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(REVIEW ARTICLE)

Unveiling the anticancer power of vinca rosea leaves: A pharmacognostical and phytochemical approach

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Abstract

Vinca rosea (Catharanthus roseus), a medicinal plant of significant pharmacological importance, has garnered attention for its potent anticancer properties. This study presents a comprehensive pharmacognostical and phytochemical evaluation of *V. rosea* leaves, emphasizing their role in cancer therapy. Pharmacognostical parameters including macroscopic, microscopic, and physicochemical characteristics were documented to ensure standardization and quality control. Phytochemical screening revealed the presence of major secondary metabolites such as alkaloids, flavonoids, tannins, and terpenoids. Notably, vinca alkaloids such as vincristine and vinblastine, known for disrupting microtubule dynamics and arresting mitosis, were identified. These compounds are clinically used in the treatment of Hodgkin's disease, leukemia, and other malignancies. The findings affirm the therapeutic potential of *V. rosea* leaves and support their further exploration as a reliable source of plant-derived chemotherapeutics. This study highlights the relevance of integrating pharmacognostic tools in identifying and validating phytoconstituents with anticancer efficacy.

Keywords: Vinca Rosea; Anticancer Activity; Vinca Alkaloids; Pharmacognosy; Phytochemical Screening

1. Introduction

1.1. Importance of Medicinal Plants in Cancer Therapy

Cancer is a leading global health concern, ranking as the second most common cause of death after cardiovascular diseases [1]. It originates from the abnormal transformation of normal cells due to genetic mutations in DNA. These mutated cells multiply uncontrollably, bypassing regulatory growth signals, invading surrounding tissues, and ultimately forming tumors [2].

Both developed and developing nations face a significant cancer burden. Globally, approximately 182 out of every 100,000 people are diagnosed with cancer annually, and 102 die from the disease [3]. According to the World Health Organization, around 14 million people are diagnosed with cancer each year, and 8 million die from it. In Iran, the prevalence is estimated at 134 cases per 100,000 individuals, with about 85,000 new cases and 55,000 cancer-related deaths reported annually [4]. Alarmingly, cancer-related mortality is projected to rise, with over 13.1 million deaths expected worldwide by 2030 [5].

Current cancer treatments, such as chemotherapy, pose challenges due to their lack of selectivity they often damage healthy cells along with cancerous ones [6]. One of the key issues in cancer therapy is eliminating tumor cells without harming normal tissues. This has led to increasing interest in discovering anticancer agents from natural sources like plants, which can offer more targeted and less toxic treatment options [7].

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Natural products are especially appealing because of their diverse bioactive compounds and relatively fewer side effects. Medicinal plants, in particular, are valuable in cancer research due to their rich chemical profiles and potential to yield new therapeutic agents [8]. These plants produce secondary metabolites—such as alkaloids, flavonoids, terpenoids, tannins, and pigments which, although not essential for plant growth, exhibit various biological activities. These include anti-inflammatory, anticancer, contraceptive, and effects on blood cells, lipid metabolism, and cardiovascular function [9].

Advancements in cancer treatment have increasingly involved natural secondary metabolites. These compounds may exert anticancer effects by inhibiting cancer-promoting enzymes, repairing DNA damage, stimulating antitumor enzyme production, boosting immune responses, or acting as antioxidants [10].

Given the severity and complexity of cancer, especially at metastatic stages where resistance to treatment is common, the need for effective therapies is urgent [11]. While conventional methods like surgery and chemotherapy remain essential, they are often associated with significant drawbacks, such as the destruction of healthy cells. Currently, over 60% of effective anticancer drugs are derived from natural sources, including plants, marine organisms, and microbes [12, 13, 14].

Numerous studies have demonstrated the efficacy of plant-based compounds in treating not only cancer but also conditions like diabetes, infertility, thyroid disorders, anemia, and psychological illnesses. As a result, identifying plant-based alternatives that can replace or enhance conventional cancer therapies remains a vital area of research [15, 16, 17].

1.2. Plants used for cancer Therapy

Many medicinal plants are known for their anticancer properties, including their active constituents and mechanisms of action. Figure 1 & Table 1 provides list of medicinal plants used in cancer therapy and their active constituents.

