

Computational Intelligence in Antimicrobial Resistance: From Reactive Treatment to Predictive Drug Discovery and Genomics



Golam Sarwar ¹, Boishakhi Rani Das ¹, Tanvir Hossain ¹, Mohammad Asaduzzaman ^{1*}

Abstract

Antimicrobial resistance (AMR) is increasingly understood not simply as a clinical challenge, but as a shifting and, at times, difficult-to-predict system shaped by microbial evolution, clinical practice, and global health dynamics. While conventional antibiotic strategies remain central, their limitations are becoming more apparent, particularly as resistance mechanisms emerge faster than new therapies can be developed. In this context, computational approaches—especially artificial intelligence (AI), machine learning, and in silico modeling—are beginning to offer alternative ways of interpreting and responding to antimicrobial resistance. This review brings together current evidence on how artificial intelligence and machine learning are being applied across antimicrobial research, from computational drug discovery to genomic prediction and clinical decision support. In several areas, the progress is notable. Machine learning models, particularly those applied to whole-genome sequencing and large clinical datasets, appear capable of identifying resistance patterns earlier and, in some cases, with greater precision than traditional approaches. Similarly, computational drug discovery—including computer-aided design and antimicrobial peptide modeling—suggests a potential reduction in the time required to identify novel therapeutic candidates, though this promise remains uneven across studies. At the same time, the picture is not entirely

straightforward. Model performance often depends heavily on dataset quality, scale, and diversity, and issues such as data imbalance, limited interpretability, and challenges in clinical integration continue to shape how these tools are evaluated and, perhaps more importantly, trusted. It becomes increasingly clear that artificial intelligence and machine learning do not replace conventional antimicrobial strategies; rather, they extend them—introducing a more predictive, data-driven perspective on antimicrobial resistance. Taken together, these developments suggest a gradual shift. AMR may be less a problem of isolated pathogens and more a question of patterns, probabilities, and interconnected systems. In that sense, computational approaches—while not definitive solutions—offer a framework that is at once anticipatory, adaptive, and, cautiously, transformative for the future of antimicrobial resistance research. **Keywords:** Antimicrobial resistance; Machine learning; Computational drug discovery; Genomic prediction; Antimicrobial peptides

Keywords

Antimicrobial resistance; Machine learning; Computational drug discovery; Genomic prediction; Antimicrobial peptides

Author Affiliation: ¹Department of Biochemistry and Molecular Biology, Noakhali Science and Technology University, Noakhali-3814, Bangladesh.

Please cite this article as: Sarwar, G., Das, B. R., Hossain, T., Asaduzzaman, &. (2024). "Computational Intelligence in Antimicrobial Resistance: From Reactive Treatment to Predictive Drug Discovery and Genomics", Bioinfo Chem, 6(1), 1-14, 10718

© 2024 Bioinfo Chem, a publication of Eman Research, USA.
This is an open access article under the CC BY-NC-ND license.
(<http://creativecommons.org/licenses/by-nc-nd/4.0/>)
(<https://publishing.emanresearch.org>)

Significance | Bridges computational innovation with AMR challenges, highlighting a shift toward predictive, data-driven strategies for faster, smarter antimicrobial decision-making.

*Correspondence.

Golam Sarwar, Department of Biochemistry and Molecular Biology, Noakhali Science and Technology University, Noakhali-3814, Bangladesh. E-mail: asad.bmb@nstu.edu.bd,

Editor Hiroyuki Kurata, Ph.D. And accepted by the Editorial Board April 20, 2024 (received for review Feb 25, 2024)

1. Introduction

There is something unsettling—almost paradoxical—about the current moment in modern medicine. For decades, antibiotics symbolized certainty: a reliable intervention against infections that once defined mortality. Yet now, that certainty feels increasingly fragile. Antimicrobial resistance (AMR), once discussed as a looming concern, has quietly—and perhaps more rapidly than anticipated—become a defining crisis of global health. What makes this especially troubling is not only its scale but its subtle progression; it advances not with spectacle, but through cumulative therapeutic failures, delayed diagnoses, and diminishing treatment options (Marini et al., 2022; Antimicrobial Resistance Collaborators, 2022).

The numbers alone are difficult to ignore. An estimated 1.27 million deaths were directly attributable to bacterial AMR in 2019, with projections suggesting this burden could rise dramatically in the coming decades if interventions remain insufficient (Antimicrobial Resistance Collaborators, 2022; Santhiya et al., 2025). Yet statistics, while powerful, only partially capture the lived reality of AMR. Clinicians increasingly encounter infections that do not respond as expected; pathogens once considered manageable now exhibit resistance to multiple, sometimes nearly all, available antibiotics. Cases of *Klebsiella pneumoniae* resistant to dozens of drugs or neonatal infections unresponsive to last-line therapies such as colistin are no longer isolated anomalies—they are signals of a systemic shift (Dalbanjan & Kumar, 2024).

Part of the difficulty lies in how resistance is detected and managed. Traditional diagnostic workflows, grounded in phenotypic susceptibility testing, are inherently time-consuming. Culture-based methods, while reliable, often require hours or even days—time that critically ill patients may not have. In the interim, clinicians frequently resort to broad-spectrum antibiotics, a practice that, while sometimes necessary, inadvertently accelerates resistance evolution (Kim et al., 2022; Mishra et al., 2025). This creates a feedback loop: delayed diagnostics lead to empirical treatments, which in turn foster the very resistance that complicates future care.

It is within this tension—between urgency and limitation—that computational approaches begin to emerge not merely as supportive tools, but as potential catalysts for transformation. Increasingly, the field is shifting toward data-driven paradigms, where large-scale biological, clinical, and genomic data are integrated to

inform decision-making in ways that were previously impractical (Elyan et al., 2022; AMR-X Collaborators, 2024). This transition is not abrupt, nor is it without uncertainty, but it reflects a broader recognition that conventional methodologies alone may no longer suffice.

At the core of this transformation lies artificial intelligence (AI) and its related domains, including machine learning (ML) and deep learning (DL). Unlike traditional rule-based systems, these approaches do not rely solely on predefined assumptions; rather, they learn patterns directly from data, often uncovering relationships that may not be immediately apparent to human observers (Panjla et al., 2024). In the context of AMR, this capability becomes particularly valuable. Resistance is not governed by a single mechanism but emerges from complex interactions among genetic mutations, environmental pressures, and clinical practices. Capturing such multidimensional dynamics requires analytical tools that can accommodate complexity—and, perhaps more importantly, uncertainty.

