



**IDENTIFICATION OF NOVEL CELLULAR AND MOLECULAR PATHWAYS FOR THE DEVELOPMENT OF PERSONALIZED DRUG TREATMENT IN PULMONARY DISEASES**

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Pulmonary diseases such as asthma and pulmonary fibrosis (PF), are high-burden conditions for which the currently available treatment is inadequate in reversing or halting the progression of disease. Lung diseases have key sex- and gender-based differences with clinical implications that impact on incidence and severity. The molecular and cellular mechanisms driven this sex-related differences have not been fully elucidated. We have recently demonstrated that sex differences in arachidonic acid metabolism (e.g. leukotriene (LT) production) and in the balance between pro-fibrotic (TGFbeta, LT, etc) and anti-fibrotic (prostaglandins, miRNA214, miRNA96, etc) mediators are responsible, at least of part, of sexual dimorphism of asthma [1, 2] and PF, respectively.

This project will aim to further investigate the molecular and cellular mechanisms related to sex dimorphism responsible of onset and progression of lung diseases. This can help in identification of personalized drug strategy which lead to practical benefits such as decreased mortality and economic burden and improve the quality of life of citizens. To this aim in vitro (epithelial cells, fibroblasts, and macrophages) and in vivo models of asthma (allergen-induced asthma) and PF (bleomycin-induced PF) will be used.

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1. Rossi A, et al., Pharmacol Res. 20;139:182-190, 2019.
2. Cerqua I, et al., Pharmacol Res. 158:104905, 2020

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