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CRISPR-Cas systems in bacteria: Beyond immunity to host interaction modulation.

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Introduction

The discovery of CRISPR-Cas systems revolutionized our understanding of bacterial immunity. Initially characterized as a defense mechanism against invading genetic elements such as bacteriophages and plasmids, CRISPR-Cas systems have since been recognized for their multifaceted roles in bacterial physiology. Recent research has illuminated their involvement in modulating host interactions, influencing virulence, biofilm formation, and immune evasion. This article explores the expanding landscape of CRISPR-Cas functions beyond adaptive immunity, emphasizing their role in host interaction modulation [1].

CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) and Cas (CRISPR-associated) proteins form a prokaryotic adaptive immune system. Upon encountering foreign DNA, bacteria incorporate short fragments (spacers) into their CRISPR arrays. These spacers are transcribed into CRISPR RNAs (crRNAs), which guide Cas proteins to recognize and cleave complementary sequences in invading genetic material. This mechanism provides sequence-specific immunity and is heritable across generations [2].

CRISPR-Cas systems in bacteria are far more than molecular sentinels against genetic invaders. Their roles in modulating host interactions, regulating virulence factors, and shaping bacterial behavior highlight their complexity and adaptability. As research continues to unravel these functions, CRISPR-Cas systems may emerge as central players in microbial ecology and host-pathogen dynamics, offering novel strategies for combating infectious diseases. While immunity remains the hallmark of CRISPR-Cas systems, emerging evidence suggests their involvement in regulating endogenous genes. In several bacterial species, CRISPR-Cas components modulate transcriptional networks, influencing traits such as metabolism, stress response, and cell communication. These regulatory roles often involve partial complementarity between crRNAs and target mRNAs, allowing for fine-tuned gene expression without complete degradation [3].

Understanding the noncanonical functions of CRISPR-Cas systems opens new avenues for therapeutic intervention. Targeting CRISPR-mediated regulatory pathways could attenuate bacterial virulence or enhance immune recognition. Moreover, engineered CRISPR-Cas tools can be used to modulate gene expression in probiotic strains, potentially improving gut health and immune function. One of the most compelling noncanonical roles of CRISPR-Cas systems is their influence on bacterial virulence. In *Francisella novicida*, a type II-B CRISPR-Cas system represses the expression of bacterial lipoproteins (BLPs) that would otherwise trigger host immune responses via Toll-like receptors (TLRs). This repression involves a complex interplay between Cas9, tracrRNA, and a small CRISPR-associated RNA (scaRNA). Similarly, in *Streptococcus pyogenes*, Cas9 downregulates the CovR/S two-component system, which controls capsule synthesis and antiphagocytic properties. Mutants lacking Cas9 exhibit reduced virulence due to impaired regulation of key surface proteins. These findings underscore the role of CRISPR-Cas systems in helping pathogens evade host defenses [4].

Biofilms are structured microbial communities that confer resistance to environmental stress and antibiotics. CRISPR-Cas systems have been implicated in biofilm regulation in species such as

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Pseudomonas aeruginosa. The type I-F system in this bacterium targets the mRNA of LasR, a master regulator of quorum sensing and biofilm formation. By modulating LasR levels, CRISPR-Cas indirectly influences biofilm architecture and host colonization. Adherence to host tissues is a critical step in bacterial pathogenesis. Studies have shown that CRISPR-Cas systems affect bacterial adhesion capabilities. For instance, *Neisseria meningitidis* and *Campylobacter jejuni* strains lacking Cas9 exhibit diminished adherence to epithelial cells, suggesting that CRISPR-Cas systems regulate surface adhesins or signaling pathways involved in host interaction [5].

Conclusion

Beyond repressing immunogenic proteins, CRISPR-Cas systems contribute to immune evasion by altering bacterial surface structures. In *Riemerella anatipestifer*, Cas9 regulates genes involved in lipopolysaccharide synthesis, affecting recognition by host immune cells. Transcriptomic analyses reveal that Cas9 deletion leads to upregulation of immune-stimulatory molecules, making bacteria more susceptible to clearance. The dual role of CRISPR-Cas systems in immunity and host interaction modulation suggests evolutionary pressure to retain and diversify these systems. Horizontal gene transfer and spacer acquisition enable bacteria to adapt rapidly to changing environments and host defenses. This versatility may explain the widespread distribution and subtype diversity of CRISPR-Cas systems across bacterial taxa

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