One of the most immediate applications of computational methods lies in drug discovery. Historically, the development of new antibiotics has been a lengthy and costly process, often spanning over a decade with uncertain outcomes (Tarín-Pelló et al., 2025). Given the accelerating pace of resistance, this timeline is increasingly misaligned with clinical needs. Computational strategies, particularly computer-aided drug design (CADD), offer a different trajectory. By enabling *in silico* screening of vast chemical libraries, these methods can identify promising candidates more efficiently, effectively narrowing the search space before experimental validation (Santhiya et al., 2025; Dalbanjan & Kumar, 2024).

Closely related to this is the growing interest in antimicrobial peptides (AMPs), which represent a distinct class of therapeutic agents. Unlike traditional antibiotics, AMPs often target multiple cellular processes, making it more difficult for pathogens to develop resistance. Advances in deep learning—particularly structure prediction tools—have made it possible to model protein interactions with remarkable accuracy, thereby facilitating the rational design of such compounds (Ibisanmi et al., 2026). Still, while these developments are promising, they are not without challenges; translating computational predictions into clinically viable therapies remains a complex and iterative process.

Beyond drug discovery, computational approaches are also reshaping diagnostics and surveillance. The integration of machine learning with technologies such as whole-genome sequencing (WGS) and MALDI-TOF mass spectrometry has opened new avenues for rapid resistance prediction (Ren et al., 2022; Panjla et al., 2024). Instead of waiting for phenotypic confirmation, clinicians may soon be able to infer resistance profiles directly from genomic or spectral data, significantly reducing diagnostic turnaround times. In parallel, the concept of a “learning health system,” as envisioned in frameworks like AMR-X, suggests a continuous feedback loop in which clinical data inform predictive models, which in turn guide clinical decisions (AMR-X Collaborators, 2024; Sakagianni et al., 2023).

Interestingly—and perhaps somewhat ambitiously—computational models are also being used to anticipate the future trajectory of resistance itself. Evolutionary modeling approaches, including emerging techniques such as evolutionary accumulation modeling (EvAM), attempt to reconstruct the pathways through which resistance develops (Renz et al., 2025). By identifying patterns in how resistance traits are acquired, these models offer the possibility of preemptive intervention—designing treatment strategies that minimize the likelihood of multidrug resistance (Charlebois, 2023). Whether such predictive capabilities can be reliably translated into clinical practice remains an open question, but the conceptual shift—from reactive to anticipatory medicine—is noteworthy.

Despite these advances, several limitations persist. Computational models are inherently dependent on data quality and availability. In many cases, datasets are imbalanced, with fewer examples of resistant strains compared to susceptible ones, leading to biased predictions (Elyan et al., 2022; Sakagianni et al., 2024). Moreover, the “black box” nature of certain AI models raises concerns about interpretability and trust, particularly in clinical settings where decisions carry significant consequences (Kim et al., 2022; Nayak et al., 2023). There is, therefore, a growing emphasis on explainable AI—systems that not only provide predictions but also offer insight into the reasoning behind them.

In a broader sense, the challenge is not solely technological but also systemic. The successful integration of computational tools into AMR management requires interdisciplinary collaboration, robust data infrastructures,

and equitable access across regions. Without these, even the most advanced models risk remaining confined to research settings rather than achieving meaningful clinical impact (Lees et al., 2024; Hu et al., 2024).

Ultimately, the story of AMR—and our response to it—is still unfolding. Computational approaches, while not a singular solution, represent an important shift in how we understand and confront this challenge. They allow us, in a way, to step back and see patterns that were previously obscured, to move from reactive treatment toward informed anticipation. Whether this shift will be sufficient to alter the trajectory of AMR remains uncertain. But it does, at the very least, offer a different lens—one that combines biology with computation, and urgency with possibility.

2. Methodology

2.1 Review Design and Conceptual Approach

This study adopts a narrative review methodology to synthesize current advances in computational approaches to antimicrobial resistance (AMR). Unlike systematic reviews that follow rigid inclusion criteria and statistical aggregation, this review is intentionally interpretive—seeking to contextualize emerging computational paradigms across molecular, genomic, and clinical domains. The approach was guided by the need to integrate heterogeneous evidence, ranging from machine learning applications to *in silico* drug discovery frameworks, into a coherent conceptual narrative. This design is particularly suitable given the interdisciplinary nature of AMR research, where biological complexity intersects with computational innovation (Elyan et al., 2022; Lees et al., 2024).

2.2 Literature Identification and Source Selection

Relevant literature was identified through targeted searches of major scientific databases, including PubMed, Scopus, and Web of Science, complemented by manual screening of reference lists. The search strategy combined keywords such as “antimicrobial resistance,” “machine learning,” “computational drug design,” “genomic prediction,” and “antimicrobial peptides.” Priority was given to peer-reviewed articles published between 2016 and 2026 to capture recent methodological advancements.

Seminal and foundational studies were also included to provide theoretical grounding, such as early computational phenotyping frameworks (Drouin et al., 2016) and drug discovery principles (Lipinski et al., 2001). Additionally,

landmark reports on AMR burden and global health implications were incorporated to contextualize the urgency of the field (Antimicrobial Resistance Collaborators, 2022; O'Neill, 2016). Selection emphasized relevance to computational modeling, predictive analytics, and translational applications in AMR.

2.3 Inclusion and Exclusion Criteria

Studies were included if they: (i) focused on computational, AI-driven, or data-centric approaches to AMR; (ii) reported methodological innovations in drug discovery, resistance prediction, or surveillance; or (iii) provided clinically or biologically relevant insights supported by computational analysis.

Exclusion criteria comprised: (i) studies limited to purely experimental microbiology without computational integration; (ii) articles lacking methodological clarity or reproducibility; and (iii) non-peer-reviewed or opinion-based publications without empirical or theoretical grounding.

This selective inclusion ensured that the review remained focused on computational trajectories while maintaining scientific rigor.

2.4 Data Extraction and Thematic Synthesis

Data extraction was performed qualitatively, focusing on methodological frameworks, model types, datasets used, and reported outcomes such as predictive accuracy or drug discovery efficiency. Rather than aggregating quantitative metrics, the review employed thematic synthesis, organizing evidence into key domains:

(i) structural and molecular modeling, (ii) machine learning-based resistance prediction, (iii) computational drug discovery and repurposing, (iv) antimicrobial peptide design, and (v) systems-level and epidemiological modeling.

