International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

Venom - A Boon for Cancer Treatment and Prevention

Divya Sharma, Ayushi Kapoor, LaxmiYadav, Vedansh Srivastava, Shailender Kumar (Assistant Professor)
Dilip Kishore Mehrotra Institute of Pharmacy, Sitapur

Date of Submission: 28-05-2025 Date of Acceptance: 08-06-2025

ABSTRACT:Cancer remains one of the leading causes of death globally, prompting ongoing efforts to discover innovative and effective therapies. Among emerging alternatives, animal venoms have gained attention for their unique biochemical compounds with potential anticancer properties. This project explores the therapeutic effects of venoms derived from snakes, scorpions, bees, ants, and spiders. These venoms are composed of bioactive proteins, peptides, and enzymes capable of inducing apoptosis, inhibiting angiogenesis, and blocking cancer cell proliferation and metastasis. The study emphasizes the pharmacological roles of specific venom components like melittin, chlorotoxin, phospholipase A2, and Solenopsis. Additionally, the potential for venom-derived compounds to serve as targeted cancer therapies especially when integrated with nanotechnology is highlighted. Although further research is essential to ensure safety and specificity, venom-based therapies represent a promising frontier in oncology.

Keywords
Venom, Cancer, Anticancer agents, Snake venom, Scorpion venom, Bee venom, Spider venom, Ant venom, Apoptosis, Angiogenesis, Metastasis, Nanotechnology, Melittin, Chlorotoxin, Solenopsis, Phospholipase A2, Targeted therapy

I. INTRODUCTION

Cancer is a major health concern across both developed and developing nations. Due to its multigenic and multicellular characteristics, it can originate in any organ or cell type, making it a highly complex disease with multiple causes. Genetic mutations play a central role in most cancer types, leading to modifications in the behaviour and function of regulatory proteins. These alterations disrupt cellular signalling pathways, enabling uncontrolled cell division and proliferation.

Globally, cancer stands as a leading cause of mortality, which has fuelled the continuous development of various treatment strategies such as chemotherapy, radiotherapy, immunotherapy, and gene therapy. Chemotherapy is still the most widely used treatment, although its long-term effectiveness is challenged by the development of drug resistance. Radiotherapy, responsible for about 40% of treatment success, aims to destroy cancer cells using radiation. However, its limitation lies in harming surrounding healthy tissue, leading to potential toxicity.

Immunotherapy has emerged as a promising avenue in recent decades. It enhances the immune system's natural ability to fight cancer by using tumour specific antigens as a target. Unlike conventional therapies, immunotherapy minimizes widespread toxicity and aims to empower immune cells to eliminate tumours. However, many cancers develop resistance through immune evasion mechanisms, requiring advanced strategies to overcome them.

Cancer begins when normal regulatory mechanisms of cell growth fail, leading to uncontrolled division and possible tumour formation. Tumours may be benign or malignant. While benign tumours usually remain localized, malignant tumours can invade nearby tissues and spread through the body via a process called metastasis.

II. DEFINITION OF CANCER

Cancer refers to a broad group of diseases characterized by abnormal cell growth with the ability to invade nearby tissues or spread to other parts of the body. These abnormal growths are called neoplasms or tumours. While many tumours form visible masses or lumps, some may grow in a more dispersed manner.Malignant tumours exhibit certain hallmark features that differentiate them from normal cells. These characteristics include:

- Uncontrolled cell growth in the absence of proper growth signals
- Continued division even when inhibitory signals are present
- Evasion of programmed cell death (apoptosis)
- Unlimited potential to replicate

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

- Ability to stimulate the formation of new blood vessels (angiogenesis)
- Invasion into surrounding tissues and the ability to spread (metastasize)

III. SIGNS AND SYMPTOMS OF CANCER

The symptoms of cancer depend largely on the location and type of the tumour. However, several general signs may be observed, though they are not specific to cancer:

