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AI BASED APPROACHES IN DEVELOPMENT OF PROTEIN KINASE INHIBITORS

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ABSTRACT

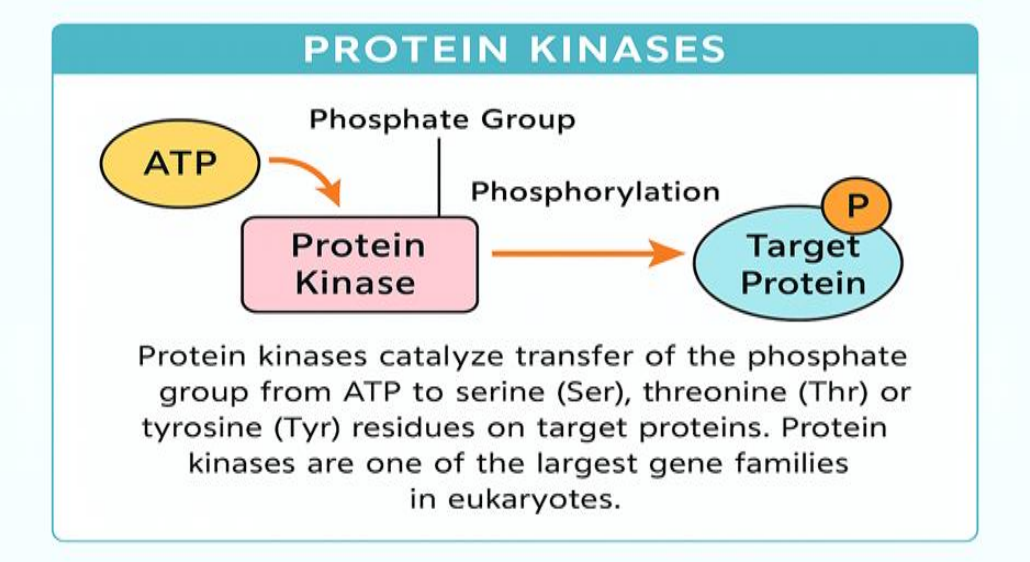
Protein kinases play a central role in regulating cellular signalling pathways, and their dysregulation is strongly associated with cancer, inflammatory disorders, and other diseases. Consequently, protein kinase inhibitors have emerged as a major class of targeted therapeutics. However, traditional drug discovery approaches remain time-consuming, costly, and limited in exploring the vast chemical and biological space. In recent years, artificial intelligence (AI) and machine learning (ML) techniques have revolutionized the development of protein kinase inhibitors by enabling faster, more accurate, and cost-effective discovery pipelines. This review highlights the integration of AI-based approaches, including quantitative structure–activity relationship (QSAR) modelling, molecular docking, molecular dynamics simulations, and deep learning frameworks, in kinase inhibitor design and optimization. Advanced methods such as 3D convolutional neural networks, network-based drug repurposing, and hybrid structure-based and data-driven models have significantly improved target prediction, binding affinity estimation, and off-target profiling. Additionally, AI-driven platforms facilitate the identification of novel kinase targets and enable drug repositioning strategies, thereby accelerating therapeutic development. Despite these advancements, challenges such as data quality, model interpretability, and generalizability remain critical barriers to widespread implementation. Future perspectives emphasize the integration of multi-omics data, explainable AI, and collaborative databases to enhance predictive performance and translational success. Overall, AI-based approaches are transforming kinase inhibitor discovery, offering promising opportunities for precision medicine and next-generation drug development.

KEYWORDS: Protein kinases, Kinase inhibitors, Artificial intelligence, Machine learning, Drug discovery, Molecular docking, QSAR modelling.

INTRODUCTION

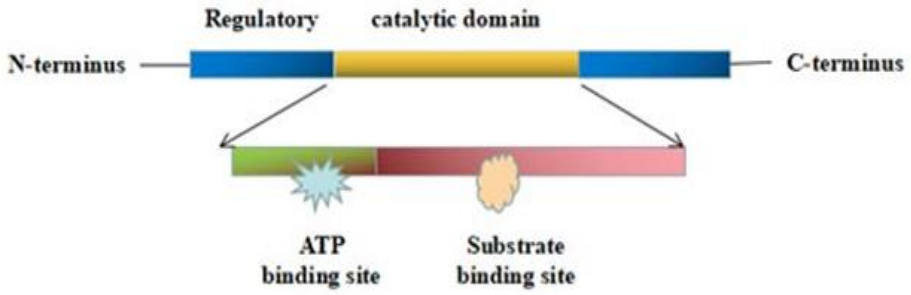
PROTEIN KINASES

- Protein kinases are enzymes that catalyse the transfer of a phosphate group from a high-energy donor molecule, typically ATP (adenosine triphosphate), to specific amino acid residues—most commonly serine (Ser), threonine (Thr), or tyrosine (Tyr) on a protein substrate. This process, called as phosphorylation, is one of the most important post-translational modifications (PTMs) in cells.
- Through phosphorylation, protein kinases regulate the structure, function, activity, localization, and interactions of target proteins. Protein kinases represent one of the largest gene families in eukaryotes with more than 518 protein kinases (478 contain a eukaryotic protein kinase [ePK] domain and 40 are atypical protein kinases [apk]) forming the human kinome.[1]