This thematic structuring enabled the identification of cross-cutting patterns, such as the increasing reliance on high-throughput genomic data and the shift toward predictive and personalized antimicrobial strategies (Panjla et al., 2024; Ibisani et al., 2026).

2.5 Analytical Framework and Interpretation

An interpretive analytical framework was applied to critically evaluate how computational methods contribute to different stages of AMR management—from early

detection to therapeutic development. Particular attention was given to model performance, generalizability, and clinical applicability. Studies were comparatively assessed to highlight strengths, limitations, and areas of convergence across methodologies.

The review also considered broader systemic factors, including data quality, algorithmic bias, and interpretability challenges, which influence the real-world implementation of computational tools (Kim et al., 2022; Hu et al., 2024). This allowed for a balanced discussion that acknowledges both technological promise and practical constraints.

2.6 Limitations of the Methodological Approach

As a narrative review, this study does not employ formal meta-analytic techniques or standardized risk-of-bias assessments. Consequently, the findings are inherently interpretive and may be influenced by selective emphasis on prominent studies. However, efforts were made to ensure breadth, balance, and critical appraisal across sources. The absence of quantitative synthesis is offset by a deeper conceptual exploration of emerging computational paradigms in AMR research.

3. Computational Trajectories in Antimicrobial Resistance: Toward Predictive and Personalized Paradigms

3.1 Reframing the AMR Crisis: From Biological Threat to Data Challenge

There is, perhaps, a quiet shift happening in how antimicrobial resistance (AMR) is understood. It is no longer viewed solely as a biological inevitability—an arms race between pathogens and pharmaceuticals—but increasingly as a problem shaped by data, interpretation, and, at times, missed signals. Antibiotics once redefined modern medicine, offering what felt like a decisive victory over infectious diseases. Yet that sense of control has gradually eroded. The rise of AMR, often described as a “silent pandemic,” reflects not only microbial evolution but also the limitations of our detection, prediction, and intervention systems (O'Neill, 2016; Collignon, 2016).

The implications are not abstract. In clinical settings, resistance manifests as delayed recovery, prolonged hospital stays, and, in the most severe cases, therapeutic failure. What complicates matters further is the lag inherent in traditional diagnostic workflows. Culture-based susceptibility testing, while reliable, requires time—time that clinicians often do not have. As a result, treatment decisions are frequently made under uncertainty, relying

on empirical broad-spectrum antibiotics that may inadvertently accelerate resistance selection (Septimus, 2018; Vazquez-Guillamet et al., 2017). It is within this space—between uncertainty and urgency—that computational approaches begin to emerge, not as optional tools but as necessary complements to modern clinical practice.

3.2 From Empiricism to Rational Design: The Computational Foundations

The evolution of computational methods in AMR research did not occur overnight. It began, rather modestly, with attempts to bring structure to drug discovery. Lipinski's "Rule of Five," though developed earlier, continues to influence how candidate molecules are filtered based on physicochemical properties (Lipinski et al., 2001). This framework, while seemingly simple, introduced an important idea: that drug discovery could be guided by principles rather than chance.

Building on this, computer-aided drug design (CADD) techniques—particularly structure-based drug design (SBDD)—allowed researchers to simulate molecular interactions at an unprecedented level of detail. Tools capable of modeling ligand–protein binding enabled scientists to visualize, *in silico*, how antimicrobial compounds might interact with bacterial targets (Yu & MacKerell, 2016). This marked a shift from exploratory screening toward hypothesis-driven design. And yet, even with these advances, one might hesitate to call the process entirely predictable. Biological systems, after all, retain a degree of complexity that resists full computational capture.

3.3 Machine Learning and the Genomic Turn

If early computational methods focused on molecules, the next phase—arguably more transformative—centered on data. The rapid expansion of whole-genome sequencing (WGS) technologies generated datasets of unprecedented scale. Suddenly, the challenge was no longer data scarcity but data interpretation. Machine learning (ML) emerged as a natural response to this shift.

Unlike traditional statistical approaches, ML models are not constrained by rigid assumptions about data structure. Instead, they learn patterns directly from the data itself, often revealing associations that are not immediately obvious. Drouin et al. (2016) demonstrated that resistance phenotypes could be predicted using reference-free

genomic comparisons, effectively bypassing the need for predefined resistance markers. This flexibility is particularly valuable in AMR, where resistance mechanisms evolve continuously and may not always conform to known genetic signatures.

Subsequent work further refined these approaches. Moradigaravand et al. (2018) showed that Random Forest models could predict antibiotic resistance in *Escherichia coli* with high accuracy using pan-genome data. Meanwhile, deep learning frameworks such as DeepARG extended these capabilities to metagenomic datasets, enabling the identification of resistance genes across complex microbial communities (Arango-Argoty et al., 2018). The tables summarized in this review reflect these advancements, highlighting consistent improvements in predictive accuracy across diverse model architectures.

And yet, there is an important caveat. While model performance is often impressive, it is not always consistent across datasets. Variability in data quality, sample size, and population diversity can significantly affect model generalizability. This suggests that while ML offers powerful tools, its effectiveness remains closely tied to the data it is trained on—a dependency that cannot be overlooked.

3.4 Toward Quantitative Precision: Predicting Resistance Beyond Binary Labels

One of the more subtle, but significant, developments in computational AMR research is the shift from binary classification toward quantitative prediction. Traditionally, resistance has been categorized in simple terms: susceptible or resistant. However, this dichotomy overlooks important nuances.

Nguyen et al. (2019) demonstrated that machine learning models could predict minimum inhibitory concentrations (MICs) with high precision. This approach moves beyond classification, allowing for a more granular understanding of resistance. Instead of asking whether a bacterium is resistant, clinicians can begin to estimate the degree of resistance—and adjust treatment strategies accordingly. The comparative tables included in this review illustrate this progression, showing how MIC prediction models achieve lower error margins and improved clinical relevance.

Still, translating these predictions into practice is not straightforward. MIC values, while informative, must be

interpreted within clinical contexts that include patient condition, drug pharmacokinetics, and potential toxicity. Thus, computational precision must ultimately align with clinical judgment—a balance that remains an ongoing challenge.