- Constant fatigue
- Unusual lumps or thickened areas under the skin
- Unexplained weight loss or gain
- Skin abnormalities such as discoloration, persistent sores, or changes in existing moles
- Changes in bowel or urinary habits
- Continuous coughing or difficulty breathing
- Swallowing problems
- Hoarseness in voice
- Frequent indigestion or discomfort post meals
- Persistent, unexplained muscle or joint aches
- Night sweats or ongoing fevers without a known cause
- Unexplained bleeding or easy bruising

IV. MECHANISM OF CANCER

In the human body, epithelial and connective tissues maintain structural and biochemical balance. Cancer develops when this balance is disrupted, often due to the replacement of normal cells by abnormal ones influenced by heavy metals or toxic compounds. These elements interfere with molecular stability, leading to changes in cellular interactions and uncontrolled bifurcation of cell structures.

This disruption can result in:

- Abnormal direction of cell division
- Accumulation of harmful metal ions that damage nearby tissues
- Enlarged channels around cancer cells that absorb nutrients rapidly, causing metabolic imbalance
- Damage to healthy neighbouring cells due to altered electric potentials and ionic flow
- Inefficient elimination of these abnormal cells by metabolic organs due to size and structure issues

Radiation exposure may also cause substitution of essential ions in blood, leading to impaired energy transfer and further disruption of cellular functions. The molecular size of these substituted compounds often prevents proper processing by capillaries and other small vessels.

V. CAUSE OF CANCER

Although the exact origins of cancer remain unclear, multiple contributing factors have been identified. While some cancers are linked to inherited genetic mutations, such cases account for less than 5% of total occurrences. For instance, mutations in BRCA1 and BRCA2 genes are known to increase the risk of breast cancer, yet they are found in a small percentage of breast cancer cases.

Most cancers result from genetic changes that occur during an individual's lifetime, typically due to environmental exposures. Common environmental factors associated with cancer risk include:

- **Tobacco smoke**: A primary cause of lung cancer and also linked to cancers of the mouth, larynx, oesophagus, bladder, and cervix.
- **Chemical exposure**: Industrial substances such as asbestos, benzene, and certain dyes have been associated with increased cancer risk.
- **Radiation**: Ionizing radiation is a known carcinogen, though the threshold level of exposure that triggers cancer is still not fully established.
- **Viral infections**: Viruses like HIV (which increases the risk of liver cancer and lymphomas) and HPV (associated with cervical, oral, and anal cancers) are known contributors.
- **Sunlight**: Prolonged exposure to UV radiation can cause skin damage and lead to skin cancer.

VI. PATHOPHYSIOLOGY OF CANCER

a. Genetics

Cancer fundamentally arises from disruptions in the mechanisms that regulate tissue growth. For a normal cell to become cancerous, genes controlling growth and differentiation must undergo changes. These alterations can happen due to chromosomal abnormalities or more commonly, due to mutations alterations in the DNA sequence.

Certain environments make these mutations more likely, such as those containing carcinogens, repetitive physical trauma, heat, ionizing radiation, or a lack of oxygen (hypoxia).

Cancers acquire several key characteristics, including:

- Evasion of apoptosis (programmed cell death)

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

- Independence from external growth signals
- Resistance to inhibitory signals
- Sustained blood supply through angiogenesis
- Unlimited replication potential
- Ability to invade surrounding tissues and metastasize

b. Epigenetics

Epigenetic changes are modifications to gene function that do not involve changes to the DNA sequence itself. These alterations can include:

- **DNA methylation** (either excessive or deficient)
- **Histone modifications**
- **Changes in chromatin structure** due to proteins like HMGA1 and HMGA2

Such modifications can silence crucial genes responsible for DNA repair. When repair genes are inactivated, DNA damage builds up, leading to a higher risk of further mutations and potential transformation into cancer cells. In some cases, the process of repairing double-stranded DNA breaks may itself lead to incomplete or faulty repair, which can also result in epigenetic silencing of essential genes. This mechanism underlines the importance of both **DNA damage** and **epigenetic disruption** in the onset and progression of cancer.

