



Computer-Aided Drug Design in the Artificial Intelligence Era: Integrated Workflows, Validation, and Translational Progress

Dr. Umaina Gazal^{1*}, K. Niharika¹

¹ Research and Development Cell, PG Center, AV College of Arts, Science & Commerce, Hyderabad, India

* Email ID: dr.umaimagazal@gmail.com

ARTICLE INFO

Article history:

Received: 07-08-2026

Received in revised form:
17-08-2026

Accepted: 12-09-2026

Available online: 13-09-2026

Keywords:

Computer-aided Drug Design

Molecular Docking

QSAR

Pharmacophore

Molecular Dynamics

ADMET

ABSTRACT

Computer-aided drug design (CADD) has developed from a supporting computational approach into an integrated component of modern drug discovery. Structure-based and ligand-based methods, including molecular docking, virtual screening, quantitative structure-activity relationship (QSAR) modelling, pharmacophore modelling, molecular dynamics (MD) simulations and in silico ADMET assessment, can reduce the experimental search space and improve lead prioritization. Artificial intelligence (AI) now extends these workflows through data-driven target identification, biomolecular structure prediction, molecular-property prediction and generative molecular design. However, computational performance does not by itself establish therapeutic activity. Docking scores are approximate, MD results depend on force fields and sampling, QSAR/AI models may fail outside their applicability domain, and predicted ADMET properties require experimental confirmation. This focused review summarizes the principal CADD methods, shows how they can be combined in a practical screening-to-validation workflow, and discusses current AI-enabled advances. The clinical translation of rentosertib, a generative-AI-designed TNK1 inhibitor for idiopathic pulmonary fibrosis, is highlighted as an instructive example. The central conclusion is that the strongest drug-discovery strategy is not AI or classical CADD alone, but an iterative combination of computational prediction, chemical reasoning and rigorous experimental validation.

1. INTRODUCTION

Drug discovery involves target identification, hit discovery, lead optimization, preclinical testing and clinical evaluation. Because experimental screening of large chemical libraries is expensive and time-consuming, computational methods are increasingly used to prioritize the most plausible targets and compounds before laboratory testing [1,2]. CADD integrates structural biology, computational chemistry, cheminformatics and biological data to guide these decisions rather than replace experiments. Two broad strategies dominate CADD. Structure-based drug design (SBDD) uses three-dimensional information about a biological target, whereas ligand-based drug design (LBDD) derives predictive information from known active and inactive compounds. In practice, the most useful workflows are hybrid: ligand similarity or pharmacophore screening may identify candidates, docking may evaluate plausible binding modes, ADMET models may remove unsuitable molecules, and MD simulations may test whether selected complexes remain dynamically stable [2], [3], [4], [5], [6]. The current transition to AI assisted drug discovery expands this framework. Machine learning (ML) and deep learning models can learn molecular representations, rank targets and compounds, predict molecular properties and generate new structures. At the same time, modern structure prediction systems such as AlphaFold 3 can model complexes containing proteins, nucleic acids, small molecules, ions and modified residues [7], [8], [9], [10], [11]. These advances are powerful, but they make validation

increasingly important because model bias, data leakage, limited chemical coverage and overinterpretation can produce apparently convincing predictions that fail experimentally [8], [9], [10], [11], [12]. The overall CADD process is illustrated in **Figure 1**. The workflow begins with target selection and preparation of structural or ligand data, followed by virtual screening, docking, QSAR or AI based prediction, and ADMET and molecular dynamics assessment. Promising candidates are then subjected to experimental validation, while the resulting biological evidence guides further lead optimization, redesign and reprioritization.