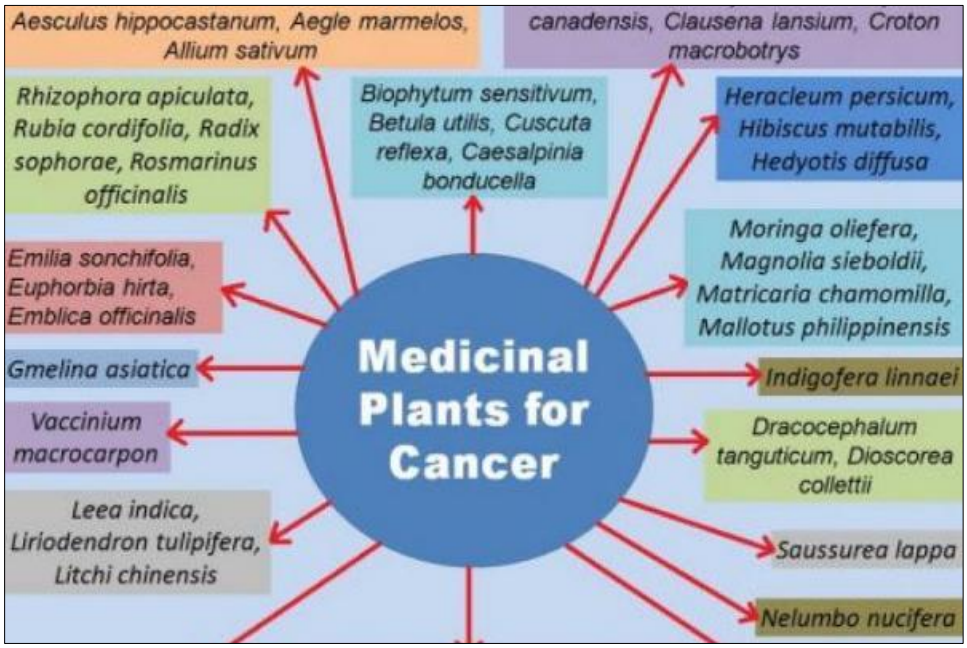

Figure 1 list of medicinal plants used in cancer therapy

Table 1 list of medicinal plants used in cancer therapy and their active constituents

Plant Name	Active Constituents	Mechanism of Action	References
Catharanthus roseus	Vincristine, Vinblastine	Inhibition of microtubule dynamics leading to cell cycle arrest and apoptosis	[18]
Curcuma longa	Curcumin	Downregulation of NF-κB, AP-1, COX-2, and EGR-1; inhibition of tumor cell invasion	[19]
Withania somnifera	Withaferin A	Induction of apoptosis; inhibition of metastasis; radiosensitization	[20]
Taraxacum mongolicum	Not specified	Induction of apoptosis via ER stress pathway; modulation of p53 expression	[21]
Zingiber officinale	6-Shogaol, 10-Gingerol	Inhibition of cancer cell proliferation and metastasis; induction of apoptosis	[22]
Moringa oleifera	Moringin, Moringa oleifera extract	Induction of apoptosis; enhancement of p53 expression; cell cycle arrest	[23]
Psidium guajava	Flavonoids, Tannins	Inhibition of AKT/mTOR signaling pathway; induction of apoptosis	[24]
Mangifera indica	Mangiferin, Polyphenols	Modulation of PI3K/AKT, AMPK, and NF-κB pathways; inhibition of cancer cell survival	[25]
Lagerstroemia speciosa	Corosolic acid	Induction of apoptosis; cell cycle arrest in liver cancer cells	[26]

1.3. Traditional and Medicinal Uses of Vinca rosea

There are numerous naturally occurring plants around us that possess medicinal value. Among them, Catharanthus roseus commonly known as vinca rosea Figure 2 is widely distributed in tropical regions. Catharanthus roseus L. is a perennial herb, native to Madagascar, and commonly found in Southern Asia and other tropical areas [26,27]. It is known by various names, including Madagascar periwinkle, bright eyes, Cape periwinkle, graveyard plant, old maid, pink periwinkle, and rose periwinkle myrtle. In Malaysia, it is locally referred to as "*Kemunting Cina*." Besides its ornamental use thanks to its attractive pink, purple, and white flowers this plant holds considerable medicinal value.

