ISSN: 2320-2831

International Journal of Pharmacy and Analytical Research (IJPAR)

IJPAR |Vol.14 | Issue 2 | Apr - Jun -2025

www.ijpar.com

DOI : https://doi.org/10.61096/ijpar.v14.iss2.2025.211-223

Review Article

Uv spectrophotometric method development for the estimation of multiple antineoplastic agents : a comprehensive analytical study

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Published on: 16 Jun 2025

Published by:
DrSriram Publications

Abstract

In terms of analytical instruments, UV-VIS spectroscopy was among the first. UV-Vis spectroscopy is a useful tool for characterizing a wide range of materials. Based on the different responses of samples and the degree of absorption or transmission of a range of beam light wavelengths, the UV-Vis provides information. It can be applied to qualitative as well as quantitative tests. Between 200 and 700 nm is the UV range. Additionally, the UV/Vis spectrum can help clarify the complexation mechanism involving templates, monomers, and cross-linkers during polymerization. This characterization procedure is rapid, easy, and reasonably priced. Many drugs are analysed by this method. Imatinib functions by blocking protein tyrosine kinases, especially BCR-ABL, which is constitutively activated in malignancies such as chronic myeloid leukemia (CML). Cisplatin mainly entails DNA damage, which kills cells. Through its binding to purine nucleotides in DNA, it creates crosslinks that obstruct transcription and DNA replication.Vincristine arrest cells in the metaphase, acting as antimicrotubule agents to prevent mitosis.

Keywords: UV-Spectrophotometer, Cancer, Imatinib, Cisplatin, Vincristine.

INTRODUCTION

Cancer

Cancer is a group of diseases characterized by uncontrolled cell growth and spread. It arises from genetic mutations disrupting normal cell regulation, leading to excessive proliferation and resistance to apoptosis. Causes include genetic predisposition, environmental factors (e.g., tobacco, radiation, chemicals), and infections (e.g., HPV, H. pylori) [1]. Cancer can develop in any tissue, with common types including lung, breast, colorectal, prostate cancer, and leukemia [2]. Advances in early detection and treatments (surgery, chemotherapy, radiation, immunotherapy, targeted therapy) have improved survival rates, yet cancer remains a leading global health challenge, responsible for nearly 10 million deaths in 2020 [3]. Research focuses on understanding cancer biology, developing new treatments, and enhancing prevention.

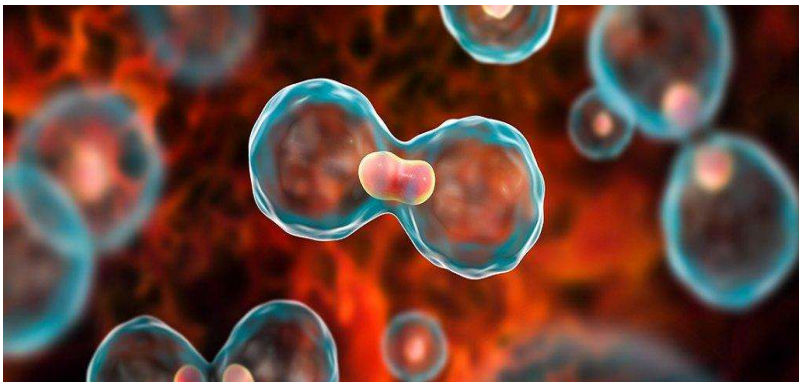

Fig 1: Cancerous Cell

Types of Cancer
Cancer is classified based on the type of cell or tissue from which it originates. The major types of cancer include:

Carcinomas
Carcinomas are the most common type of cancer, arising from epithelial cells that line the body's internal and external surfaces. Examples include:
- Adenocarcinoma – Found in glandular tissues such as the breast, prostate, pancreas, and lungs.
- Squamous Cell Carcinoma – Originates in the squamous epithelial cells found in the skin, lungs, and esophagus.
- Basal Cell Carcinoma – A slow-growing cancer that arises in the basal cells of the skin [4].

Sarcomas
Sarcomas originate in connective tissues such as bones, muscles, fat, and cartilage. Examples include:
- Osteosarcoma – A bone cancer that primarily affects children and young adults.
- Liposarcoma – A cancer of fat tissue that commonly affects the limbs and abdomen.
- Leiomyosarcoma – Arises from smooth muscle tissue, often affecting the uterus, stomach, or intestines [5].