c. **Metastasis**

Metastasis refers to the spread of cancer cells from the original tumour to distant organs. These secondary tumours are called **metastatic tumours**, while the original is termed the **primary tumour**.
Almost all cancers could metastasize, especially in their advanced stages. Cancer cells can spread through the bloodstream, the lymphatic system, or both. The general steps of metastasis include:

- **Local invasion** into nearby tissues
- **Intravasation** (entry into blood or lymph vessels)
- **Circulation** through the body
- **Extravasation** (exit into distant tissues)
- **Proliferation and angiogenesis** at the new site

d. **Metabolism**

Cancer cells display altered energy metabolism compared to normal cells. Typically, healthy cells derive most of their energy through **oxidative phosphorylation**, whereas many cancer cells rely heavily on **glycolysis**, even in the presence of oxygen a phenomenon known as the **Warburg effect**.

Although glycolysis is common in most cancers, some cancers (e.g., **leukaemia**, **lymphoma**, and **endometrial cancer**) still depend largely on oxidative phosphorylation. In rare cases, cancer cells use **glutamine** as their primary energy source, especially for DNA and RNA synthesis due to its nitrogen content.
Cancer stem cells are often more reliant on **oxidative phosphorylation** or **glutamine metabolism**. Because of these differences, metabolic therapies like the **ketogenic diet** (low-carb) are sometimes proposed as supportive treatments to target cancer metabolism.

VII. DIAGNOSIS OF CANCER

Early and accurate diagnosis of cancer is crucial to prevent its progression and spread. Detecting specific cancer-related genes is key for early intervention and better outcomes.
The common diagnostic techniques used for identifying cancer include:

1. **Biopsy** – Removing tissue samples for examination
2. **Histopathological analysis** – Microscopic evaluation of cells and tissue structure
3. **Radiography** – Using X-rays for internal imaging
4. **Computed Tomography (CT)** – Detailed cross-sectional imaging of body tissues
5. **Magnetic Resonance Imaging (MRI)** – Soft tissue imaging using magnetic fields
6. **Molecular biology techniques** – Genetic and protein-based tests for cancer biomarkers

VIII. VENOM

Snake venom is a biologically active substance secreted by specialized glands and delivered through fangs to immobilize and digest prey. While snakebites remain a major global health threat with over 100,000 deaths and 400,000 disabilities each year, as per WHO the biochemical complexity of snake venom has also positioned it as a rich source of potential therapeutic agents.

In traditional medicine systems like those in China, snake venom has long been used (e.g., snake wine) for healing purposes. Modern pharmacology has also derived drugs from venom components. One notable example is **Captopril**, an antihypertensive drug developed from a bradykinin-potentiating peptide (BPP5a) found in Bothrops jararaca venom. Snake venoms offer a diverse arsenal of bioactive molecules with promising pharmaceutical potential, especially for cancer therapy.

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

IX. TYPES OF VENOM

Venom is produced by specialized glands and is introduced into other organisms via mechanisms like fangs (in snakes and spiders), stingers (in bees and ants), or harpoons (in marine animals like cone snails). It serves primarily as a defence mechanism or for hunting prey.

Biologically, venom can be classified by its effects:

- **Neurotoxic** – affects the nervous system
- **Cardiotoxic** – targets the heart
- **Haemolytic** – damages red blood cells
- **Digestive** – breaks down tissues
- **Haemorrhagic** – causes bleeding
- **Algo genic** – induces pain

X. BASIC COMPOSITION OF VENOM

Chemically, venom is a toxic cocktail of alkaloids, terpenes, polysaccharides, bio-genic amines (histamine), organic acids (formic acid), and amino acids, although the majority are peptides and proteins. Venom composition is highly variable between species, and the peptides composing the venom will determine the nature of the venom. Not only is the venom of every animal or insect different, but a subtle difference also exists between different species and between juveniles and adults, even among an organism of same species but of different geographical regions. Approximately 90%–95% of venom's dry weight is composed of mini-proteins called peptides. These peptides are directed against a wide variety of pharmacological targets, making them an invaluable source of ligands for studying the properties of these targets in different experimental paradigms. A number of these peptides have been used in vivo for proof-of-concept studies, with several having undergone preclinical or clinical development for the treatment of pain, diabetes, multiple sclerosis, cardiovascular diseases, and cancer.