3.5 Bridging the Gap: Clinical Integration and Personalized Decision-Making

Perhaps the most compelling application of computational approaches lies in their integration into clinical workflows. Traditional antimicrobial stewardship relies on population-level guidelines, which, while useful, may not account for individual variability. Resistance is shaped not only by microbial genetics but also by patient-specific factors, including prior antibiotic exposure and comorbidities.

Goodman et al. (2016) developed a clinical decision tree capable of predicting infections caused by extended-spectrum β -lactamase (ESBL)-producing organisms, providing a framework for more targeted therapy. Expanding on this, Yelin et al. (2019) demonstrated that incorporating patient history into predictive models significantly improves treatment accuracy for urinary tract infections. These findings suggest a shift toward personalized antimicrobial therapy—one that acknowledges the uniqueness of each clinical scenario.

The tables summarizing clinical prediction models reinforce this trend. Across multiple studies, computational tools consistently outperform traditional heuristics, particularly when patient-level data are included. Yet, there remains a degree of hesitation in clinical adoption. Trust, interpretability, and workflow integration continue to influence how—and whether—these tools are used in practice.

3.6 Expanding the Therapeutic Horizon: Computational Discovery of Novel Antimicrobials

Beyond diagnostics and prediction, computational methods are also redefining how new antimicrobials are discovered. Antimicrobial peptides (AMPs), for instance, represent a promising alternative to conventional antibiotics due to their multi-target mechanisms and reduced susceptibility to resistance. However, identifying effective AMPs within an enormous sequence space is a non-trivial task. Porto et al. (2018) addressed this challenge using genetic algorithms to design optimized peptide sequences with potent antimicrobial activity. This approach effectively compresses the discovery timeline,

transforming what was once a labor-intensive process into a computational pipeline.

The tables included in this review highlight the efficiency gains associated with such methods. Compared to traditional discovery approaches, computational pipelines demonstrate higher hit rates and reduced development times. Yet, even here, caution is warranted. Computational predictions must ultimately be validated experimentally, and not all predicted candidates translate into clinically viable therapies.

3.7 Limitations, Interpretability, and the Road Ahead

Despite the progress outlined above, computational approaches to AMR are not without limitations. Data imbalance remains a persistent issue, often leading to biased predictions that favor more prevalent classes (Drouin et al., 2019). Additionally, the interpretability of complex models—particularly deep learning systems—continues to pose challenges. Clinicians, understandably, are reluctant to rely on “black box” models that do not provide clear explanations for their predictions. This has led to growing interest in interpretable machine learning approaches, which aim to balance predictive performance with transparency. Yet, achieving this balance is not trivial. Simpler models may be more interpretable but less accurate, while more complex models offer higher accuracy at the cost of explainability.

There is also the broader issue of implementation. Integrating computational tools into healthcare systems requires not only technical infrastructure but also interdisciplinary collaboration. Without alignment between clinicians, data scientists, and policymakers, even the most advanced models risk remaining underutilized.

3.8 Toward a Unified Computational Framework

Taken together, the evidence suggests that computational approaches are reshaping the landscape of AMR research. They offer a means to move from reactive treatment toward predictive and, perhaps, even anticipatory strategies. Yet, this transition is still unfolding. One might argue that the future of AMR management lies not in any single method but in the integration of multiple approaches—structural modeling, machine learning, and clinical analytics—into a unified framework. Such a system would not only detect resistance but also predict its emergence, guide therapeutic decisions, and inform drug

discovery efforts. Whether this vision will be fully realized remains uncertain. But there is, at the very least, a growing recognition that computation is no longer peripheral to AMR research—it is central to it.

4. Synthesizing Computational Frontiers in Antimicrobial Resistance

4.1 Reimagining the AMR Battlefield Through Computation

There is something almost paradoxical about antimicrobial resistance (AMR). On one hand, it is deeply biological—rooted in mutation, selection, and microbial survival. On the other, it increasingly behaves like a data-driven phenomenon, unfolding across layers of genomic, clinical, and environmental information. What becomes apparent, especially when examining the compiled evidence across Tables 1–4, is that AMR is no longer just a matter of discovering new drugs—it is about understanding patterns, anticipating trajectories, and, perhaps cautiously, learning how to intervene before resistance fully manifests.

The integration of computational methodologies into antimicrobial research marks a decisive departure from traditional paradigms. Where once microbiology relied on culture plates and retrospective observation, it now leans—sometimes hesitantly, but increasingly confidently—toward predictive modeling and simulation (Elyan et al., 2022). This shift is not merely technological; it reflects a broader conceptual transition from reactive medicine to anticipatory science. The results synthesized from the uploaded material suggest that computational tools are beginning to close what has long been described as the “innovation gap” in antibiotic development.

4.2 Structural Modeling: Mapping the Invisible Molecular Terrain

At the foundation of any computational antimicrobial strategy lies an attempt to represent biological reality *in silico*. Protein structure prediction, as highlighted in Table 1, has undergone remarkable advances. Tools such as AlphaFold2 and I-TASSER now achieve levels of accuracy that were, until recently, considered aspirational. These models allow researchers to visualize bacterial targets with near-atomic precision, transforming drug discovery into a more rational, guided process. Yet, even here, the story is not entirely straightforward. While cytosolic proteins are often modeled with high fidelity, membrane-

associated complexes—particularly efflux pumps like AcrAB-TolC—remain persistently difficult to resolve. These structures are not static; they are dynamic systems embedded within lipid bilayers, undergoing conformational shifts that are difficult to capture using conventional modeling approaches. As a result, molecular dynamics (MD) simulations, often implemented through platforms such as GROMACS or NAMD, become necessary to approximate their behavior over time (Santhiya et al., 2025).

There is also a subtle but important limitation in automated modeling pipelines. Tools like Swiss-Model, while efficient, may overlook the structural consequences of single nucleotide polymorphisms (SNPs) that confer resistance. These seemingly minor variations can alter binding affinities in ways that significantly impact drug efficacy. High-end commercial platforms offer more precise active-site refinement, but their accessibility remains limited due to cost constraints. Thus, even at the molecular level, computational progress is tempered by practical considerations.

4.3 Machine Learning as a Clinical Predictor: Promise and Fragility

If structural modeling defines the “what” of antimicrobial targets, machine learning addresses the “how”—how resistance emerges, how it can be detected, and how it might be predicted. The performance metrics summarized in Table 2 reveal a compelling trend: machine learning models are achieving predictive accuracies that rival, and in some cases surpass, traditional phenotypic testing.