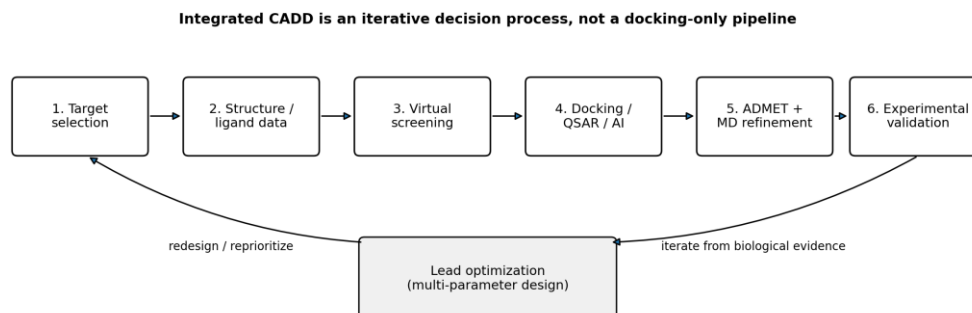


Figure 1. Integrated CADD workflow showing target selection, virtual screening, computational prediction, ADMET and molecular dynamics refinement, experimental validation, and iterative lead optimization.

2. CORE CADD METHODS AND THEIR COMPLEMENTARY ROLES

2.1 Molecular Docking

Molecular docking predicts possible ligand orientations within the binding site of a biological target and ranks the resulting poses using a scoring function. It is widely applied in virtual screening and interaction analysis because it enables rapid evaluation of large numbers of candidate molecules. However, docking scores should not be interpreted as direct measurements of binding affinity. Factors such as protein preparation, protonation state, retained water molecules, receptor flexibility and the selected scoring function can substantially influence the predicted results. A reliable docking protocol should therefore include redocking of a known ligand when available, examination of key interacting residues, comparison with reference compounds and validation using complementary computational or experimental approaches [3], [4], [5].

2.2 Quantitative Structure Activity Relationship

Quantitative structure activity relationship (QSAR) modelling establishes relationships between molecular descriptors or learned molecular representations and experimentally measured biological activity or physicochemical properties. The reliability of a QSAR model depends strongly on the quality and diversity of the dataset, appropriate separation of training and test sets, external validation and definition of the applicability domain. A high training set R^2 value or classification accuracy alone does not demonstrate predictive reliability if the model performs poorly when applied to new chemical structures [13], [14].

2.3 Pharmacophore Modelling

Pharmacophore modelling provides an interpretable representation of the structural and chemical features required for biological activity. These features commonly include hydrogen bond donors and acceptors, aromatic regions, hydrophobic groups and ionizable centres. Pharmacophore models can be used to identify molecules containing the essential interaction features required for target recognition and are particularly useful for virtual screening and scaffold hopping [15]. When combined with docking or QSAR, pharmacophore modelling can provide an additional level of confidence during compound prioritization.

2.4 Molecular Dynamics Simulation

Molecular dynamics (MD) simulation complements static docking by examining the behaviour of proteins and ligand protein complexes over time. Frequently analysed parameters include root mean square deviation (RMSD), root mean square fluctuation (RMSF), radius of gyration, hydrogen bond occupancy and persistence of important molecular contacts. MD simulation can help determine whether a docked binding

pose remains stable and can reveal important conformational changes that may not be evident from a single static structure. Nevertheless, the results depend on factors such as force field selection, system preparation, simulation duration and conformational sampling [6]. Free energy calculations may further strengthen compound prioritization, although they require greater computational resources.

2.5 ADMET and Drug Likeness Prediction

Absorption, distribution, metabolism, excretion and toxicity (ADMET) prediction is an important component of early drug discovery because strong target binding alone does not guarantee that a compound will possess acceptable pharmacokinetic or safety properties. Computational tools can provide early estimates of solubility, permeability, metabolic stability, cytochrome P450 interactions, distribution and potential toxicity liabilities. Platforms such as SwissADME and pkCSM demonstrate the usefulness of rapid in silico assessment during compound prioritization [16], [17]. However, predicted ADMET values should be clearly reported as computational estimates and should not be presented as experimentally measured pharmacokinetic or toxicological properties.

Physicochemical guidelines such as the Rule of Five can assist in evaluating oral drug likeness, although such criteria should be considered useful screening guidelines rather than absolute requirements [18]. Compounds containing reactive groups or structures associated with assay interference should also be examined carefully before further development [19]. Together, docking, QSAR, pharmacophore modelling, MD simulation and ADMET prediction provide complementary information that can improve the reliability of candidate selection when used as part of an integrated CADD workflow. A comparative overview of the principal computational approaches used in integrated CADD studies, including their inputs, outputs, strengths, limitations and validation requirements, is presented in **Table 1**.