Among the earliest plant-derived compounds used in cancer therapy are the vinca alkaloids [28]. The milky sap from the stems of Catharanthus roseus contains over 70 different indole alkaloids. Of these, two major anti-neoplastic compounds vinblastine and vincristine are widely known [29]. Vincristine is primarily used in the chemotherapy regimen for Hodgkin's lymphoma, while vinblastine is employed in the treatment of childhood leukemia. These alkaloids disrupt cell division by halting the mitotic process at the metaphase stage. However, they are associated with side effects including peripheral neuropathy, hair loss, hyponatremia, and constipation [30].

In addition to cancer treatment, Catharanthus roseus is traditionally used for managing conditions such as hypertension, diabetes, blood cancers, malaria, non-small-cell lung cancer, and memory enhancement. The plant also exhibits antimicrobial, antioxidant, anti-diarrheal, hypolipidemic, and wound-healing properties.

Historically, the genus Catharanthus was established by Carl Linnaeus, derived from the Greek words katharos (pure) and anthos (flower). The Scottish botanist George Don identified the species as Catharanthus roseus, which sparked debate over its botanical classification. Initially, in 1759, Linnaeus had named it Vinca rosea [31]. In 1828, German botanist Heinrich Gottlieb Ludwig Reichenbach proposed the genus Lochnera, and it was later renamed Lochnera rosea by Austrian botanist Stephan Ladislaus Endlicher in 1838. However, British botanist William Stearn later confirmed Catharanthus roseus as the correct and accepted name. He also pointed out that Lochnera was invalid because it was too similar to Lochneria, a name already published in 1777 by the naturalist Giovanni Antonio Scopoli [32].

Figure 2 *Vinca rosea* flower

2. Pharmacological Activities of Vinca rosea

2.1. Anti-Neoplastic Activity

The leaves and stems of Vinca rosea are rich in alkaloids with potent anti-cancer and anti-tumor properties. Compounds like vinblastine and vincristine disrupt tumor growth by targeting cell division. Vinblastine is effective against conditions like Hodgkin's disease and choriocarcinoma, while vincristine is used to treat pediatric leukemia. Marketed under names like Velban and Oncovin, these alkaloids hinder mitosis by interfering with microtubule formation during metaphase. Semi-synthetic derivatives, including vinorelbine and vinflunine, enhance therapeutic outcomes by targeting tubulin in cancer cells [33].

2.2. Anti-Diabetic Activity

Ethanolic extracts from the flowers and leaves of Vinca rosea exhibit blood sugar-lowering effects comparable to glibenclamide. These effects are attributed to enhanced hepatic glucose utilization. A 1:1 dichloromethane:methanol extract administered orally (500 mg/kg) in streptozotocin-induced diabetic rats over 7–15 days demonstrated a hypoglycemic effect of 48.6–57.6%. Extended treatment up to 30 days offered complete protection from STZ-induced diabetes. Enzymes like glycogen synthase and dehydrogenases showed improvement, indicating better glucose metabolism and reduced lipid peroxidation [34].

2.3. Anti-Microbial Activity

Vinca rosea contains natural chemotherapeutic agents that offer broad-spectrum antimicrobial activity. These plant compounds are valuable in developing new antibiotics, especially against resistant bacterial strains [35].

2.4. Anti-Oxidant Properties

Roots of pink and white varieties contain ethanolic extracts with antioxidant potential, as evidenced by assays such as hydroxyl, superoxide, DPPH, and nitric oxide radical scavenging activities [36].

2.5. Anti-Helminthic Activity

The plant displays significant anti-parasitic activity against helminths affecting humans and animals. Ethanol extracts at 250 mg/mL demonstrated efficacy in models using Pheretima posthuma, with piperazine citrate serving as the reference standard [37].

2.6. Wound Healing Potential

Daily oral administration of 100 mg/kg ethanolic extract promoted wound contraction and reduced epithelialization time in rats. This was marked by increased tensile strength, dry weight, and hydroxyproline content, indicating accelerated healing [38].

