Viral infections of the respiratory tract are the most common precipitants of acute asthma exacerbations. Exacerbations are only poorly responsive to current asthma therapies and new approaches to therapy are needed. Viruses, most frequently human rhinoviruses (RV), infect the airway epithelium, generate local and systemic immune responses, as well as neural responses, inducing inflammation and airway hyperresponsiveness. Using in vitro and in vivo experimental models the role of various proinflammatory or anti-inflammatory mediators, antiviral responses and molecular pathways that lead from infection to symptoms has been partly unravelled. In particular, mechanisms of susceptibility to viral infection have been identified and the bronchial epithelium appeared to be a key player. Nevertheless, additional understanding of the integration between the diverse elements of the antiviral response, especially in the context of allergic airway inflammation, as well as the interactions between viral infections and other stimuli that affect airway inflammation and responsiveness may lead to novel strategies in treating
and/or preventing asthma exacerbations. This review presents the current knowledge and highlights areas in need of further research.