Anticancer Agent: Information on Important Herbal Drugs

Gajanan Narayan Mngalvedhe[1], Manoj Rajaram Dhotre[2], Ajay Sanjay Gawade[3], Vaibhav Ramhari Sonavne[4],Nilesh Pralhad Deore Shreekant Dattatray Dhanayat[6] ,Dattu Babasaheb Somase[7], Shreyash Diliprao Dhat[8]

Abstract

Since the number of fatalities rises day by day and year by year, cancer has almost completely destabilised human society. Many studies have been conducted in an attempt to discover a cancer treatment, but to no avail. Herbs have been proven to be effective anticancer agents, and their value in managing and treating cancer cannot be understated. The current evaluation is an honest attempt to assemble the most promising plant-based anticancer medicines and highlight their key cancer-curing potentials.

Keyword*: Anticancer, Herbas, etc…*

1. INTRODUCTION

As long as human civilisation, plants have been used as medicines.Around 60% of anti-cancer medications are derived from plants, including taxol from Taxus brevifolia and camptothecin from Camptotheca acuminata, among others. The management of cancer is still lagging, and new medications for cancer prevention and treatment must be found immediately. The use of plants to treat cancer is still a possibility in this situation.[1]

The incidence and mortality of cancer have recently achieved a high plateau and are a significant global public health issue.

Many studies are devoted to finding novel cancer therapies, and many of these studies concentrate on plant-derived chemicals with therapeutic promise that have been utilised extensively in conventional medicine.[2]

A group of cells exhibit the characteristics of uncontrolled growth, invasion, and occasionally metastasis in cancer, a class of disorders. These three malignant characteristics set cancer apart from benign tumours, which are self-contained, do not penetrate, and do not spread. Most malignancies develop tumours, but some do not, such as leukaemia.

Even foetuses can develop cancer, but the risk for the more prevalent types tends to rise with age. 13 percent of all fatalities are due to cancer. The American Cancer Society estimates that 7.6 million individuals worldwide passed away from cancer in 2007.Other than people, animals and plants can also develop cancer.

Almost all malignancies are brought on by anomalies in the altered cells' genetic code. The effects of carcinogens like tobacco smoke, radiation, chemicals, or pathogenic agents may be to blame for these anomalies. Some genetic mutations that increase the risk of developing cancer might occur at random due to mistakes in DNA replication or can be inherited, making all cells vulnerable to them from birth. Only some people acquire cancer after exposure to a known carcinogen, which may be explained by complex interactions between carcinogens and the host genome. More and more, it is understood that new elements of the genetics of cancer pathogenesis, such as DNA methylation and microRNAs, are crucial.

Two general types of genes are frequently affected by the genetic alterations identified in cancer. Oncogenes that promote cancer are frequently activated in cancer cells, giving them new characteristics such accelerated growth and division, resistance to programmed cell death, disregard for normal tissue boundaries, and the capacity to colonise various tissue settings. Because tumour suppressor genes are frequently deactivated in

cancer cells, those cells lose their capacity for routine activities such precise DNA replication, cell cycle regulation, orientation and adherence within tissues, and communication with immune system defence cells.

The tissue from which the malignant cells arise, the original tumour, as well as the normal cell type they most closely resemble, are typically used to classify cancer. They are, respectively, histology and location. The early sign of malignancy can be symptoms or radiographic imaging abnormalities, but a definite diagnosis usually needs the histologic examination of a tissue sample specimen by a pathologist. Depending on the precise type, location, and stage of the cancer, it is usually treatable and in some cases curable.

Cancer is typically treated with a mix of surgery, chemotherapy, and radiotherapy once it has been diagnosed. Treatments are getting more tailored to the many types of cancer as research advances. Targeted treatment medications that act selectively on observable molecular abnormalities in specific malignancies while minimising harm to normal cells have made major strides. The type of cancer and its stage, or degree of spread, have the biggest effects on a patient's prognosis.

the illness. Moreover, histologic grading and the presence of particular molecular markers might be helpful in evaluating the prognosis as well as specific therapy.