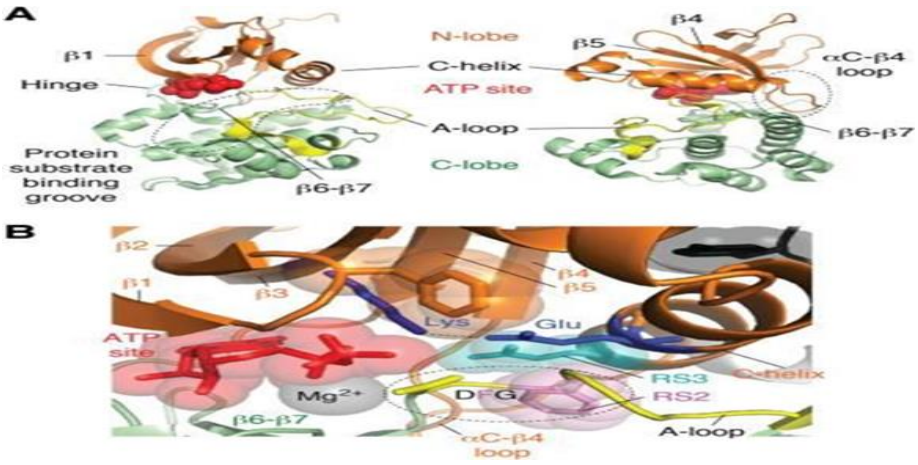


PROTEIN KINASE STRUCTURE

- Protein kinases share a conserved 3D catalytic domain of 250–300 amino acids.
- Domain has a larger α-helical C-terminal lobe and a smaller β-sheet N-terminal lobe.
- The two lobes are linked by a peptide scaffold, forming a deep groove that binds ATP and the peptide substrate.
- The ATP-binding region can rotate between **"**on" and "off" states depending on ATP binding and enzyme activation.



- **N-lobe:** 5-stranded β-sheet (β1–β5) + at least one α-helix
- **C-lobe:** mainly α-helical + small β6–β7 sheet, two lobes connected by a hinge region with flexibility.
- ATP-binding cleft lies between the two lobes. ATP phosphates sit under the Gly-rich loop (β1–β2). Phosphates interact with β3 lysine.
- Mg^{2+} ions stabilize ATP and connect it to the C-lobe. Hinge region forms two H-bonds with ATP adenine.
- Inter- lobal interface contains key catalytic elements: αC-helix + Activation loop (A-loop).
- **A-loop:** dynamic, often phosphorylated, regulates activity, aids substrate recognition.
- Some kinases (e.g., PINK1) use N-lobe for substrate binding too.
- Interface finalized by αC–β4 loop interacting with C-lobe β-sheet and αE-helix.[2]



FUNCTIONS:[3]

1) Control of Cellular Functions, Growth and Development

Protein kinases play a role in controlling what cells do how they grow and how they develop. They do this by adding or removing a group from other proteins.

2) Cell Signalling Pathways

Protein kinases help send signals inside cells. These signals tell cells to do things like grow make things copy themselves become different types of cells or die.

3) Control of Protein Activation and Inactivation

Protein kinases turn proteins on or off by adding or removing groups. They do this to one-third of all the proteins in a cell.

4) Control of Enzyme Function

When protein kinases add groups to enzymes it can change the shape of the enzymes. This changes how well the enzymes work and how they interact with things in the cell.

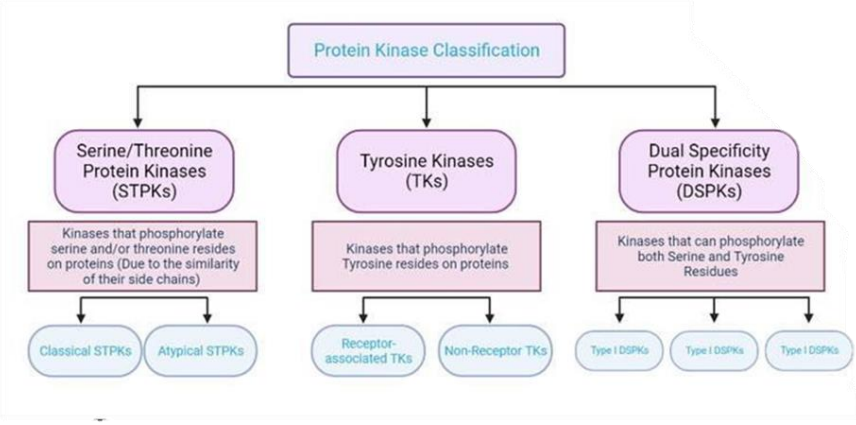
5) Cell Homeostasis

Protein kinases need to be controlled so that cells work properly. If protein kinases do not work right it can cause diseases like cancer or problems with how the body works.

