

## Herbal Treatment of Cancer: A Review

Navneet Kumar Verma<sup>\*1</sup>, Asheesh Kumar Singh<sup>2</sup>, Pratibha Kasaudhan<sup>3</sup>, Sejal Srivastava<sup>3</sup>, Nalini Paswan<sup>3</sup>

<sup>1</sup>Associate Professor, Buddha Institute of Pharmacy, GIDA, Gorakhpur, UP, India-273209 Affiliated to Dr. APJ Abdul Kalam Technical University, Lucknow, Uttar Pradesh, India

<sup>2</sup>Professor & Director, Buddha Institute of Pharmacy, GIDA, Gorakhpur, UP, India-273209 Affiliated to Dr. APJ Abdul Kalam Technical University, Lucknow, Uttar Pradesh, India

<sup>3</sup>Student of B. Pharmacy, Buddha Institute of Pharmacy, GIDA, Gorakhpur, UP, India-273209 Affiliated to Dr. APJ Abdul Kalam Technical University, Lucknow, Uttar Pradesh, India

### ABSTRACT

The term "cancer" refers to a broad range of disorders that can occur anywhere at any moment, but all share the unchecked proliferation of aberrant cells. This article has been made to review some medicinal plants used for the treatment of cancer. Cancer is a group of diseases characterised by uncontrolled growth and spread of abnormal cells. If the spread is not controlled, it can result in death. Mutations in genes can cause cancer by accelerating cell division rate or in habit in normal controls on the system such as cell cycle arrest or programmed cell death. As a mass of cancerous cells grows, it can develop into a tumour. As plant-derived anticancer medicines, taxans, vinca, alkaloids, podophylotoxin, and camptothecin have all been employed in clinical settings. New technologies are emerging to develop the area for the new technologies involve nanoparticles for nanomedicines which aim to increase anticancer activities of plant-derived drugs by managing the release of the compound and researching new methods for administration. Herbal medicines have a vital role in the prevention and treatment of cancer.

**Keywords:** Herbal Treatment, Cancer, Uncontrolled Growth.

**\*Corresponding Author**

**Navneet Kumar Verma**

Buddha Institute of Pharmacy, GIDA, Gorakhpur, UP,  
India-273209



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### INTRODUCTION

Cancer is described as an uncontrolled cell growth due to loss of differentiation that spreads to other tissues and organs through metastasis. Chewing tobacco, drinking alcohol in combination, smoking, and being exposed to certain chemical carcinogens are all known to cause cancer. According to recent estimates, herbal medications are used by 13% to 63% of cancer patients. As a result, there is a growing demand for adverse drug interactions in medical oncology, thus it is essential to understand these interactions. Clinical trials have shown that CHM (Chinese Herbal Medicine) can increase tumour response, improve survival, and lessen the chemotherapy's toxicity. CHM can also increase effectiveness while reducing toxic reaction. These raised the feasibility combination of chemotherapy and herbal medicines, although much remained to be investigated in this area [1-3].

#### General scenario of herbal medicine

With a lengthy history of changing practices, herbal therapy is used all across the world today. Animal or herbal remedies are still heavily used in their early stages in many nations, including Greece. Ayurveda, traditional Chinese medicine (TCM), and allopathic medicine in India. However, the majority of these nations abandoned this custom. Since China is a well-known nation and had a dominant publishing system in the ancient world, it has preserved many ancient books and continues to practise herbal medicine now [3].

#### Major challenge

In medicinal chemistry of herbal medicine is a very complicated. There should be new initiatives that must be explored to overcome these kinds of obstacles. This perspective highlights the efforts against cancers.

#### Pharmacological characters

The difference between synthetic drugs and natural chemical drugs, natural chemical drugs is of stronger medical armaments, low- rate of acquired drug-induced resistance in clinical trials as well as high therapeutic index.

#### From herbal medicine to natural chemical drug

There are many differences between natural chemical drugs and herbal medicine. However, a deeper understanding of herbal medicine may help us discover more effective natural chemical drugs. To clarify our vision on this matter, the major characters of TCM must be introduced.

### **Drug development transformation**

Facing the situation of cost surge, high risk and low productivity in modern drug development, creative study for technology and science can provide unprecedented insights into therapeutics against cancer [4].

