

that cross-reactivity between these allergens may not be critical in our study population. However, confirming this would require inhibition studies using the sera of workers sensitized to these different allergens.²

Studies in patients and animals have indicated that parasites can alternately decrease or increase atopy and/or allergies, depending on variables such as parasite load, species, and the time kinetics of the infection.^{3,4} In the study by Daschner et al,⁵ a negative association was found between acute parasitism by *Anisakis simplex* and allergic sensitization to aeroallergens. This is in line with the hygiene hypothesis, which suggests that infections by pathogens such as helminths may reduce the incidence of allergic disease. In contrast, we did not find a negative association between *Anisakis* sensitization and sensitization to aeroallergens. As Daschner¹ suggests, this may illustrate the importance of the route of sensitization, because the former study was conducted among patients with acute parasitism after ingestion of parasitized fish, whereas our study focuses on subjects who may have been exposed to *Anisakis* proteins in various ways: through ingestion, skin contact, or inhalation as well as previous infection with live larvae. Such variables may influence the subsequent response to other allergens in various ways, which are currently being investigated in our laboratory.

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Eosinophilic bronchitis and asthma with sputum eosinophilia are different entities

To the Editor:

We read with interest the article "Eosinophilic bronchitis in asthma: a model for establishing dose-response and relative potency of inhaled corticosteroids,"¹ wherein the authors demonstrate that sputum eosinophilia of more than 2.5% in patients with uncontrolled asthma could be a useful model for assessing and performing dose-response and relative potency studies on different inhaled corticosteroid compounds and delivery devices. However, we disagree with the use of the term *eosinophilic bronchitis* (EB) for describing sputum eosinophilia in asthma. Airway inflammation with eosinophils is now reported to occur not only in asthma but also in other airway diseases, such as chronic cough, atopic cough, episodic symptoms without asthma, allergic rhinitis, and chronic obstructive pulmonary disease.² Eosinophilic inflammation exists in central and peripheral airways in both asthma and EB and has been demonstrated on the basis of biopsy and bronchoalveolar lavage fluid results.² Whether EB represents a distinct clinical condition or is a precursor of asthma is unclear, but EB differs from asthma in several important ways.³

In comparison with asthmatic patients, those with EB experience the symptom of cough alone and usually have normal lung function and airway responsiveness.^{2,4} Although both conditions are associated with reticular basement membrane thickening and infiltration with similar numbers of subepithelial T lymphocytes, mast cells, and macrophages,⁵ infiltration of airway smooth muscle by mast cells that express IL-4 and IL-13 is a feature of asthma but not of EB.^{2,6} Also, the production of vascular endothelial growth factor and airway permeability are increased in asthma but not in EB, and this might explain the airway hyperresponsiveness in patients with asthma but not in those with EB.⁴ In contrast to asthma, the most common outcome in EB is continuing disease, and complete resolution is rare, with female sex, smoking, and prolonged eosinophilic airway inflammation being predictors of a rapid decrease in FEV₁.⁷ Until the pathophysiology and long-term outcome of EB are fully characterized, it would be better not to use these terms interchangeably to avoid further confusion of terminology.

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Reply

To the Editor:

We thank Drs Agarwal and Gupta¹ for their correspondence regarding our article entitled "Eosinophilic bronchitis in asthma: a model for establishing dose-response and relative potency of inhaled corticosteroids."² The issue they raise is one of semantics. By suggesting that our use of the term *eosinophilic bronchitis in asthma* might be confusing to the reader, they demonstrate the adoption of an essentialist rather than a nominalist philosophy. Essentialists use words in an abstract fashion to imply that diseases be classified in a logical sequence of mutually exclusive categories and talk of diseases as if they exist as real entities. In this case the *words* eosinophilic bronchitis are displaced by the *concept* of eosinophilic bronchitis as a disease. We believe that essentialist definitions have no place in science.

We prefer to use a nominalist approach in science, in which words are used to mean what they say and not refer to abstract concepts. The original description of eosinophilic bronchitis without asthma came from our group in Hamilton,³ and we continue to hold the view that *eosinophilic bronchitis* is a nominal term used to describe an eosinophilic cellular infiltrate of the bronchial wall. As such, the term is not specific to any single disease entity and can occur in asthma (most commonly),^{4,5} occupational lung disease,⁶ smoking-related chronic obstructive pulmonary disease,^{7,8} or a number of other airway inflammatory conditions.

Induced sputum analysis measures inflammatory cells within the airway lumen. To get to the lumen, cells need to transgress through the airway wall, and the presence of sputum eosinophilia is therefore an appropriate measurement to detect the presence of eosinophilic bronchitis.⁹ Drs Agarwal and Gupta apparently failed to appreciate that the original publication from our group,³ as well as

subsequent publications^{10,11} on the entity of eosinophilic bronchitis in patients with chronic cough, normal spirometric results, and airway responsiveness used the term *eosinophilic bronchitis without asthma* to characterize these patients.

The use of the term *eosinophilic bronchitis in asthma* in our recent study is justified on the basis that asthma is characterized by the presence of variable airflow limitation, airway hyperresponsiveness, and eosinophilic airway inflammation. In this study the primary outcome was a decrease in sputum eosinophil counts as a surrogate marker of eosinophilic bronchitis. Changes in spirometric results, airway hyperresponsiveness, exhaled nitric oxide levels, or patient symptom scores, although all valid alternative outcomes in asthma trials, were not the primary outcome in this study. We therefore believe that the title of our article, "Eosinophilic bronchitis in asthma: A model for establishing dose-response and relative potency of inhaled corticosteroids," is nominally accurate, clinically appropriate, and devoid of any confusion to the reader.

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