2.7. Hypolipidemic Activity

Leaf juice of Vinca rosea helped reduce serum cholesterol, triglycerides, LDL, and VLDL levels, contributing to anti-atherosclerotic effects. These benefits are likely due to flavonoids and vinpocetine-like compounds with strong antioxidant effects [39].

2.8. Anti-Diarrheal Activity

In Wistar rats, ethanolic extracts of Vinca leaves administered at 200 and 500 mg/kg showed dose-dependent inhibition of castor oil-induced diarrhea and slowed gastrointestinal transit (charcoal meal test), supporting its traditional use for managing diarrhea [40].

2.9. Anti-Ulcer Activity

Alkaloids like vincamine and vindoline possess anti-ulcer effects. While vincamine also offers neuroprotective and cerebrovasodilatory properties, high doses may cause gastric lesions in animal studies, indicating the need for dose optimization in therapeutic use [41].

3. Mechanism of Action

The cytotoxic activity of vinca alkaloids primarily arises from their ability to bind to tubulin and disrupt the normal function of microtubules, particularly those forming the mitotic spindle, resulting in cell cycle arrest at the metaphase stage [42]. Apart from their interference with microtubules, these compounds also exhibit various other biochemical effects, though many of these occur only at concentrations that are not clinically relevant. Nevertheless, vinca alkaloids, like other antimicrotubule agents, impact both cancerous and normal cells during non-mitotic phases due to the involvement of microtubules in a range of cellular processes [43].

Vinca alkaloids bind to tubulin at sites distinct from those targeted by taxanes, colchicine, podophyllotoxin, and guanosine-5′-triphosphate (GTP) [44]. This binding is characterized by rapid kinetics and reversibility. Evidence supports the presence of two specific vinca-binding sites per tubulin dimer [45]. Approximately 16–17 high-affinity binding sites are believed to be present at the ends of each microtubule. Binding at these regions interferes with microtubule assembly. Interestingly, at lower drug concentrations, vinca alkaloids do not significantly reduce microtubule mass but instead suppress the dynamics of microtubule growth and shrinkage by forming a “kinetic cap” at the growing end, leading to functional suppression [46].

At such low doses, vinca alkaloids primarily inhibit microtubule dynamics at the ends of mitotic spindles, causing metaphase arrest before any noticeable loss in microtubule polymer mass occurs [47]. Additionally, vinca alkaloids demonstrate anti-angiogenic properties by suppressing endothelial cell proliferation, migration, and adhesion to extracellular matrix components like fibronectin at picomolar concentrations (0.1–1.0 pmol/L) [48]. Notably, these effects are selective, sparing fibroblasts and certain lymphoid tumor cells.

When combined with antibodies targeting vascular endothelial growth factor (VEGF), low-dose vinblastine (VBL) significantly enhances anti-tumor efficacy—even in tumors unresponsive to direct cytotoxic action of the drug [49]. Ultimately, vinca alkaloids induce mitotic arrest and apoptosis by binding to tubulin, inhibiting its polymerization, and destabilizing microtubules—mechanisms central to the actions of vincristine (VCR) and related compounds [50].

3.1. Toxicity

Despite their structural similarities, vinca alkaloids exhibit markedly different toxicity profiles. One common adverse effect across this class is peripheral neurotoxicity, with vincristine (VCR) presenting the highest neurotoxic potential [51]. This neurotoxicity typically manifests as a symmetric sensory-motor and autonomic polyneuropathy, mainly due to axonal damage and impaired axonal transport likely triggered by disruption of microtubule function [52]. Although central nervous system effects are rare due to limited brain penetration, instances of confusion, mood changes, hallucinations, agitation, insomnia, seizures, coma, and inappropriate antidiuretic hormone secretion have been noted. Rarely, cases of laryngeal paralysis have been reported. The most effective intervention remains the reduction or discontinuation of the drug, as no antidotes (including thiamine, folinic acid, pyridoxine, or vitamin B12) have shown definitive efficacy [53].

All vinca alkaloids share similar neurotoxic symptoms, but VCR exhibits the most severe cases, whereas vinblastine (VBL) and vinorelbine (VRL) show milder toxicity. In contrast, the major dose-limiting side effect of VBL, vinorelbine, and vindesine (VDS) is neutropenia, while thrombocytopenia and anemia are less frequently encountered. VCR is

seldom associated with hematologic toxicity; however, severe myelosuppression has been observed in cases of excessive drug exposure or liver impairment [54].

