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The seventh Scandinavian COPD Research Symposium was held on 18–19 November 2016 at Scandic Holmenkollen Park Hotel, Oslo, Norway. Since the first meeting in 2004, the purpose of these Symposia have been to allow young researchers from Denmark, Finland, Norway and Sweden to present their research, thereby also facilitating networking, collaboration and stimulating future research in the field of COPD. Ten young scientists from our countries presented their research, and three state-of the art lectures covered the areas Biomarkers and Inflammation in COPD, E-cigarettes and COPD, and Imaging in COPD. Within the topics of each state-of-the-art the lecturers held in-depth discussions with the participants on the second day of the meeting. The meeting was generously supported by grants from Boehringer Ingelheim, which also made publication of this supplement to European Clinical Respiratory Journal possible.

State-of-the art abstracts

Biomarkers and inflammation in COPD

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There is a need for biomarkers in COPD – in order to evaluate disease activity, to characterise disease phenotypes and endotypes and to evaluate risk of exacerbations and complications such as comorbidities. Not surprisingly, markers of systemic inflammation have been the target of research as COPD is seen as a disease where inflammation has been part of the definition until now and where easily accessible biomarkers are available.

The main biomarkers studied have been fibrinogen hs-CRP, white blood cell count and differential count (eosinophils), as well as by-products resulting from inflammation, not least markers of matrix remodelling and breakdown. Most studies have shown that markers of systemic inflammation have independent prognostic value regarding exacerbations, hospitalisation and mortality, although studies with fairly dubious associations can be found. As a result of studies on fibrinogen, the US Food and Drug Administration (FDA) has decided to approve fibrinogen as the first biomarker in COPD for enriching controlled trials using exacerbations as primary outcome. None of these markers of systemic inflammation seem to reflect ongoing disease activity (when measured as FEV1 decline), but design of studies of FEV1 decline have inherent biases as a result of the variety of trajectories that can lead to clinical COPD. Many of the associations found may also reflect the well-known relationship between smoking and systemic inflammation. Lately, eosinophils have been seen as ‘the new black’ in COPD research. Fairly limited prospective data exist to evaluate the value of eosinophils in predicting response to inhaled (and systemic) corticosteroids in COPD. The issue of determining relevant cut-offs is also unresolved.

Systemic inflammation is now known to be present in a subset of COPD patients, to be associated with poor prognosis, but is not associated with subsequent FEV1 decline. Other biomarkers may have larger potential in this respect. Overall, this research field is still fairly open.

E-cigarettes and COPD

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