Leukemias
Leukemia is a type of blood cancer that originates in the bone marrow and affects white blood cells. Major types include:
- Acute Lymphoblastic Leukemia (ALL) – Common in children, affecting immature lymphocytes.
- Acute Myeloid Leukemia (AML) – Affects myeloid cells, common in adults.
- Chronic Lymphocytic Leukemia (CLL) – Progresses slowly and mainly affects older adults.
- Chronic Myeloid Leukemia (CML) – Associated with the Philadelphia chromosome abnormality [6].

Lymphomas
Lymphomas develop in the lymphatic system and are divided into:
- Hodgkin Lymphoma (HL) – Characterized by the presence of Reed-Sternberg cells.
- Non-Hodgkin Lymphoma (NHL) – A diverse group of lymphatic cancers, including diffuse large B-cell lymphoma and follicular lymphoma [7].

Myelomas
Myeloma affects plasma cells, which produce antibodies in the bone marrow. The most common type is Multiple Myeloma, which leads to weakened bones and impaired immune function [8].

Brain and Central Nervous System (CNS) Cancers
These cancers arise in the brain or spinal cord. Examples include:
- Glioblastoma Multiforme (GBM) – An aggressive brain tumor with poor prognosis.
- Meningioma – A tumor that develops in the meninges, usually benign but sometimes malignant.
- Medulloblastoma – A common childhood brain cancer [9].

Skin Cancers

Skin cancers arise from different layers of the skin and include:

- Melanoma – The deadliest form, originating from melanocytes (pigment-producing cells).
- Basal Cell Carcinoma (BCC) – A slow-growing skin cancer.
- Squamous Cell Carcinoma (SCC) – More aggressive than BCC and can spread to other parts of the body [10].

Gastrointestinal Cancers

These cancers develop in the digestive system and include:

- Esophageal Cancer – Squamous cell carcinoma and adenocarcinoma are the main subtypes.
- Gastric (Stomach) Cancer – Often associated with Helicobacter pylori infection.
- Colorectal Cancer – One of the most common and preventable cancers with screening [11].

Current scenario in cancer

Incidence and Mortality Statistics

In 2025, the U.S. expects 2.04 million new cancer cases and over 618,000 deaths. Prostate, lung, and colorectal cancers are most common in men; breast, lung, and colorectal in women.Globally, breast cancer incidence is rising by 1–5% annually in many countries. High-HDI nations show declining mortality, but low-HDI regions face growing burdens. By 2050, global cancer cases may rise 38%, with deaths increasing by 68%.

Advances in Cancer Treatment

Recent advances in cancer care include personalized cancer vaccines detecting early-stage cancers through blood protein markers. The NHS introduced a seven-minute cancer injection, streamlining treatment compared to traditional IV infusions. Cell and immunotherapies are revolutionizing treatment, bringing hope for potential cures. Immunotherapy combinations like nivolumab and ipilimumab show success in advanced melanoma.CAR T-cell and CAR-NK therapies reprogram immune cells to target cancer more precisely.

Disparities in Cancer Detection and Survivorship

Cancer detection and survivorship outcomes vary significantly across populations. The CA-125 test is less effective in detecting ovarian cancer in Black and Native American patients. This may delay diagnosis and reduce survival rates among these groups. Childhood cancer survivors are nearly three times more likely to face age-related illnesses. Their elevated health risks highlight the need for tailored long-term medical care.

Future Directions

Looking ahead, experts anticipate major breakthroughs in spatial transcriptomics and sub-cellular high-resolution imaging, enhancing our understanding of cancer progression and resistance.Advancements in artificial intelligence are also expected to revolutionize cancer detection and treatment, leading to more precise diagnoses and personalized therapies.

In summary, 2025 has seen significant advancements in cancer research and treatment, offering hope for improved patient outcomes.However, challenges such as healthcare disparities and long-term survivorship issues remain, necessitating continued efforts to address these concerns [12].