6) Therapeutic Agents

Because protein kinases are so important, for how cells work and can cause disease scientists study them to find medicines. They especially look at the part of the protein kinase where it binds with a molecule called ATP.

CLASSIFICATION OF PROTEIN KINASES



[1] Serine/Threonine-Specific Protein Kinases (STPKs)

Enzymes that phosphorylate serine or threonine –OH groups in proteins. Major post-translational modification regulating eukaryotic cell functions. Involved in cell growth, division, differentiation, metabolism, and signaling.

TYPES OF STPKs

[A] Classical Serine/Threonine Kinases

Possess a highly conserved ~250 aa catalytic domain. Require conserved motifs for ATP binding and phosphate transfer.

- Protein Kinase A (PKA)
- Protein kinase C (PKC)
- Cyclin-Dependent Kinases (CDKs)
- Mitogen-Activated Protein Kinases (MAPKs)

[B] Atypical Serine/Threonine Kinases

Atypical STPKs are protein kinases that lack classical kinase motifs and are activated without Ca^{2+} or DAG [diacylglycerol]. And playing key roles in specialized signaling such as cell polarity and growth.

The two main members of this subclass are:

- PKC ζ (PKC zeta)
- PKCι/λ (PKC iota/lambda).[4]

[2] Tyrosine Kinases (TKs)

Structure: Contain a conserved catalytic core with a glycine-rich ATP-binding pocket and an essential aspartic acid residue for catalysis

Function: Catalyze phosphorylation of tyrosine residues on target proteins.

TYPES

1. Receptor Tyrosine Kinases (RTKs)
2. Non-Receptor Tyrosine Kinases (NRTKs)[6]

[3] Dual-specificity protein kinases (DSPKs)

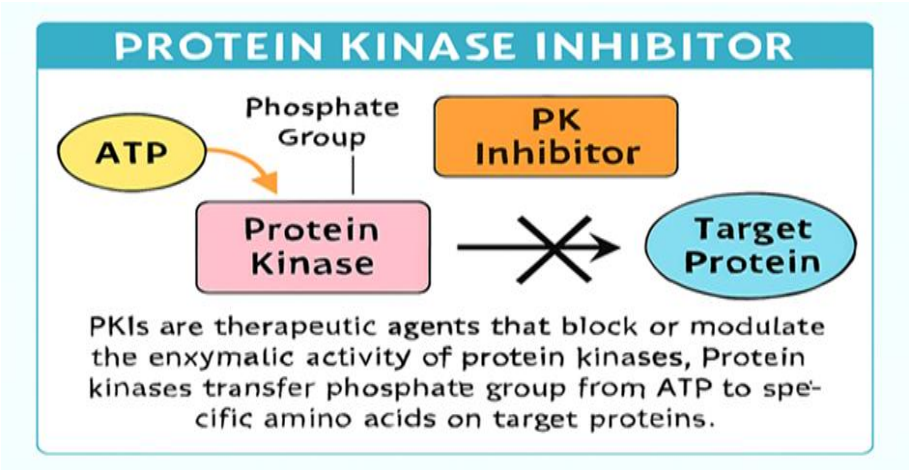
- Phosphorylate both serine/threonine and tyrosine residues
- Provide versatility in substrate recognition and regulation. Act as integrators of multiple signaling pathways.

Functions

- Regulate cell cycle progression.
- Involved in DNA damage repair.
- Mediate cellular stress responses.
- Help maintain cellular homeostasis.[7]

PROTEIN KINASE INHIBITORS (PKIs)

Protein kinase inhibitors (PKIs) are therapeutic agents that block or modulate the enzymatic activity of protein kinases, the enzymes responsible for transferring phosphate groups from ATP to specific amino acids (serine, threonine, or tyrosine) on target protein.



Types of Kinase Inhibitors

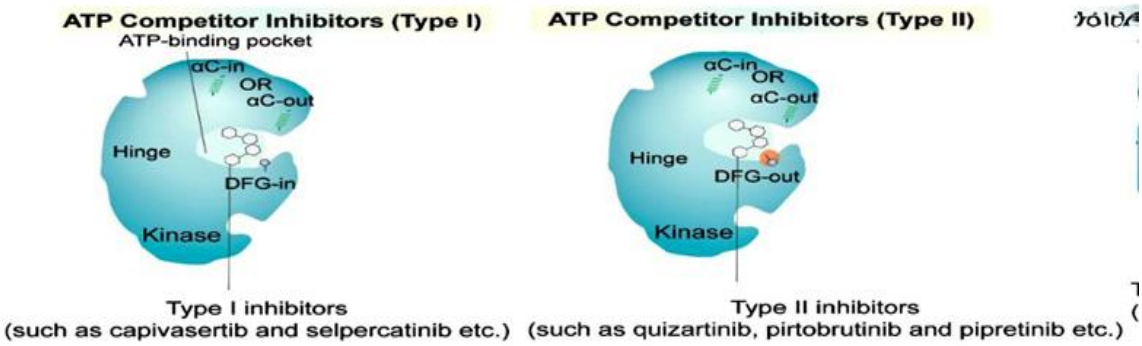
- ATP-Competitive Inhibitors
- Allosteric Inhibitors
- Covalent Inhibitors