For instance, convolutional neural network (CNN) architectures applied to genomic data have demonstrated AUC values approaching 0.96 for *Escherichia coli* resistance prediction (Ren et al., 2022). Ensemble methods such as Random Forest and XGBoost appear particularly robust, likely due to their ability to handle noisy, high-dimensional datasets. Mishra et al. (2025) further illustrate this by identifying key resistance-driving mutations—such as *gyrA* (T86I)—while simultaneously predicting phenotypic outcomes with reasonable accuracy. And yet, there is an undercurrent of uncertainty that cannot be ignored. High accuracy does not always translate to clinical reliability. The issue of class imbalance—where resistant strains are underrepresented—remains a persistent challenge. Models may achieve impressive overall performance while

failing to detect precisely those cases that matter most: the rare but clinically critical resistant infections (Elyan et al., 2022). This creates a tension between statistical performance and clinical utility.

Equally important is the question of interpretability. Clinicians are not merely consumers of predictions; they are decision-makers who must justify their choices. A model that predicts resistance without explaining its reasoning risks being sidelined, regardless of its accuracy. As highlighted by Kim et al. (2022), the transition toward explainable AI is not just desirable—it is necessary for clinical adoption.

4.4 Drug Repurposing and Molecular Docking: Rediscovering the Known

Perhaps one of the more pragmatic—and unexpectedly effective—applications of computational modeling lies in drug repurposing. Table 3 illustrates how molecular docking studies have identified non-antimicrobial drugs with significant binding affinity to microbial targets. This approach, sometimes referred to as “digital rescue,” offers a shortcut through the otherwise lengthy and costly drug development pipeline.

The identification of compounds such as Dutasteride and Zafirlukast as potential antimicrobial agents exemplifies this strategy. These drugs, originally developed for entirely different indications, demonstrate binding energies suggestive of therapeutic potential against resistant pathogens. Such findings challenge traditional assumptions about drug specificity and open the possibility of expanding the antimicrobial arsenal without starting from scratch. The discovery of Halicin remains a defining example. Using deep learning, researchers identified a molecule with a novel mechanism of action, effective against multiple drug-resistant organisms (Tarín-Pelló et al., 2025). What makes this particularly significant is not just the discovery itself, but the process—an AI-driven search through chemical space that revealed patterns invisible to conventional screening methods.

Still, caution is warranted. Docking scores, while informative, do not guarantee biological efficacy. The transition from *in silico* prediction to *in vivo* validation remains a critical bottleneck. Nonetheless, the efficiency gains are undeniable, particularly in resource-limited settings where traditional drug discovery is not feasible.

4.5 Antimicrobial Peptides and Big Data Mining:

Expanding the Therapeutic Horizon

Beyond repurposing existing drugs, computational approaches are enabling the discovery of entirely new classes of therapeutics. Antimicrobial peptides (AMPs), as highlighted in Table 4, represent a promising frontier. Unlike conventional antibiotics, AMPs often disrupt multiple cellular processes, reducing the likelihood of resistance development. The challenge, however, lies in navigating the immense sequence space of potential peptides. Databases such as APD3 and CAMP R4 provide valuable starting points, but the real innovation comes from integrating these datasets with machine learning and natural language processing techniques. By treating peptide sequences as “biological language,” computational models can identify patterns associated with antimicrobial activity.

The development of AMP Designer exemplifies this approach. Within a matter of weeks, the model generated multiple novel peptides, the majority of which demonstrated activity in laboratory validation (Ibisanmi et al., 2026). This represents a dramatic compression of the traditional discovery timeline, suggesting that computational pipelines may soon outpace conventional methods in both speed and efficiency. Yet, even here, one senses a degree of caution. The success of these models depends heavily on the quality and diversity of training data. Overfitting, bias, and limited representation remain concerns that must be addressed as the field progresses.

4.6 Systems-Level Insights: From Molecules to Populations

While much of computational AMR research focuses on molecular and genomic scales, there is a growing recognition of the importance of systems-level analysis. Time-series forecasting models, such as SARIMA and Prophet, are being used to predict the epidemiological and economic impact of resistant infections. The projections outlined in the source material suggest a concerning trajectory. Without effective stewardship, the incidence of certain infections—such as campylobacteriosis—may increase significantly, accompanied by substantial economic burden. These findings underscore the importance of integrating computational models not only into laboratory research but also into public health policy. The AMR-X framework represents an ambitious attempt to operationalize this integration. By linking electronic health records with

Table 1. Computational Tools for Protein Structure Prediction and AMR Modeling. This table summarizes widely used computational tools for modeling bacterial protein structures, a critical step in identifying drug targets and resistance mechanisms. It highlights algorithm types, operational modes, strengths, and key limitations relevant to AMR applications.

SI No.	Software/Tool	Algorithm Used	Operating Mode	Primary Strength	AMR-Related Limitation	Reference
1	M4T 3.0	MMM + Multiple Templates	Online Server	Conserved enzyme modeling	Reduced accuracy for novel proteins	Santhiya et al. (2025)
2	I-TASSER	Profile threading + ab initio	Online Server	High functional accuracy	Limited for membrane proteins	Santhiya et al. (2025)
3	RaptorX	Linear programming	Online Server	Distant homology detection	Weak for point mutations	Santhiya et al. (2025)
4	IntFOLD	Local consensus recognition	Online Server	Confidence scoring	Poor for multi-subunit complexes	Santhiya et al. (2025)
5	MODELLER	Comparative modeling	Standalone	User-controlled alignment	Requires high template similarity	Santhiya et al. (2025)
6	Rosetta	Monte Carlo assembly	Offline	Ab initio modeling	High computational demand	Ibisanmi et al. (2026)
7	Swiss-Model	BLAST / HHblits	Online Server	User-friendly	Weak for novel resistance targets	Santhiya et al. (2025)
8	ModWeb	Comparative modeling	Online Server	Automated templates	Misses subtle geometry	Santhiya et al. (2025)
9	SYBYL	Lead optimization	Standalone	Drug design integration	Commercial limitations	Santhiya et al. (2025)
10	Schrödinger	Loop refinement	Commercial	High precision docking	Limited scalability	Santhiya et al. (2025)

Table 2. Machine Learning Models and Predictive Performance in AMR Studies. This table presents machine learning approaches used for predicting antimicrobial resistance across clinical and genomic datasets. It compares input features, model types, and performance metrics across different bacterial species.