Pathophysiology of Cancer

The development of cancer involves a series of genetic mutations that disrupt normal cell regulatory mechanisms. These mutations can result from various factors, including environmental exposures, lifestyle choices, and inherited genetic predispositions. The accumulation of these mutations leads to uncontrolled cell division, resistance to cell death, and the ability to invade surrounding tissues and metastasize to distant sites.

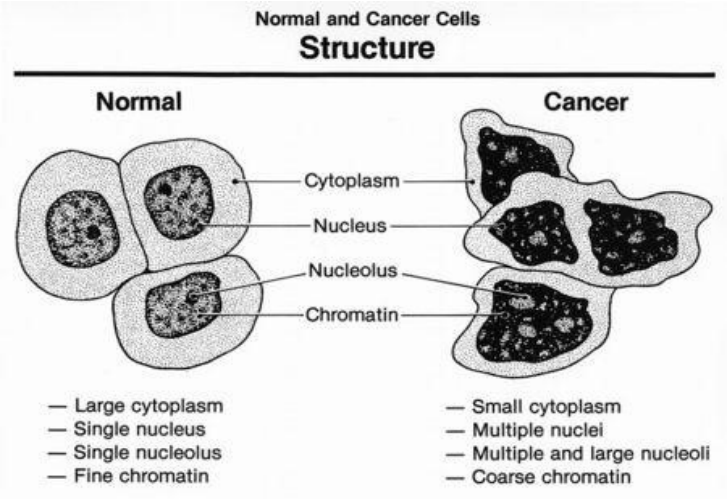

Fig 2: Differences between normal cell and cancer cell

The development of cancer involves multiple stages:

1. **Initiation:** Genetic mutations occur due to factors like carcinogen exposure or inherited genetic defects.
2. **Promotion:** Mutated cells undergo clonal expansion, driven by growth signals and a supportive tumor microenvironment.
3. **Progression:** Further genetic alterations lead to increased malignancy, enabling invasion and metastasis [13].

Chronic inflammation plays a significant role in cancer development. Persistent inflammatory conditions can increase the risk of cancers such as gastric, colon, and hepatic cancers. The tumor microenvironment, comprising various cell types, cytokines, and extracellular matrix components, influences tumor growth and response to therapy [14].

Advancements in Treatment

Recent research has led to the development of novel therapeutic strategies targeting specific molecular pathways involved in cancer progression. These include:

- Targeted Therapies: Drugs designed to interfere with specific molecules essential for tumor growth and progression.
- Immunotherapies: Treatments that enhance the body's immune system to recognize and attack cancer cells more effectively.
- Epigenetic Therapies: Approaches that modify gene expression without altering the DNA sequence, thereby reversing abnormal gene activation or silencing in cancer cells.
- RNA-based Interventions: Techniques that utilize RNA molecules to modulate gene expression or directly target cancer-specific genetic abnormalities [16].

These innovative treatments have opened new avenues for precision medicine, offering more personalized and effective options for patients [17].

Ongoing research continues to deepen our understanding of cancer biology, leading to the development of more effective and personalized treatments.

Anti-cancer drugs

Anticancer drugs, also known as chemotherapeutic agents, are designed to inhibit or destroy cancer cells. These drugs work through various mechanisms, such as interfering with DNA replication, inhibiting cell division, or targeting specific molecular pathways involved in cancer progression.

Cancer continues to be one of the leading causes of mortality worldwide, prompting an urgent need for effective diagnostic and therapeutic strategies. The development of anticancer agents plays a critical role in oncology, offering targeted, cytotoxic, and immunomodulatory effects to combat tumor growth. However, the accurate quantification and quality assessment of these chemotherapeutic agents are essential to ensure their safety, efficacy, and regulatory compliance.

According to the World Health Organization (WHO), cancer accounts for nearly one in six deaths worldwide, with incidence and mortality rates steadily increasing, particularly in low- and middle-income countries. Advances

in molecular biology and genomics have revolutionized cancer therapy, enabling the development of targeted treatments, immunotherapeutics, and personalized medicine strategies. Among these, chemotherapeutic agents continue to play a vital role in first-line and combination regimens for a wide range of cancers.