2. Research on Cancer

The first time that cancer was linked to faulty genes was around 1980, and since then, cancer research has grown significantly, substantially reinforcing the underlying principles of contemporary biology. Unraveling the human genome sequence was one of those extraordinary achievements, primarily made possible by cancer research. Many of the remarkable discoveries into the genetic circuits that control developmental processes have also come from cancer research. Many biological fields, including virology, cell biology, classical and molecular genetics, epidemiology, biochemistry, and clinical sciences, collaborate closely in their search for treatments to halt the aberrant cell development that is a hallmark of malignant cells.

3. Role of Plant Based Drugs

The treatment of cancer has benefited greatly by chemicals obtained from plants. Almost every civilisation on earth has acknowledged the healing potential of plants. Folk remedies were primarily generated from natural product extracts in the nineteenth and preceding centuries, especially those that were derived from botanical species.

Around 50% of all prescribed medications in industrialised countries today are made from natural compounds, primarily from plants and microorganisms, which are readily available sources. It is believed that terrestrial plants provide a distinctive and renewable resource for the identification of possible novel medications and biological entities due to the structural and biological diversity of their constituents.

The goals of using plants as sources of therapeutic agents:

In order to create patentable substances with higher activity and/or lower toxicity, such as metformin, nabilone, oxycodon (and other narcotic analgesics), which are based, respectively, on morphine, taxol, podophyllotoxin, etc., it is necessary to produce bioactive compounds with novel or well-known structures as lead compounds for semisynthesis.

To use agents as pharmacologic tools, e.g., lysergic acid diethylamide

. To use the whole plant or part of it as a herbal remedy,

to separate bioactive substances for direct pharmacological usage, such as vinblastine, vincristine, morphine, digoxin, etc.

4. Development of Anticancer Drugs from Plants

There are thought to be 500,000 higher flowering plant species that live in terrestrial settings. Several species have just been skimmed over in terms of their potential for pharmacological and medicinal use. The potential for usage as innovative medicinal agents has only been thoroughly explored in less than 1% of these species.

Historically, the development of anti-cancer medications involved extensive testing of synthetic compounds against animal tumour models, mainly murine leukaemia. The majority of the drugs identified during the first two decades of cancer chemotherapy (1950–1970) interacted with DNA or its precursors, preventing the production of new genetic material or harming DNA irreparably. There have been numerous claims made about how plants can be used to treat cancer.[3]

The development of drugs from medicinal plants has been crucial in the fight against cancer. Between 1940 and 2002, 40% of all anticancer medications were natural products either directly or indirectly, and 8% were considered natural product mimics.[4]

5. Epipodophyllotoxins

Etoposide and teniposide, semi-synthetic derivatives of epipodophyllotoxins, were isolated from Podophyllum peltatum L. and Podophyllum emodi for use in the creation of anticancer medications.[5] The mayapple is the name given to Podophyllum peltatum most frequently, but it is also known as Devil's apple, hog apple, Indian apple, umbrella plant, wild lemon, and American mandrake in some places.

The American Indians and early colonists utilised podophyllotoxin, which is isolated from the mandrake plant (also known as the may apple; Podophyllum peltatum L.), as a folk treatment because of its emetic, cathartic, and anthelmintic properties. The mayapple is a perennial tree found in Canadian and Eastern American woodlands that belongs to the Berberidaceae family, which also includes the barberry. The stem fork of the two-leaved plants often produces a single, tiny white flower (typically in May, thus the name). The sole portion of the plant that isn't dangerous is the flower, which grows into a pulpy, lemon-yellow berry in late summer. The long, thin rhizome of the plant, which is a horizontal underground stem from which the roots sprout, is both the most lethal and the most beneficial due to its high concentrations of the anticancer substances podophyllotoxin and alpha and beta peltatin. Etoposide and teniposide, two of the derivatives of podophyllotoxin (podofilox), are all cytostatic (antimitotic) glucosides. Cells going through mitosis (division) are particularly susceptible to the effects of podofilox, an extract from the mayapple. Podophyllotoxin interacts with tubulin at a different place than the vinca alkaloids do. In standard concentrations, etoposide and teniposide have little impact on microtubular structure or function.[6][7]

The plant's extracts are being utilised in topical treatments for various skin malignancies and genital warts brought on by the human papilloma virus (HPV). You shouldn't try to self-medicate with mayapplerhizome powder because of its potent purgative effects and poisonous chemical composition. The FDA considers this plant's use to be "unsafe."