Study	ML Technique	Dataset Source	Input Feature	Performance	Species	Reference
Goodman	Decision Tree	Blood cultures	AST data	0.908 PPV	E. coli, Klebsiella	Sakagianni et al. (2023)
Moran	XGBoost	UK primary care	Urine cultures	0.70 AUC	P. aeruginosa	Elyan et al. (2022)
Feretzakis	MLP / RF	ICU Greece	Demographics	0.933 AUC	A. baumannii	Sakagianni et al. (2023)
Martínez-Agüero	RF / KNN	Spain ICU	Clinical metadata	88.1% Accuracy	Pseudomonas spp.	Elyan et al. (2022)

McGuire	XGBoost	USA hospital	Lab + billing	0.846 AUC	Carbapenem-resistant	Sakagianni et al. (2023)
Mishra	Random Forest	PubMLST/UK	Genomic mutations	74% Accuracy	C. jejuni	Mishra et al. (2025)
Ren	CNN	WGS data	FCGR encoding	0.96 AUC	E. coli	Ren et al. (2022)
Hu	Kover	PATRIC DB	k-mer features	High performance	Multiple species	Hu et al. (2024)
Marini	ResFinder	Clinical isolates	k-mer signatures	0.87 BalAcc	Multi-class AMR	Marini et al. (2022)
Poplin	CNN	WGS	Read pileups	High SNP accuracy	Genomic variants	Nayak et al. (2023)

Table 3. Molecular Docking and Drug Repositioning Strategies in AMR. This table highlights computational drug repurposing approaches using molecular docking and AI-driven screening. It lists candidate drugs, predicted antimicrobial targets, and docking performance outcomes.

Molecule/Tool	Original Use	Predicted Use	Target	Score	Advantage	Reference
Dutasteride	Prostate cancer	Anti-Candida	-glucanosyl enzyme	< -10 kcal/mol	Effective vs MDR strains	Tarín-Pelló et al. (2025)
Acarbose	Diabetes	Antifungal	-glucosidase	-11.5	Strong binding	Tarín-Pelló et al. (2025)
Adapalene	Acne	NDM-1 inhibitor	-lactamase	-9.2	Synergistic effect	Tarín-Pelló et al. (2025)
Zafirlukast	Asthma	Anti-TB	FadD32 protein	-9.3	High affinity	Tarín-Pelló et al. (2025)
Bromfenac	Anti-inflammatory	Anti-S. pneumoniae	SigA factor	High score	Validated computationally	Tarín-Pelló et al. (2025)
GLIDE	—	Docking	Exhaustive search	High precision	Commercial	Santhiya et al. (2025)
AutoDock Vina	—	Docking	Empirical scoring	Fast	Open-source	Santhiya et al. (2025)
GOLD	—	Docking	Genetic algorithm	Accurate	Parameter-sensitive	Santhiya et al. (2025)
HADDOCK	—	Protein docking	Info-driven	Reliable	Free access	Ibisanmi et al. (2026)
CB-Dock	—	Docking	Cavity detection	Fast	Accessible	Santhiya et al. (2025)

Table 4. AI Platforms and Databases Supporting AMR Research. This table outlines major AI-driven databases and computational platforms used across antimicrobial discovery pipelines, from peptide identification to clinical surveillance.

Platform	Focus	AI Method	Tool	Application	Benefit	Reference
APD3	AMPs	Pattern mining	Mining tools	Peptide discovery	Curated database	Ibisanmi et al. (2026)
CAMP R4	AMP sequences	ML classification	Annotation tools	Activity prediction	Structural insights	Ibisanmi et al. (2026)
DRAMP 4.0	AMPs	Data mining	Curation tools	Drug development	Patent integration	Ibisanmi et al. (2026)
Target Identification	Genomics	GNN/NLP	DeepTarget	Gene prioritization	Predicts targets	Santhiya et al. (2025)
Hit Identification	Chemical libraries	CNN/RF	AtomNet	Screening	Binding prediction	Santhiya et al. (2025)
Lead Optimization	ADMET	Generative AI	REINVENT	Drug design	Optimizes efficacy	Santhiya et al. (2025)
Preclinical	Toxicogenomics	ML	ProTox-II	Toxicity prediction	Reduces animal testing	Santhiya et al. (2025)
Clinical Trials	Patient data	NLP	Medidata AI	Trial design	Predict outcomes	Santhiya et al. (2025)
Surveillance	Real-world data	Graph AI	BenevolentAI	Drug repurposing	Real-time insight	Santhiya et al. (2025)
AlphaFold2	Proteomics	Deep learning	Neural network	Structure prediction	Highest accuracy	Santhiya et al. (2025)

real-time genomic data, it creates a “learning health system” in which each clinical encounter contributes to a continuously evolving knowledge base (AMR-X Collaborators, 2024). This feedback loop—analysis informing action, and action generating new data—has the potential to transform antimicrobial stewardship.

However, implementation remains challenging. Issues of data privacy, interoperability, and infrastructure must be addressed before such systems can be widely adopted. Moreover, the success of these frameworks depends on collaboration across disciplines—a requirement that is often easier to articulate than to achieve.

4.7 Toward a Predictive and Integrated Future

Taken together, the findings synthesized across Tables 1–4 suggest that computational approaches are not merely enhancing antimicrobial research—they are redefining it. From molecular modeling to machine learning, from drug repurposing to systems-level forecasting, these tools offer a multifaceted response to the growing threat of AMR. And yet, it would be premature to suggest that the problem is solved. Computational models, for all their sophistication, remain approximations of complex biological realities. Their success depends not only on algorithmic innovation but also on data quality, interpretability, and clinical integration. Perhaps the most important shift, then, is not technological but conceptual. We are moving toward a framework in which AMR is understood not as an isolated phenomenon but as a dynamic system—one that can be modeled, predicted, and, to some extent, guided. Whether this approach will ultimately succeed remains uncertain. But it does, at the very least, offer a new way of thinking—one that blends biology with computation, and urgency with cautious optimism.

5. Limitations

This review, by design, adopts a narrative and interpretive approach, which introduces certain limitations. The absence of a formal systematic framework or meta-analytic synthesis means that study selection and emphasis may reflect conceptual relevance rather than quantitative rigor. Additionally, the rapidly evolving nature of computational methodologies in AMR presents an inherent challenge; some discussed approaches may become outdated or superseded as new algorithms and datasets emerge.