[1] ATP-Competitive Inhibitors

- These inhibitors compete directly with ATP for binding to the ATP-binding site of kinases. Since kinases rely on ATP to transfer phosphate groups, blocking ATP binding prevents their activity.
- ATP-Competitive Inhibitors are commonly categorised into Type I and Type II inhibitors.
- **Type I**: Bind active kinase (DFG-in) at the ATP pocket. These inhibitors form the largest group of kinase inhibitors. Eg: Capivasertib, Capmatinib

  Type II: Bind inactive kinase (DFG-out); these is more selective due to structural variability.

Eg: Quizartinib, Pirtobrutinib, Selpercatinib

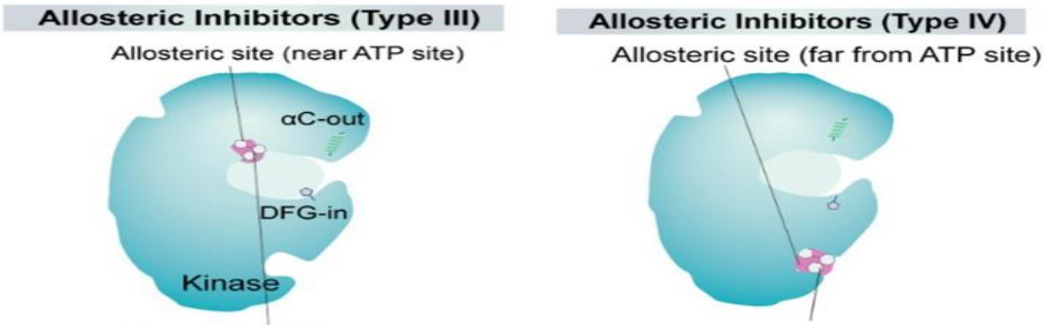


[2] Allosteric Inhibitors

Allosteric kinase inhibitors are a class of competitive inhibitors that bind to sites distinct from the ATP-binding pocket of kinases. Unlike ATP-competitive inhibitors, they do not directly interact with the hinge region of the ATP-binding site. These inhibitors are categorized as Type III or Type IV.

Type III: Bind adjacent to ATP pocket, often more selective.

Type IV: Bind topologically distinct sites and modulating kinase conformation.



TYPE III inhibitors (selumetinib) **TYPE IV inhibitors (asciminib)**

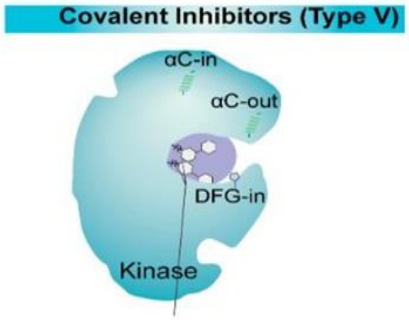
[3] Covalent Inhibitors

Covalent inhibitors are small molecules that permanently inactivate target proteins by forming a covalent bond with a specific amino acid, often in the protein's active site. They typically consist of a reactive "warhead" and a "guidance system" for selective binding. This irreversible interaction results in a long-lasting therapeutic effect.

Type V: Covalent inhibitors form bonds with specific amino acids (usually cysteine) in the kinase, blocking its activity. This covalent attachment effectively blocks kinase activity, either irreversibly or reversibly.

Irreversible inhibitors: Bind permanently, increasing potency and can overcome drug resistance.
Eg: Futibatinib, Mobocertinib, Zanubrutinib

Reversible covalent inhibitors: Bind temporarily, reducing side effects while maintaining effective action. Eg: Rilzabrutinib.[8]



TYPE V inhibitors (futibatinib)

Table: Different protein tyrosine kinases and serine/threonine kinases and their role in the cellular functions and diseases.[5]