6. Paclitaxel

Members of the yew family include Taxus brevifolia and Taxus baccata (Taxaceae). The common name for T. baccata is English yew. Although T. brevifolia is more frequently recognised to as the Pacific Yew, it is also sometimes called the Western or American yew. It is the sole authorised source of paclitaxel (Taxol), an anticancer medication that has drawn a lot of interest, according to the FDA. Yews produce vivid red arils, which each hold a single seed. The only portions of the plant that don't contain toxic taxine alkaloids are the arils, which are fairly delicious.

Paclitaxel's anticancer characteristics were first found in the 1960s as a consequence of a vast plant-screening programme conducted by \sthe National Cancer Institute (NCI). Clinical trials to determine its safety began in 1983 after researchers further defined its precise functions (it prevents the breakdown of microtubules, a type of cell structure) in 1979. Other yew species, like the American yew and T. cuspidata, the Japanese Yew, have the compound in smaller amounts. The English yew includes docetaxel, a comparable substance that is sold under the brand name Taxotere. The discovery of paclitaxel, which was originally separated from the bark of a western yew tree in 1971, has shown to be a successful medication for the treatment of breast and ovarian malignancies.[8]

By attaching to microtubules, paclitaxel, marketed by Bristol-Myers Squibb as Taxol, prevents their depolymerization (molecular breakdown) into tubulin.[9][10] This indicates that paclitaxel prevents a cell from dismantling the mitotic spindle during mitosis (cell division). The spindle prevents the cell from dividing into daughter cells, in contrast to medications like colchicine and the Vinca alkaloids that prevent mitosis by preventing the formation of the spindle in the first place. Ovarian and advanced breast cancers are the two conditions for which paclitaxel is most effective when administered intravenously (it irritates skin and mucous membranes on contact).

7. Docetaxel

Docetaxel and Taxotere are both trademarks of Rhone-Poulenc Rorer.It works similarly to paclitaxel in that it stabilises the microtubule bundles to stop the mitotic spindle from being damaged, although clinical trials show that it is nearly twice as effective.Docetaxel, which is also administered intravenously, is being evaluated on non-lymphoma, Hodgkin's malignant melanoma, and carcinomas of the bladder, cervix, lung, and ovaries.

8. Beta-lapachone and Lapachol

Lapachol, a naphthoquinone that may be isolated from the lapacho tree (Tabebuia avellanedae), a member of the catalpa family, is the precursor of a quinone (Bignoniaceae). ß-Lapachone inhibits DNA topoisomerase I similarly to camptothecin and topotecan.

Researchers have discovered that this substance has potential antiviral and anticancer effects. Beta-lapachone and other topoisomerase inhibitors appear to be effective against a number of malignancies, including lung, breast, colon, prostate, and malignant melanoma. Due to its toxicity, beta-lapachone use in humans has been restricted. Beta-lapachone has been discovered to be more harmful than 3-allyl-beta-lapachone, which suggests that 3-allyl-beta-lapachone may be more beneficial.

Beta-lapachone affects DNA replication in order to function. The chromosomal DNA is unwound by an enzyme called topoisomerase I. The cell must unwind the chromosomes in order to utilise the genetic material for protein synthesis. Beta-lapachone prevents the cell from producing proteins by keeping the chromosomes tightly wrapped. The cell thus halts its growth. Cancer cells are more susceptible to topoisomerase inhibition than normal cells because of their considerably quicker pace of growth and reproduction. Moreover, beta-lapachone slows the progression of HIV-1 by interfering with its replication.

9. Colchicine

The autumn crocus contains an alkaloid that slows or prevents cell division by preventing mitosis. In particular, it prevents spindles from forming as the nuclei divide. Usually, the cell would divide into two new cells, each of which would contain a single set of chromosomes, using its spindle fibres to arrange its chromosomes, copy them, and do so.

Colchicine prevents the formation of spindle fibres, which prevents the cell from moving its chromosomes. The cell may copy some or all of the chromosomes in the end, but because it is unable to split, it never does. Compared to normal cells, cancer cells divide significantly more quickly, making them more vulnerable to poisoning. by mitotic inhibitors such as vincristine and vinblastine from the Vinca alkaloids, paclitaxel, and colchicine.