Table 1. Comparison of major computational approaches used in an integrated CADD study.

Method	Typical input	Main output	Strength	Key limitation	Minimum validation
Docking	Target structure + ligands	Binding pose; relative rank	Fast structure-based screening	Approximate scoring; receptor flexibility	Redocking, reference ligand, interaction inspection
QSAR / ML	Curated compounds + measured endpoints	Activity/property prediction	Learns trends across chemical series	Dataset bias; extrapolation risk	External test set; applicability domain
Pharmacophore	Known ligands and/or complexes	Essential 3D interaction features	Scaffold hopping; interpretable filters	Model depends on input actives/alignment	Decoy/enrichment testing; active-set recovery
MD simulation	Prepared molecular system	Dynamic stability and conformational behaviour	Adds time and flexibility	Force-field and sampling dependence	Replicates, convergence and trajectory metrics
ADMET prediction	Molecular structure	PK/toxicity risk estimates	Early removal of liabilities	Endpoint- and model-dependent uncertainty	Use multiple tools; experimental confirmation
Generative AI	Training data + objectives/constraints	Novel candidate structures	Explores large chemical space; multi-objective design	Validity, novelty, synthesizability, bias	Retrospective benchmarks + prospective synthesis/assays

3. ARTIFICIAL INTELLIGENCE AS AN EXTENSION OF CADD

3.1 AI Applications in Drug Discovery

Artificial intelligence is most useful when it extends the decision-making capacity of established CADD methods rather than being considered a replacement for conventional computational approaches. Current applications include target prioritization using omics and literature data, prediction of protein and molecular

structures, activity and ADMET prediction, retrosynthesis support, image based phenotypic screening and de novo molecular generation [7], [8], [9], [10]. Recent developments have also shifted computational drug discovery from traditional molecular descriptors toward graph neural networks, transformers, diffusion models and other generative architectures that can optimize several molecular properties simultaneously [8], [9], [10].

3.2 Alphafold 3 and Structure Prediction

AlphaFold 3 represents an important development in structure-based drug discovery because it can predict joint structures of biomolecular complexes involving proteins, nucleic acids, small molecules, ions and modified residues [11]. Its ability to model protein ligand interactions can support structural analysis and early drug discovery when experimentally determined structures are unavailable. However, a predicted complex should not be considered equivalent to an experimentally solved structure. Confidence metrics, local binding site quality, possible alternative conformations and available experimental structural information should therefore be evaluated before using predicted structures for medicinal chemistry decisions.

3.3 Generative AI in Molecular Design

Generative molecular design extends conventional virtual screening by allowing computational systems to propose new molecular structures according to predefined objectives. Such models can simultaneously consider predicted potency, selectivity, solubility, metabolic stability and synthetic accessibility. This approach provides an opportunity to explore chemical space beyond compounds already available in screening libraries. However, optimization based on imperfect predictive models can also amplify modelling errors. Generated molecules should therefore be evaluated for chemical validity, novelty, synthetic feasibility, stereochemistry, undesirable structural features, applicability domain and prospective experimental activity [9], [12]. Generative AI should consequently be viewed as a tool for prioritizing and designing candidates that still require rigorous computational and experimental validation.

4. TRANSLATIONAL EXAMPLE: RENTOSERTIB

4.1 AI Enabled Discovery and Preclinical Development

Rentosertib, formerly known as INS018_055 and ISM001-055, is a small molecule inhibitor of TRAF2 and NCK interacting kinase (TNIK) and represents an important example of the clinical translation of AI based drug discovery. An AI supported platform was used to identify TNIK as a potential antifibrotic target, followed by generative molecular design and conventional medicinal chemistry optimization to develop a suitable candidate. Preclinical investigations demonstrated antifibrotic activity in several experimental models, while subsequent Phase I studies provided initial evidence regarding safety, tolerability and pharmacokinetic behaviour. The development programme reported approximately 18 months from target discovery to preclinical candidate nomination [20].