A. Hyaluronidase

Hyaluronidase is present in snakes, scorpions, and bee venom. Hyaluronidase is considered an endoglycosidase because it degrades the b-N-acetyl-glycosaminidase linkages in hyaluronic acid (HA) polymers. The adhesive properties of HA hold cells together; there-fore, once they are hydrolysed, other venom components can penetrate the cells. As a result, hyaluronidase is known as a spreading factor. It damages the extracellular matrix at the site of a bite, leading to severe morbidity. Hyaluronidase also helps in rapid spreading of other toxins by destroying the integrity of the extracellular matrix of the tissue.

B. Phospholipase

A2Phospholipase A2 (PLA2) plays an important role in many biological events, including cell signalling and cell growth as well as generation of proinflammatory lipid mediators such as prostaglandin and leukotrienes. The PLA2 enzyme hydrolyses the sn-2 acyl ester bond of various phospholipids to produce free fatty acids and Lys phospholipids. Mammalian PLA2 plays important roles in various biological processes such as phospholipid metabolism, homeostasis of cellular membranes, host defence, and signal transduction. The Ca2+-dependent secretory PLA2 present in snake venom not only serves as a digestive enzyme, but also plays an important role in immobilizing the prey. PLA2 has other pharmacological properties such as antiplatelet, anticoagulant, haemolytic, neurotoxic, and myotoxic effects. PLA2 has been classified into two broad groups -

1. 1PLA2, which is found mainly in the venoms of cobras, kraits, and sea snakes.
2. 2PLA2, which is found in venoms of vipers and pit vipers.

C. Cholinesterase

Cholinesterase attacks the nervous system, relaxing the muscles to the point at which the victim has very little or no control. Cholinesterase plays a lead role in the cholinergic system, where it functions in the rapid termination of nerve impulse transmission. Its high reactivity toward organophosphorus compounds suggests that exogenous cholinesterase can serve as an effective therapeutic agent in the treatment of prophylaxis and organophosphorus poisoning.

D. L-Amino Acid Oxidase

L-amino acid oxidase (LAAO) is a dimeric flavoprotein that contains a noncovalently bound famine adenine dinucleotide (FAD) as a cofactor. It constitutes 1%–9% of the total venom protein and is responsible for the light yellowish colour of the venom. LAAO catalyses the stereospecific deamination of an l-amino acid substrate to an a-keto acid, along with the production of ammonia and hydrogen peroxide. LAAO from snake venom has been shown to induce apoptosis in mammalian endothelial cells, possibly due to the production of high concentrations of hydrogen peroxide.

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

E. Metalloproteinase

The metalloproteinase enzyme belongs to the family of zinc endopeptidases that degrade protein of the extracellular matrix and components of the haemostatic system. Metalloproteinase could disrupt micro vessels, which are then responsible for provoking local and systemic haemorrhagic and contribute to other pathways that lead to local tissue damage. Metalloproteinase might also provecy to toxic to end othelial cells.

F. Histamine

Histamine has been demonstrated to be involved in cell proliferation, embryonic development, and tumour growth. Histamine causes widespread peripheral vasodilation as well as increased permeability of the capillaries. Increased capillary permeability results in marked loss of plasma from the circulation. The signalling pathways triggered by histamine are mediated through its binding to the cognate histamine receptors (H1, H2, H3, and H4). In cancer cells, histamine receptors can be differentially expressed in different cancer types such as those of the brain, colon, and breast. Venoms of various species of snakes, scorpions, and honeybees have shown a detectable presence of histamine in various degrees. Honeybee venom showed the highest titters to histamine.