10. Vincristine and Vinblastine

The plant known as the Madagascar periwinkle yields vinca alkaloids. They are extracted from the pink periwinkle plant Catharanthus roseus G. Don and are naturally occurring or partially manufactured nitrogenous bases. There are four main vinca alkaloids that are used in medicine:Vincristine, vinblastine, vinorelbine, and vindesine (VBL, VRL, VDS), however only VCR, VBL, and VRL are permitted for usage in the US.

Vinca alkaloids primarily cause metaphase arrest by interfering with the function of microtubules, particularly those microtubules that make up the mitotic spindle apparatus, and by interacting with tubulin. They can perform a wide range of other metabolic processes, though, which might or might not be connected to their effects on microtubules. Many adverse consequences, many of which do not involve microtubule disruption, only manifest after treatment ofcells with dosages of the vinca alkaloids that are not clinically relevant. Yet, because microtubules play a significant role in a number of nonmitotic processes, vinca alkaloids and other antimicrotubule drugs also have an impact on both benign and malignant cells during the nonmitotic cell cycle.

The binding sites on tubulin that the vinca alkaloids bind to are distinct from those used by taxanes, colchicine, podophyllotoxin, and guanosine 5' triphosphate. Rapid binding is possible, as well as reversal. There are two vinca alkaloid binding sites for every mole of tubulin dimer, according to the evidence currently available.

11. Developments Towards Newer Anticancer Agents

Biologically active substances from higher plants have been essential in the early development of modern medicine in giving treatments for diseases and suffering. For instance, the 1932 British Pharmacopoeia organic monographs

over 70% of the items are made from plants. Nonetheless, the use of therapeutic compounds originating from plants has drastically decreased (mainly) in economically developed countries since the development of synthetic medicinal chemistry. The body of an animal is more poisonous to synthetic medications. Along with harming healthy cells in the body, they cause significant side effects that are not only long-lasting but could endanger the patient's life.

In Asia, ganoderma lucidum, also known as Lingzhi in Japan or Reishi in China, has been utilised to promote health. for millennia. It is regarded as a natural remedy that supports longevity and preserves a person's vitality. Recent pharmacologic investigations have verified its beneficial therapeutic effects in patients with hepatitis, hyperglycemia, chronic bronchitis, cancer, muscular dystrophy, arteriosclerosis, hypertension, hypercholesterolemia, and leukopenia. Recently, the fruiting bodies, mycelia, and spores have drawn increasing attention as both new medication sources and home cures.[12][13] Both in vitro and in vivo studies have proven that G. lucidum has anti-cancer properties [14]. s. Moreover, cancer patients have begun using Ganoderma in conjunction with chemotherapy due to its anti-cancer properties [15]. In rodent models, Ganoderma's efficacy in benign prostatic hyperplasia has already been documented [16].

The herbal medication used in China For the treatment of liver cancer, radix sophorae is frequently used as an anti-carcinogenic and anti-metastatic drug.

Leachianone A, isolated from Radix sophora, demonstrated strong cytotoxic action against the human hepatoma cell line HepG2 in vitro, with an IC50 value of 3.4 mg/ml following a 48-hour treatment, according to Cheung et al. Both intrinsic and extrinsic apoptosis pathways were implicated in its mode of action. Further evidence of its anti-tumor impact was provided in vivo by a 17–54% decrease in tumour size in nude mice bearing HepG2;

no toxicity to the heart or liver tissues was seen. The isolation of Leachianone A from Radix sophora and the molecular basis for its anti-proliferative effect on HepG2 cells are described in this paper for the first time [19].

Punica granatum, the pomegranate tree, and its fruit, in particular, have a long past in ethnomedicine and serve as a reservoir of phytochemicals with potential therapeutic benefits. The seed, juice, peel, leaf, flower, bark, and roots are just a few anatomical divisions of the fruit or tree that each have intriguing pharmacologic properties. Juice and peels, for instance, have strong antioxidant properties. Juice, peels, and oil, on the other hand, all have weak estrogenic properties and may be of interest for the therapy of menopausal symptoms and their aftereffects. Juice, peels, and oils have also been shown to have anticancer properties, such as inhibiting tumour cell growth, cell cycle, infiltration, and angiogenesis.[20]

A pentacyclic triterpene known as betulinic acid is a typical secondary metabolite of plants, mainly from the Betula species (Betulaceae).

