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When medications stop working: Rethinking drug design.

Laura Carlos *

Weldon School of Biomedical Engineering, Purdue University, USA

***Correspondence to:** Laura Carlos, Weldon School of Biomedical Engineering, Purdue University, USA, E-mail: Laura.c@purdue.edu

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Introduction

Modern medicine has made remarkable strides in treating diseases once considered incurable. Yet, a persistent and growing challenge threatens these advances: drug resistance. Whether in cancer, infectious diseases, or chronic conditions, medications that once worked effectively can lose their potency over time. This phenomenon demands a fundamental shift in how we approach drug development. Rethinking drug design is no longer optional—it's essential for sustaining therapeutic efficacy and improving patient outcomes. Drug resistance arises when pathogens, cancer cells, or even the human body adapt to evade the effects of medications. In cancer, resistance can be intrinsic (present before treatment) or acquired (develops during therapy). In infectious diseases, overuse and misuse of antibiotics have accelerated the emergence of resistant strains like MRSA and drug-resistant tuberculosis [1, 2].

Conventional drug design often follows a linear path: identify a target, screen compounds, optimize leads, and test efficacy. While this method has yielded many successful drugs, it struggles to keep pace with evolving resistance mechanisms. High attrition rates in clinical trials due to unforeseen resistance, Static targeting that fails to account for biological variability, Limited personalization, ignoring patient-specific factors. Moreover, the time and cost involved—often exceeding a decade and billions of dollars—make it difficult to respond rapidly to emerging threats. To overcome these limitations, researchers are embracing innovative strategies that integrate technology, biology, and systems thinking [3, 4].

SBDD uses 3D models of target proteins to design molecules that fit precisely. This approach allows for: Targeting mutant proteins in resistant strains, Designing multi-target drugs to reduce escape pathways, Predicting binding affinity and off-target effects. Analyzing vast datasets to predict resistance patterns, Generating novel compounds using generative models, Optimizing drug combinations through reinforcement learning [5, 6].

Sequential drug administration to delay resistance, Dose modulation based on real-time biomarkers, Combination therapies targeting multiple pathways. In lung cancer, resistance to EGFR inhibitors like erlotinib led to the development of osimertinib, which targets the T790M mutation. Researchers now use liquid biopsies to monitor resistance and adjust treatment dynamically. New antibiotics like zoliflodacin are designed to bypass traditional resistance mechanisms. Additionally, phage therapy and CRISPR-based antimicrobials offer precision targeting of resistant bacteria [7, 8].

In HIV and hepatitis C, protease inhibitors and polypharmacology approaches have improved outcomes by targeting multiple viral components simultaneously. Nanoparticles can deliver drugs directly to resistant cells, bypassing efflux pumps and enhancing intracellular accumulation. Real-time monitoring of drug levels and biomarkers enables personalized dosing and early detection of resistance. Though in its infancy, quantum computing promises to simulate complex molecular interactions, revolutionizing drug design speed and accuracy [9, 10].

Conclusion

When medications stop working, the consequences are dire—not just for individual patients but for global health. Rethinking drug design is not merely a scientific challenge; it's a moral imperative. By embracing innovation, personalization, and adaptability, we can build a future where resistance

is not a dead end but a detour toward smarter, more resilient therapies.

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