

between the alveoli and the mouth. Therefore, in EBC samples, not only volatiles, but also several other mediators with no volatile characteristics can be found and have been reported, including adenosine, different interleukins (-4, -5, -8), interferon- γ , *etc.* [3–5]. Regarding markers of lipid peroxidation, EBC contains isoprostanes and thiobarbituric acid-reactive substances [2].

The authors are right in saying that there are methodological limitations to this type of sampling. However, this is mainly due to the limited understanding of solute formation and dilution of samples, and the accuracy of some of the currently available methods for measuring mediators in EBC. I agree that ambient air may influence the levels of exhaled biomarkers in EBC and this is shown for hydrogen peroxide [6]. Volatility may be a problem when measuring mediators from EBC, not because the sampling relies on this characteristic, but because if a molecule is volatile it is very hard to figure out the result of its equilibration between the gas and the fluid phase while breath condensation is ongoing. A good example is the ammonia measurement [7, 8].

Despite the misinterpretation of exhaled breath condensate, I believe that this review is a valuable source of knowledge and references on lipid peroxidation, with detailed information on the limitations and advantages of the current measuring methods.

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From the authors:

We thank I. Horváth for his kind comments on our review "Biomarkers of lipid peroxidation, airway inflammation and asthma" [1], and agree that we have been imprecise with the use of the term "breath condensate". The paper would be improved by replacing this term with "exhaled breath" in relation to ethane, pentane and nitric oxide measurements.

While there is an intuitive explanation for the presence of volatile substances in exhaled breath, the mechanisms by which nonvolatile substances enter expired breath are poorly understood and need to be further investigated. 8-iso-prostaglandin F_{2 α} and malondialdehyde have been measured in breath condensate as markers of lipid peroxidation [2]; however, it is, as yet, unknown whether this medium can be used for reliable measurement of antioxidant defences. Analysis of total and oxidised glutathione concentrations in induced sputum indicates that sputum supernatant is suitable for this purpose [3]. Hence, we stand by our conclusions in this area.

At the moment both sputum induction and breath condensate collection are promising techniques. The most useful sampling technique remains to be determined and this is an important area of future research. Comparison of both sampling methods in a head-to-head study is needed to resolve this issue.

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Noneosinophilic asthma

To the Editors:

We read with interest the article by BUIST [1] on similarities and differences between asthma and chronic obstructive pulmonary disease. We would like to make some comments on the nature of inflammation in asthma, which the author has

mentioned to be predominantly eosinophilic. Patients have been noted to have severe asthma or suffer an exacerbation without an increase in the eosinophil population in the airways [2]. Based on several studies from 1995 onwards with data on eosinophil levels (cut-off values 2–4%) on bronchial biopsy specimens, bronchoalveolar lavage fluid and sputum of asthmatic