

drug efficacy, and that our study was designed so as not to uncover differences between montelukast and beclomethasone. However, the situation is quite the opposite. Meta-analyses can serve as secondary sources of information when clinical trials of adequate size are lacking. They are clearly inferior to prospective, well-designed studies of adequate size. Our study was rigorously designed, randomized, placebo controlled, and conducted to compare the treatment effects of montelukast and beclomethasone on control of asthma using previously validated end points.² In addition, our study included a placebo group to verify that the trial had assay sensitivity (ie, the ability to detect a difference between treatments if it was present). Both drugs were superior to placebo in all end points measured. What this study adds to the literature is an approach that allows data from individual patients to be reflected in the treatment comparisons. This approach provides physicians far more information about the effects of a drug than can be gleaned from a mean value alone. By comparing the distribution of responses in the 2 treatment groups, we were able to provide assurance that the similarity in treatment effect is indeed present in the entire population.

The writers suggest that the study was of short duration. They note that the onset of action of inhaled steroids is slower than that of montelukast and are concerned that this might have influenced the result. It did not. As they suspect, there was an advantage of montelukast initially; however, the similarity in treatment response was present from week 1 onward (see Figure 2 in our original article). In addition, both drugs improved the proportion of days that patients were rescue free (increase of 17.6% for both montelukast and beclomethasone), a measure also reported in previous studies of asthma.³

Finally, Stemple and Stoloff note that our patients had moderate asthma and would have benefited from combination asthma therapy. We agree. However, the purpose of this study was to compare the effects of montelukast and beclomethasone on daily asthma control in patients not on controller therapy. It is clear from our results that montelukast and beclomethasone each provide improved asthma control in such patients. We refer the correspondents to other studies that have demonstrated the additive effects of montelukast and inhaled corticosteroids^{4,5} and agree that combination therapy is a reasonable strategy for such patients.

Elliot Israel, MD

*Director of Respiratory Therapy and Pulmonary Clinical Research
Brigham & Women's Hospital
Associate Professor of Medicine, Harvard Medical School
75 Francis St
Boston, MA 02115*

REFERENCES

1. Stoloff SW, Stempel DA. Study design is a major determinant of whether equivalence or superiority is demonstrated. *J Allergy Clin Immunol* 2003;111:1413.
2. Malmstrom K, Rodriguez-Gomez G, Guerra J, Villaran C, Pineiro A, Wei LX, et al. Oral montelukast, inhaled beclomethasone, and placebo for chronic asthma: a randomized, controlled trial. *Ann Intern Med* 1999;130:487-95.
3. Busse W, Raphael GD, Galant S, Kalberg C, Goode-Sellers S, Srebro S, et al. Low-dose fluticasone propionate compared with montelukast for first-line treatment of persistent asthma: a randomized clinical trial. *J Allergy Clin Immunol* 2001;107:461-8.
4. Laviolette M, Malmstrom K, Lu S, Chervinsky P, Pujet J-C, Peszek I, et al. Montelukast added to inhaled beclomethasone in treatment of asthma. *Am J Respir Crit Care Med* 1999;160:1862-8.
5. Simons FE, Villa JR, Lee BW, Teper AM, Lyttle B, Aristizabal G, et al. Montelukast added to budesonide in children with persistent asthma: a randomized, double-blind, crossover study. *J Pediatr* 2001;138:694-8.
doi:10.1067/mai.2003.1503

Clinical laboratory assessment of IgE

To the Editor:

We would like to comment on 2 recent articles entitled "Clinical and laboratory-based methods in the diagnosis of natural rubber latex"¹ and "Clinical laboratory assessment of IgE-dependent hypersensitivity."²

In these articles the performances of Food and Drug Administration–cleared latex-specific IgE serology were summarized in Table II in the first article and Table VI in the second article.

In both occasions we were somewhat surprised to find our results³ in these tables referred to as Turjanmaa et al⁴ in the former and Ownby et al⁵ in the latter. In fact, we believe that the values presented by the authors (Immuno-CAP sensitivity, 97%; Immuno-CAP specificity, 86%; AlaSTAT sensitivity, 100%; AlaSTAT specificity, 33%) resulted from our investigation that was published, as one of the first, in this Journal.

Didier G. Ebo, MD

Wim J. Stevens, MD, PhD

Chris Bridts, MT

Luc S. De Clerck, MD, PhD

*Department of Immunology-Allergy-Rheumatology
University Antwerp
Universiteitsplein 1
2610 Antwerpen
Belgium*

REFERENCES

1. Hamilton RG, Peterson EL, Ownby DR. Clinical and laboratory-based methods in the diagnosis of natural rubber latex. *J Allergy Clin Immunol* 2002;110:S47-56.
2. Hamilton RG, Adkinson NF Jr. Clinical laboratory assessment of IgE-dependent hypersensitivity. *J Allergy Clin Immunol* 2003;111:S687-701.
3. Ebo DG, Stevens WJ, Bridts CH, De Clerck LS. Latex-specific IgE, skin testing, and lymphocyte transformation to latex in latex allergy. *J Allergy Clin Immunol* 1997;100:618-23.
4. Turjanmaa K, Palosuo T, Alenius H, Leynadier F, Autegarden JE, Andre C, et al. Latex allergy diagnosis: in vivo and in vitro standardization of a natural rubber latex extract. *Allergy* 1997;52:41-50.
5. Ownby DR, Magera B, Williams PB. A blinded, multi-center evaluation of two commercial in vitro tests for latex-specific IgE quantification. *Ann Allergy Asthma Immunol* 2000;84:193-6.
doi:10.1067/mai.2003.1519

Reply

To the Editor:

I would like to extend my sincere apologies to Drs Ebo, Stevens, Bridts, and De Clerck¹ for 2 errors involving the referencing of tables in 2 of our articles. Drs Ebo et al were correct in identifying the same error in our referencing of