

Correspondence

Desensitization to itraconazole

To the Editor:

In addition to the report of Bittleman et al. (J Allergy Clin Immunol 1994;92:270-1), we would like to report a case of successful desensitization to itraconazole.

A 30-year-old woman with hay fever, asthma, and atopic eczema had persistent sinusitis. A right ethmoidectomy was performed in December 1993. Sinus pain and a purulent yellow discharge from the right nostril recurred a few months later, and these symptoms failed to respond to lengthy courses of oral antibiotics. Subsequently, a repeat right ethmoidectomy was performed in September 1994. Fungal balls were removed, from which both *Aspergillus* and *Curvularia* species were cultured. Treatment was begun with itraconazole, 100 mg twice daily. This medication was tolerated well for 1 week, after which a confluent erythematous rash erupted over her face, neck, and shoulders. The rash faded within a few days of the itraconazole being stopped and recurred a few hours after the patient resumed taking the drug. In June 1996 we decided to attempt desensitization to itraconazole because there had been little clinical improvement. Results of skin prick tests performed with a solution of itraconazole (10 mg/ml, prepared for intravenous use) were negative. Desensitization proceeded with the protocol of Bittleman et al.; however, the drug was given in the form of a diluted solution rather than as crushed tablets, which greatly simplified preparation and administration of the schedule. Desensitization proceeded uneventfully. The patient tolerated a 6-week course of itraconazole, 100 mg twice daily, without ill effects. Further sinus surgical drainage was performed in July, during the course of itraconazole. *Curvularia* was seen on histologic examination of the resected specimen, but no fungal species were cultured. She has subsequently made an excellent clinical recovery.

We present this case because the negative skin prick test results suggest that the hypersensitivity reaction to itraconazole was not IgE-mediated and because the procedure originally described has been significantly simplified by the availability of an oral solution of itraconazole.

Richard Douglas

Denis Spelman

Dan Czarny

Robyn O'Hehir

Department of Allergy and Clinical Immunology

Department of Microbiology and Infectious Diseases

Alfred Hospital Commercial Road

Prahran (Melbourne) Victoria

3181 Australia

1/8/78565

Inhaled mite allergen

To the Editor:

I was interested in the article by Sakaguchi et al. on inhaled mite allergen (J Allergy Clin Immunol 1996;97:1040-4). We previously reported an estimate of the inhaled allergen intake of 1-month-old infants.¹ There were some critical differences in our study compared with their study in that we used a pump with a minute volume the same as that of the infant to replicate the expiratory/inspiratory cycle. In addition, we found a threefold variation in the measured allergen intake with this instrument when we altered the direction of the sampler's nose by 180 degrees. We have, on repeated occasions, presented correlations between surfaces and airborne allergen and indeed currently have a paper submitted, which addresses the differences in airborne allergen "intake" when using a constant steady sampling system similar to that of Sakagu-

chi et al. compared with a plate dust-settling method. It appears that the best correlation in our study with the Casella sampler (Casella, Bedford, U.K.) in school children who had asthma was between airborne allergen intake overnight and the pillow surface sample. We have also demonstrated and published the success of bedding covering arrangements in reducing both surface allergen and airborne allergen and producing a significant reduction in nonspecific bronchial reactivity in mite-sensitive asthmatic children.²⁻⁴

Sakaguchi et al. report the results in nanograms per gram of dust, which theoretically is not the best method for looking for a correlation between surface and air samples. If it is the movement of the surface that makes the allergen airborne, it would be logical to relate the mite allergen sample to a standard surface sample area, as discussed extensively in the article they quote. It is also interesting, and I suspect it can be related to both humidity and the hygroscopic properties of the fecal pellets, that there is likely to be some difference between the surface/air correlations in very dry climates (e.g., Perth, West Australia) and humid-wet climates (e.g., West of England). We have demonstrated that use of covers on the bed can very significantly reduce mite allergen exposure, and this method would be preferable to removing the bedding, as Sakaguchi et al. suggest, because it has been established that recolonization of the bedding will occur over a period of months.

Fleming Carswell, MD, PhD

University of Bristol

Department of Child Health

Royal Hospital for Sick Children

St. Michael's Hill

Bristol BS2 8BJ

United Kingdom

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Reply

To the Editor:

In response to Dr. Carswell's letter, we also found changes in mite major allergen levels detected when we altered the direction of inlet of filter in the air sampler. Throughout this study, we set up the inlet in the vertical position.

We did not find a good correlation between the mite levels in the air and in bedding cotton, cotton dust, and floor dust. Price et al.¹ also reported that there was no correlation between the level of Der 1 in carpets and the level in the air. Tovey et al.² reported that there was little correlation between the mite allergen concentration in the air and the levels in floor and bedding dust. The airborne allergen levels might be influenced by several factors: the level of the allergens present on the floor, bedding, and furniture; ventilation; and human activity in the room. These factors vary in each home.