Community-acquired pneumonia (CAP) remains a major cause of hospitalization and significant mortality worldwide. In addition to *Streptococcus pneumoniae*, which is the most common cause of pneumonia, intracellular *Legionella* pathogens are also a significant etiological agent of pneumonia. *Legionella* bacteria are present in various natural environments, including rivers, lakes, and soils, where they proliferate within the cells of free-living protozoa of the genus *Acanthamoeba, Vermamoeba,* and *Tetrahymena.* Protozoa are not only a source of essential nutrients for *Legionella*, but also protect the bacteria from unfavourable environmental conditions and increase their resistance to antibiotics, osmotic and thermal stress. Bacteria, after getting into the natural environment, colonize equipment and water distribution systems, which can be a source of contaminated water-air aerosol that is dangerous to human health and life. Acquired in the course of evolution with protozoa, the ability of *Legionella* bacteria to infect human macrophages and break their killing mechanisms leads to the development of acute pneumonia called Legionnaires' disease. The incidence of Legionnaires' disease has increased significantly in the European Union and the United States over the last decade, indicating that the disease has the potential to become a serious health problem, especially in the context of an aging population, climate change, and increasing pressure on water resources. All of the 70 known *Legionella* species are considered potentially pathogenic to humans, but two species, *L. pneumophila* and *L. longbeachae*, are responsible for the majority of confirmed cases of Legionnaires' disease. Although recent studies have shown that *L. longbeachae* is more virulent than *L. pneumophila* in a mouse model of Legionnaires' disease, little is still known about the biology and infection process of this species. The project assumes that the high virulence of *L. longbeachae* strains is conditioned by the presence of specific determinants of the bacterial cell envelope (lipopolysaccharide, lipids, proteins), which determine their increased ability to interact with host cells. The bacteria presumably modify the composition of the vesicles released from their surface, promoting molecules with high pathogenic potential. The project also assumes that bacteria, after contact with protozoan and macrophage cells, change their gene expression profile, promoting those responsible for increased pathogenic potential, in particular genes for the synthesis of surface structures. Comparative analysis at the transcriptome level of the bacteria released from protozoan and macrophage cells will identify those genes that specifically adapt to human cells. Identification of key virulence markers of *L. longbeachae* will provide insight into complex host-pathogen interactions, potentially facilitating the development of diagnostic and therapeutic tools for Legionnaires' disease.