Abstract

Bacteria belonging to the genus *Legionella*, upon entering the human body from the aquatic environment, induce legionellosis. This disease occurs in two clinical forms: Pontiac fever, resembling influenza-like symptoms, and severe pneumonia known as Legionnaires' disease. Demographic and climatic changes have made *Legionella* spp. one of the most important waterborne pathogens, both in terms of frequency and severity of infections. Although there are over 70 species and 90 serogroups of *Legionella* in aquatic ecosystems, approximately 90% of legionellosis cases worldwide are caused by *L. pneumophila* sg 1. The serogroup of *L. pneumophila* is determined based on the structure of lipopolysaccharide (LPS). The structure of *L. pneumophila* sg 1 LPS influences the physicochemical properties of these bacteria's surfaces, determines their ability to interact with host cells, and modulates the immune response to infection. Clinical strains of *L. pneumophila* sg 1 and mutants lacking *O*-acetyl groups in the sugars of the core region and *N*-methyl groups of legionaminic acid in the polysaccharide part of LPS were used for the study. Disruptions in LPS synthesis led to differences in the lipid profile of *L. pneumophila* sg 1. Measurement of bacterial surface polarity using the fluorescent dye probe (prodan) revealed differences in the hydrophobicity of the strains. *O*-acetyl groups of the polysaccharide part of LPS play a crucial role in the early stage of *L. pneumophila* sg 1 interaction with host cells before the bacteria enter the interior of eukaryotic cells. Studies on the adhesive abilities of bacteria to phagocytic and non-phagocytic cells showed that LPS modifications promote interactions between *L. pneumophila* sg 1 and macrophages. Research on the induction of pro-inflammatory cytokines showed that not only lipid A but also the polysaccharide part of *L. pneumophila* sg 1 LPS affects the level of TNF-α, and the presence of *O*-acetyl groups acylating the rhamnose core may be one of the ways for these bacteria to evade the human immune system's control. Understanding the complex mechanisms of modulating the structure, function, and interaction mode of *L. pneumophila* sg 1 LPS with host cells is crucial in designing new drugs and evaluating new therapeutic strategies to protect against infections caused by *L. pneumophila* sg 1.

Keywords: *Legionella pneumophila* sg 1, lipopolysaccharide, lipids, adhesion, pro-inflammatory cytokin

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