

the two could not be identical. Especially for our purpose, the placebo encasings were made of tight woven cotton of a good quality, and according to the manufacturer they looked very much like the active encasings. All encasings were delivered directly from the manufacturer to the patients, none of the patients had ever had encasings before, and none of them knew which kind of encasing they had. The parents did the dust sampling themselves; the investigators never came to the patients' homes and never saw the encasings. Thus, the blinding was complete and no breakage of the blinding occurred.

The randomization is described in the Design section of our article. The intended matching was successful. In addition, because we compared changes from baseline and did not restrict the analyses to direct comparisons between the 2 groups, it was not critical whether the 2 groups were completely identical.

We regret that our description of asthma symptom scores has led to misunderstanding. The maximum daytime asthma symptom score was 3 per day; likewise, the maximum nighttime score was 3 per night. This means that for a 2-week period the possible maximum daytime score was 42 and the possible maximum nighttime score was also 42. Each of the reported mean daytime scores (3.33 and 1.62) describes the mean of the *total* (the sum) of the symptom scores during the 2-week period. These figures are not statistically different from each other.

The data presented in the previous poster were preliminary data, and we later found minor errors in the calculations. Because of Journal requirements, we had to limit the amount of information in our article, and we were therefore unable to include data on PEF values or lung function. As described, no significant differences were found between the 2 groups with respect to PEF or FEV<sub>1</sub>, either at baseline or during the follow-up period, though a greater, not statistically significant, improvement was seen in the active intervention group. The baseline data on house dust mite (HDM) allergen concentrations was presented in our Table I.

The refereed large study by Custovic et al<sup>3</sup> was published only as an abstract, and it is difficult to compare their results with ours. Only 65% of their patients were mite-sensitive, and because there was no effect on the mite exposure, a clinical effect could hardly be expected.

Our study was a double-blind, controlled study that included a sufficient number of children with documented asthma and HDM allergy, and for the first time it was proved by bronchial challenge with HDM allergen extract that the patients had asthma caused by HDM allergy. And a 1-year follow-up adjusts for seasonal variation. We found both a significant perennial reduction in HDM allergen exposure and a significant clinical relevant reduction in the need for inhaled steroids. We agree that mattress and pillow encasings should not be a standard recommendation for all patients with asthma, but we find that our study documents an effect of allergen-impermeable mattress and pillow encasings in children with asthma and proven HDM allergy.

In our opinion, it makes no sense to include measures without a proven effect on HDM exposure in a review on

the effect of HDM control measures for asthma. In addition, it seems evident that patients included in such studies should have proven HDM allergy causing asthma symptoms. We therefore hope that this will be considered in future studies and reviews.

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#### Clarification of terminology in patients with eosinophilic and noneosinophilic hyperplastic rhinosinuitis

##### To the Editor:

The article by Steinke et al<sup>1</sup> looks at 58 patients undergoing elective sinus surgery and analyzes the histopathology for expression of cysteinyl leukotrienes (CysLTs). Throughout the article, I found their terminology for the 3 populations analyzed confusing. The first group (n = 32), patients with chronic hyperplastic eosinophilic sinusitis (CHS), was the subgroup that showed significantly greater expression of CysLTs. Why was the "E" left out of the abbreviation? It would be clearer throughout the article if these patients were referred to as CHES. In the title the author's leave "eosinophil" out of chronic hyperplastic sinusitis-nasal polyposis but subsequently refer to the subgroup of chronic hyperplastic sinusitis without eosinophils as patients with noneosinophilic polyps (n = 14). Does the title refer to patients with polyps regardless of the presence of eosinophils? From the data presented, I interpreted that these 2 polyp groups, those with and without eosinophilia, are clinically indistinguishable, and histopathologic evaluation was required to subdivide the groups into those with and without eosinophilia because it could not be done clinically preoperatively.

I suggest that a more straightforward terminology would refer to these 2 subgroups of patients with chronic hyperplastic rhinosinuitis with polyps as eosinophilic chronic hyperplastic rhinosinuitis (ECHRS) or, if you like, chronic hyperplastic eosinophilic sinusitis (CHES), whereas those patients with polyps but without eosinophilia could be designated as

noneosinophilic chronic hyperplastic rhinosinusitis (non-ECHRS). These are just suggestions, and I am sure there are a number of ways to designate these patients more clearly than the abbreviations used in the current article.

