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From the authors:

We thank C. Persson and L. Uller for their interesting correspondence regarding our recent research paper on the link between systemic and airway eosinophilia and asthma control [1].

As there has been a huge controversy on the role of eosinophils in asthma, the purpose of our paper [1] was to report our clinical experience on the relationship between eosinophils and asthma. In our study the link between eosinophil counts and asthma control is significant but not so strong, which is in keeping with the concept of concordant and discordant disease when relating symptoms to inflammation [2]. In fact only airway eosinophilia was directly associated with poor Asthma Control Questionnaire, with an r coefficient of 0.16, while the blood cell count did not. As those patients who combined high blood and sputum eosinophils had worse asthma control, our interpretation is that blood eosinophils contribute to mount an intense airway eosinophil infiltration. The role of airway eosinophils in poor asthma control is further supported by the fact that they contribute in determining the level of bronchial hyperresponsiveness, a hallmark of asthma pathophysiology [3, 4]. Having said this, we entirely agree that just looking at cell counts does not provide a complete picture of the cell role in pathophysiology. As strongly suggested and convincingly argued by C. Persson and L. Uller, primary eosinophil lysis in the airway is likely to be an essential contributor to the intensity of airway eosinophilic inflammation and, thereby, poor asthma control. Therefore, the relationship with asthma control could have been stronger if we had looked at eosinophil activation. As stated in our paper we also recognise that some patients who were called non-eosinophilic, based on eosinophils contained in the airway lumen, may have been misclassified due to the persistence of eosinophils in the airway wall and engaged in the scavenging process by macrophages [5].

We would like to emphasise again that behind the results reported here our strategy was to really improve asthma care and management in a university hospital. Very much influenced by the letter by PAVORD *et al.* [6] and the study by GREEN *et al.* [7], 10 years ago we set up an asthma clinic in which we embarked on inducing sputum as a routine practice to monitor eosinophilic inflammation and adjust inhaled corticosteroids (ICS) prescription accordingly. For centres experienced in using induced sputum the recent European Respiratory Society/American Thoracic Society guidelines recommends that induced sputum and clinical criteria be used to guide treatment in adults with severe asthma, rather than by clinical criteria alone [8]. Although detailed treatment adjustment based on sputum cell count was left at the discretion of the clinician, we found in our prospective cohort a reduction in exacerbation rate by 42% (from 0.86 per patient per year to 0.50 per patient per year) in the year following the visit to asthma clinic. Though we are aware this finding has to be interpreted with caution because of the recall bias, we feel that it is reassuring, even more so it was not subordinated to a huge increase in ICS prescription. In addition it was reassuring that the patients for whom the clinician did not feel the need to prescribe an ICS after their asthma clinic visit, did not report any exacerbation in the following year. This suggests that using the mere cell count to assess inflammation in clinical practice may still carry some value and help phenotype asthma patients [9]. This assumption was indeed further supported by the DREAM study results where the efficacy of mepolizumab was partly dependent on the blood eosinophil count [10].

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Assessing airway and blood eosinophilic inflammation in clinical practice helps to phenotype asthmatics http://ow.ly/zdhyE

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Manuscript cited in "Specific inhalation challenge in the diagnosis of occupational asthma: consensus statement"

To the Editor:

On page 1579 of the manuscript entitled "Specific inhalation challenge in the diagnosis of occupational asthma: consensus statement", a consensus statement published in the June 2014 issue of the *European Respiratory Journal*, it is stated: "…the duration of exposure should preferably be increased from one day to the next with, for instance, a cumulative exposure limited to <30 min on the first challenge day," after which reference numbers 11, 13 and 57 are cited [1].

I am the co-author of reference 11 and in this text we say, in table 2 (page 234), to limit exposure to 1 min on the first day, 5 min on the second day and 30 min on the third day [2].

So, our reference was wrongly quoted. Exposing subjects for 30 min on the first day can lead to severe reactions, which can explain the relatively high frequency of such reactions as referred to in the paper referenced as 57 [3].

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