G. Melittin

Melittin is a small protein containing 26 amino acid residues. It is the principal toxic component of bee venom. Melittin is a potent hemolytic29 that causes mast cell degranulation and activates PLA2, increasing calpain activity and cell necrosis in hepatocellular carcinoma.30 The melittin enzyme exhibits antimicrobial activities and proinflammatory effects,31 in addition to inducing perturbations in the cell membrane and damage to enzyme system. Melittin inhibits cell growth through enhancement of death receptor expression in human ovarian cancer cells.

H. Piperidine

The major chemical components in the venom of red imported fire ants, Solenopsis invicta Buren, are 6-alkyl-2 methyl or alkenyl piperidines.33 Over 95% of Solenopsis invicta venom is composed of piperidine alkaloid components.34 These alkaloids function primarily as defensive compounds, but may also possess antibacterial, antifungal, insecticidal, and haemolytic properties. These piperidine alkaloids (i.e., alkylpyridines) have been classified as Solenopsis, Solenopsis, dehydrosolenopsis, depending on the relative configuration of their substituents, length, and unsaturation of the alkyl chain. Tetra Ponera are tricyclicalkaloids from the pseudo-Myrmicine ants ofthegenusTetra Ponera,which has shown cytotoxic activities. The effect of the size of the alkyl chainonthecy to toxicity is comparable to that observedfor the Solenopsis, a group of 6-alkyl-2 meth-yl-piperidines that are the main constituentsof the venom of fire ants.The non-naturallong alkyl derivative is cytotoxic against HT29cells,whereastheshort-chainderivativeisinactive.

Pharmacological action of snake venom

Numerous toxins found in snake venom have been extensively studied and formulated into drugs to treat serious conditions such as cancer, high blood pressure, and blood clotting disorders. For example, venoms from rattlesnakes and other pit vipers can significantly affect blood vessels, alter blood cell function, and interfere with clotting mechanisms.

Venom can also impact the nervous and respiratory systems. The intensity and nature of the effects depend on several factors, including the species of the snake, the amount and type of venom injected, and the site of envenomation. Additional variables like the victim's age, general health, and body size also influence the outcome.

In both humans and animals, snake venom typically lowers blood pressure, often leading to **hypotension** and shock. In severe cases, death may occur within a few hours to a couple of days, with most fatalities reported between 18 to 32 hours after envenomation.

Mechanism of venom

Snake venom contains enzymes and other molecules that disrupt multiple physiological systems. These toxins can:

- Affect the vascular system by altering blood pressure and causing haemorrhages
- Interfere with nervous signals, leading to paralysis or muscle dysfunction
- Disrupt cellular membranes, enzyme systems, and the immune response
- Trigger inflammatory and cytotoxic pathways that damage tissues and organs

The venom components act in a highly coordinated manner, allowing the snake to immobilize its prey quickly and begin digestion even before ingestion. This powerful mechanism

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

is now being repurposed in controlled ways for targeted therapies, particularly in cancer treatment.

XI. VENOM AS POTENTIAL ANTI CANCER DRUG

Animal venoms have long been explored as therapeutic tools, with numerous compounds isolated from venomous species showing anti-cancer properties. These include **proteins, enzymes, and peptides** derived from snakes, scorpions, bees, ants, and spiders. Research has shown that these venom components can:

- Induce apoptosis (programmed cell death) in cancer cells
- Disrupt tumour signalling pathways
- Inhibit angiogenesis (formation of new blood vessels) that feed tumours
- Block cancer cell adhesion and migration

The biodiversity found in venoms offers a unique set of molecular templates for developing novel cancer therapies. Natural venom-derived compounds often show greater selectivity and lower side effects than synthetic drugs. As a result, several of these substances are in various stages of **preclinical and clinical development** for cancer treatment.

