**Identification of potential biomarkers useful in differentiation COPD from asthma**

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Chronic obstructive pulmonary disease (COPD) is a heterogenous disorder, resulting in progressive and irreversible lung ventilation alteration caused by exposure to cigarette smoke and inhaled toxic particles and gases. In COPD, chronic inflammation leads to the release of different inflammation mediators: chemokines, cytokines, growth factors, proteases and protease inhibitors.

In this study we investigate the possibility of using the above listed factors as markers in differentiating COPD from other pathological condition of the lung (e.g .asthma), or differentiate between different COPD patient categories.

Methods: Twenty-nine biomarker candidates were analyzed in the plasma samples obtained from controls, COPD and asthma patients by using Luminex magnetic beads based immune assays. The 29 analytes were divided into a 21plex, a 4 plex, a 2 plex and two single plex. The samples were incubated overnight with the beads and the analytes were quantified with a Luminex MagPix instruments.

Results: Out of the twenty-nine marker candidates analyzed in this study we found that the level of 13 inflammatory mediators: CCL-2, CXCL-1, CXCL-8, CXCL-9, CXCL-10, CXCL-11, FGF-a, MMP-8, MMP-10, MMP-13, SLPI, TNF-α, and TIMP-1 were significantly in COPD patients compared to the control group.

When we compered the level of these analytes between the control group and patients classified as GOLD1, GOLD2, GOLD3 and GOLD4 according to GOLD scale, we could not detect significant differences between the control and patient groups, or the analyte levels differed slightly.

The level of CCL-2, CXCL-8, CXCL-10, CXCL-11, MMP-13, SLPI and TIMP-1 differed significantly between the controls and asthma patients. But the level of these analytes did not differ significantly in COPD and asthma patients

Comparing the analyte levels of the COPD an asthma group, we could not detect significant differences between the two groups.

Conclusion: Further measurements are required as the number of patients enrolled in this study is still not sufficient to define the marker set which will be able to discriminate between “healthy” control and COPD patients with high accuracy.

We are also planning to verify other inflammation factors which be more useful in the classification of patients in categories according to the GOLD stage.

We have to analyze other inflammation mediators as well, because the selected twenty-nine inflammatory reaction components could not differentiate between COPD and asthma.

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