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From the authors:

We thank R.P. Young and co-workers for their comments on our recent article [1]. As we highlighted in our article, there are two recent reports indicating that single-nucleotide polymorphisms in the advanced glycosylation end product-specific receptor (*AGER*) gene, which encodes the receptor for advanced glycation end-products (RAGE), are associated with changes in measurements of airflow obstruction [2, 3]. The findings reported by R.P. Young and co-workers in their correspondence add to these earlier studies and shed light on the genetic basis by which cigarette smoke exposure leads to chronic obstructive pulmonary disease (COPD) in some individuals, while "resistant smokers" maintain normal lung function.

Our finding that circulating levels of soluble RAGE (sRAGE) are lower in COPD subjects than healthy controls has since been reproduced in a study reported recently by MINIATI *et al.* [4]. Within an individual, circulating levels of sRAGE may be determined by polymorphisms in the *AGER* gene, but are also susceptible to environmental factors, especially as plasma sRAGE levels are very low during acute exacerbations of COPD and rise during convalescence [1]. There is now a need for longitudinal studies to define the relationship between polymorphisms in the *AGER* gene and circulating levels of sRAGE in patients with COPD, and to assess the extent to which this predicts rate of decline in lung function over time.

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Can dog allergen alone, if combined with indoor pollution, be responsible for asthma in children?

To the Editors:

We read with interest the article by Carlsten *et al.* [1] showing the increasing risk of incident asthma in a high-risk birth cohort after early co-exposure to dog allergen (Can f 1) and nitrogen dioxide (NO_2) or environmental tobacco smoke. The topic is highly relevant because most studies on the interaction between allergens and air pollution regard outdoor environments and very few articles have been published on the possible allergen–pollutant relationship in indoor places.

Nevertheless, we think that other limitations to the study should be considered in addition to those already acknowledged by the authors. In their study, they referred to the article of McConnell *et al.* [2] that showed a significant association between "bronchitis symptoms" and particulate matter only in the subset of asthmatic children who owned dogs. However, McConnell *et al.* [2] examined the relationship of both dog and cat ownership with air pollution, and reported that effects

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