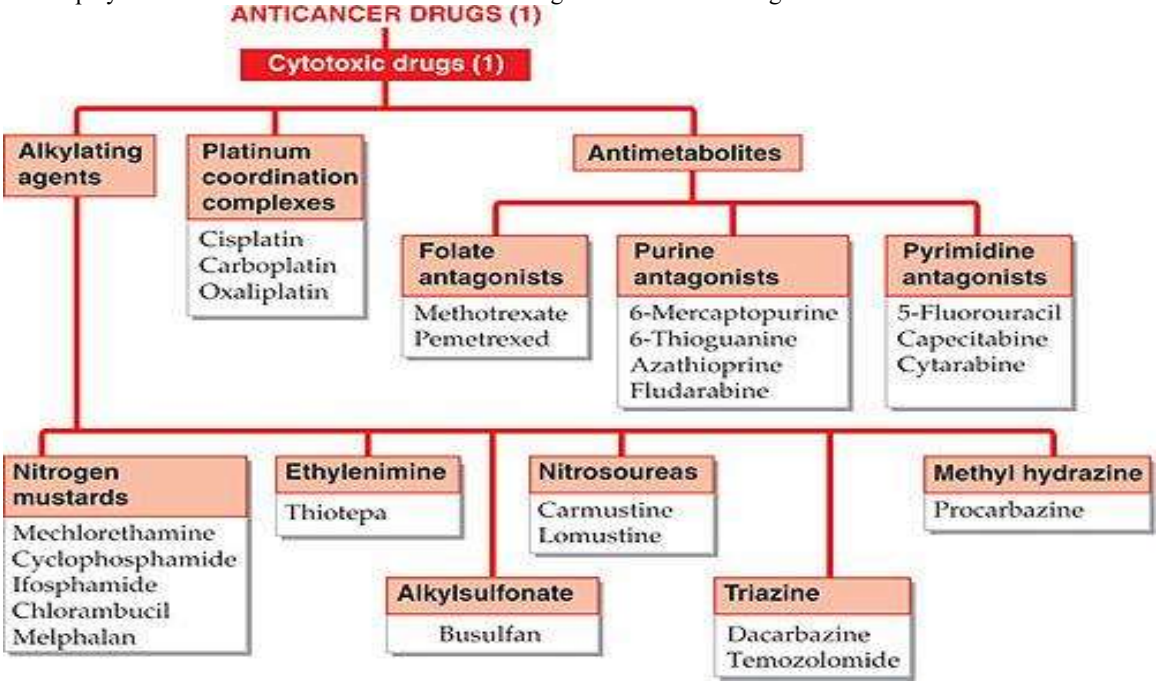

Fig 3: Classification of Anti-Cancer Agents

Drug profile
Imatinib
Drug Profile
Generic Name: Imatinib
Brand Names: Gleevec (USA), Glivec (Europe)
Drug Class: Tyrosine kinase inhibitor (TKI)
IUPACName:4-[(4-Methylpiperazin-1-yl)methyl]-N-[4-methyl-3-[(4-pyridin-3-ylpyrimidin-2-yl)amino]phenyl]benzamide
Chemical Formula: $C_{29}H_{31}N_7O$
ATC Code: L01EA01
Approved By: FDA (2001)

Mechanism of Action (MOA)
Imatinib is a selective inhibitor of the **BCR-ABL tyrosine kinase**, which is formed due to the Philadelphia chromosome translocation (t[9;22]) in chronic myeloid leukemia (CML). It also inhibits other tyrosine kinases, such as **c-KIT** (in gastrointestinal stromal tumors, GIST) and **PDGFR** (platelet-derived growth factor receptor). By blocking ATP binding, it prevents phosphorylation of substrates required for tumor cell proliferation and survival, leading to apoptosis of malignant cells [18].

Indications
- Chronic Myeloid Leukemia (CML)(Philadelphia chromosome-positive)
- Acute Lymphoblastic Leukemia (ALL)(Philadelphia chromosome-positive)
- Gastrointestinal Stromal Tumors (GIST)(c-KIT positive)

Pharmacokinetics
- Absorption: Orally bioavailable (~98%).
- Metabolism: Primarily in the liver via CYP3A4.
- Half-life: ~18 hours (parent drug), ~40 hours (active metabolite).
- Excretion:Feces (~68%) and urine (~13%).

