Chronic Obstructive Pulmonary Disease (COPD) is one of the most common chronic diseases worldwide. Based on the data gathered in the Malopolska region of Poland, it is estimated that approximately 22% of Polish population may suffer from this disease. The main cause of the disease is cigarette smoking, but toxic gases, fumes and, to a lesser extent, genetic predisposition also play a role.

Like every other disease, COPD does not have the same clinical course in every patient. In the 1990's researchers discovered that in the airways of more or less 40% of the patients suffering from COPD, despite being non-allergic, cells typical for allergic asthma can be found. These cells are called eosinophils.

Subsequent studies showed that this group of patients was more likely to require treatment in hospital due to severe worsening of symptoms. Risk of disease worsening can be reduced by using a group of medications called inhaled steroids. However, therapy of COPD patients with inhaled steroids may cause serious lung infection (pneumonia). Therefore, other therapies, without such side effects are needed.

Reasons for increased eosinophil numbers in the airways of COPD patients are unknown. One of the possible causes is activation of a layer of cells covering bronchi from the inside by cigarette smoke and infections. This leads to release of a group of substances which act by attracting and stimulating groups of cells that can turn into eosinophils, or intensify this transformation. These substances are called epithelial alarmins.

In order to study these phenomena, biological samples will be obtained from patients with COPD and healthy non-smoking and smoking subjects. These biological samples are blood and mucus coughed up from lower airways. In addition, a subset of study participants will undergo bronchoscopy, i.e. will have a thin, flexible tube inserted into their lower airways to collect small fragments of the inner layer of the bronchi. The levels of substances that may be involved in eosinophil accumulation in the airways will be measured in these samples. Moreover, in the laboratory settings, blood and airway cells will be stimulated with substitutes for viruses and bacteria to determine if infections may lead to rise in eosinophil numbers by increasing production of epithelial alarmins.

Understanding these mechanisms will allow development of new, safer and more effective ways to treat patients with COPD, resulting in improvement in their health and reduction of costs associated with frequent hospitalizations due to disease exacerbations.