Ziziphus mauritiana Lam. (Rhamnaceae), which was gathered in Zimbabwe, was extracted by Pisha et al. in 1995. The specific cytotoxicity of the extract against human melanoma cells was shown to be ethyl acetate-soluble (MEL-2). Then, betulinic acid was shown to have low toxicity and be efficacious in vivo using athymic mice injected with human melanomas.

Additional biochemical research revealed that the mechanism by which betulinic acid causes apoptosis[21] Additionally continuing is pre-clinical development for a topical formulation.

Different researchers have demonstrated that turmeric has a wide range of pharmacological characteristics, including anti-inflammatory, anti-carcinogenic, and anti-oxidant properties. According to Yasmin et al. (1998), turmeric also stimulates lymphocytes and causes tumour cells to undergo apoptosis.[22] Swiss albino mice were used to test the methanolic extract of Glinus lotoides' anticancer effects on Dalton's ascitic lymphoma (DAL). a considerable increase in the average survival rate

In comparison to the control group, peritoneal cell count in normal mice and time of tumor-bearing mice were both detected.[24] The main diterpenoid in the extract of Andrographis paniculata, andrographolide, has demonstrated cytotoxic effect against the cells KB (human epidermoid carcinoma) and P-388 (lymphocytic leukaemia).

In M1 (mouse myeloid leukaemia) cells, the methanol extract of Andrographis paniculata aerial parts and some of the isolated components displayed growth inhibitory and differentiation activities.[25] In several cell lines of human tumour cells, -hydroxyisovalerylshikonin (HIVS), which was extracted from the plant Lithospermum radix (roots of Lithospermum erythrorhizon), induces apoptosis.

The induction of apoptosis may be significantly aided by the inhibition of PLK-1 (polo-like kinase 1) activity by -HIVS through inhibition of tyrosine kinase activity.[26] A new lappadilactone and seven sesquiterpene lactones were isolated from Saussurea lappa using bioassay-directed fractionation, and they were found to be effective cytotoxic agents against certain human cancer cell lines. The most effective cytotoxicity was shown by lappadilactone, dehydrocostus lactone, and costunolide against Hep-G2, OVCAR-3, and HeLa cell lines.[27] Significant levels of polyphenolic chemicals are present in litchi fruit pericarp (LFP) extract, which also displays potent antioxidant action against the in vitro oxidation of fat. This study established the LFP extract's anticancer effectiveness against human breast cancer both in vitro and in vivo and clarified how it works.[28] Significant levels of polyphenolic chemicals are present in litchi fruit pericarp (LFP) extract, which also displays potent antioxidant action against the in vitro oxidation of fat. This study established the LFP extract's anticancer effectiveness against human breast cancer both in vitro and in vivo and clarified how it works.[29]

When CD lignan combination was administered for nine days at 300 mg/kg, i.p., Ehrlich ascites carcinoma and CA-51 showed 53% and 54%, respectively, tumour shrinkage.days in the CA-51 paradigm and 400 mg/kg, i.p. for the same duration in the mice bearing Ehrlich ascites cancer. At 22 mg/kg and 20 mg/kg, it was comparable to 5-fluorouracil.

In Korea, pine needles (Pinus densiflora Siebold et Zuccarini) have a long history of use as a traditional medicine food that promotes health.

Antioxidant, antimutagenic, and antitumor properties were evaluated in vitro and/or in animals by Kwak et al., to study their possible anticancer effects (2006). When compared to normal cells (HDF) in 3-(4, 5-

dimethylthiazol2-yl)-2, the proliferation of cancer cells (MCF-7, SNU-638, and HL60) was significantly inhibited by PNE exposure. When CD lignan combination was administered at 300 mg/kg, i.p. for nine 5-diphenyltetrazolium bromide assays, tumour regression was seen in Ehrlich ascites carcinoma and CA-51 at rates of 53% and 54%, respectively. In in vivo anticancer investigations, rats treated with the mammary carcinogen 7, 12-dimethylbenz [a] anthracene (DMBA, 50 mg/kg body weight) or mice injected with Sarcoma-180 cells were fed diets enriched with freeze-dried pine needle powder (5%, wt/wt). Pine needle addition in the two model systems reduced tumorigenesis. In the DMBA-induced breast tumour model, rats fed with pine needles had significantly reduced blood urea nitrogen and aspartate aminotransferase levels. Their findings showed that pine needles had potent anticancer, antimutagenic, and antiproliferative effects on cancer cells as well as antitumor effects in vivo, and they suggest their potential usefulness in cancer prevention [30]