Dosage & Administration
- CML: 400–600 mg/day (chronic phase); up to 800 mg/day (accelerated/blast phase).
- GIST: 400 mg/day; can be increased to 800 mg if needed.
- Route: Oral (tablet form).

Cisplatin
Drug Profile
Generic Name: Cisplatin
Brand Names: Platinol, Platinol-AQ
Drug Class: Platinum-based chemotherapy agent (Alkylating agent)
IUPAC Name:cis-di amine di chloro platinum
Chemical Formula: $(NH_3)_2Cl_2$

Mechanism of Action
Cisplatin is an alkylating agent that exerts its cytotoxic effects by binding to DNA in cancer cells, leading to the formation of DNA cross-links. This impairs DNA replication and transcription, causing apoptosis (programmed cell death) in rapidly dividing cells. Cisplatin primarily exerts its effects in the S-phase of the cell cycle.

Indications
Cisplatin is used to treat various types of cancer, including:
- Testicular cancer
- Ovarian cancer
- Bladder cancer
- Lung cancer (non-small cell)

Dosage and Administration
- Intravenous administration is the most common method of delivery.
- The dosage varies depending on the type of cancer, the patient's overall health, and other treatment regimens.
- Typically, doses range from 50 to 120 mg/m² depending on the specific indication.
- It is usually administered every 3 to 4 weeks, although this can vary based on the cancer being treated.

Pharmacokinetics
- Absorption- Cisplatin is given intravenously, so it directly enters the bloodstream.
- Distribution- Cisplatin is widely distributed in tissues, including the kidneys, liver, and lungs, and is concentrated in the tumor cells.
- Metabolism- Cisplatin is not extensively metabolized in the liver; rather, it acts directly on cellular DNA.
- Excretion- Excreted primarily through the kidneys in the form of unchanged drug, with a half-life of approximately 1 hour in the plasma.

Precautions
- Regular monitoring of renal function (creatinine, BUN) and audiometric tests (hearing function) is recommended.
- Hydration should be maintained before and after administration to prevent nephrotoxicity.
- Pre-treatment with antiemetic is often necessary to prevent nausea and vomiting.

Vincristine
Drug Profile
Generic Name: Vincristine
Brand Name(s): Oncovin,VincasarPFS
Drug Class: Antineoplastic (Chemotherapy drug),Vinca Alkaloid
IUPAC Name: Methyl(5β,12β,19α)-3-hydroxy-16-methoxy-1-methyl-6,7-didehydroindole[2′,3′:3,4]pyrido[1,2-b]indol-20(1H)-one-16-carboxylate
Chemical Formula: $C_{46}H_{56}N_4O_{10}$

Mechanism of Action
Vincristine is a vinca alkaloid chemotherapy drug that disrupts the formation of microtubules ,structures essential for cell division. It binds to tubulin and inhibits micro tubule polymerization, preventing the formation of the mitotic spindle necessary for cell division. This leads to the accumulation of cells in metaphase, ultimately resulting in cell death[19].

Indications

Vincristine is used in the treatment of various cancers, including:

- Acute lymphoblastic leukemia(ALL)
- Hodgkin lymphoma
- Non-Hodgkin lymphoma
- Wilms tumor
- Rhabdomyosarcoma
- Solid tumors (often in combination with other chemotherapy drugs) & Certain sarcomas

Vincristine is frequently used as part of multi-agent chemotherapy regimens[19].

Dosage and Administration

Vincristine is usually administered intravenously(IV) in a clinical setting. The typical dosing regimen for adults and children is calculated based on body surface area(BSA).

Usual Adult Dose: 1.4–2.0mg/m² IV, administered once weekly (or as part of a multi-drug regimen).

Pediatric Dose: The dosing in children depends on the specific chemotherapy

Vincristine should be administered slowly over10-15 minutes to avoide extravasation (leakage in to surrounding tissues).

➢ Serious Side Effects:

- Neurotoxicity (increased risk of neurological dysfunction)
- Extravasation injury(tissue damage if the drug leaks outside the vein)
- Autonomic dysfunction (e.g., bladder dysfunction) [20].