4.2 Phase 2a Clinical Evidence

Further clinical evidence provides a more balanced assessment of the potential of AI enabled drug discovery. In a randomized Phase 2a study published in 2025, 71 patients with idiopathic pulmonary fibrosis were assigned to placebo or one of three rentosertib treatment regimens for 12 weeks. The incidence of treatment emergent adverse events was generally similar across the treatment groups. Among the secondary lung function outcomes, patients receiving 60 mg rentosertib once daily showed a mean change in forced vital capacity of +98.4 mL, compared with -20.3 mL in the placebo group [21]. Although these findings are encouraging, the relatively small number of participants and short treatment duration mean that the efficacy and long-term safety of rentosertib require confirmation in larger clinical studies of longer duration. This example demonstrates the potential of AI to accelerate selected stages of drug discovery while emphasizing that conventional pharmacology, toxicology and controlled clinical evaluation remain essential for establishing therapeutic efficacy and safety.

5. VALIDATION: WHAT MAKES A CADD STUDY SCIENTIFICALLY CONVINCING?

5.1 Importance of Validation in CADD

A major limitation of many in silico studies is not the selection of computational software but the interpretation of the generated results. A favourable docking score, a stable RMSD profile or a predicted low toxicity value should not be considered sufficient evidence of therapeutic efficacy. Reliable CADD studies should combine evidence obtained from complementary computational approaches and clearly distinguish between prediction and experimental confirmation. Each method should be interpreted according to its specific strengths and limitations, while the final selection of promising candidates should be supported by multiple independent criteria.

5.2 Practical Validation Criteria

The major validation requirements for different stages of an integrated CADD workflow are summarized in Table 2. These criteria emphasize appropriate target selection, careful structural preparation, validation of computational protocols, assessment of model reliability and experimental confirmation of prioritized compounds.

Table 2. Practical validation checklist for a submission quality CADD study.

Checkpoint	Minimum information / good practice
Biological target	Explain disease relevance and biological mechanism, and use validated target evidence where possible.
Protein structure	Report the source or PDB identifier, prediction method, structural quality, missing residues, protonation state and binding site definition.
Docking	State the software and version, grid or binding site, ligand preparation and scoring method. Redock a known ligand or benchmark the protocol where possible.
QSAR / AI	Describe data curation, training and test sets, performance metrics, external validation and applicability domain, and minimize analogue or data leakage.
ADMET	Report results as predicted values, compare relevant endpoints using appropriate tools and clearly indicate uncertainty.
Molecular dynamics	Report the force field, solvent system, ions, equilibration procedure, simulation duration and trajectory analyses. Stability alone should not be interpreted as evidence of therapeutic efficacy.
Hit selection	Apply multiple criteria including molecular interactions, predicted potency, ADMET properties, novelty, synthetic feasibility and chemical alerts.
Experimental validation	Confirm prioritized compounds using biochemical or cellular assays and, where appropriate, pharmacokinetic, toxicity and in vivo studies.

6. CURRENT LIMITATIONS AND FUTURE DIRECTIONS

6.1 Current Limitations

Several limitations remain common to both classical and AI assisted CADD approaches. First, biological systems are dynamic and highly context dependent, whereas many computational models simplify protein conformations, solvent effects, cellular environments and disease heterogeneity. Such simplifications may reduce the accuracy of predictions when computational results are transferred to experimental or clinical settings.

Second, the reliability of computational and AI models is strongly influenced by the quality, diversity and coverage of the underlying data. Sparse, biased or poorly curated datasets can produce models that appear accurate during development but perform poorly when applied to new chemical structures [19]. In addition, inappropriate separation of training and test datasets may lead to overly optimistic estimates of model performance.

Third, commonly used computational benchmarks may not accurately represent prospective medicinal chemistry applications, particularly when structurally similar compounds occur in both training and test datasets. Finally, high predicted potency does not necessarily indicate that a compound will become a

successful drug. Poor selectivity, solubility, permeability, metabolic stability, toxicity or synthetic accessibility can still limit further development.

