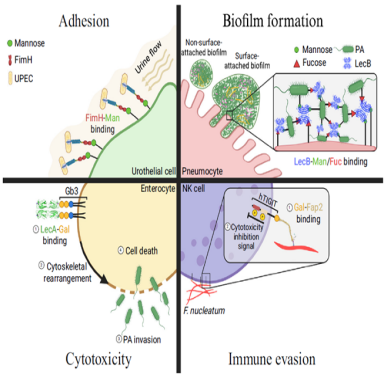

Bacterial lectins: multifunctional tools in pathogenesis and possible drug targets

Description

Glycans are vital macromolecules with diverse biological roles, decoded by lectins â?? specialized carbohydrate-binding proteins crucial in promoting pathogenesis. With the rise in antimicrobial resistance, targeting lectins with inhibitors presents a promising opportunity to enhance the hostâ??s ability to clear the pathogen. The WHO identifies bacterial antimicrobial resistance (AMR) as a critical global health challenge, necessitating innovative strategies that also target non-antibiotic pathways. Ongoing research continues to uncover a growing range of functions for bacterial lectins in pathogenesis, such as host recognition and adhesion, biofilm formation, cytotoxicity, and host immune evasion, with individual lectins often playing multiple roles in these processes. Ongoing research continues to uncover a growing range of functions for bacterial lectins in pathogenesis, such as host recognition and adhesion, biofilm formation, cytotoxicity, and host immune evasion, with individual lectins often playing multiple roles in these processes.

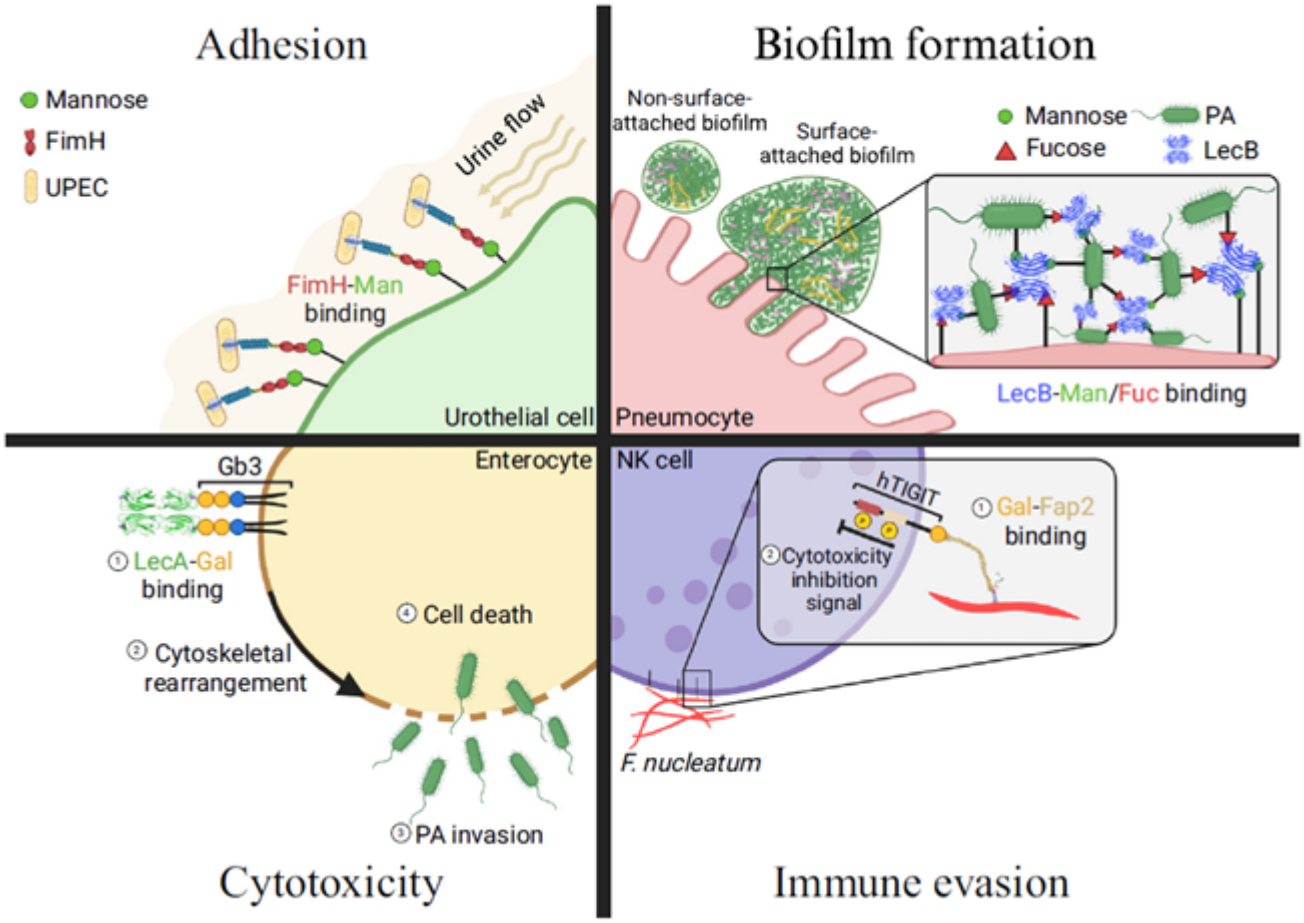

Figure. The roles of bacterial lectins in pathogenesis (selected examples). Adhesion. Uropathogenic Escherichia coli (UPEC) uses its FimH adhesin to bind to mannose-presenting uroplakin on urothelial cells, facilitating attachment and helping the bacteria resist clearance by urine flow during miction. Biofilm formation. Pseudomonas aeruginosa (PA) forms and stabilizes its biofilms by using its mannose-and fucose-binding LecB to crosslink the glycans present on biofilm exopolysaccharides, on the host, or the bacterial surfaces. Cytotoxicity. LecA from PA binds the glycosphingolipid Gb3, thereby rearranging the cytoskeleton of enterocytes and increasing their susceptibility to PA invasion which leads to cell death. Immune evasion. Fusobacterium nucleatum uses its galactose-sensitive adhesin Fap2 to inhibit natural killer (NK) cell effector functions by binding the human T cell immunoreceptor with immunoglobulin and immunoreceptor tyrosine-based inhibition motif domain (hTIGIT) and triggering a biochemical cascade to downregulate NK cell function.

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