places (schools, day care centers, means of transport, etc), passive transfer constitutes the exclusive modality of dog or other animal allergens contamination.[4] Finally, several studies have shown that amounts of pet allergens passively transferred in pet-free environments are of sufficient magnitude to induce allergic sensitization in susceptible, atopic individuals and triggering allergic symptoms in already and highly pet-sensitized subjects.[5]

In the article of Vredegoor et al, the authors have not considered this particular aspect. In other words, dog owners of both groups frequently or intensively exposed to other dogs elsewhere should not be included in the study or, alternatively, their clothes should be removed or washed before entering indoors.[6,7] It is likely that considerably amounts of Can f 1 found in indoor environments (on the floor and in the air) do not belong to dogs who live in that environment. This possibility is a crucial point in the hypoallergenic dog group because *in vitro* assays are not able to distinguish Can f 1 produced by the hypoallergenic dogs from Can f 1 produced by unknown dogs in other settings (and not necessarily from hypoallergenic breeds) passively transferred indoors. Exactly inverse considerations can be done for indoor environments of nonhypoallergenic dog breeds potentially contaminated by Can f 1 produced by unknown hypoallergenic dogs passively transferred indoors.

In conclusion, the evaluation of Can f 1 amounts indoors without considering the percentages of allergen carried at home through clothing or other items constitutes a serious bias in defining some dog breeds as hypoallergenic.

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Reply

To the Editor:

We thank Liccardi et al[1] for raising an interesting topic for discussion—the issue of passive transport of allergens. The topic was raised because of some findings described in our study on Can f 1 levels in so-called hypoallergenic dog breeds.[2] Passive transport and transfer of allergens from one to another environmental compartment is indeed an underestimated source of allergen exposure that can trigger allergic symptoms, even in environments in which the primary source of the allergen is absent.[3] However, we believe that passive transfer did not dramatically influence the results of our study.

We did not attempt to establish possible passive transfer of allergens in our study. However, we did ask participants whether their homes were visited by other dogs during the 28-day period of exposure measurements. The homes that were visited by other dogs, and had additional primary allergen sources for a short period in the home, had no significantly higher Can f 1 levels than did homes that were not visited by other dogs (Table I). This suggests that the main source of allergen exposure in a house is the dog living there.

Second, by analyzing material directly taken from the dog,[2] we have shown that so-called hypoallergenic breeds produce as much Can f 1 as the nonhypoallergenic breeds. This similar production is reflected in the results of the environmental sampling, suggesting that the environmental exposure is not dramatically influenced by an external allergen source.

How much dog allergen present in a house is a result of passive transfer? A study by Egmar et al[4] reported in homes without dogs no differences in Can f 1 exposure between people who had dog contact and people who had not. The same study showed that homes with dogs had Can f 1 levels that were on average 300 times higher. In an ongoing study on exposure in homes (with and without pets) and schools in our group, we found that the pet allergen load in classrooms is higher than in homes without pets, but significantly lower than in homes with pets (manuscript in preparation). The classrooms studied were daily occupied by children of which up to 65% had a dog. Considering these findings, we believe that a dog, as a primary source of dog allergens, contributes much more to the environmental allergen burden found in homes

TABLE I. Can f 1 levels (geometric mean)

	Homes visited by other dogs	Homes not visited by other dogs	*P* value
No.	45	105	
Can f 1 in floor dust (μg/g)	26.4	32.8	.353
Can f 1 in floor dust (μg/m^2)	10,123	10,120	1.000
Can f 1 in settled airborne dust (μg/m^2)	6.6	5.9	.594

than does passive transfer from public spaces to the home environment. The additional contribution of passive transfer would not have altered our results significantly.

In conclusion, we agree with Liccardi et al[1] that passive transfer of pet allergens occurs and can contribute to total exposure, but we believe that a dog living in the house is the primary source of, and major contributor to, Can f 1 exposure. For people not living with a dog in the house, passive transfer of Can f 1 allergen will be a relatively more important source of allergen exposure.

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The crucial task of defining a healthy immune response toward respiratory infections

To the Editor:

In a recent publication, King et al[1] obtained heterogeneous populations of total primary lung cells from patients with chronic obstructive pulmonary disease (COPD) and smokers with normal lung function and stimulated them *ex vivo* with a common bacterium in COPD, that is, nontypeable *Haemophilus influenzae* (NTHi). These assays mainly showed that resident lung T cells of patients with COPD produce more TNF-α and IL-13 than do those of smokers without COPD, albeit similar IFN-γ.[1] The authors conclude that this type of T-cell–derived cytokine response promotes inflammation and impedes bacterial clearance in COPD. However, some issues are worth commenting on.

First, most of the T cells that reside in the lungs are memory T cells, which respond more rapidly and vigorously to their cognate antigens than do naive T cells.[2] NTHi is a common bacterium in COPD, and NTHi-specific memory T cells, expected to exist in the lungs of patients with COPD but not in the lungs of healthy subjects, will be stimulated by NTHi only in patients with COPD. Second, the lungs of patients with COPD contain higher numbers of antigen-presenting cells (APCs), such as macrophages, B cells, and langerin$^+$ dendritic cells, than do the lungs of healthy subjects.[3] The higher APCs-to-T cells ratio in patients with COPD may cause increased cytokine production by lung T cells. This bias could be overcome if lung T cells were isolated and stimulated by NTHi at a single APC-to-T cell ratio for all subjects.

Despite the fact that the study designed by King et al was biased toward increased stimulation of lung T cells in COPD, the master effector cytokine of T helper cells, that is, IFN-γ, was produced at similar levels between patients with COPD and smokers without COPD. This was unexpected, as the numbers of IFN-γ expressing T_H1 cells are higher in COPD. However, experimental respiratory rhinoviral infection induces lower pulmonary IFN-γ levels in patients with COPD than in healthy smokers and secondary bacterial infection.[4,5] Furthermore, animal emphysema models and *in vitro* functional immunologic assays suggest that cigarette smoke possesses proinflammatory, yet immunosuppressive properties.[6] Taken together the data obtained by King et al with these data underscore the notion that antigen-specific lung T-cell responses are less efficient in clearing pathogens in patients with COPD than in healthy subjects.

But the question that we as respiratory physicians are really interested in answering is "Do patients with COPD get sicker because their lung T-cell responses toward respiratory pathogens fail to clear pathogens?" A scanty immune response would increase vulnerability to pathogens, yet an overly immune response could cause damage.[7] Whether scanty T cell–mediated antigen-specific immune responses in patients with COPD are "unhealthy" or whether they represent a mechanism used by the respiratory system to reduce the impact of persistent/frequent respiratory infections would be an excellent area of future research.

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