Table No. 1 Promising anticancer compound isolated from different species of animals

Species	Compound	Target	Application
Agkistrodon acutus	Contortrostatin	Target integrins	Inhibit tumour growth and angiogenesis
Indian Russell viper	DrCT-1	Apoptosis through G1 phase arrest of the cell cycle	Antiproliferative, cytotoxic
Naja Naja atra	Cardiotoxin-3[44]	Inhibits the cellular proliferation and induces apoptosis of various cancer cells	Breast Cancer, leukaemia cells, colorectal cancer
Apis mellifera	Melittin[47]	Inhibit calmodulin activity; downregulates Erk and the Akt signalling pathway	Inhibit cell growth and clonogenicity of human and murine leukemic cells
Acanth Scurria gemesiana	Gomesin[46]	Cytotoxic and antitumor activities	Breast cancer and colon carcinoma

Snake Venom

The therapeutic value of snake venom was first recognized by Claude Bernard, often called the father of physiology. Several enzymes found in snake venom have demonstrated **anticancer activity**. For example, venom from Vipera lebentina turnica has shown to trigger cell death in ovarian cancer cells by suppressing key pathways like **NF-κB** and **STAT3**, and promoting pro-apoptotic proteins such as **Bax** and **Caspase-3**, while reducing the anti-apoptotic **Bcl-2** protein. Cobra venom, particularly due to its **phospholipase activity**, has shown potential in targeting cancer cells. Studies involving venom from snakes like Bothrops netwide and Naja Naja revealed strong cytotoxic effects against various cancer models including **melanoma**, **sarcoma**, and **leukaemia**. Furthermore, a component called **Contortrostatin** (from the southern copperhead snake) has been shown to inhibit breast cancer cell migration and lung colonization, suggesting its strong anti-metastatic potential.

Scorpion Venom

Scorpion venom is a highly complex mixture, rich in biologically active molecules. It includes **neurotoxins**, **protease inhibitors**, **mucopolysaccharides**, and **histamine-related peptides**, which affect ion channels like sodium, potassium, and chloride in cells.

Some of these components have demonstrated promising anticancer effects in both lab and animal studies. One of the most researched molecules is **Chlorotoxin**, extracted from the Lei

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

urus quinquestriatus species. This peptide has shown specific binding to **glioma cells** a type of brain tumour while sparing normal brain cells. Chlorotoxin works by inhibiting **MMP-2**, an enzyme that facilitates tumour invasion, thus limiting the spread of cancerous cells.

Applications of Scorpion Venom-
Potential therapeutic application

- Antibacterial
- Antifungal
- Analgesic
- Anticancer
- Antiviral

Bee Venom

Bee venom contains a potent combination of proteins, the most notable being **melittin** and **phospholipase A2 (PLA2)**. Melittin makes up nearly half of the dry weight of bee venom and is known for its **antimicrobial, pro-inflammatory, and cytotoxic properties**.

In cancer research, melittin has been found to disrupt cancer cell membranes, induce necrosis, and activate immune responses through cytokine stimulation. It affects various types of cancer including **leukaemia**, **lung**, **liver**, **prostate**, **bladder**, and **breast** cancers. Another component, **Apamin**, acts as a neurotoxin by interfering with potassium channels and modulating nervous system responses. Collectively, bee venom also exhibits **anti-thrombotic** properties that contribute to its therapeutic applications.

Ant Venom

Ants, particularly species like Solenopsis invicta (red imported fire ants), produce venom rich in alkaloids such as **Solenopsis A**, which displays strong **anti-angiogenic** activity. This compound can block pathways involved in the formation of new blood vessels a crucial process for tumour growth and survival.