Gastrointestinal issues are also a concern, often resulting from autonomic nervous system dysfunction. Common symptoms include abdominal discomfort, bloating, constipation, and paralytic ileus—particularly in VCR-treated patients or when high doses of other vinca alkaloids are used [55]. VBL is more likely than VRL to cause mucositis, although VCR is also implicated. Other GI symptoms include nausea, vomiting, and diarrhea. Vinca alkaloids are vesicants and may cause substantial tissue injury upon extravasation [56]. Additional complications such as acute cardiac ischemia, chest pain, fever of unknown origin, pulmonary reactions, hepatic toxicity, Raynaud's phenomenon, and hand-foot syndrome have also been documented [57].

These medications should be avoided during pregnancy or breastfeeding due to potential teratogenic effects. Patients undergoing treatment are advised against receiving live vaccines. VCR, in particular, can suppress immune function, increasing vulnerability to infections [58]. It is crucial that healthcare providers are informed of concurrent medications and any underlying conditions such as viral infections (chickenpox, herpes zoster), kidney or liver diseases, muscle disorders, or gout [59]. Ultimately, toxicity is determined by both drug concentration and treatment duration, with evidence suggesting that surpassing a critical concentration threshold plays a pivotal role in adverse effects [60].

4. Conclusion

Vinca alkaloids are integral to many combination chemotherapy protocols due to their unique mechanism of action, which differs from DNA-alkylating agents and does not exhibit cross-resistance. Besides their prominent role in cancer therapy, they also display potential in treating conditions like diabetes, hypertension, and microbial infections. Their primary antitumor mechanism involves the inhibition of cell division, leading to apoptosis.

The four major vinca alkaloids used clinically are vinblastine (VBL), vinorelbine (VRL), vincristine (VCR), and vindesine (VDS). Among them, VCR, VBL, and VRL are FDA-approved in the United States. Vinflunine, a newer synthetic derivative, is approved in Europe for second-line treatment of transitional cell carcinoma of the urothelium (TCCU) and is under investigation for other cancer types [62]. As a class, vinca alkaloids remain among the most extensively used anticancer agents globally. Ongoing research continues to explore new therapeutic applications and improved formulations of these compounds.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] World Health Organization. Preventing Chronic Diseases: A Vital Investment. Geneva, Switzerland: World Health Organization; 2005.

[2] Smeltzer SC, Bare BG, Hinkle JL, Cheever KH. Brunner and Suddarth's Textbook of Medical Surgical Nursing. 12th ed. London, England: Wolters Kluwer; 2010:205–231.

[3] Kumar V, Abbas A, Aster J. Robbins Pathologic Basis of Disease. 9th ed. Tehran, Iran: Arjomand; 2014.

[4] Mousavi SM, Gouya MM, Ramazani R, Davanlou M, Hajsadeghi N, Seddighi Z. Cancer incidence and mortality in Iran. Ann Oncol. 2009;20:556–563.

[5] Rafieian-Kopaie M, Nasri H. On the occasion of World Cancer Day 2015: the possibility of cancer prevention or treatment with antioxidants: the Ongoing Cancer Prevention Researches. Int J Prev Med. 2015;6:108. doi:10.4103/2008-7802.169077.

[6] Lachenmayer A, Alsinet C, Chang CY, Liovit JM. Molecular approaches to treatment of hepatocellular carcinoma. Dig Liver Dis. 2010;42:264–272.

[7] Newman DJ, Cragg GM. Natural products as sources of new drugs over the last 25 years. J Nat Prod. 2007;70:461–477.

[8] Mansouri E, Kooti W, Bazvand M, et al. The effect of hydro-alcoholic extract of Foeniculum vulgare Mill on leukocytes and hematological tests in male rats. Jundishapur J Nat Pharm Prod. 2015;10:e18396

[9] Kooti W, Ghasemiboroon M, Asadi-Samani M, et al. The effects of hydro-alcoholic extract of celery on lipid profile of rats fed a high fat diet. Adv Environ Biol. 2014;8:325–330.