Table 1: Drug interactions & safety profile table

Drug	Drug interaction	Food interactions	Contraindica-tions	Side effects
Imatinib	CYP3A4 inhibitors/inducers (e.g., ketoconazole, rifampicin), warfarin, erythromycin, carbamazepine	Grapefruit juice (increases plasma concentration)	Hypersensitivity to imatinib or excipients	Nausea, vomiting, fluid retention, rash, muscle cramps, fatigue, hepatotoxicity
Cisplatin	Nephrotoxic drugs (e.g., aminoglycosides), ototoxic drugs, loop diuretics, paclitaxel (↑ neurotoxicity)	Avoid alcohol (enhances nephrotoxicity risk)	Pre-existing renal impairment, hearing impairment, hypersensitivity to platinum	Nausea, vomiting, nephrotoxicity, ototoxicity, myelo suppression, electrolyte disturbances
Vincristine	CYP3A4 inhibitors (e.g., itraconazole), phenytoin (↓ phenytoin levels), digoxin (↓ efficacy)	High-fat meals may affect absorption	Demyelinating conditions, Charcot-Marie-Tooth disease, severe hepatic impairment	Constipation, neuropathy, alopecia, jaw pain, SIADH, abdominal cramps

Table 2: Validated UV Spectrophotometric Parameters of Selected Anticancer Drugs

Drug	Author Name	Journal Name	Title Name	Analytical Conditions
Imatinib	Patel S. A., Patel N. J., Patel S. A.	Der PharmaChemica	Development and Validation of UV Spectrophotometric Method for Estimation of ImatinibMesylate in Bulk and Tablet Dosage Form.	**λmax:** 265nm **Concentration:** 1-10 **Absorbance:** 0.1-0.9 **SolventUsed:** Methanol

				[22]
Enzalutamide	Zamir, G. K., Patel, M. P., & Patel, N. J.	Analytical Chemistry: An Indian Journal.	Validated UV Spectroscopic Methods for Determination of Enzalutamide in Pure and Pharmaceutical Dosage Form.	**λmax:** 236nm **Concentration:** 3-15 **Absorbance:** 0.2-1.0 **Solvent Used:** Methanol [23]
Erlotinib	Sereya, K., Latha, S. T., Kamalakannan, D., Jambulingam, M., AnandaThangadurai, S., &Anilkumar, M.	International Journal of Pharmaceuticals and Health Care Research	A new UV spectrophotometric method development and validation for Erlotinib by derivative spectroscopy.	**λmax:** 246nm **Concentration:** 2-10 **Absorbance:** 0.1- 0.6 **Solvent Used:** Methanol [24]
Febuxostat	Suhagia, B. N., Patel, I. S., & Shah, N. S	Oriental Journal of Chemistry,	Simple and validated ultraviolet spectrophotometric method for the estimation of Febuxostat in bulk and pharmaceutical dosage forms.	**λmax:** 275nm **Concentration:** 10-70 **Absorbance:** 0.3- 1.2 **Solvent Used:** 0.1 N NaOH [25]
Capecitabine	Goud, E. R., Reddy, L. S. S., Latha, K. V., &Mukkanti, K.	World Journal of Pharmaceutical Research	Development and validation of UV method for capecitabine analysis.	**λmax:** 303nm **Concentration:** 10-30 **Absorbance:** 0.2- 0.7 **Solvent Used:** Phosphate buffer pH 7.4 [26]

Dasatinib	Reddy T. P., Devi D. R.	Journal of Chemical and Pharmaceutical Research	Development and Validation of UV Spectrophotometric Method for Determination of Dasatinib in Bulk and Pharmaceutical Dosage Form.	**λmax:** 315nm **Concentration:** 2-20 **Absorbance:** 0.2- 1.8 **Solvent Used:** Acetonitrile:Water (1:1) [27]
Sorafenib Tosylate	Rao K. M., Goud K. K., Kumar R. B.	Journal of Chemical and Pharmaceutical Research	Development and Validation of UV Spectrophotometric Method for Sorafenib Tosylate in Bulk and Tablet Formulation.	**λmax:** 265nm **Concentration:** 5-25 **Absorbance:** 0.3- 1.5 **Solvent Used:** Methanol [28]
Temozolomide	Patel S. A., Patel N. J., Patel S. A.	Der PharmaChemica	Development and Validation of UV Spectrophotometric Method for Estimation of Temozolomide in Bulk and Tablet Dosage Form.	**λmax:** 328nm **Concentration:** 10-60 **Absorbance:** 0.3- 1.8 **Solvent Used:** 0.1 N HCl [29]
Bortezomib	Kale D. S., Rode M. A., Upadhye A. M.	Research Journal of Pharmacy and Technology	Method Development and Validation for Estimation of Bortezomib by UV-VIS Spectrophotometric Method.	**λmax:** 270nm **Concentration:** 2-20 **Absorbance:** 0.1- 1.0 **Solvent Used:** Methanol [30]