India's "wonder shrub" ashwagandha is frequently utilised in Ayurveda medicine and health products that tout their For nine 5-diphenyltetrazolium bromide assays, CD lignan combination was given at a dose of 300 mg/kg, intravenously. Ehrlich ascites carcinoma and CA-51 both saw tumour regression at rates of 53% and 54%, respectively. In in vivo cancer research, mice and rats given Sarcoma-180 cell injections or the mammary carcinogen 7, 12-dimethylbenz [a] anthracene (DMBA, 50 mg/kg body weight) were given meals enhanced with freeze-dried pine needle powder (5%, wt/wt). In the two model systems, adding pine needles decreased tumorigenesis. Rats given pine needles exhibited considerably lower blood urea nitrogen and aspartate aminotransferase levels in the DMBA-induced breast cancer model. Their results demonstratedresearch suggested that pine needles showed strong anticancer, antimutagenic, and antiproliferative effects on cancer cells in addition to antitumor activities in vivo.[31]

12. Conclusion

Finding naturally occurring compounds that can suppress, delay, or reverse the multistage carcinogenesis process has received a lot of interest in recent years.

The best anticancer medications are those with few adverse effects, induce apoptosis, and specifically target cancer cells.

13. References

[1] . Verma M, Singh SK, Bhushan S, SharmaVK, DattP, et al., (2008) In vitro cytotoxic potential of Polyalthia longifolia on human cancer cell lines and induction of apoptosis through mitochondrial-dependent pathway in HL-60 cells. Chem Biol Interact. 171(1): 45-56.
[2] Shi M, Cai Q, Yao L, Mao Y, Ming Y, et al., (2006) Antiproliferation and apoptosis induced by curcumin in human ovarian cancer cells. Cell Biol Int. 30(3): 221-6.
[3] Hartwell JL (1971) Plants used against cancer- A survey. Lloydia. 34(4): 204-55.
[4] Newman DJ, Cragg GM, Snader KM (2003) Natural products as sources of new drugs over the period 1981-2002. J Nat Prod. 66(7): 1022-37.
[5] Cragg GM, Schepartz SA, Suffness M, Grever MG (1993) The taxol supply crisis. New NCI policies for handling the large scale production of novel natural product anticancer and anti-HIV agents. J Nat Prod. 56(10): 1657-68.
[6] Hande KR (1998) Etopside: four decades of development of a topoisomerase II inhibitor. Eur J Cancer. 34(10): 1514-21.
[7] Chabner BA, Lango DL (2001) Topoisomerase II inhibitors: the epipodophyllotoxins, m–AMSA and the ellipticine derivatives. Cancer Chemotheraphy and Biotherapy: Principles and Practice. 3rd (Edn), Lippoincott Williams and Wilkins, Philadelphia.
[8] Wani MC, Taylor WHC, Coggon ME, Phail MC (1971) Plant antitumor agents. The isolation and structure of taxol, a novel antileukemic and antitumor agent from Taxus brevifolia .J Am Chem Soc. 93(9): 2325-7.
[9] Schiff PB, Fant J, Horwitz SB (1979) Promotion of microtubule assembly in vitro by taxol. Nature. 277(5698): 665-7.
[10] Horwitz SB (2004) Personal recollections on the early development of taxol. J Nat Prod. 67(2): 136-8.
[11] Moudi M, Go R, Yien CSK, Nazre M (2013) Vinca Alkaloids. Int J Prev Med. 4(11): 1231–5.