6.2 Future Directions

Future CADD systems are expected to place greater emphasis on Mult objective optimization, uncertainty estimation, active learning and iterative experimental validation. Rather than optimizing only predicted biological activity, future computational platforms may simultaneously consider potency, selectivity, pharmacokinetic properties, toxicity and synthetic feasibility.

Explainable AI is also likely to become increasingly important because medicinal chemists need to understand not only which compounds receive high predictive scores, but also which molecular features and biological evidence contribute to those predictions. Advances in cloud computing and high-performance computing may enable screening of larger chemical libraries and more extensive molecular simulations. At the same time, integration with automated synthesis and experimental assay platforms may shorten the design, synthesis, testing and analysis cycle.

Despite these technological advances, prospective validation using previously untested compounds and clinically relevant biological endpoints will remain essential. The future value of AI assisted CADD will therefore depend not only on increasingly sophisticated computational models but also on their ability to generate reproducible predictions that can be confirmed experimentally. Recent literature further demonstrates the rapid integration of computational screening, AI and experimental validation in contemporary drug discovery [22], [23], [24]. Generative approaches increasingly incorporate deep learning, de novo molecular generation and improved strategies for evaluating chemical validity and synthesizability [25], [26], [27]. AI is also being extended toward multi target design and prospective molecular discovery, with recent studies demonstrating the value of experimentally validated generative workflows [28], [29], [30].

Beyond discovery pipelines, evidence that AI adoption improves operational performance in information-technology-intensive work supports the broader translation of such computational platforms into practice [31]. As AI-assisted discovery systems move into regulated clinical and industrial workflows, the governance of algorithmic agency becomes a deployment consideration for such systems [32].

7. CONCLUSION

Computer aided drug design has become an important component of modern drug discovery by integrating structural biology, cheminformatics, molecular modelling, pharmacokinetic prediction and artificial intelligence. Molecular docking, QSAR, pharmacophore modelling, molecular dynamics simulation and ADMET prediction provide complementary information for identifying and prioritizing promising compounds. However, the predictive value of each method depends on the quality of the input data, model assumptions, validation strategy and applicability to the chemical and biological system being studied. Artificial intelligence extends conventional CADD by improving molecular representation, structure prediction, property prediction and generative molecular design. Nevertheless, AI based predictions should not be considered substitutes for experimental evidence. Computationally prioritized compounds require confirmation through appropriate biochemical, cellular, pharmacokinetic, toxicity and, where necessary, in vivo and clinical studies.

The development of rentosertib illustrates the potential of AI assisted drug discovery to accelerate selected stages of target identification and candidate development, while also demonstrating the continued importance of conventional experimental and clinical validation. Therefore, the most reliable future strategy is an integrated and iterative workflow in which computational predictions guide experiments, experimental results refine computational models and multiple independent methods are used to reduce uncertainty. Continued advances in model validation, explainability, data quality and prospective experimental testing will be essential for translating CADD and AI based predictions into safe and effective therapeutic candidates.

ACKNOWLEDGEMENT

The authors gratefully acknowledge the Research & Development Cell, AV College of Arts & Commerce, PG Center, Hyderabad, India, for providing the academic support and facilities necessary for the preparation of this review article.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

FUNDING

No specific funding was reported for this review.

DATA AVAILABILITY

No new datasets were generated or analysed in this narrative review.

AUTHOR CONTRIBUTIONS

All authors contributed to the conceptualization, literature review, manuscript preparation, critical revision, and approval of the final manuscript.