Kinase Target	Role in cellular function and disease	Representative inhibitors	Mechanism of action	Clinical applications
FLT3 Kinase [class 3 RTK]	Essential for haematopoiesis; FLT3 mutations drive AML progression.	Sorafenib, quizartinib, gilteritinib	Inhibit FLT3 activation, blocks survival & growth signals in leukemic cells, leads to death.	Acute myeloid leukemia (AML)
BCR-ABL fusion kinase [non RTK]	Promotes uncontrolled cell proliferation in CML via constitutive tyrosine kinase activity.	Imatinib, nilotinib, ponatinib	Blocks ATP-binding to BCR-ABL, inhibiting leukemia cell proliferation.	Chronic myeloid leukemia (CML)
VEGFR (vascular endothelial growth factor receptor) [RTK]	Key regulator of angiogenesis, supporting tumour vascularization.	Sorafenib, sunitinib, pazopanib	Blocks VEGFR activation, reducing tumour blood supply.	Renal cell carcinoma (RCC), hepatocellular carcinoma (HCC), colorectal cancer
ALK (anaplastic lymphoma kinase)	Regulate neural development, cell growth. Drives tumorigenesis in NSCLC and lymphoma through fusion proteins.	Crizotinib, ceritinib, lorlatinib	Targets ALK fusion-proteins, stopping the cancer cells from growing.	NSCLC, anaplastic large cell lymphoma (ALCL)
Multi-target RTKs	Regulate multiple oncogenic pathways simultaneously.	Sorafenib, sunitinib, Lenvatinib	Inhibit multiple RTKs, targeting angiogenesis and tumour growth.	Hepatocellular carcinoma, thyroid cancer, RCC
Serine/threonine kinases (STKs)	Regulate cell cycle progression, apoptosis, and DNA repair.	Trametinib, dabrafenib, everolimus, temsirolimus	Inhibit MEK, BRAF, or mTOR pathways, blocking tumour proliferation and survival.	Melanoma (BRAF-mutant), renal cell carcinoma, breast cancer
p38 MAP kinase [MAPK]	Involved in inflammation, apoptosis, and oncogenesis; dysregulated in inflammatory diseases and cancer.	SB203580, VX-745	Inhibit p38 MAPK, reducing inflammation and cell survival.	Inflammatory diseases, cancer
Cyclin-dependent kinase (CDK)	Regulates cell cycle progression; dysregulated in cancer and neurodegenerative diseases.	Palbociclib, alvocidib, seliciclib	Inhibit CDK activity, blocking tumour cell cycle progression.	Cancer

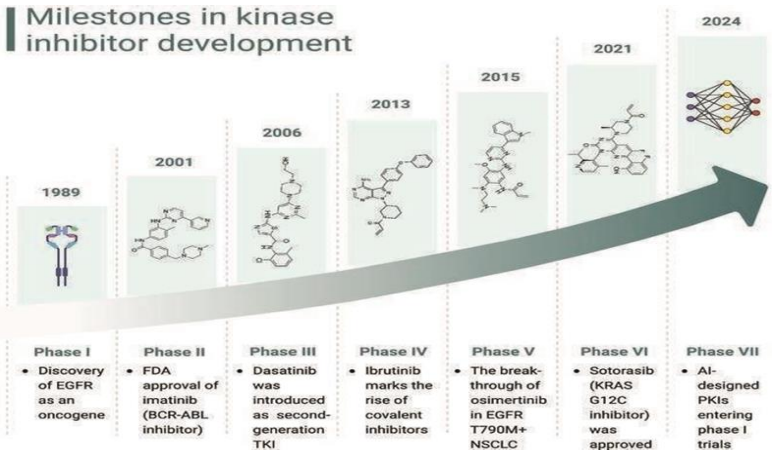
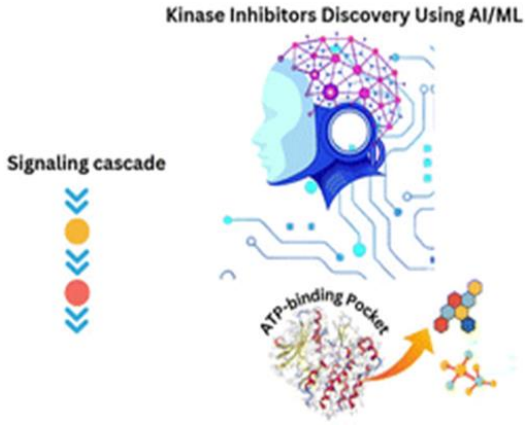


Fig. 1: Milestones in kinase inhibitor development.

INTRODUCTION TO AI IN DRUG DISCOVERY

Artificial Intelligence (AI) is the ability of machines to mimic human intelligence for tasks like learning and decision-making. It helps analyze complex data and generate predictions that guide research and problem-solving.

AI and ML accelerate kinase drug discovery by analyzing large chemical and biological datasets to predict inhibitor selectivity and optimize lead compounds. They enhance traditional methods by enabling faster, more precise, and data-driven design of next-generation kinase inhibitors.



