

In some cases, prick and intradermal tests with the native drug (i.e., not coupled to a carrier) have proved to be valid methods in the diagnosis of immediate reactions to TMP.^{1,3,4} Significant IgE levels to TMP have been previously detected by radioimmunoassay in three patients, as far as we are aware. In two of them TMP was coupled to bis-oxirane-activated Sepharose,¹ and in the other TMP-HSA was coupled to nitrocellulose paper disks.⁴ Inhibition assay was performed in the first two cases, and results were positive.¹ We have detected specific IgE to TMP using both techniques. The positive result of the RAST inhibition with TMP supports the specificity of our findings and the validity of both methods in detecting specific IgE to TMP.

Finally, we would like to highlight the importance of testing TMP and SMX independently to try to identify the offending drug in the reaction not only for scientific purposes but for therapeutic considerations for the patient. TMP was strictly forbidden for our patient, but sulfonamide therapy may be of benefit to him in the future.

Despite the fact that SMX is more frequently involved than TMP in adverse reactions to cotrimoxazole, hypersensitivity to TMP sometimes happens, and we should be aware of this in order not to underdiagnose this condition.

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Corticosteroids and IgE

To the Editor:

The study of Zieg et al. (*J ALLERGY CLIN IMMUNOL* 1994;95:222-30) provides interesting new data on the increase in serum IgE after 1 week of treatment with oral glucocorticoids. The rise in IgE was due to a polyclonal activation of IgE synthesis. The authors point out in the discussion that the rise in serum IgE was not deleterious, because there was a concomitant improvement in pulmonary function tests. This is in line with the

clinical experience of favorable therapeutic effects of short courses of oral prednisone.

We investigated whether the effects on IgE synthesis can also be found with inhaled corticosteroids. Moreover, we tested a longer period of administration, 2.5 years. We used stored serum samples from a recently completed multicenter study¹ of 274 patients with obstructive airways disease (mainly patients with asthma, some with chronic obstructive pulmonary disease). Mean age was 40 years, mean FEV₁ was 66% of predicted value, and geometric mean PC₂₀ was 0.27 mg/ml. Seventy-three percent were allergic (skin test). Extensive data on the allergic status of the study group have been reported earlier.² Serum samples were drawn at the start and end of the study from patients who continued to receive double-blind medication for 2.5 years. Serum samples were not drawn within a month of a course of oral corticosteroids. Thus 134 paired measurements were obtained. All patients inhaled a β_2 -agonist (terbutaline, 2000 μ g daily) with either a corticosteroid (beclomethasone 4 times daily, 200 μ g, $n = 56$) or an anticholinergic (ipratropium, 160 μ g daily, $n = 40$), or placebo ($n = 38$).

The significant improvements in FEV₁ and PC₂₀ with inhaled corticosteroids have been reported elsewhere.¹ Median total IgE (Pharmacia, The Netherlands) at the start of the study was 157 IU/ml in the steroid group as compared with 106 IU/ml in the other two groups. No significant change in total serum IgE was found: median change was 1 IU/ml in all three groups.

The discrepancy between the findings of Zieg et al. and our results can be due to a difference in IgE synthesis-enhancing effect between prednisone and beclomethasone. It can also be due to the difference in total systemic dose between oral corticosteroids (20 mg) and inhaled corticosteroids (800 μ g). Finally, it is possible that an effect found within 1 week of steroid treatment does not persist after prolonged treatment. Although interesting from a scientific point of view, we are unsure whether the finding of IgE synthesis enhancement by steroids has any clinical relevance.

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