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Opinion Article

Lactobacillus Supplementation for the Respiratory Tract

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DESCRIPTION

Intestinal supplementation with *Lactobacillus* has been shown to promote respiratory health; however immunobiotic *Lactobacillus's* direct action on the respiratory mucosa may regulate local immunity of the respiratory tract. It has been identified that *lactobacillus* administered intranasally can enhance the respiratory immune response more than *lactobacillus* administered orally. Due to a lack of substrate, *Lactobacillus* administered *via* the nasal route typically does not result in SCFA (Short-Chain Fatty Acids) production. There are primarily two components to the potential methods *via* which they control respiratory immunity. *Lactobacillus* components can be detected by PRR's (Pattern Recognition Receptor) in the respiratory tract, which eventually caused subsequent pathways to be activated. For instance, nasal priming with *L. rhamnosus* CRL1505 peptidoglycan elevates lung TNF- and IL-10 levels and upregulates TLR2 and TLR9 expression, which is comparable to intranasal delivery of entire bacteria. While this is happening, nasal priming with *L. rhamnosus* CRL1505 peptidoglycan can improve the TLR3/RIG-I-activate antiviral immune response by enhancing IFN- and NK cell activity, resulting in increased viral clearance and reducing lung tissue damage.

Peptidoglycan Recognition Proteins (PGRPs), a category of PRRs that mediates bactericidal action, can also detect lung peptidoglycan. For example, active PGRP2 may cause neutrophil recruitment in the lung tissue of mice infected with *S. pneumoniae*. It is important to observe that not all *Lactobacillus* species' peptidoglycans function as protective agents. In immunodeficient mice, nasal treatment of the *L. rhamnosus* CRL534 peptidoglycan does not increase resistance to *S. pneumoniae* infection. This demonstrates that the *Lactobacillus* protective effect is strain-specific. Furthermore, several mechanisms may be used to activate PRR's when *Lactobacillus* is administered by nasal. Even if one PRR is eliminated, another pathway may still be allowed to serve as a secondary protection. For instance, it demonstrates that *L. plantarum* BAA-793 only begins to have a protective function against *pneumonia* virus infection when both NOD2 and TLR2 are turned out. Therefore, a major element of the protective function provided by intranasal *Lactobacillus* administration may include components of *Lactobacillus* that activate PRR's.

The ability of *Lactobacillus* attached to host cells and prevent pathogen adhesion or binding is the primary. In terms of the bacterium, studies have also shown that *Lactobacillus* can directly prevent the adherence of bacteria to respiratory epithelial cells. The adherence of *S. pyogenes* to pharyngeal epithelial cells can be prevented by *L. rhamnosus* Kx151A1, *L. reuteri* PTA-5289, and *L. salivarius* LMG9477. Furthermore, administering *L. murinus* CNCM I-5314, a *eubacterium* from the murine lung, intravenously can operate as a barrier to prevent *S. pneumoniae* from colonising the lung tissue. When it comes to viruses, *Lactobacillus* binds rapidly to the viral receptor molecule to prevent the virus from penetrating the host cell. Angiotensin-converting enzyme 2 is the receptor molecule for the SARS-CoV-2 spike glycoprotein, and lipopeptides generated by *L. curvatus*, *L. sakei*, and *L. lactis* can bind to it. This may prevent the virus from infecting host cells.

CONCLUSION

In addition to preventing pathogenic bacteria from sticking and adhering, *Lactobacillus* also directly exhibits antibiotic activity. *In vitro*, several *Lactobacillus* species have an antibacterial impact on group A *Streptococcus*. Similar to *L. rhamnosus* Kx151A1, *L. reuteri* PTA-5289 also greatly reduces *S. pyogenes' in vitro* hemolytic activity. Additionally, some of the proteins that *Lactobacillus* secretes have antibacterial properties. Reuterin, a substance released by *L. reuteri*, shows broad-spectrum antibacterial activity.

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