AI APPROACHES IN DRUG DISCOVERY

1. MACHINE LEARNING

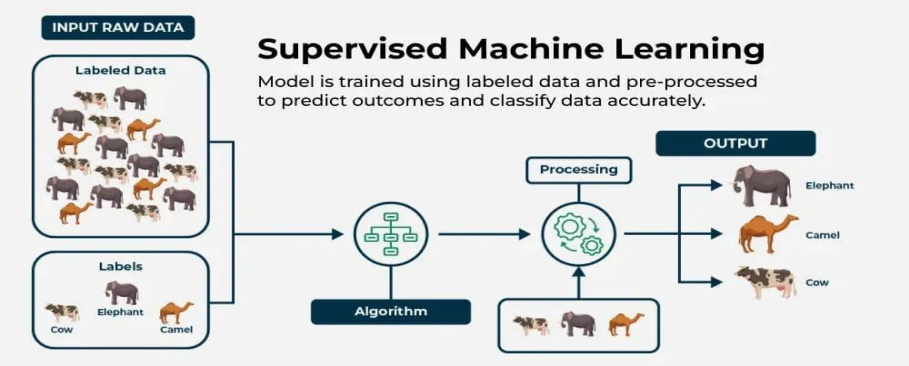
Machine learning can be described as a branch of artificial intelligence whereby a computer is able to learn through data. Unlike in rule-based AI, in ML the system learns through experience drawn from the data.[9]

DIFFERENT ML MODELS

(a) Supervised learning

Supervised learning is a type of machine learning in which a model is trained using labeled data—data for which both the input and the correct output are already known.

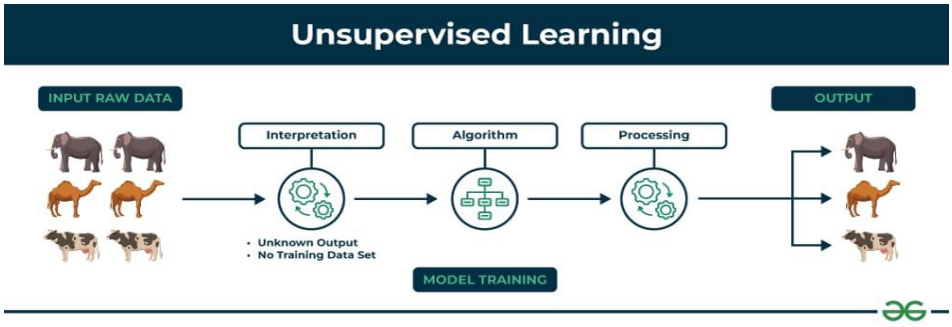
The algorithm learns the relationship between these inputs and outputs during training, so it can correctly predict the label for new, unseen data.



(b) Unsupervised learning

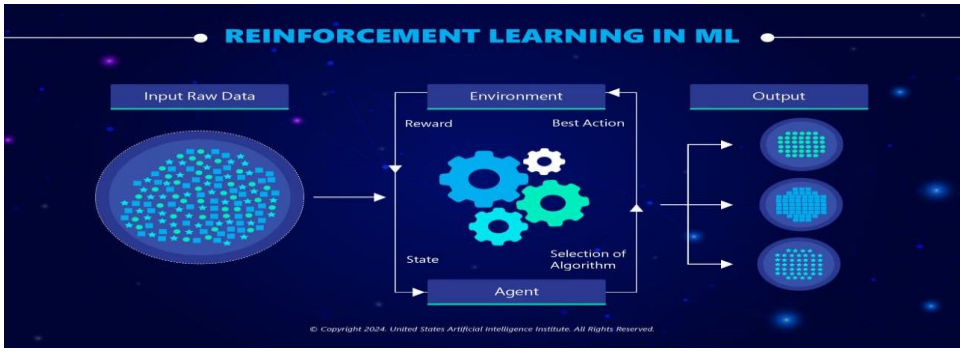
Unsupervised learning is a type of machine learning whereby the algorithms are able to learn based on unlabeled data. In such algorithms, no output variable is presented to the computer.

In other words, unlike in supervised learning, here the algorithm attempts to find patterns within the data rather than providing examples with the right answer.



(c) Reinforcement learning

Reinforcement learning is a type of machine learning where an agent learns by interacting with an environment. It requires actions and is rewarded or punished for its results. Over many trials, the agent learns which actions lead to the best long-term rewards. This helps it gradually improve its decision-making to achieve a desired goal.