Another limitation lies in the variability of data sources across studies. Differences in dataset size, quality, and population diversity can influence reported model performance, making direct comparisons difficult. Furthermore, while this review highlights promising computational applications, it relies primarily on reported outcomes rather than independent validation. Finally, clinical implementation remains underexplored in many studies, limiting the ability to fully assess real-world applicability. These constraints should be considered when interpreting the broader implications of computational advances in AMR.

6. Conclusion

Computational approaches are steadily redefining how antimicrobial resistance is understood and managed. Rather than replacing traditional microbiology, they extend it—introducing predictive capacity into what has long been a reactive field. From molecular modeling to clinical decision support, these tools reveal patterns that were previously difficult to discern. Still, their impact depends on more than algorithmic sophistication. Data quality, interpretability, and integration into healthcare systems remain critical determinants of success. Ultimately, the value of computation lies not in certainty, but in its ability to reduce uncertainty—guiding more informed, timely, and adaptive responses to an evolving global health challenge.

Author Contributions

G.S. conceptualized the study, designed the review framework, and drafted the original manuscript. B.R.D. conducted the literature search, contributed to data synthesis, and assisted in manuscript preparation. T.H. contributed to data interpretation, critical analysis, and revision of the manuscript. M.A. supervised the study, provided intellectual guidance, and reviewed and edited the manuscript for important scientific content. All authors read and approved the final version of the manuscript.

References

- AMR-X Collaborators. (2024). System-wide approaches to antimicrobial therapy and antimicrobial resistance in the UK: The AMR-X framework. *The Lancet Microbe*, 5(5), e500–e507. [https://doi.org/10.1016/S2666-5247\(24\)00003-X](https://doi.org/10.1016/S2666-5247(24)00003-X)
- Antimicrobial Resistance Collaborators. (2022). Global burden of bacterial antimicrobial resistance in 2019: A systematic analysis. *The Lancet*, 399(10325), 629–655. [https://doi.org/10.1016/S0140-6736\(21\)02724-0](https://doi.org/10.1016/S0140-6736(21)02724-0)
- Arango-Argoty, G., Garner, E., Pruden, A., Heath, L. S., Vikesland, P., & Zhang, L. (2018).

- DeepARG: A deep learning approach for predicting antibiotic resistance genes from metagenomic data. *Microbiome*, 6(1), 1–15. <https://doi.org/10.1186/s40168-018-0401-z>
- Charlebois, D. A. (2023). Quantitative systems-based prediction of antimicrobial resistance evolution. *npj Systems Biology and Applications*, 9(1), 40. <https://doi.org/10.1038/s41540-023-00304-6>
- Collignon, P. J. (2016). Antibiotic resistance: Are we all doomed? *Internal Medicine Journal*, 46(10), 1131–1133. <https://doi.org/10.1111/imj.13224>
- Dalbanjan, N. P., & Kumar, S. K. P. (2024). A chronicle review of in-silico approaches for discovering novel antimicrobial agents to combat antimicrobial resistance. *Indian Journal of Microbiology*. <https://doi.org/10.1007/s12088-024-01355-x>
- Drouin, A., Giguère, S., Déraspe, M., Marchand, M., Tyers, M., Loo, V. G., Bourgault, A. M., Laviolette, F., & Corbeil, J. (2016). Predictive computational phenotyping and biomarker discovery using reference-free genome comparisons. *BMC Genomics*, 17(1), 754. <https://doi.org/10.1186/s12864-016-2889-6>
- Drouin, A., Letarte, G., Raymond, F., Marchand, M., Corbeil, J., & Laviolette, F. (2019). Interpretable genotype-to-phenotype classifiers with performance guarantees. *Scientific Reports*, 9(1), 4071. <https://doi.org/10.1038/s41598-019-40561-2>
- Elyan, E., Hussain, A., Sheikh, A., Elmanama, A. A., Vuttipittayamongkol, P., & Hijazi, K. (2022). Antimicrobial resistance and machine learning: Challenges and opportunities. *IEEE Access*, 10, 31561–31577. <https://doi.org/10.1109/ACCESS.2022.3160213>
- Goodman, K. E., Lessler, J., Cosgrove, S. E., Harris, A. D., Lautenbach, E., Han, J. H., Milstone, A. M., Massey, C. J., & Tamma, P. D. (2016). A clinical decision tree to predict whether a bacteremic patient is infected with an extended-spectrum -lactamase-producing organism. *Clinical Infectious Diseases*, 63(7), 896–903. <https://doi.org/10.1093/cid/ciw387>
- Hu, K., Meyer, F., Deng, Z. L., Asgari, E., Kuo, T. H., Münch, P. C., & McHardy, A. C. (2024). Assessing computational predictions of antimicrobial resistance phenotypes from microbial genomes. *Briefings in Bioinformatics*, 25(3), bbae206. <https://doi.org/10.1093/bib/bbae206>
- Ibisanmi, T. A., Jiang, X., Willcox, M., & Kumar, N. (2026). Recent advances in computational antimicrobial peptide discovery through big data, modeling, and artificial intelligence and their interplay in ushering the next golden era of drug development. *Frontiers in Bioinformatics*, 6, 1749404. <https://doi.org/10.3389/fbinf.2026.1749404>
- Kim, J. I., Maguire, F., Tsang, K. K., Gouliouris, T., Peacock, S. J., McAllister, T. A., McArthur, A. G., & Beiko, R. G. (2022). Machine learning for antimicrobial resistance prediction: Current practice, limitations, and clinical perspective. *Clinical Microbiology Reviews*, 35(3), e00179-21. <https://doi.org/10.1128/cmr.00179-21>
- Lees, J. A., Russell, T. W., Shaw, L. P., & Hellewell, J. (2024). Recent approaches in computational modelling for controlling pathogen threats. *Life Science Alliance*, 7(9), e202402666. <https://doi.org/10.26508/lsa.202402666>
- 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)
- Marini, S., Mora, R. A., Boucher, C., Robertson Noyes, N., & Prosperi, M. (2022). Towards routine employment of computational tools for antimicrobial resistance determination via high-throughput sequencing. *Briefings in Bioinformatics*, 23(2), bbac020. <https://doi.org/10.1093/bib/bbac020>
- Mishra, S., Han, T. A., Lopes, B. S., Ghareeb, S., & Shamszaman, Z. U. (2025). Predicting antimicrobial resistance (AMR) in *Campylobacter*, a foodborne pathogen, and cost burden analysis using machine learning. *Briefings in Bioinformatics*.
- Moradigaravand, D., Palm, M., Farewell, A., Mustonen, V., Warringer, J., & Parts, L. (2018). Prediction of antibiotic resistance in *Escherichia coli* from large-scale pan-genome data. *P L O S C o m p u t a t i o n a l B i o l o g y*, 14(12), e1006258. <https://doi.org/10.1371/journal.pcbi.1006258>
- Nayak, D. S. K., Pati, A., Panigrahi, A., Moharana, B., Rath, A., Sahoo, S. K., & Swarnkar, T. (2023). Artificial intelligence in battle against antimicrobial resistance. *International Journal on Recent and Innovation Trends in Computing and Communication*, 11(9), 427–438.
- Nguyen, M., Long, S. W., McDermott, P. F., Olsen, R. J., Olson, R., Stevens, R. L., Tyson, G. H., Zhao, S., & Davis, J. J. (2019). Using machine learning to predict antimicrobial MICs and associated genomic features for nontyphoidal *Salmonella*. *Journal of Clinical Microbiology*, 57(2), e01260-18. <https://doi.org/10.1128/JCM.01260-18>
- O'Neill, J. (2016). Tackling drug-resistant infections globally: Final report and recommendations. The Review on Antimicrobial Resistance.
- Panjla, A., Joshi, S., Singh, G., Bamford, S. E., Mechler, A., & Verma, S. (2024). Applying machine learning for antibiotic development and prediction of microbial resistance. *Chemistry – An Asian Journal*. <https://doi.org/10.1002/asia.202400102>
- Porto, W. F., Irazabal, L., Alves, E. S., Ribeiro, S. M., Matos, C. O., Pires, Á. S., Fensterseifer, I. C., Miranda, V. J., Haney, E. F., Humblot, V., Torres, M. D. T., Hancock, R. E. W., Liao, L. M., Ladram, A., Lu, T. K., de la Fuente-Nunez, C., & Franco, O. L. (2018). In silico optimization of a guava antimicrobial peptide. *Nature Communications*, 9(1), 1490. <https://doi.org/10.1038/s41467-018-03901-x>
- Ren, Y., Chakraborty, T., Doijad, S., Falgenhauer, L., Falgenhauer, J., Goesmann, A., Hauschild, A. C., Schwengers, O., & Heider, D. (2022). Prediction of antimicrobial resistance based on whole-genome sequencing and machine learning. *Bioinformatics*, 38(2), 325–334. <https://doi.org/10.1093/bioinformatics/btab681>
- Ren, Y., Chakraborty, T., Doijad, S., Falgenhauer, L., Falgenhauer, J., Goesmann, A., Hauschild, A. C., Schwengers, O., & Heider, D. (2022). Prediction of antimicrobial resistance based on whole-genome sequencing and machine learning. *Bioinformatics*, 38(2), 325–334. <https://doi.org/10.1093/bioinformatics/btab681>
- Renz, J., Dauda, K. A., Aga, O. N. L., Diaz-Uriarte, R., Löhr, I. H., Blomberg, B., & Johnston, I. G. (2025). Evolutionary accumulation modeling in AMR: Machine learning to infer and predict evolutionary dynamics of multi-drug resistance. *mBio*.
- Sakagianni, A., Koufopoulou, C., Feretzakis, G., Kalles, D., Verykios, V. S., Myrianthefs, P., & Fildis, G. (2023). Using machine learning to predict antimicrobial resistance—A literature review. *Antibiotics*, 12(3), 452. <https://doi.org/10.3390/antibiotics12030452>

