The aim of this project is to investigate the role of *Acinetobacter baumannii* ADP-heptose in the pathogenesis of pneumonia of this etiology. Our hypothesis is that ADP-heptose is an important molecule of bacterial origin that allows the host cell's to recognizen the*A. baumannii* infection). The response of human lung epithelial cells to ADP-heptose has not yet been studied, although its receptors, namely ALPK1 (Alpha-protein kinase 1) and TIFA (TRAF interacting protein with forkhead associated domain), are expressed by human lung epithelial cells and human alveolar macrophages. Additionally, there is a lack of knowledge about the influence of ADP-heptose during *A. baumannii* infection on the innate immune response, especially in the pathogenesis of pneumonia of this etiology. The lack of knowledge in these matters necessitates the investigation of the effect of ADP-heptose on the host immune response. Especially since infections with multi-drug resistant strains of *A. baumannii* have become an extremely important clinical problem in intensive care units (ICU) around the world, as noticed by the World Health Organization (WHO). WHO classified *A. baumannii* in the group of pathogens for which the development of new antibacterial agents should have the highest priority - the critical priority. In addition, the American Society for Infectious Diseases has identified a group of bacteria, including *A. baumannii*, that cause nosocomial infections with the acronym ESKAPE, which precisely means "escape" the effects of antimicrobial drugs. In the ICU *A. baumannii* is the most common cause of mechanical ventilation associated pneumonia (VAP) and sepsis. Infection with this bacterium significantly increases the risk of death in patients. Due to the difficulties in treating infections caused by this bacterium due to increasing resistance, therapies modulating the immune system response may become a chance for patients. *A. baumannii* can develop resistance to colistin, which often turns out to be the last resort in the treatment of patients, through the loss of lipopolysaccharide (LPS), which indicates that a different PAMP may be crucial in inducing an immune response. ADP-heptose, which is an important metabolite of the inner core biosynthetic pathway of LPS among Gram-negative bacteria, appears to be one of the candidates for this. Interestingly, it was only recently discovered that ADP-heptose can act as PAMP and its pattern recognition receptor (PRR) is ALPK1, which, when activated, in turn activates TIFA phosphorylation. In response to infections, the ALPK1/TIFA signaling axis results in the initiation of an NF-κB-dependent pro-inflammatory immune response.
In our project, we plan to determine the role of ADP-heptose in the response of human myeloid and lung epithelial cells by selected strains of *A. baumannii*. As a first step, we plan to detect ADP-heptose delivery by *A. baumannii* to host cells and to evaluate NF-κB activation through its potential receptors: ALPK1 and TIFA. Next, we will assess the effect of ADP-heptose on the immune response, in particular on inflammasome activation. The expression of genes related to the inflammasome pathway and activation of inflammasomes protein concentration will be investigated. We will also study the effect of this nucleotide sugar *in vivo* in response to *A. baumannii* infection (pneumonia), which we will assess by determining the live bacteria in the lungs of infected mice, the variable number of cells and the concentration of selected cytokines.

By determining the effect of ADP-heptose on the host immune response, we expect to learn about the role of *A. baumannii* ADP-heptose and its receptor pathway in pulmonary immunity response to this pathogen. Thus, we hope to discover an important mechanism for host recognition of *A. baumannii* through PAMP detection, which in turn may indicate a new approach to the development of antimicrobial strategies through immunotherapy.