Another promising group of molecules from ant venom is **Tetraponerines**, tricyclic alkaloids derived from Tetra Ponera ants. These have shown cytotoxic effects against cancer cell lines like **HT29**. Solenopsis also interferes with the **PI3K/Akt** signalling pathway, which is often overactive in cancer, making it a valuable target for treatment. Interestingly, its structure is like known anticancer drugs like **Miltefosine** and **Peritonise**, supporting its potential for drug development.

Spider Venom

Spider venom is a highly diverse chemical mixture composed of low molecular weight compounds, **acyl polyamines**, **linear peptides**, and **cysteine rich proteins**. These compounds primarily affect neural and muscular functions in prey but are now being explored for their **antitumor effects**.

One notable example is **Gomes in**, a peptide extracted from the spider Acanth Scurria gomesiana. Gomes in has shown potent anticancer activity by inducing membrane disruption and energy imbalance in cancer cells. Another peptide, **Psalm toxin 1 (PcTX1)**, isolated from Psalmopoeus cambridgei, selectively blocks **acid-sensing ion channels (ASICs)**, which are highly expressed in aggressive brain cancers like glioblastoma. These findings suggest that spider venoms hold immense potential as **targeted therapies for difficult-to-treat tumours**.One notable example is **Gomes in**, a peptide extracted from the spider Acanth Scurria gomesiana. Gomes in has shown potent anticancer activity by inducing membrane disruption and energy imbalance in cancer cells. Another peptide, **Psalm toxin 1 (PcTX1)**, isolated from Psalmopoeus cambridgei, selectively blocks **acid-sensing ion channels (ASICs)**, which are highly expressed in aggressive brain cancers like glioblastoma. These findings suggest that spider venoms hold immense potential as **targeted therapies for difficult-to-treat tumours**.

Table No. 2 List of Synthesized analogs from Venom

Name	Peptide	Species	Target protein	Disease	Clinical Stage	Company
TM-601	Chlorotoxin	Leiurus quinquertiums	Cl channel	Brain tumor	Phase II	Trans molecular Inc.
TM-701	Chlorotoxin	Leiurus quinquertiums	Cl channel	Chronic monotherapy & pharmaceutical sensitizer	Preclinical	Trans molecular Inc.

International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 3 May–June 2025, pp: 1171-1179 www.ijprajournal.com ISSN: 2456-4494

				coadninitered drug cocktails for cancer		
Alfimeprase	fibrolase	Agkistrodon contortrix	Fibrin	Metastatic ovarian cancer	Phase II	Novello Inc.

XII. CONCLUSIONS

This review highlights the immense therapeutic potential of various venom components in cancer treatment. Several venom-derived molecules are currently undergoing clinical and preclinical trials, indicating their promise as natural anticancer agents. However, concerns remain regarding their possible toxicity to normal cells, making it essential to evaluate their selectivity and safety in detail.

One of the most innovative approaches involves coupling venom peptides with **nanoparticles** for targeted drug delivery, minimizing damage to healthy tissues while maximizing anticancer efficacy. Interdisciplinary research combining **biotechnology, pharmacology, and nanoscience** could pave the way for the development of next-generation venom-based drugs.

Nature continues to be an invaluable source of novel bioactive compounds. In fact, nearly 74% of modern anticancer drugs are derived from or inspired by natural sources. Compared to synthetic compounds, natural products offer higher chemical diversity and more selective biological activity. Thus, the discovery of venom-based agents adds a significant dimension to the ongoing search for more effective, safer cancer treatments. In conclusion, early diagnosis, and access to effective treatment especially for cancers like breast, cervical, and oral cancers must remain a healthcare priority. With continued research and innovation, venom-based therapies have the potential to revolutionize the field of oncology.

REFERENCES

[1]. Baskar R, Lee KA, Yeo R, Yeoh KW. Cancer and radiation therapy: current advances and future directions. Int J Med Sci. 2012;9:193–9.

[2]. Jain D, Kumar S. Snake venom: a potent anticancer agent. Asian PacJ Cancer Prev. 2012;13:4855–60.

