The adipokine leptin: a pleiotropic molecule in the human respiratory tract

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Leptin, a 16-kd adipocyte-derived hormone originally described in metabolism regulation, plays a pleiotropic role in the immune system and inflammation.[1] Leptin exerts its action through the leptin receptor (Ob-R), present in several tissues, human respiratory tract included (Figure 1). Leptin is a survival cytokine for human neutrophils and eosinophils,[2,3] other than for other cytotypes, included lung carcinoma cells.[4] The following findings highlight the specific role of leptin both in the lung and in the nasal tract. We firstly find that *ex-vivo* leptin expression is increased and co-localized with lymphocytes T inflammatory cells, in bronchial mucosa of chronic obstructive pulmonary disease (COPD) patients and it is associated with COPD severity, airway inflammation and airflow obstruction.[5] On the other side, previous our *in vitro* and *ex-vivo* results show that the leptin/leptin receptor pathway is decreased in the bronchial epithelium of subjects with mild, uncontrolled, untreated asthma, whereas RBM thickness and TGF-beta 1 are increased, when compared with healthy volunteers.[6] In addition, in another our *in vitro* study, we assess that leptin increases adenocarcinoma cell line proliferation and the pathway with its receptor is increased by the flavonoid apigenin (4,5,7,-trihydroxyflavone).[7] Furthermore, our recent *in vitro* results report that the leptin/leptin receptor pathway is involved in human nasal epithelial homeostasis in allergic rhinitis and its expression is restored by Fluticasone Furoate in presence of the allergens.[8] In conclusion, in the submucosa, leptin might act as a cytokine-like mediator capable of playing a role in airway inflammation in chronic obstructive pulmonary disease with a potential impact on the severity of the disease; in the epithelium, the leptin/leptin receptor pathway is involved both in airway and in nasal epithelial homeostasis, in asthma and in allergic rhinitis, promoting also, in a cancer context, epithelial cell proliferation. Its expression decreases in subjects with uncontrolled and severe asthma and in presence of allergen exposure and is inversely correlated with airway remodelling, and cancer cell apoptosis.

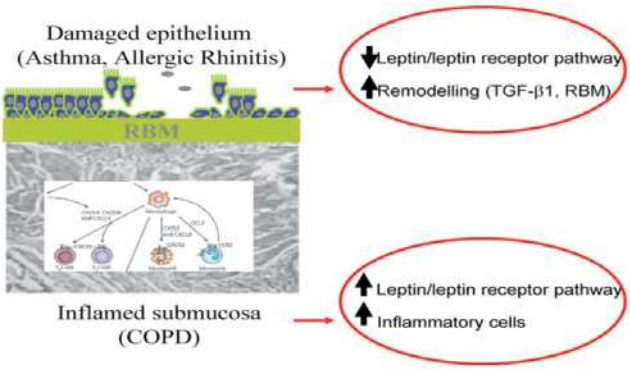

Figure 1. Leptin in human respiratory tract.

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