Finally, the authors designate the third group as control subjects that provided "healthy control sinus tissue" obtained at the time of surgery from the ostiomeatal complex. In the "Methods" section the authors state that polyp tissue was obtained from subjects (n = 58) referred to the University of Virginia Health System for sinus surgery. Actually, there were 32 ECHRS subjects, 14 non-ECHRS subjects (total of 46 polyp subjects), and 12 control subjects. I would be reluctant to designate these control subjects as having "healthy sinus tissue" because presumably, as listed in the "Methods" section, they had "a medical history consistent with chronic sinusitis ... and documented disease in their sinuses, as shown through computed tomographic scanning." I suggest that these patients are not healthy control subjects; rather, they are patients with non-polypoid chronic rhinosinusitis. However, I am confused by the inclusion criteria in the "Methods" section and the authors' referral to these patients as healthy control subjects and ask for clarification.

I agree with the authors that better strategies of subgrouping patients with chronic hyperplastic rhinosinusitis are required, and a basic separation on the basis of histopathologic presence of eosinophils is supported by the reduced presence of CystLT expression in the noninfectious, noneosinophilic subgroups compared with the ECHRS group.

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#### Reply

##### To the Editor:

Dr Ferguson<sup>1</sup> clearly delineates the need for a consistent nomenclature in addressing the study of chronic sinusitis in her comments on our article.<sup>2</sup> Historically, sinusitis was divided into 3 categories on the basis of disease duration, with *chronic sinusitis* referring to patients whose symptoms were of greater than 6 weeks' duration. At the time this terminology was developed, all sinusitis was thought to be infectious, and this led to the prominent use of antibiotics and surgical drainage as treatment. It is now clear, however, that the majority of patients with chronic sinusitis do not have an infectious disorder, and this has led to the need to develop more appropriate terminology to describe the myriad of conditions that make up chronic sinusitis.

There are 4 major pathophysiologic processes responsible for chronic sinusitis. A small subset of these patients does, in fact, have *chronic infectious sinusitis*. This typically involves patients with underlying humoral immune deficiencies, HIV, Kartagener syndrome, and cystic fibrosis. In contrast, most patients with chronic sinusitis have an inflammatory disorder with prominent hyperplasia of immune cells. *Chronic inflammatory sinusitis* is thought to result from chronic or recurrent occlusion of the sinus ostia caused by viral rhinitis, allergic rhinitis, anatomic predisposition, or other causes. These processes lead to recurrent acute bacterial infections, possibly in association with barotrauma of the sinus cavities and damage to the respiratory epithelium, ciliary destruction, mucous gland and goblet cell hyperplasia, bacterial colonization, and, ultimately, the chronic inflammatory changes. Eosinophils are not a feature of chronic inflammatory sinusitis, although this disorder might produce nasal polyps. Chronic inflammatory sinusitis is generally responsive to surgical interventions. Because it is defined by the prominent expression of eosinophils and to help distinguish it from chronic inflammatory sinusitis, the other immune inflammatory disease perhaps should be referred to as *chronic hyperplastic eosinophilic sinusitis*, as suggested by Dr Ferguson. This disease is frequently associated not only with nasal polyps but also with asthma, atopy, aspirin sensitivity, and, as described in our article, the overproduction of cysteinyl leukotrienes. We believe that chronic hyperplastic eosinophilic sinusitis represents the majority of patients seen with chronic sinusitis, and in contrast to chronic inflammatory sinusitis, it does not respond well to surgery. The final condition associated with chronic sinusitis is *allergic fungal sinusitis*. This presumably represents a severe variant of chronic hyperplastic eosinophilic sinusitis associated with the colonization of fungi within the sinus cavities and the presence of an IgE- and T<sub>H</sub>2-like lymphocyte-mediated allergic inflammatory response.

Although commonly used, we believe that the term *rhinosinusitis* is problematic and should be avoided. This term is derived from the use of clinical criteria to diagnose sinusitis, including such symptoms as nasal congestion, rhinorrhea, and purulent nasal drainage, criteria that clearly reflect the presence of nasal and not sinus disease. The problem with this approach is made clear by studies that use objective criteria, such as the computed tomographic scan, that have established that these clinical criteria are little better than random in predicting the presence and severity of sinusitis.<sup>3-5</sup> This lumping of rhinitis and sinusitis in the same term also leads to the misconception that this is a single disease. Although rhinitis might contribute to the development of sinusitis, especially acute infectious and chronic inflammatory sinusitis, chronic rhinitis and chronic sinusitis are clearly quite distinct disorders with unique pathologies, and therapies and we believe these terms should remain separate.

To clarify Dr Ferguson's last question, our control tissue consisted of sinus tissue obtained from the ostia of