2. DEEP LEARNING

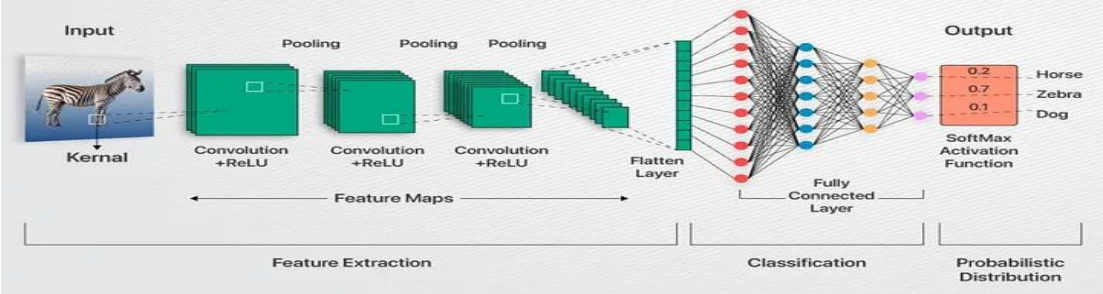
Deep Learning is an approach used by machine learning that involves the application of neural networks to automatically learn and recognize patterns in data. Feature extraction is not needed because it learns directly from its raw data like images, texts, or molecules.[10]

DEEP LEARNING MODELS

(a) Convolutional Neural Network (CNN)

Convolutional Neural Network (CNN) is a deep learning network that uses convolution filters to detect hierarchical features in input data. This type of network is very effective in learning spatial patterns like image or molecular structures.

FUNCTIONS: Pattern Recognition Predictive Models Development.



(b) Recurrent Neural Networks (RNNs)

A Recurrent Neural Network (RNN) is a neural architecture designed to process sequential data by maintaining an internal memory across time steps. This allows it to learn temporal patterns and dependencies in time-ordered information.

FUNCTIONS: Predicting Future States Analysis of Biological Sequences.[11]

(c) Graph Neural networks (GNNs):

The Graph Neural Network (GNN) is a deep learning framework that learns data represented as networks or graphs containing nodes and edges. GNNs learn from message exchange among nodes to uncover the relationship between the nodes of a graph.

FUNCTIONS: Drug Response Prediction Molecular Structure Interpretation.

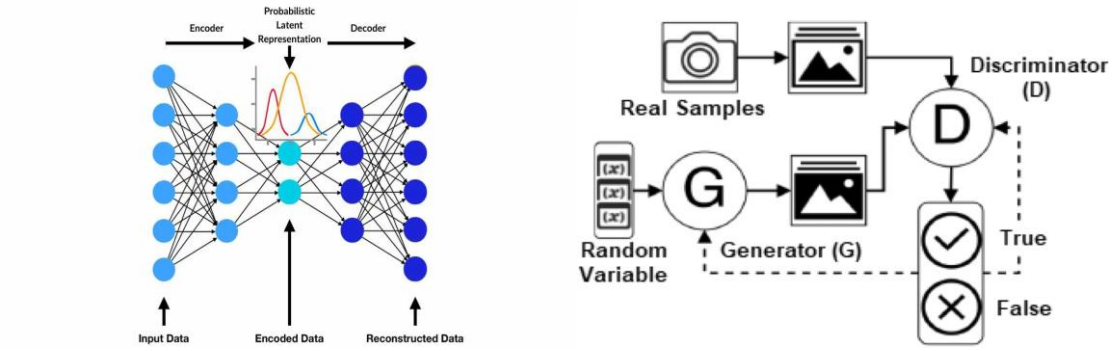
3. GENERATIVE MODELS FOR MOLECULAR DESIGN

Generative models in molecular design learn the underlying chemical patterns and rules from large molecular datasets. They then generate new, novel molecules that follow these learned patterns and may exhibit desirable drug-like or biological properties.[10]

TYPES

[a] Variational Autoencoders (VAEs)

Variational Autoencoder is considered among various types of generative deep learning algorithms used to generate new similar data samples based on training samples through the encoding of input data (e.g., an image) into the probability distribution in the latent space by means of two parameters – the mean vector (μ) and variance; then decode random samples in this latent space.



[b] Generative Adversarial Networks (GANs)

A Generative Adversarial Network (GAN) may be referred to as probably the most popular approach to building generative neural networks since it comprises two parts, each of which performs certain functions: the first one acts like an artist who creates art and the second like an inspector that evaluates this art.

AI/ML applications in kinase inhibitor development

1. Target Identification

AI analyzes biological data to find potential drug targets, such as proteins, associated with a disease.

2. Virtual Screening

Machine learning models evaluate large databases of chemical molecules to predict their potential biological activity against a specific target.

3. Lead Optimization

AI/ML helps refine drug candidates by improving their chemical properties, bioactivity, and structure to increase effectiveness and reduce side effects.

4. Structure-Activity Relationship (QSAR) modeling

AI, particularly deep learning, is used to build sophisticated models that predict how changes in a molecule's structure affect its activity.