[10] Kooti W, Hasanzadeh-Noohi Z, Sharafi-Ahvazi N, Asadi-Samani M, Ashtary-Larky D. Phytochemistry, pharmacology, and therapeutic uses of black seed (Nigella sativa). Chin J Nat Med. 2016;14:732–745.

[11] Sakarkar DM, Deshmukh VN. Ethnopharmacological review of traditional medicinal plants for anticancer activity. Int J Pharm Tech Res. 2011;3:298–308.

[12] Valastyan S, Weinberg RA. Tumor metastasis: molecular insights and evolving paradigms. Cell. 2011;147:275–292.

[13] sadi-Samani M, Kooti W, Aslani E, Shirzad H. A systematic review of Iran's medicinal plants with anticancer effects. J Evid Based Complementary Altern Med. 2016;21:143–153.

[14] Kooti W, Farokhipour M, Asadzadeh Z, Ashtary-Larky D, Asadi-Samani M. The role of medicinal plants in the treatment of diabetes: a systematic review. Electron Physician. 2016;8:1832–1842

[15] Kooti W, Ghasemiboroon M, Ahangarpoor A, et al. The effect of hydro-alcoholic extract of celery on male rats in fertility control and sex ratio of rat offspring. J Babol Univ Med Sci. 2014;16(4):43–49.

[16] Kooti M, Ghasemiboroon M, Asadi-Samani M, et al. The effect of alcoholic extract of celery leaves on the delivery rate (fertilization and stillbirths), the number, weight and sex ratio of rat off spring. AENSI. 2014;8:824–830.

[17] Kooti W, Mansouri E, Ghasemiboroon M, Harizi M, Ashtary-Larky D, Afrisham R. The effects of hydroalcoholic extract of Apium graveolens leaf on the number of sexual cells and testicular structure in rat. Jundishapur J Nat Pharm Prod. 2014;9:e17532.

[18] Qu, Y., Safonova, O., & De Luca, V. (2019). Completion of the canonical pathway for assembly of anticancer drugs vincristine/vinblastine in Catharanthus roseus. The Plant Journal, 97(2), 257-266.

[19] Giordano, A., & Tommonaro, G. (2019). Curcumin and cancer. Nutrients, 11(10), 2376.

[20] Dutta, R., Khalil, R., Green, R., Mohapatra, S. S., & Mohapatra, S. (2019). Withania somnifera (Ashwagandha) and withaferin A: Potential in integrative oncology. International journal of molecular sciences, 20(21), 5310.

[21] Li, X. H., He, X. R., Zhou, Y. Y., Zhao, H. Y., Zheng, W. X., Jiang, S. T., ... & Han, S. Y. (2017). Taraxacum mongolicum extract induced endoplasmic reticulum stress associated-apoptosis in triple-negative breast cancer cells. Journal of Ethnopharmacology, 206, 55-64.

[22] Zick, S. M., Djuric, Z., Ruffin, M. T., Litzinger, A. J., Normolle, D. P., Alrawi, S., ... & Brenner, D. E. (2008). Pharmacokinetics of 6-gingerol, 8-gingerol, 10-gingerol, and 6-shogaol and conjugate metabolites in healthy human subjects. Cancer Epidemiology Biomarkers & Prevention, 17(8), 1930-1936.

[23] Charoensin, S. (2014). Antioxidant and anticancer activities of Moringa oleifera leaves. J. Med. Plants Res, 8(7), 318-325.

[24] Mailoa, M. N., Mahendradatta, M., Laga, A., & Djide, N. (2014). Antimicrobial activities of tannins extract from guava leaves (Psidium guajava L.) on pathogens microbial. International journal of scientific & technology research, 3(1), 236-241.

[25] Pardo-Andreu, G. L., Paim, B. A., Castilho, R. F., Velho, J. A., Delgado, R., Vercesi, A. E., & Oliveira, H. C. (2008). Mangifera indica L. extract (Vimang®) and its main polyphenol mangiferin prevent mitochondrial oxidative stress in atherosclerosis-prone hypercholesterolemic mouse. Pharmacological Research, 57(5), 332-338.

[26] Stohs, S. J., Miller, H., & Kaats, G. R. (2012). A review of the efficacy and safety of banaba (Lagerstroemia speciosa L.) and corosolic acid. Phytotherapy Research, 26(3), 317-324.