[3]. Lai D, Visser-Grieve S, Yang X. Tumour suppressor genes in chemotherapeutic drug response. Biosci Rep. 2012;32:361–74.

[4]. Barnett GC, West CM, Dunning AM, Elliott RM, Coles CE, Pharoah PD, Burnet NG. Normal tissue reactions to radiotherapy: towards tailoring treatment dose by genotype. Nat Rev Cancer. 2009;9:134–42.

[5]. Kruger C, Greten TF, Korangy F. Immune based therapies in cancer. Histol Histopathol. 2007;22:687–96.

[6]. Geissler M, Weth R. Immunotherapy: new insights. Praxis. 2002;91:2236–46.

[7]. Orentas RJ, Lee DW, Mackall C. Immunotherapy targets in paediatric cancer. Front Oncol. 2012;2:3.

[8]. Hammerstrom AE, Cauley DH, Atkinson BJ, Sharma P. Cancer immunotherapy: sipuleucel- T and beyond. Pharmacotherapy. 2011; 31:813–28.

[9]. Armugam A, Cher CD, Lim K, Koh DC, Howells DW, Jeyaseelan K. A secretory phospholipase A2-mediated neuroprotection and anti-apoptosis. BMC Neurosci. 2009; 10:120.

[10]. Habermann E, Reiz KG. On the biochemistry of bee venom peptides, melittin and apamin. Biochem Z. 1965;343:192–203.

[11]. Arimura T, Niwa K, Mitani N, HagiwaraI, Kawaida T, Shimazu H. A resected case of triple cancer in the uterus, lung and thyroid. Nihon Kyobu Geka Gakkai. 1989;37:1233–7.

[12]. Muller GJ. Scorpionism in South Africa. A report of 42 serious scorpion envenomation. S Afr Med J. 1993;83: 405–11.

[13]. Ernst DS, Eisenhauer E, Wainman N, Davis M, Lohmann R, Baetz T, Belanger K, Smylie M. Phase II study of peritonise in previously untreated patients with metastatic melanoma. Invest New Drugs. 2005; 23:569–75.

[14]. Kuhn-Nentwig L, Schaller J, Nentwig W. Biochemistry, toxicology, and ecology of the venom of the spider Cupiennius salei (Ctenidia). Toxicon. 2004; 43:543–53.

[15]. Swenson S, Ramu S, Markland FS. Ant angiogenesis and RGD-containing snake

venom disintegrants. Curr Pharm Des.2007;13:2860–71.
[16]. Cohen O, Kronman C, Chitlaru T, Ordentlich A, Velan B, Shafferman A. Effect of chemical modification of recombinant human acetylcholinesterase by polyethylene glycol on its circulatory longevity. Biochem J. 2001;357:795–802.
[17]. Pawelek PD, Cheah J, Coulombe R, Mache roux P, Ghisla S, Vrielink A. The structure of L-amino acid oxidase reveals the substrate trajectory into an enantiomerically
[18]. conserved active site. EMBO J. 2000;19:4204–15.
[19]. Wei JF, Wei XL, Mo YZ, He SH. Induction of mast cell accumulation, histamine release and skin edema by N49 phospholipase A2. BMC Immunol. 2009;10:21.
[20]. Arora AS, de Groen PC, Croall DE, Emori Y, Gores GJ. Hepatocellular carcinoma cells resist necrosis during anoxia by preventing phospholipase-mediated calpain activation. J Cell Physiol. 1996;167, 434–42
[21]. Sumikura H, Andersen OK, Drewes AM, Arendt-Nielsen L. A comparison of hyperalgesia and neurogenic inflammation induced by melittin and capsaicin in humans. Neurosci Lett. 2003;337, 147–50.
[22]. Fitzgerald KT, Flood AA. Hymenoptera stings. Clin Tech Small Anim Pract. 2006;21, 194–204.