5. Predicting and overcoming resistance

AI can analyze resistance mutations to help design next-generation inhibitors that are effective against mutated kinases.[10]

LITERATURE REVIEW

Gilvary et al. (2020) developed a machine learning and network-based framework to identify new therapeutic indications for small molecules. The study employed an XGBoost-based machine learning model known as CATNIP, combined with a drug–drug similarity network and extensive feature engineering using chemical, biological, and pathway data. The results demonstrated strong predictive performance, achieving an AUC of 0.841, and successfully identified novel drug repurposing opportunities. These findings highlight the effectiveness of machine learning approaches in advancing drug repositioning efforts.[11]

Ashraf et al. (2022) conducted a comprehensive study aimed at modeling and designing TTK inhibitors by integrating multiple computational approaches. The methodology combined 3D-QSAR techniques, including CoMFA and CoMSIA, with molecular docking, molecular dynamics simulations, MM/PBSA calculations, and ADMET analysis to evaluate the effectiveness and drug-like properties of potential compounds. The study successfully identified three

compounds NDC1, NDC7, and NDC9—as promising TTK inhibitors. These compounds demonstrated stable binding interactions with the target protein, favourable energy profiles, and satisfactory drug-likeness characteristics, highlighting their potential for further development.[12]

De Simone et al. (2022) proposed a machine learning–driven framework for repositioning kinase inhibitors by integrating multiple computational techniques. The study employed various machine learning classifiers, including Support Vector Machines (SVM), Random Forests (RF), and Neural Networks (NN), along with molecular fingerprints, binding site similarity analysis, and docking studies. The findings demonstrated that combining machine learning approaches with binding-site similarity significantly enhances the efficiency of repurposing kinase inhibitors. This integrated strategy not only improves prediction accuracy but also increases the overall reliability of identifying potential drug candidates for new therapeutic applications.[13]

Kanev et al. (2023) conducted a study titled "Predicting the Target Landscape of Kinase Inhibitors Using 3D Convolutional Neural Networks." The research employed advanced computational approaches, including 3D convolutional neural networks, transfer learning, and Random Forest regression that integrates both protein and ligand fingerprints. The study demonstrated that structure-aware deep learning models significantly enhance the prediction of kinase inhibitor potency across diverse datasets, highlighting the effectiveness of combining spatial molecular information with machine learning techniques for improved drug-target interaction predictions.[14]

Almukadi et al. (2023) conducted a study titled "Combining Machine Learning and Structure-Based Approaches to Develop Oncogene PIM Kinase Inhibitors." The research utilized multiple machine learning models, including Random Forest (RF), Support Vector Machine (SVM), and XGBoost, along with structure-based techniques such as molecular docking, molecular dynamics simulations, and virtual screening. The study successfully identified potent and stable PIM-1 inhibitors, demonstrating that integrating machine learning–guided screening with docking and molecular dynamics simulations is an effective strategy for discovering and validating promising kinase inhibitors.[15]

Iqbal et al. (2025) conducted a study titled "Integrating Machine Learning and Structure-Based Approaches for Repurposing Potent Src Kinase Inhibitors." The research combined machine learning models such as Support Vector Machine (SVM) and Random Forest (RF) with structure-based techniques including molecular docking, molecular dynamics simulations, MMGBSA/MMPBSA binding energy calculations, and toxicity prediction. The study successfully repurposed FDA-approved drugs, namely orlistat and acarbose, identifying them as safe and stable Src kinase inhibitors with potential applications in the treatment of inflammatory disorders.[16]

Jindi Huang et al. (2024) conducted a large-scale comparison of machine learning methods for the profiling prediction of kinase inhibitors. The study employed a benchmark dataset specifically designed for kinase profiling prediction to ensure reliable evaluation. It involved the calculation of molecular representations to effectively capture chemical features relevant for modeling. Additionally, various machine learning (ML) and deep learning (DL) algorithms were selected for assessment and model construction. The findings highlight that standardized and cleaned data from sources such as ChEMBL Version 29, PubChem, Binding DB, and ZINC database enabled the creation of a high-quality dataset with clear active and inactive labels, improving prediction accuracy.[17]

Hyerim Kim et al. (2023) explored artificial intelligence-driven new drug discovery targeting serine/threonine kinase for cancer treatment. The study employed methodologies such as viability assays, cell cycle analysis using PI flow cytometry, and AI-aided drug repurposing techniques. PI flow cytometry was used to accurately determine cell cycle distribution across G0/G1, S, and G2/M phases in MDA-MB-231 and A549 cells. The approach enabled detailed evaluation of cellular responses following treatment with STK33-23. The findings demonstrated that this method effectively identifies dose-dependent cell cycle arrest and changes in cell proliferation.[18]

LITERATURE REVIEW CONCLUSION

We concluded that Protein kinases play a key role in many diseases, especially cancer, making them important drug targets. The reviewed studies (Literature) shows that AI-based approaches such as machine learning [Support vector machine, Random forest, Neural networks] and deep learning [convolutional neural network] when combined with QSAR, molecular docking, and molecular dynamics studies, improve drug discovery by accurately predicting activity, selectivity, and safety. These methods also help in overcoming drug resistance and repurposing existing drugs. Overall, AI-integrated strategies accelerate the development of effective and safer therapies for cancer and other complex diseases.

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