Asthma is the most common chronic airway disease of childhood. Although most asthma cases can be well-controlled, patients with severe asthma show poor response to the treatment and thus suffer from chronic airway inflammation that leads to respiratory symptoms (e.g. coughing, dyspnea). These patients require high doses of anti-inflammatory drugs that generate multiple side effects, such as obesity, osteoporosis, and type 2 diabetes. Additionally, although only 10% of patients have severe asthma, this group accounts for up to 50–80% of all asthma-related costs. Therefore, new drugs are urgently needed for patients with severe asthma to control airway inflammation and prevent symptoms exacerbations. The possible treatment strategy involves the use of microRNAs that are responsible for silencing gene expression. The most promising candidate for miRNA-based therapy is miRNA-223-3p that control many proinflammatory genes. So far, the expression of miRNA-223-3p has been associated with neutrophilic immune response and has been analyzed mainly in *in vitro* cultures that, however, do not reflect cell-type specific interactions. Clinical samples or tissues from animal models, in turn, analyzed the bulk gene expression and thus lack the expression profile of individual cell populations. Therefore, the exact cell-type specific expression and the role of miRNA-223-3p in the pathogenesis of asthma still remain elusive. We hypothesize that miRNA-223-3p shows cell-type specific activity, and its decreased expression leads to chronic allergic inflammation in patients suffering from severe asthma. Identifying the role of miRNA-223-3p in different cell populations will, therefore, significantly increase the current state of knowledge of the pathogenesis of severe asthma. Therefore, we aim to identify genes that are regulated by miRNA-223-3p in the lungs and peripheral blood. For this, we plan to analyze the whole gene expression of individual cell populations in lungs and peripheral blood from asthmatic and control rats with and without miRNA-223-3p knockout. This will allow us to verify if the activity of miRNA-223-3p is distributed equally in different cell populations of lungs and peripheral blood. Additionally, it will also allow us to verify if, upon allergic inflammation, some cell types show disturbed expression of miRNA-223-3p-regulated genes. Furthermore, we aimed to administer a synthetic miRNA-223-3p analog to the lungs of allergic animals to verify the therapeutic potential of this miRNA. Although miRNAs were previously administered to animal models of asthma, no study so far used aerosol and functionalized nanoparticles as carriers. Synthetic miRNA-223-3p analog might be used in the future to develop an effective drug to reduce allergic inflammation and control clinical symptoms of severe asthma.