REFERENCES

- [1] Talevi, A. (2024). Computer-aided drug discovery and design: Recent advances and future prospects. *Methods in Molecular Biology*, 2714, 1–20. https://doi.org/10.1007/978-1-0716-3441-7_1
- [2] Sliwoski, G., Kothiwale, S., Meiler, J., & Lowe, E. W., Jr. (2014). Computational methods in drug discovery. *Pharmacological Reviews*, 66(1), 334–395. <https://doi.org/10.1124/pr.112.007336>
- [3] Ferreira, L. G., dos Santos, R. N., Oliva, G., & Andricopulo, A. D. (2015). Molecular docking and structure-based drug design strategies. *Molecules*, 20(7), 13384–13421. <https://doi.org/10.3390/molecules200713384>
- [4] Pinzi, L., & Rastelli, G. (2019). Molecular docking: Shifting paradigms in drug discovery. *International Journal of Molecular Sciences*, 20(18), 4331. <https://doi.org/10.3390/ijms20184331>
- [5] Trott, O., & Olson, A. J. (2010). AutoDock Vina: Improving the speed and accuracy of docking with a new scoring function, efficient optimization, and multithreading. *Journal of Computational Chemistry*, 31(2), 455–461. <https://doi.org/10.1002/jcc.21334>
- [6] AlRawashdeh, S., & Barakat, K. H. (2024). Applications of molecular dynamics simulations in drug discovery. *Methods in Molecular Biology*, 2714, 127–141. https://doi.org/10.1007/978-1-0716-3441-7_7
- [7] Vamathevan, J., Clark, D., Czodrowski, P., et al. (2019). Applications of machine learning in drug discovery and development. *Nature Reviews Drug Discovery*, 18(6), 463–477. <https://doi.org/10.1038/s41573-019-0024-5>
- [8] Singh, S., Kaur, N., & Gehlot, A. (2024). Application of artificial intelligence in drug design: A review. *Computers in Biology and Medicine*, 179, 108810. <https://doi.org/10.1016/j.combiomed.2024.108810>
- [9] Wang, K., Huang, Y., Wang, Y., You, Q., & Wang, L. (2024). Recent advances from computer-aided drug design to artificial intelligence drug design. *RSC Medicinal Chemistry*, 15(12), 3978–4000. <https://doi.org/10.1039/D4MD00522H>
- [10] Du, Y., Jamasb, A. R., Guo, J., et al. (2024). Machine learning-aided generative molecular design. *Nature Machine Intelligence*, 6, 589–604. <https://doi.org/10.1038/s42256-024-00843-5>
- [11] Abramson, J., Adler, J., Dunger, J., et al. (2024). Accurate structure prediction of biomolecular interactions with AlphaFold 3. *Nature*, 630, 493–500. <https://doi.org/10.1038/s41586-024-07487-w>
- [12] Gangwal, A., Ansari, A., Ahmad, I., Azad, A. K., & Wan Sulaiman, W. M. A. (2024). Current strategies to address data scarcity in artificial intelligence-based drug discovery: A comprehensive review. *Computers in Biology and Medicine*, 179, 108734. <https://doi.org/10.1016/j.combiomed.2024.108734>
- [13] Cherkasov, A., Muratov, E. N., Fourches, D., et al. (2014). QSAR modeling: Where have you been? Where are you going to? *Journal of Medicinal Chemistry*, 57(12), 4977–5010. <https://doi.org/10.1021/jm4004285>
- [14] Tropsha, A. (2010). Best practices for QSAR model development, validation, and exploitation. *Molecular Informatics*, 29(6–7), 476–488. <https://doi.org/10.1002/minf.201000061>
- [15] Yang, S. Y. (2010). Pharmacophore modeling and applications in drug discovery: Challenges and recent advances. *Drug Discovery Today*, 15(11–12), 444–450. <https://doi.org/10.1016/j.drudis.2010.03.013>

