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Perspective

The Immune Evasion and Drug Resistance of Tuberculosis Bacteria

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DESCRIPTION

The immune system's efforts to destroy invading pathogens, Tuberculosis (TB) bacteria have developed practical ways to evade the body's defenses, allowing the disease to persist. Once TB bacteria enter the lungs, they are engulfed by immune cells called macrophages. However, instead of being destroyed, the bacteria can survive within these cells, creating a protective environment for themselves. They achieve this by inhibiting the fusion of phagosomes (compartments that contain the bacteria) with lysosomes (organelles that contain enzymes for pathogen destruction). TB bacteria can alter the host's immune response, interfering with signalling pathways to weaken the immune system's effectiveness. This strategy allows the bacteria to remain dormant within the body for years, often without symptoms, and then reactivate if the immune system becomes weakened.

Avoiding initial detection and manipulating macrophages

TB bacteria enter the body through the respiratory tract, typically when an infected person coughs or sneezes. Once inhaled, these bacteria make their way to the lungs, where they encounter macrophages, immune cells that act as the body's first line of defense. Macrophages are designed to recognize and engulf foreign invaders, but TB bacteria have a unique, waxy cell wall that makes it hard for the immune system to detect and attack them. This cell wall consists of a lipid-rich layer that masks the bacterial cell, helping it blend in and avoid early detection. When TB bacteria do get engulfed by macrophages, they don't get destroyed right away. Instead, they manipulate these cells to create a safe environment for themselves. TB bacteria interfere with the usual process of phagocytosis, where macrophages ingest and break down pathogens. Normally, engulfed bacteria would be trapped in an acidic compartment called the phagolysosome, where they would be destroyed. However, TB bacteria release proteins that prevent the fusion of the phagosome (the compartment containing the bacteria) with the lysosome (which contains digestive enzymes). By preventing this fusion, TB bacteria create a protected space where they can survive and even multiply within the macrophages.

Suppressing immune response in granuloma formation and evasion

In addition to creating a safe space within macrophages, TB bacteria can also interfere with the immune response on a broader level. They secrete molecules that disrupt the signalling pathways needed to activate other immune cells. For example, TB bacteria can inhibit the release of certain cytokines-chemical messengers that alert the immune system to the presence of a pathogen. Without these signals, other immune cells, like T cells, may not be fully activated or recruited to the infection site, allowing TB bacteria to evade a coordinated immune attack. One of the characteristic of TB infection is the formation of granulomas, which are clusters of immune cells that surround infected macrophages to contain the bacteria. At first, granulomas are a defense mechanism intended to wall off the bacteria and prevent them from spreading. However, TB bacteria can manipulate this structure to their advantage. Within the granuloma, TB bacteria enter a dormant state, meaning they stop actively multiplying and remain hidden. This dormancy allows TB to persist in the body for years without causing symptoms, which is why people can carry TB in a latent form. When the immune system weakens due to factors like malnutrition, stress, or co-infections these bacteria can reactivate, leading to active TB disease. While granulomas contain TB bacteria for a time, they can eventually break down, allowing the bacteria to spread throughout the lungs or even to other organs. TB bacteria can weaken the structure of granulomas by causing immune cells within them to release enzymes that degrade surrounding tissue. This breakdown enables the bacteria to escape and infect new cells, often worsening the infection and making it harder to control.

Advancing of drug therapy

TB bacteria's strategies to evade immune response also make them resilient against certain antibiotics. Their ability to enter a

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dormant state within granulomas means that many antibiotics, which are effective only against actively replicating bacteria, do not work well. Additionally, the lipid-rich cell wall of TB bacteria not only shields them from immune attacks but also blocks the penetration of some antibiotics, making the bacteria inherently resistant to many drugs. This is why TB treatment is lengthy, typically requiring multiple antibiotics taken over six to nine months.

CONCLUSION

Leprosy granulomas reflect the complex interplay between host immunity and bacterial survival strategies. By dissecting the cellular and molecular determinants of bacterial load, researchers can identify novel interventions to improve disease control and patient outcomes. Understanding the dynamics within leprosy granulomas offers essential insights into the mechanisms of immune evasion and persistence of *Mycobacterium leprae*. By targeting these pathways, innovative therapeutic approaches can be developed to enhance bacterial clearance and modulate immune responses. Such advancements hold the potential to reduce transmission, improve treatment efficacy, and mitigate long-term complications.