Human respiratory viruses: rhinoviruses (HRVs) and coronaviruses (HCoVs) cause 50-60% and 10-15% airway infections, respectively, in healthy individuals. They can pose a serious threat to patients with chronic respiratory diseases and evoke heavy asthma exacerbations. Studies show that the lungs vascular endothelium, after infection with HRV16 rhinovirus, produces a strong and rapid inflammatory and, above all, antiviral response. Additionally, the mechanisms of intracellular antiviral resistance are activated - increased expression of the enzymes: 2'5'-oligoadenylate synthetase (OAS-1) and protein kinase R (PKR). These The main aim of the project is to assess whether rhinoviruses may limit coronaviral infection of the lung vascular endothelium through the interferon-dependant mechanisms. Specific objectives of the project encompass: (1). The effect of HRV16 on IFN-dependant activation of 2'-5'-oligoadenylate synthase 1 (OAS-1) and protein kinase R (PKR) in the lung vascular endothelium. (2). The effect of HCoV229E on the inflammatory activation and cell death of the lung vascular endothelium. (3). The effect of HRV16 on the prevention and/or limitation of the infection of the lung vascular endothelium with HCoV229E. (4). The possible involvement of HRV16-induced OAS-1 and PKR-dependant mechanisms in the prevention and/or limitation of the infection of the lung vascular endothelium with HCoV229E.
We believe that HRV primary infection can protect endothelial cells from infection with the 229E coronavirus by dynamically activating the antiviral response. Better understanding of viral infection of endothelium will open novel possibilities of effective therapy based on intracellular antiviral machinery to prevent virus-induced exacerbation of respiratory diseases.