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Perspective

Exploring the Role of Bacterial Metabolic Interactions in Antibiotic Resistance and Tolerance

Tom Jensen*

Department of Immunology and Microbiology, University of Copenhagen, Copenhagen, Denmark

DESCRIPTION

Antibiotic resistance has emerged as a critical global health challenge, threatening the effectiveness of commonly used antibiotics and complicating the treatment of bacterial infections. Traditional research has primarily focused on understanding the genetic basis of resistance. However, recent studies have shed light on the significant role played by bacterial metabolic interactions in contributing to antibiotic resistance and tolerance and it explore the mechanisms through which bacterial communities exchange metabolites, fostering a protective environment against antibiotics and leading to increased resistance and tolerance.

Metabolic cooperation in biofilms

Bacteria commonly exist in nature as biofilms, complex microbial communities encased in a self-produced extracellular matrix. Within these biofilms, different bacterial species engage in metabolic cooperation to enhance their survival. Some bacteria may produce metabolites that provide essential nutrients to neighbouring species, promoting their growth and overall fitness. In this context, antibiotic resistance can arise due to a combination of factors like competition for nutrients, spatially protected microenvironments, and the exchange of antibiotic-degrading enzymes among bacterial community members. Metabolic cooperation allows certain bacteria to endure stress conditions imposed by antibiotics. While some members may actively resist antibiotics through genetic mechanisms, others benefit from the protective environment created by their metabolically active neighbors. This cooperative behavior increases the likelihood of survival for resistant subpopulations, leading to persistent infections and treatment failure.

Nutrient competition and antibiotic tolerance

Bacterial metabolic interactions can involve competition for limited nutrients within their environment. When antibiotics are present, some bacteria may undergo metabolic changes, reducing their growth rate or becoming metabolically dormant to conserve energy. This altered metabolic state allows them to better withstand the stress of antibiotic exposure. Meanwhile, other bacteria may take advantage of the reduced competition for nutrients and thrive, leading to an overall decrease in the efficacy of antibiotics. Furthermore, the presence of neighbouring metabolically active bacteria can indirectly protect dormant bacteria from antibiotic action. By creating a physical barrier or consuming antibiotics, the active members provide a shield against antibiotic penetration, making it more challenging for antibiotics to reach and kill the dormant bacteria. This process contributes to the phenomenon known as "tolerance" and makes complete eradication of bacterial populations much more difficult.

Metabolite-mediated antibiotic degradation

Bacterial metabolic interactions can also facilitate the degradation of antibiotics through enzymatic processes. Certain bacteria produce enzymes capable of breaking down antibiotics into non-toxic components, effectively neutralizing their antimicrobial properties. These enzymes may act intracellular or be secreted into the extracellular environment, enabling them to interact with neighbouring bacteria. Horizontal gene transfer, a mechanism through which bacteria exchange genetic material, allows the transfer of antibiotic resistance genes and associated enzymes between bacterial species. As a result, the presence of antibiotic-degrading enzymes can spread within the bacterial community, leading to a collective resistance and tolerance to specific antibiotics.

Induction of antibiotic resistance genes

Metabolic interactions can influence the expression of antibiotic resistance genes within bacterial communities. Some metabolites present in the environment act as signaling molecules, inducing the expression of resistance genes in response to antibiotic exposure. This activation of resistance genes can lead to the emergence of antibiotic-resistant bacterial subpopulations. For

Correspondence to: Tom Jensen, Department of Immunology and Microbiology, University of Copenhagen, Copenhagen, Denmark, E-mail: tomjen@edu.dk

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instance, the presence of certain nutrients may trigger the up-regulation of efflux pump genes, which are responsible for pumping antibiotics out of bacterial cells.

This phenomenon can significantly reduce the intracellular concentration of antibiotics, rendering them ineffective against the resistant bacteria. As a consequence, the community becomes enriched with antibiotic-resistant strains, exacerbating the overall resistance problem.

Impact of antibiotic use on metabolic interactions

The widespread and inappropriate use of antibiotics has far-reaching consequences on microbial ecosystems. Prolonged antibiotic exposure can disrupt natural bacterial communities and alter their metabolic interactions.

The selective pressure exerted by antibiotics may favor the survival of antibiotic-resistant strains, leading to a shift in the composition of microbial populations.

In the context of antibiotic resistance, the use of broad-spectrum antibiotics is particularly concerning. These antibiotics target a wide range of bacterial species, including beneficial ones, further disrupting the balance of microbial communities.

As susceptible bacteria are eliminated, antibiotic-resistant strains have less competition, allowing them to proliferate and dominate the community. Consequently, this dominance can enhance metabolic interactions that promote antibiotic resistance and tolerance.

Targeting bacterial metabolic interactions for novel therapies

The growing understanding of bacterial metabolic interactions in antibiotic resistance has opened new avenues for developing innovative treatment strategies. One potential approach is to disrupt these interactions within biofilms. By targeting key metabolic pathways or communication mechanisms, it may be possible to weaken the protective biofilm structure and enhance the susceptibility of bacteria to antibiotics. Several potential interventions are being explored, including the use of specific enzyme inhibitors that prevent the degradation of antibiotics, thus preserving their antimicrobial activity. Additionally, targeted disruption of quorum sensing, a communication mechanism used by bacteria to coordinate behaviors, could reduce the formation and stability of biofilms, making bacteria more susceptible to antibiotics.

Bacterial metabolic interactions play a critical role in antibiotic resistance and tolerance, highlighting the complexity of microbial communities and their response to antibiotic pressure. As our understanding of these interactions deepens, new therapeutic approaches could be developed to mitigate antibiotic resistance and improve treatment outcomes. Furthermore, promoting the responsible use of antibiotics remains paramount to preserving the efficacy of these life-saving drugs in combating bacterial infections. By addressing the multifaceted nature of antibiotic resistance, we can work towards a sustainable and effective approach to combat bacterial infections in the future.