Description for the general public

The main aim of the project is to assess if respiratory virus such as human rhinovirus (HRV) may infect and directly act on human vascular endothelium, what effect HRV may exert on human vascular endothelium and finally what may be the significance for the development of allergic inflammation and airway remodeling. These processes are crucial for asthma development. Asthma exacerbations evoked by HRV lead to the development of inflammation within bronchial mucosa and airway remodeling which includes decreased epithelial integrity, basement membrane and smooth muscle cells thickening and increased bronchial vasculature. It is believed that HRV infections may facilitate airway remodeling and angiogenesis through the induction of proangiogenic and proinflammatory factors production by bronchial epithelium and immune cells. So far, the direct effect of HRV on human vascular endothelium has not been investigated. Results of the project will allow to answer the following questions: if HRV may infect vascular endothelium, thus influencing the development of allergic inflammation and angiogenesis and airway remodeling through the induction of chemokines/cytokines release and if in asthma patients, there is a higher susceptibility of vascular endothelium to HRV-induced generation of proinflammatory and proangiogenic factors as well as the development of allergic inflammation, angiogenesis and airway remodeling as compared to healthy people. Present therapeutic approach on asthma is based on the affecting of allergic bronchial inflammation initiated by the bronchial epithelium infection and damage. We believe that proving of the direct infection of vascular endothelium by HRV in the pathogenesis of inflammation and airway remodeling may lead to the development of novel therapies focused on the interactions of virus with vascular endothelium.