- [16] Daina, A., Michielin, O., & Zoete, V. (2017). SwissADME: A free web tool to evaluate pharmacokinetics, drug-likeness and medicinal chemistry friendliness of small molecules. *Scientific Reports*, 7, 42717. <https://doi.org/10.1038/srep42717>
- [17] Pires, D. E. V., Blundell, T. L., & Ascher, D. B. (2015). pkCSM: Predicting small-molecule pharmacokinetic and toxicity properties using graph-based signatures. *Journal of Medicinal Chemistry*, 58(9), 4066–4072. <https://doi.org/10.1021/acs.jmedchem.5b00104>
- [18] Lipinski, C. A., Lombardo, F., Dominy, B. W., & Feeney, P. J. (2001). Experimental and computational approaches to estimate solubility and permeability in drug discovery and development settings. *Advanced Drug Delivery Reviews*, 46(1–3), 3–26. [https://doi.org/10.1016/S0169-409X\(00\)00129-0](https://doi.org/10.1016/S0169-409X(00)00129-0)
- [19] Baell, J. B., & Holloway, G. A. (2010). New substructure filters for removal of pan assay interference compounds (PAINS) from screening libraries and for their exclusion in bioassays. *Journal of Medicinal Chemistry*, 53(7), 2719–2740. <https://doi.org/10.1021/jm901137j>
- [20] Ren, F., Aliper, A., Chen, J., et al. (2025). A small-molecule TNIK inhibitor targets fibrosis in preclinical and clinical models. *Nature Biotechnology*, 43, 63–75. <https://doi.org/10.1038/s41587-024-02143-0>
- [21] Xu, Z., Ren, F., Wang, P., et al. (2025). A generative AI-discovered TNIK inhibitor for idiopathic pulmonary fibrosis: A randomized phase 2a trial. *Nature Medicine*, 31, 2602–2610. <https://doi.org/10.1038/s41591-025-03743-2>
- [22] Sadybekov, A. V., & Katritch, V. (2023). Computational approaches streamlining drug discovery. *Nature*, 616, 673–685. <https://doi.org/10.1038/s41586-023-05905-z>
- [23] Janet, J. P., Mervin, L., & Engkvist, O. (2023). Artificial intelligence in molecular de novo design: Integration with experiment. *Current Opinion in Structural Biology*, 80, 102575. <https://doi.org/10.1016/j.sbi.2023.102575>
- [24] Tropsha, A., Isayev, O., Varnek, A., Schneider, G., & Cherkasov, A. (2024). Integrating QSAR modelling and deep learning in drug discovery: The emergence of deep QSAR. *Nature Reviews Drug Discovery*, 23, 141–155. <https://doi.org/10.1038/s41573-023-00832-0>
- [25] Gangwal, A., & Lavecchia, A. (2024). Unleashing the power of generative AI in drug discovery. *Drug Discovery Today*, 29(6), 103992. <https://doi.org/10.1016/j.drudis.2024.103992>
- [26] Pang, C., Qiao, J., Zeng, X., Zou, Q., & Wei, L. (2024). Deep generative models in de novo drug molecule generation. *Journal of Chemical Information and Modeling*, 64(7), 2174–2194. <https://doi.org/10.1021/acs.jcim.3c01496>
- [27] Gangwal, A., Ansari, A., Ahmad, I., Azad, A. K., Kumarasamy, V., Subramanian, V., & Wong, L. S. (2024). Generative artificial intelligence in drug discovery: Basic framework, recent advances, challenges, and opportunities. *Frontiers in Pharmacology*, 15, 1331062. <https://doi.org/10.3389/fphar.2024.1331062>
- [28] Cichońska, A., Ravikumar, B., & Rahman, R. (2024). AI for targeted polypharmacology: The next frontier in drug discovery. *Current Opinion in Structural Biology*, 84, 102771. <https://doi.org/10.1016/j.sbi.2023.102771>
- [29] Atz, K., Cotos, L., Isert, C., et al. (2024). Prospective de novo drug design with deep interactome learning. *Nature Communications*, 15, 3408. <https://doi.org/10.1038/s41467-024-47613-w>
- [30] Filella-Merce, I., Molina, A., Díaz, L., et al. (2025). Optimizing drug design by merging generative AI with a physics based active learning framework. *Communications Chemistry*, 8, 238. <https://doi.org/10.1038/s42004-025-01635-7>
- [31] Lalitha Kumari, P., & Pandey, J. K. (2021). Impact of artificial intelligence on employees' performance in information technology sector. AIP Conference Proceedings, 2587(1). <https://doi.org/10.1063/5.0150403>
- [32] Pandey, J. K., & Kumar, R. (2025). Governing the algorithmic agent: Confronting overt and covert challenges to justice and the future of work. *International Journal of Law Management & Humanities*, 8(4), 1974–1984. <https://doi.org/10.1000/IJLMH.1110648>