[27] Sharma Sk. "Medicinal Plants used in Ayurveda". New Delhi:Rashtriya Ayurveda Vidyapeeth, Ministry of Health and Family Welfare, Govt of India (1998): 193.

[28] The Wealth of India-Raw Materials New Delhi: Publication and Information Directorate, Council of Scientific and Industrial Research 3 (1985): 391-395

[29] Brogan C. Alkaloids cancer treatments. 2010. Jun 7, [Cited on 2010 Sep 23]. Available from:http://www.Vinca alkaloids\AlkaloidsCancer Treatment Livestrong_com.mh.

[30] Catharantus Roseus–periwinkle. Tropiclab Inc.www.tropilab.com/periwinkle.html(accessed 20 July 2008).

[31] Johnson IS, Armstrong JG, Gorman M, Burnett JP. The vinca alkaloids: a new class of oncolytic agents.Cancer Res.1963;23:1390–427

[32] Asma N., et al. "An updated Review on Catharanthus Roseus:Phytochemical and Pharmacological Analysis". Indian Research Journal of Pharmacy and Science 3.2 (2016): 631-653

[33] Chattopadhyay RR., et al. "Hypoglycemicand antihyperglycemic effect of leaves of Vinca rosea Linn". Indian Journal of Physiology and Pharmacology 35.3 (1991): 145-151

[34] Prajakta J Patil and Jai S Ghosh. "Antimicrobial Activity of Catharanthus roseus –A Detailed Study". British Journal of Pharmacology and Toxicology 1.1 (2010): 40-44

[35] Alba Bhutkar MA, Bhise SB. Comparative Studies on Antioxidant Properties of Catharanthus Rosea and Catharanthus. International Journal of Pharmaceutical Techniques, 2011, 3(3): 1551-1556

[36] Swati Agarwal, Simi Jacob, Nikkita Chettri, Saloni Bisoyi, Ayesha Tazeen, Vedamurthy AB, Krishna V, Joy Hoskeri H.Evaluation of In-vitro Anthelminthic Activity of Catharanthus roseus Extract. International Journal of Pharmaceutical Sciences and DrugResearch, 2011, 3(3): 211-213

[37] Nayak BS.,et al. "Evaluation of wound-healing potential of Catharanthus roseus leaf extract in rats". Fitoterapia 78.7-8 (2007): 540-544.

[38] Yogesh P. "Evaluation of hypolipidemic activity of leaf juice of Catharanthus roseus (Linn.)". Acta Poloniae Pharmaceutica -Drug Research 68.6 (2011) :927-935.

[39] Mithun Singh Rajput, Veena Nair, Akansha Chauhan.Evaluation of Antidiarrheal Activity of Aerial Parts of Vinca major in Experimental Animals. Middle-East Journal of Scientific Research.2011, 7 (5): 784-788

[40] Hassan KA., et al. "In vivo anti-diarrheal activity of the ethanolic leaf extract of Catharanthus roseus Linn. (Apocyanaceae) in Wistar rats". African Journal of Pharmacy and Pharmacology 5.15 (2011): 1797-1800

[41] Babulova A, Machova J, Nosalova V. Protective action of vinpocetine against experimentally induced gastric damage in rats. Arzneimittel forschung, 2003, 43:981-985. 14) P. P. Pillay, C. P. M. Nair, and T. N. Santi Kumari. Lochnera rosea as a potential source of hypotensive and other remedies.Bulletin of Research Institute of the University of Kerala, 1959, 1:51–54

[42] Himes RH. Interactions of the catharanthus (Vinca) alkaloids with tubulin and microtubules. Pharmacol Ther. 1991;51:257–67. doi: 10.1016/0163-7258(91)90081-v. [DOI] [PubMed] [Google Scholar]

[43] Downing KH. Structural basis for the interaction of tubulin with proteins and drugs that affect microtubule dynamics. Annu Rev Cell Dev Biol. 2000;16:89–111. doi: 10.1146/annurev.cellbio.16.1.89. [DOI] [PubMed] [Google Scholar]

