Obesity has emerged as an important risk factor for inflammatory airway diseases. There has been a remarkable increase in the prevalence of obesity worldwide as a consequence of the modern eating habits associated with a sedentary lifestyle. Along with the growing prevalence of obesity and metabolic syndrome, a concomitant rise in the incidence of inflammatory airway diseases, such as, asthma, COPD, rhinitis has been observed in the last few years. Nevertheless, clinically, obesity is known to increase disease severity, impair the efficacy of medications, and worsening disease control in patients with inflammatory airway diseases.

Mechanisms underlying obesity-related inflammatory airway diseases are not well comprehended yet. Distribution and location of adipose tissue and its adipokine release are determinant factors influencing this correlation. Furthermore, hormones involved in glucose homeostasis and in the pathogenesis of obesity are likely to directly or indirectly link obesity and inflammatory airway diseases. Recently, clinical and basic studies highlighted the association between adipokines or insulin with pro-inflammatory mediators and showed that in accordance with other obesity-associated diseases, low-grade inflammation may be determinant for the pathogenesis of inflammatory airway diseases in obese patients. One key factor in clarifying the association between inflammatory airway diseases and obesity is to understand the intercommunication between the respiratory epithelium and the adipocytes, and to know how insulin and adipokines mediate this conversation.

Mucins play essential roles by regulating mucociliary clearance, protecting airway mucosa from bacteria and toxins, and by aiding the maintenance of airway hemostasis under normal and inflammatory conditions. Major secreted and membrane-bound mucins, are overexpressed in inflammatory airway diseases such as asthma, chronic bronchitis, chronic obstructive pulmonary disease, and rhinosinusitis. They lead to airway obstruction, increasing susceptibility to infection, and decreasing pulmonary function. In addition, it is also regulated by stimulating several inflammatory cytokines and hormones. Therefore, we focused on the relationship between obesity-related pathologic conditions (increased adipokines, hyperinsulinemia) and the regulation of major mucin genes expression. Through review of our previous studies, we want to talk about the changes in mucin gene expression in obesity, as one of the major causes of the severity of airway disease in obese patients.