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Synthetic biology approaches to antiviral drug design.

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Introduction

The global burden of viral diseases—from HIV and hepatitis to emerging threats like SARS-CoV-2 and Nipah virus—has intensified the need for innovative antiviral strategies. Traditional drug development, while effective in many cases, often struggles to keep pace with rapidly mutating viruses and complex host-pathogen interactions. Synthetic biology, a multidisciplinary field that combines engineering principles with molecular biology, offers a transformative approach to antiviral drug design. By enabling the rational construction of biological systems and tools, synthetic biology is revolutionizing how we discover, optimize, and deliver antiviral therapies [1].

These platforms reduce reliance on chemical synthesis and enable rapid response to emerging threats. Synthetic biology is pushing boundaries with living therapeutics—engineered organisms that detect and respond to viral infections in vivo. Gut bacteria can be engineered to sense viral markers and secrete antiviral peptides or immune modulators. T cells and macrophages can be modified to recognize and eliminate virus-infected cells, offering potential for chronic infections like HIV and hepatitis. Synthetic biology involves the design and construction of new biological parts, devices, and systems—or the redesign of existing ones—for useful purposes. It integrates principles from genetics, bioengineering, computer science, and chemistry to create programmable biological functions. In antiviral drug design, synthetic biology enables the creation of novel molecules, biosensors, delivery platforms, and even living therapeutics that can detect, inhibit, or destroy viruses with high specificity and adaptability [2].

These approaches offer dynamic, self-regulating therapies that adapt to disease progression. Viruses evolve rapidly, often developing resistance to conventional drugs. Synthetic biology addresses this challenge through: Designing drugs that target multiple viral sites simultaneously reduces the likelihood of resistance. Modular systems can be reprogrammed to target new mutations or strains. Synthetic biology can identify and modulate host factors essential for viral replication, offering broad-spectrum solutions. These strategies enhance durability and reduce the need for frequent drug redesign. Despite its promise, synthetic biology in antiviral drug design faces challenges: Engineered organisms and gene-editing tools must be rigorously tested to prevent off-target effects or environmental release. Novel therapies require new frameworks for approval and monitoring. One of the most promising applications of synthetic biology is the rational design of antiviral agents. Unlike traditional drug discovery, which often relies on screening natural compounds, synthetic biology allows for the de novo design of molecules tailored to specific viral targets. Engineered peptides can mimic host receptors or viral binding domains, competitively inhibiting viral entry. For example, synthetic decoy receptors have been developed to block SARS-CoV-2 spike protein binding to ACE [3].

Synthetic biology enables the design of small interfering RNAs (siRNAs), antisense oligonucleotides, and CRISPR-Cas systems that target viral genomes for degradation or editing. These are short, synthetic nucleic acid sequences that bind to viral proteins with high affinity, acting as inhibitors or diagnostic tools. These molecules can be optimized for stability, specificity, and delivery using computational modeling and high-throughput screening. CRISPR-Cas systems, originally developed for genome editing, have been repurposed as powerful antiviral tools. Synthetic biology has enabled the adaptation of CRISPR-Cas13 and Cas12 systems to target RNA and DNA viruses, respectively. Cas13 can be programmed to

cleave viral RNA, offering a direct method to suppress replication in viruses like influenza, Zika, and SARS-CoV-2. Cas12 systems have shown promise in targeting herpesviruses and hepatitis B virus. These platforms can be delivered via viral vectors or lipid nanoparticles and offer modularity for rapid adaptation to new viral strains. Early and accurate detection is critical for effective antiviral intervention. Synthetic biology has enabled the development of biosensors that detect viral nucleic acids, proteins, or host biomarkers with high sensitivity [4].

These RNA-based sensors activate gene expression in response to specific viral RNA sequences, enabling low-cost diagnostics. Portable, paper-based diagnostics using freeze-dried synthetic circuits can detect viruses like Ebola and Zika in resource-limited settings. SHERLOCK and DETECTR platforms use CRISPR enzymes to detect viral RNA or DNA with single-molecule sensitivity. These tools support rapid, decentralized testing and surveillance, essential for outbreak control. Synthetic biology also enables the engineering of microbial cell factories—such as *E. coli*, yeast, and algae—to produce antiviral compounds at scale. Microbes can be programmed to produce monoclonal antibodies, interferons, and viral antigens for therapy or vaccination. Pathways for antiviral compounds like ribavirin or artemisinin can be reconstructed in microbes for sustainable production. Engineered microbes can produce VLPs that mimic viral structures without genetic material, useful for vaccines and immunotherapy [5].

Conclusion

Synthetic biology is redefining antiviral drug design by enabling the rational, programmable creation of molecules, systems, and organisms that combat viral infections with unprecedented precision. From CRISPR-based antivirals and biosensors to engineered microbial factories and living therapeutics, this field offers a versatile and scalable toolkit for current and future viral threats. With continued investment, collaboration, and ethical stewardship, synthetic biology will play a pivotal role in building a resilient and responsive global health infrastructure.

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