Our research focuses on the immunopathogenesis of respiratory syncytial virus (RSV) infections in early childhood, and the human immune defenses that protect the healthy infant from severe illness. RSV is the most common respiratory virus resulting in hospital admission in the first two years of life, and severe RSV infection in early life is associated with airway hyper-reactivity and asthma development later in life. The only preventive option is "RSV immunoprophylaxis" via passive immunization with palivizumab, a humanized monoclonal antibody specific to the RSV fusion protein. However, this therapy is very costly and we currently do not have the means to identify which apparently healthy infants are in fact at risk for severe RSV disease. Palivizumab prophylaxis is currently restricted to children with congenital disorders and those that are born very premature. The overwhelming number of RSV-related hospital admissions occurs in babies who cannot be identified as at-risk by available methods.

We study the complex virus-host interactions with an emphasis on human innate immune responses to RSV using primary blood mononuclear cells. One of the highlights of our research is the discovery that newborn infants and very young children are unable to mount robust innate antiviral immune defenses, which is mainly due to an impaired response of plasmacytoid dendritic cells in early life. In addition, we are investigating the role of common genetic variants (single nucleotide polymorphisms) in the outcome of pediatric RSV infections. A better understanding of the underlying immunological and genetic factors that determine the outcome of RSV infections is pivotal to clinical management and prevention of severe RSV infection in children. In the long term, we hope this research will help to find new strategies to identify children at risk, who may benefit from, but currently do not qualify for, RSV immunoprophylaxis.