### **CELL CYCLE OF CANCER**

A "cell division cycle" is a series of synchronised events that includes the processes of duplicating DNA and dividing a cell. It is known that cells can be controlled by at least two different types of mechanisms: a series of protein phosphorylations that relay a cell from one stage to the next and a series of checkpoints that keep track of when crucial events are completed and postpone progression to the following stage if necessary. A family of kinases that is tightly regulated is used as the first type of control. The periodic "cyclin" subunit associates with its partner "cyclin-dependent kinase" (CDK) to create an active complex with particular substrate specificity. Kinase activation typically necessitates association with a second subunit that is transiently expressed at the appropriate time of the cell cycle. The activity of CDK-cyclin complexes is finely tuned by regulatory phosphorylation and dephosphorylation, ensuring a clearly defined transition between cell cycle stages. Checkpoint control, a different sort of cell cycle regulation, is more controlling. [5] It is not a crucial component of the mechanism that advances the cycle. Checkpoints in the cell cycle detect errors in crucial processes including DNA replication and chromosomal segregation. Signals are sent to the machinery that controls cell cycle progression when checkpoints are engaged, such as when DNA is not duplicated or is damaged. The advancement of the cycle is delayed as a result of these signals until the threat of mutation has passed. The scope of checkpoint function is less clear than that of process-essential components like CDKs because checkpoint function is not necessary in every cell cycle. The relationship between the cell cycle and cancer is intuitive on the surface: the cell cycle regulates cell proliferation, and cancer is a condition caused by uncontrolled cell proliferation. Fundamentally, all tumours allow for an excessive number of cells to exist. Gain-of-function mutations were the first genetic changes to be linked to the emergence of cancer. A group of "oncogenes," which are mutant variations of typical cellular "protooncogenes," are defined by these alterations[6]. Protooncogenes' byproducts have a role in signalling systems that encourage cell division. However, the ability of a particular oncogene to transform a cell can be redundant (a mutation in one of several genes can result in transformation) or cell type-specific (mutations would change some cells but not others). This shows that there are numerous, unique genetic modification pathways that might result in cancer, but that not every pathway affects every type of cell in the same manner. Recently, it has become more and more clear how important loss-of-function mutations are to the development of cancer. Initially, it was thought that mutations in these so-called "tumour suppressor" genes played a significant part in inherited cancer susceptibility. People heterozygous for mutations at the locus have normal phenotypes because loss of function of a tumour suppressor gene requires inactivation of both copies of the gene. Loss-of-function tumour suppressor mutations can therefore be passed through the gene pool without having a negative impact, in contrast to gain-of-function mutations. However, because only one mutational event is necessary to inhibit the synthesis of any functional gene product, people who are heterozygous for tumour suppressor mutations are more likely to acquire cancer. Tumour suppressor gene mutations now appear to be highly likely to promote and maybe even be necessary for a significant percentage of both spontaneous and hereditary forms of cancer. But how does a normal cell use the tumour suppressor gene product? There is some evidence that numerous tumour suppressor genes encode proteins that negatively control cell cycle progression, even though this is a subject for more study. For instance, it would be expected that the loss of activity of the tumour suppressor gene product pRb would release E2F transcriptional activators without the need for phosphorylation and circumvent a typical negative regulation controlling entry into the cycle. Similar results would result from the loss of the tumour suppressor gene product p16, which would allow E2Fs to be released by increasing pRb phosphorylation. Additionally, the tumour suppressor gene product p53, which is activated in response to checkpoints sensing DNA damage and probably also chromosome damage, can stop the continuation of the cell cycle at multiple points. If p53 were to disappear, this brake on cycling would no longer exist [7].

### **ANTICANCER HERBAL DRUGS**

#### **1. Vinca Alkaloids**

##### **Mechanism of action**

The mechanism of action of vinca alkaloids is to arrest dividing cell in metaphase by binding tubulin and preventing its polymerization into microtubules. This is also the proposed mechanism of causing neuropathy by inhibiting anterograde and retrograde axonal transport, thereby causing axonal degeneration. Toxicity is dose-dependent and is associated with a sensor motor neuropathy.

##### **Pathogenesis**

Vinca alkaloids bind to tubulin, stop it from polymerizing into microtubules from soluble dimers, and interfere with normal mitotic spindle function. In the rat sciatic nerve, misorientation of microtubules and neurofilaments has been

noted. Microtubules play a role in axoplasmic transport in addition to the mitotic spindle. Axoplasmic flow disruption caused by the disintegration of micro/neurotubules may be the first physiological lesion in vinca alkaloid-induced neuropathy, which later results in axonal degeneration. In fact, vincristine caused axoplasmic transport to be blocked at varying doses in experimental models, along with the loss of microtubules and axonopathy.



**Fig 1. Vinca alkaloids**

### **Therapeutic use**

Vinca alkaloids such as vinblastine, vinorelbine, vindesine, and vincristine are used in the treatment of lymphomas, acute lymphocytic leukemia, and solid tumors [6].

### **1.Epipodophyllotoxin**

Epipodophyllotoxins are semisynthetic derivatives of Mayapple plant. It induces single- and double-strand breaking of DNA, inhibiting or altering DNA synthesis.

### **MECHANISM OF ACTION**

A DNA single- and double-strand break-causing epipodophyllotoxin. Most effective during the S and G2 phases of cell division; cell cycle dependent and phase-specific. DNA re-ligation is inhibited by etoposide's inhibition of DNA topoisomerase II. This results in significant mistakes in DNA synthesis during the premitotic stage of cell division, which may cause the cancer cell to undergo apoptosis. Etoposide develops anti-tumor action when topoisomerase II alpha isoform is inhibited. Although the medication has the ability to inhibit the beta isoform as well, this target's suppression is not linked to the anti-tumor efficacy. Instead, it is linked to the cancer-causing impact [8].

### **Pharmacokinetics**

Variably absorbed from the GI tract. Rapidly distributed, low concentrations in CHF. Protein binding;97% metabolized in the liver. Primarily excreted in urine. Not removed by hemodialysis. Half-life; 3-12 hr.

### **Therapeutic use**

Epipodophyllotoxin is used in the Leukemias, testicular cancer lymphomas, small cell carcinoma of the lung[8].

### **CONCLUSION**

In both wealthy and underdeveloped nations, cancer is gaining in popularity. The WHO reported in 2007 that 7.6 million people died from cancer-related illnesses in 2005, the majority of whom resided in low-income nations. As a result, there is a huge need for a cancer treatment and prevention. Drugs with anticancer activity that are chemically generated have been produced, and it is important to screen them for any useful information. This review provided some of the plants having anticancer action for different types of cancer, which may also aid in conservation.

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