Irinotecan	Rao K. M., Goud K. K., Kumar R. B.	Journal of Chemical and Pharmaceutical Research	Development and Validation of UV Spectrophotometric Method for Irinotecan in Bulk and Tablet Formulation.	**λmax:** 254nm **Concentration:** 5-25 **Absorbance:** 0.2- 1.2 **Solvent Used:** Acetonitrile:Water (1:1) [31]
Vinorelbine	Patel V. J., Mehta R. S., Gajjar A. K.	Research Journal of Pharmacy and Technology	UV Spectrophotometric Method Development and Validation of Vinorelbine in Bulk and Injectable Formulation.	**λmax:** 275nm **Concentration:** 2-12 **Absorbance:** 0.2- 1.0 **Solvent Used:** Phosphate Buffer pH 3.5 [32]
Cabazitaxel	Sharma R., Singh P., Kapoor A	Asian Journal of Pharmaceutical Analysis	UV Spectrophotometric Determination of Cabazitaxel in Pharmaceutical Dosage Form	**λmax:** 275nm **Concentration:** 2-12 **Absorbance:** 0.2- 1.0 **Solvent Used:** Phosphate Buffer pH 3.5 [33]
Ixabepilone	Kulkarni R. R., Deshpande S. G.	International Journal of Research in Pharmacy and Science	Development and Validation of UV Spectrophotometric Method for Ixabepilone in Bulk and Formulations.	**λmax:** 273nm **Concentration:** 3-15 **Absorbance:** 0.2- 0.9 **Solvent Used:** Methanol [34]

Pemetrexed	Singh D., Mishra A., Tiwari A.	Asian Journal of Pharmaceutical Analysis	UV Spectrophotometric Method Development and Validation for Pemetrexed in Bulk and Injectable Form.	**λmax:** 284nm **Concentration:** 5-25 **Absorbance:** 0.2- 1.3 **Solvent Used:** Methanol [35]
Fludarabine	Maheshwari R. K., Chavada S	Indian Journal of Pharmaceutical Sciences	Development of a UV Spectrophotometric Method for Estimation of Fludarabine in Pharmaceutical Dosage Form.	**λmax:** 261nm **Concentration:** 1-10 **Absorbance:** 0.1- 0.8 **Solvent Used:** Water [36]
Mitoxantrone	Rao D. V., Pasha M. K.	International Journal of Pharmaceutical Sciences and Research	UV Spectrophotometric Method Development and Validation for Mitoxantrone Hydrochloride.	**λmax:** 609nm **Concentration:** 0.5-10 **Absorbance:** 0.05- 0.9 **Solvent Used:** Methanol:Water(1:1) [37]
Oxaliplatin	Thakur V. D., Rajopadhye B. K.	Asian Journal of Research in Chemistry	UV Spectrophotometric Determination of Oxaliplatin in Bulk and Pharmaceutical Dosage Forms.	**λmax:** 285nm **Concentration:** 1-10 **Absorbance:** 0.1- 0.9 **Solvent Used:** 0.1N HCl [38]

CONCLUSION

UV-Visible spectrophotometry, owing to its simplicity, cost-effectiveness, and sensitivity, has emerged as a widely accepted analytical technique for the estimation of anticancer drugs in pharmaceutical formulations and biological matrices. The present study aims to develop and validate UV spectrophotometric methods for a series of clinically relevant anticancer drugs, analyzing their spectral characteristics, λmax values, solvents used, absorbance patterns, and concentration ranges. This comprehensive validation not only aids in quality control but also supports the ongoing pursuit of therapeutic precision in cancer treatment.

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