Legionella rods ubiquitous in the aqueous and soil environments become dangerous to human health and life after transmission to artificial systems of water supply. Human infection is most frequently caused through inhalation of bacteria-infected water distributed as a water-air aerosol by a condition system, cooling towers, and industrial and medical facilities. In human organisms, the bacteria can cause different severity infections: from flu-like self-limiting infections to severe bilateral pneumonia, which may be lethal in immunocompromised subjects. The most prevalent clinical form of *Legionella* infection is Legionnaires' disease. The results of etiological investigations of pneumonia indicate that these bacteria are responsible for 2%-16 % of incidence and from 14%-37% of severe cases with fatality over 30%. The pathogenesis of Legionnaires' disease is based on the bacterial ability to penetrate and proliferate within the host cell. Owing to their exceptional adaptive abilities, *Legionella* rods have evolved a number of unique traits facilitating replication in the nutrient-rich but extremely adverse environment of human macrophages. Despite the expanding knowledge of the biology and pathogenicity of this microorganism, it is still unknown why only one species, i.e. *L. pneumophila* serotype 1 is responsible for over 90% of laboratory confirmed legionellosis cases, which diverges from the prevalence of the bacteria in the environment. Our project assumes that the special structure of the surface components, mainly LPS of *L. pneumophila* serotype 1, which determines the specific interactions with the host cell can be involved in the increased virulence of strains. Therefore, the main objective of the project is structural analysis of the surface components (LPS and phospholipids) of clinical *L. pneumophila* isolates and their mutants defective in the synthesis of the O-specific chain. The modern spectral methods employed in the project will allow exploration of *Legionella* pathogenicity-important structures of native LPS molecules in wild-type and mutants strains. The biophysical methods will facilitate precise monitoring and analysis, at the level of single cells, of molecular interaction between the pathogen and host cell. Additionally, we intend to determine the role of LPS in modulation of cell immune response to infection with *L. pneumophila* serotype 1. Prospective results of the project may have clinical importance for evaluation of Legionnaires' disease prognosis. Elucidation of the complex mechanism of *L. pneumophila* pathogenicity is the basis for the development of a strategy for prevention and treatment of atypical human pneumonia.