[44] Correia JJ, Lobert S. Physiochemical aspects of tubulin-interacting antimitotic drugs. Curr Pharm Des. 2001;7:1213–28. doi: 10.2174/1381612013397438. [DOI] [PubMed] [Google Scholar]

[45] Jordan MA, Thrower D, Wilson L. Effects of vinblastine, podophyllotoxin and nocodazole on mitotic spindles. Implications for the role of microtubule dynamics in mitosis. J Cell Sci. 1992;102:401–16. doi: 10.1242/jcs.102.3.401. [DOI] [PubMed] [Google Scholar]

[46] Toso RJ, Jordan MA, Farrell KW, Matsumoto B, Wilson L. Kinetic stabilization of microtubule dynamic instability in vitro by vinblastine. Biochemistry. 1993;32:1285–93. doi: 10.1021/bi00056a013. [DOI] [PubMed] [Google Scholar]

[47] Tantray, J., Patel, A., Prajapati, B. G., Kosey, S., & Bhattacharya, S. (2024). The use of lipid-based nanocarriers to improve ovarian cancer treatment: an overview of recent developments. Current Pharmaceutical Biotechnology, 25(17), 2200-2217.

[48] Vacca A, Iurlaro M, Ribatti D, Minischetti M, Nico B, Ria R, et al. Antiangiogenesis is produced by nontoxic doses of vinblastine. Blood. 1999;94:4143–55. [PubMed] [Google Scholar]

[49] Klement G, Baruchel S, Rak J, Man S, Clark K, Hicklin DJ, et al. Continuous low-dose therapy with vinblastine and VEGF receptor-2 antibody induces sustained tumor regression without overt toxicity. J Clin Invest. 2000;105:R15–24. doi: 10.1172/JCI8829. [DOI] [PMC free article] [PubMed] [Google Scholar]

[50] Wang LG, Liu XM, Kreis W, Budman DR. The effect of antimicrotubule agents on signal transduction pathways of apoptosis: A review. Cancer Chemother Pharmacol. 1999;44:355–61. doi: 10.1007/s002800050989.

[51] Rowinsky EK, Donehower RC. Paclitaxel (taxol) N Engl J Med. 1995;332:1004–14. doi: 10.1056/NEJM199504133321507.

[52] Rowinsky EK, Donehower RC. Paclitaxel (taxol) N Engl J Med. 1995;332:1004–14. doi: 10.1056/NEJM199504133321507. [DOI] [PubMed] [Google Scholar]

[53] Gregory RK, Smith IE. Vinorelbine: A clinical review. Br J Cancer. 2000;82:1907–13. doi: 10.1054/bjoc.2000.1203. [DOI] [PMC free article] [PubMed] [Google Scholar]

[54] Joel S. The comparative clinical pharmacology of vincristine and vindesine: Does vindesine offer any advantage in clinical use? Cancer Treat Rev. 1996;21:513–25. doi: 10.1016/0305-7372(95)90015-2. [DOI] [PubMed] [Google Scholar]

[55] McGuire SA, Gospe SM, Jr, Dahl G. Acute vincristine neurotoxicity in the presence of hereditary motor and sensory neuropathy type I. Med Pediatr Oncol. 1989;17:520–3. doi: 10.1002/mpo.2950170534. [DOI] [PubMed] [Google Scholar]

[56] Hoff PM, Valero V, Ibrahim N, Willey J, Hortobagyi GN. Hand-foot syndrome following prolonged infusion of high doses of vinorelbine. Cancer. 1998;82:965–9. doi: 10.1002/(sici)1097-0142(19980301)82:5<965::aid-cncr23>3.0.co;2-y. [DOI] [PubMed] [Google Scholar]

[57] Johnson IS, Armstrong JG, Gorman M, Burnett JP., Jr The vinca alkaloids: A new class of oncolytic agents. Cancer Res. 1963;23:1390–427. [PubMed] [Google Scholar]

[58] Jackson DV, Jr, Bender RA. Cytotoxic thresholds of vincristine in a murine and a human leukemia cell line in vitro. Cancer Res. 1979;39:4346–9.

[59] Tantray, J., Patel, A., Parveen, H., Prajapati, B., & Prajapati, J. (2025). Nanotechnology-based biomedical devices in the cancer diagnostics and therapy. Medical Oncology, 42(2), 50.