pitta*, helping to stabilize metabolism and immunity during cancer therapy.[21]
- ***Ushna Virya* (Potency):** *Ushna virya* (hot potency) substances are known in Ayurveda to enhance *Agni* (digestive fire), thereby accelerating metabolism and facilitating the efficient elimination of *ama* (toxins).[22,23] This detoxifying action makes them especially valuable in disease management, including chronic conditions like cancer. Herbs like *Ashwagandha* (*Withania somnifera*), which possess *Uṣhṇa Virya* exhibit significant anticancer potential. Among its various phytochemicals, Withaferin A, a potent withanolide, has been shown to induce apoptosis (programmed cell death) selectively in cancer cells. Thus, the *Ushna Virya* helps in suppression of proliferation of neoplasmas.
- ***Madhura Vipaka* (Post-Digestive Effect):** *Madhura Vipaka* ensures long-term tissue regeneration and prevents excessive depletion caused by chemotherapy or radiation. It also has an immunomodulatory effect, improving the body's natural defence against HPV infections. *Madhura Vipaka* is strengthening and nourishing, supporting tissue repair in degenerative diseases.[24]
- **Laghu Guna:** Helps in easy assimilation of its bioactive compounds, enhancing its efficacy at a cellular level.
- ***Snigdha Guna*:** Protects healthy tissues from oxidative damage, which is beneficial during chemotherapy and radiation therapy. Withaferin A reduces oxidative stress and lipid peroxidation, preventing further DNA damage in cervical cancer cells. *Laghu* and *Snigdha* properties help in disease modulation and tissue repair.

Fig.1 Phytochemicals from *Withania somnifera*

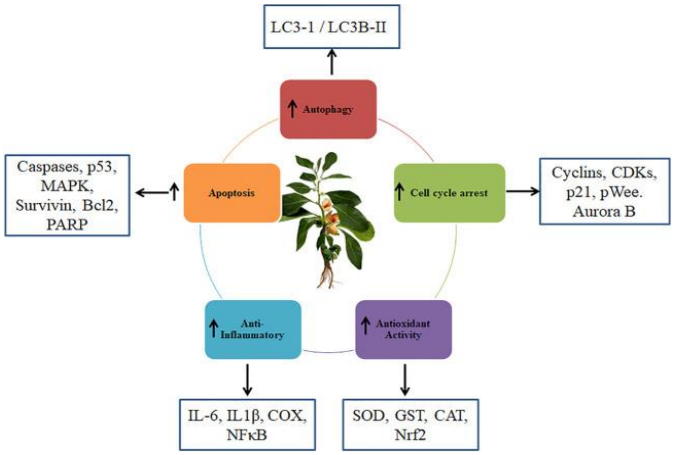

Fig.2 Pleiotropic Effects of *Ashwagandha* in Cancer Modulation

DISCUSSION

Several in vitro studies have evaluated the anticancer effects of Withaferin A on cervical cancer cell lines, including HeLa, SiHa and CaSki.[25,26,27] Withaferin A downregulates HPV oncogenes, restores p53 function, and induces apoptosis via reactive oxygen species generation and mitochondrial disruption in xenograft models, it also demonstrated dose-dependent cytotoxicity, leading to a significant reduction in cell viability. It promotes apoptosis through increased caspase-3 activity and DNA fragmentation and induces cell cycle arrest, particularly at the G2/M phase. These findings suggest that Withaferin A can effectively disrupt cell proliferation and survival in cervical cancer cells. A study reported that Withaferin A significantly inhibited HeLa cell proliferation at micromolar concentrations, further supporting its therapeutic potential in vitro.[28]

In vivo studies using animal models have confirmed the anticancer efficacy of Withaferin A in cervical cancer. In tumor xenograft models, administration of Withaferin A resulted in a notable reduction in tumor volume.A study demonstrated that Withaferin A treatment reduced tumor volume by 60% in cervical cancer xenograft models.[28]

Histopathological analysis revealed an increase in apoptotic markers and a significant reduction in angiogenesis within tumor tissues. Importantly, Withaferin A exhibited minimal systemic toxicity, as no major adverse effects were observed in normal tissues of treated animals.[29]

However, challenges remain in translating these finding to clinical practice, key issues include low oral availability, variability in extract composition, and the need for standardized dosing protocols while the safety profile in preclinical models appear acceptable, well designed clinical trials are necessary to confirm efficacy and establish optimal dosing in humans. Future research should focus on enhancing extract formulations and conducting rigorous human studies to fully validate *Ashwagandha* role in cancer prevention and treatment. The Ayurvedic properties of *Ashwagandha* directly correlate with its modern pharmacological actions against cervical cancer. Withaferin A, the primary active compound, aligns with traditional descriptions of its *Tikta -Kashaya Rasa, Ushna Virya,* and *Rasayana Karma*, which collectively contribute to its anticancer, apoptotic, and immunomodulatory effects.

CONCLUSION

Ashwagandha (*Withania somnifera*) holds significant potential as an adjuvant therapy in the management of cervical cancer. Preclinical studies shows that it enhances the efficacy of conventional treatments such as chemotherapy and radiation, while simultaneously mitigating their side effects like nausea, headache, weakness, hair fall, etc. Its antioxidant, immunomodulatory, and pro-apoptotic properties position it as a valuable adjunct in reducing treatment-related morbidity and improving patient outcomes.

As the field of integrative oncology gains global recognition, *Ashwagandha* is increasingly viewed as a bridge between traditional Ayurvedic wisdom and modern biomedical science. It is as a natural, well-tolerated, and potentially potent therapeutic agent making it suitable for inclusion in complementary cancer care protocols.

However, while in vitro and in vivo studies provide a compelling basis for its therapeutic role, robust clinical validation through well-designed human trials is necessary. Advancing this research could unlock a plant-based therapeutic option with the potential to reshape supportive care and enhance treatment outcomes in one of the world's most prevalent malignancy.

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