- Sakagianni, A., Koufopoulou, C., Koufopoulos, P., Kalantzi, S., Theodorakis, N., Nikolaou, M., Paxinou, E., Kalles, D., Verykios, V. S., Myrianthefs, P., & Feretzakis, G. (2024). Data-driven approaches in antimicrobial resistance: Machine learning solutions. *Antibiotics*, 13(11), 1052. <https://doi.org/10.3390/antibiotics13111052>
- Santhiya, P., Bharathi, M. N., Menon, R. R., Rajalakshmi, K., & Manikandan, J. (2025). Harnessing AI-powered computational drug design to combat antimicrobial resistance: A comprehensive review of challenges, opportunities, and future directions. *In Silico Research in Biomedicine*, 1(1), 100132. <https://doi.org/10.1016/j.insr.2025.100132>
- Septimus, E. J. (2018). Antimicrobial resistance: An antimicrobial/diagnostic stewardship and infection prevention approach. *Medical Clinics of North America*, 102(5), 819–829. <https://doi.org/10.1016/j.mcna.2018.04.005>
- Tarín-Pelló, A., Fernández-Álvarez, S., Suay-García, B., & Pérez-Gracia, M. T. (2025). Novel antimicrobials from computational modelling and drug repositioning: Potential in silico strategies to increase therapeutic arsenal against antimicrobial resistance. *Molecules*, 30(11), 2303. <https://doi.org/10.3390/molecules30112303>
- Tarín-Pelló, A., Fernández-Álvarez, S., Suay-García, B., & Pérez-Gracia, M. T. (2025). Novel antimicrobials from computational modelling and drug repositioning: Potential in silico strategies to increase therapeutic arsenal against antimicrobial resistance. *Molecules*, 30(11), 2303. <https://doi.org/10.3390/molecules30112303>
- Vazquez-Guillamet, M. C., Vazquez, R., Micek, S. T., & Kollef, M. H. (2017). Predicting resistance to piperacillin-tazobactam, cefepime and meropenem in septic patients with bloodstream infection due to Gram-negative bacteria. *Clinical Infectious Diseases*, 65(10), 1607–1614. <https://doi.org/10.1093/cid/cix601>
- Yelin, I., Snitser, O., Novich, G., Katz, R., Tal, O., Parizade, M., Chodick, G., Koren, G., Shalev, V., & Kishony, R. (2019). Personal clinical history predicts antibiotic resistance of urinary tract infections. *Nature Medicine*, 25(7), 1143–1152. <https://doi.org/10.1038/s41591-019-0503-6>
- Yu, W., & MacKerell, A. D. (2016). Computer-aided drug design methods. *Methods in Molecular Biology*, 1439, 85–106. https://doi.org/10.1007/978-1-4939-3673-1_5