ECRONICON OPEN ACCESS

EC PULMONOLOGY AND RESPIRATORY MEDICINE

Editorial

Do we Understand Chronic Inflammatory Lung Diseases?

Michael Roth*

Pulmonary Cell Research/Pneumology, Department of Biomedicine/Internal Medicine, University and University Hospital Basel, Basel, Switzerland

***Corresponding Author**: Michael Roth, Pulmonary Cell Research/Pneumology, Department of Biomedicine/Internal Medicine, University and University Hospital Basel, Basel, Switzerland. E-Mail: Michael.roth@usb.ch

Received: November 15, 2018; **Publisshed:** November 19, 2018

Chronic inflammatory lung diseases including asthma, COPD and lung fibrosis are increasing worldwide with unknown reason [1]. Global initiatives such as GINA (asthma), GOLD (COPD) and GIFT (Global Initiative for Fibrosis Treatment) aim to develop global strategies for the diagnosis, management and prevention for the respective disease. Following new discoveries in clinical and basic research, the classification schemes for chronic inflammatory diseases were adapted over the years. However, the improvement of the therapies remains slow, because the etiology of the diseases is not well understood [2-4].

A major aim of the above named initiatives is to characterise each patient better, based on clinical and biological parameters to provide personalised medicine. Clinical parameters are the main tools to describe the disease phenotypes, and can be used to subgroup the patients within each disease for specific therapeutic strategies. Despite of many studies using "Big Data", no biomarker for the diagnosis and staging of chronic inflammatory lung diseases have been found. Surprisingly, many Big Data studies only confirmed biomarkers of chronic inflammatory lung diseases that have been known and used for years [5]. This indicates that perhaps we have missed some crucial mechanisms underlying the etiology of these diseases.

A major problem of Big Data studies on genomics, transcriptomics, proteomics etc. is the timing of the sample collection. In most studies, it had not been determined if the suggested biomarkers are stable, or to which extend they vary over the course of the disease. Future studies should take into account that biomarkers might be affected by the seasons, circadian rhythm, or life style [6,7]. Studies in asthma suggested that several biomarkers used for the phenotyping of patients were affected by gender [8]. Genetic, genome wide association, and epidemiological studies suggested a genetic predisposition for chronic inflammatory lung diseases and several susceptibility genes have been described for asthma and COPD. However, it remains unknown how these susceptibility genes lead to the disease [5,9].

The influence of life style and environment on the etiology of chronic inflammatory lung diseases is a relative new field of investigation. An increasing number of studies indicated that both can alter the mechanisms that regulate gene activity and protein function. Importantly, these effects in chronic inflammatory lung diseases could become irreversible, and be passed on to the next generation [10]. We are just starting to understand how these epigenetic events are working, for example, by the modification of the DNA structure or of histones; both alter the transcription of genes [11].

It remains an opened question if different environmental factors (triggers) lead to the same pathogenesis, or if what we defined as asthma or COPD has to be further classified. Today, these diseases are mainly defined by their clinical symptoms. This view might be challenged by the results of ongoing Big Data and Omic studies, putting the origin of the diseases in the focus. Future studies have to answer the question if biomarkers or epigenetic events are causative or the results of the disease.

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Volume 7 Issue 12 December 2018