[12] Nahata A (2013) Ganoderma lucidum: A Potent Medicinal Mushroom with Numerous Health Benefits. Pharmaceut Anal Acta. 4: e159
[13] Gao JJ, Nakamura N, Min BS, Hirakawa A, Zuo F, et al., (2004) Quantitative determination of bitter principles in specimens of Ganoderma lucidum using high-performance liquid chromatography and its application to the evaluation of Ganoderma products. Chem Pharm Bull. 52(6): 688-95.
[14] Nahata A, Saxena A, Suri N, Saxena AK, Dixit VK (2013) Sphaeranthus indicus induce apoptosis through mitochondrial-dependent pathway in HL-60 cells and exerts cytotoxic potential on several human cancer cell lines. Integr Cancer Ther. 12(3): 236-47.
[15] Chi HJK, Amalini CJ, Karen SB, Marcus PG, Lynnette RF (2013) Anticancer activities of Ganoderma lucidum: active ingredients and pathways. Funct Food Health Dis. 3(2): 48-65.
[16] Nahata A, Dixit VK (2012) Ganoderma lucidum is an inhibitor of testosterone induced prostatic hyperplasia in rats. Andrologia. 44(1): 160-74.
[17] Nahata A, Dixit VK (2011) Sphaeranthus indicus attenuates testosterone induced prostatic hypertrophy in albino rats. Phytother Res. 25(12): 1839-48.
[18] Nahata A, Dixit VK (2012) Ameliorative effects of Stinging Nettle (Urtica dioica) on testosterone-induced prostatic hyperplasia in rats. Andrologia. 44(1): 396-409.
[19] Cheung CSF, Chung KKW, Lui JC, Lau CP, Hon PM, et al., (2007) Leachianone A as a potential anti-cancer drug by induction of apoptosis in human hepatoma HepG2 cells. Cancer Lett. 253(2): 224-35.
[20] Lanksy EP, Robert AN (2007) Punica granatum (pomegranate) and its potential for prevention and treatment of inflammation and cancer. J Ethnopharmacol. 109(2): 177–206.
[21] Pisha E, Chai H, Lee IS, Chagwedera TE, Farnsworth NR, et al., (1995) Discovery of betulinic acid as a selective inhibitor of human melanoma that functions by induction of apoptosis. Nat Med. 1(10): 1046-51.
[22] Yasmin H, Mazumdar T, Mittal P, Chakravarty AK (1998) Turmeric mediated activation of lymphocytes and induction of apoptosis of tumor cells. J Immunol Cell Biol. 16(5): 221-32.
[23] Gupta NK, Nahata A, Dixit VK (2010) Development of Spectrofluorimetric Method for the determination of curcumin. Asian J Traditional Med. 5(1): 12-8.
[24] Kavimani S, Manisenthilkumar KT, Ilango R, Krishnamoorthy G (1999) Effect of the methanolic extract of Glinus lotoides on Dalton's ascitic lymphoma. Biol Pharm Bull. 22(11): 1251-2.
[25] Kumar A, Sridevi K, Kumar NV, Nanduri S, Rajagopal S (2002) Hypericin in cancer treatment: more light on the way. Int J Biochem Cell Biol. 34(3): 221-41.
[26] Masuda Y, Nishida A, Huri K, Hirabayashi T, Kajimoto S, et al., (2003) β-Hydroxyisovalerylshikonin (HIVS) induces apoptosis in human leukemia cells by inhibiting the activity of a Polo-like kinase 1(PLK-1). Oncogene. 22(7): 1012-23.
[27] Ming SC, Syu Wan Jr, Jaw DM, Jin LJ, Hee LG (2003) Cytotoxic sesquiterpene lactones the root of Saussurea lappa. J Nat Prod. 66(9): 1175-80.
[28] Wang X, Yuan S, Wang J, Lin P, Liu G, et al., (2006) Anticancer activity of litchi fruit pericarp extract against human breast cancer in vitro and in vivo. Toxicol Appl Pharmacol. 215(2): 168–78.
[29] Singh SK, Shanmugavel M, Kampasi H, Singh R, Mondhe DM, et al., (2007) Chemically Standardized Isolates from Cedrus deodara Stem Wood having Anticancer Activity. Planta Med. 73(6): 519-26.
[30] Kwak CS, Moon SC, Lee MS (2006) Antioxidant, antimutagenic, and antitumor effects of pine needles (Pinus densiflora). Nutr Cancer. 56(2): 162-71.
[31] Widodo N, Kaur K, Shrestha BG, Takagi Y, Ishii T, et al., (2007) Selective killing of cancer cells by leaf extract of Ashwagandha: identification of a tumor-inhibitory factor and the first molecular insights to its effect. Clin Cancer Res. 13(7): 2298-306.
[32] Sharma M, Agrawal SK, Sharma PR, ChadhaBS, KhoslaMK, et al., (2010) Cytotoxic and apoptotic activity of essential oil from Ocimum viride towards COLO 205 cells. Food Chem Toxicol. 48(1): 336-44.