and EASI scores for all subjects from visit 1 to visit 3 (P = .0005, .009, .01), indicating that sleep improved as skin condition improved (Table IV). Although skin condition was related to sleep, subject perception of itch was not; the pruritus score was not correlated with any sleep variable (Table IV). These contrasting findings suggest either that subjects had difficulty accurately judging itch, particularly at night, or that the relationship between skin inflammation and sleep were not mediated by altering the subject's experience of itch.[10]

Clearly, more work is needed to establish the mechanisms by which sleep disturbance and skin disease are related, and the optimal course of treatment to maximize sleep improvement. Further research is needed to examine the relationship of AD and sleep using actigraphy as an objective measurement for longer treatment periods. This becomes essential because subjects appear to have difficulty recalling nocturnal itch and its effect on sleep parameters. Also, additional investigation of the subject's perception of both daytime and nocturnal itching and sleep symptoms would clarify individual response to nocturnal itching and, ultimately, long-term sleep patterns.

Harvey L. Leo, MD[a]
Bruce G. Bender, PhD[b,c,d]
Susan B. Leung, RN[a]
Zung Vu Tran, PhD[b]
Donald Y. M. Leung, MD, PhD[a,c]
[a]*Division of Pediatric Allergy and Immunology*
[b]*Division of Pediatric Behavioral Health*
National Jewish Medical and Research Center
1400 Jackson Street
Denver, CO 80206
[c]*Department of Pediatric*
[d]*Department of Psychiatry*
University of Colorado Health Sciences Center
Denver, Colo

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Sugar glider allergy: Identification of serum specific IgE

To the Editor:

Sugar gliders are small marsupial possums that are becoming popular pets in North America, Europe, and Japan. Their common name reflects the fact that they have a predilection for sweet food and have a gliding membrane similar to that of many flying squirrels. Sugar gliders are actually mammals in the subclass *Marsupialia,* family *Petauridae*. This family contains 11 species of possums, some of which can glide like the sugar glider. Their scientific name is *Petaurus breviceps*. *Petaurus* means tightrope walker or rope dancer, and *breviceps* means short head.[1] Geographically, they are found in Northern and Eastern Australia, Tasmania, Indonesia, and Papua-New Guinea.[2]

A patient presented to our clinic with complaints of worsening asthma and contact urticaria to his pet sugar glider. We sought to identify immunologically active allergens from the sugar glider that could explain these symptoms.

Our patient was a 49-year-old white man with a history of mild intermittent asthma and perennial allergic rhinitis with seasonal exacerbation in the winter. He was allergic to both cat hair and mountain cedar. Significantly, he complained of an immediate, erythematous, papular, pruritic rash whenever the sugar glider came in contact with his skin. In addition, he developed more severe and persistent asthma symptoms, requiring an emergency department visit and oral steroids, since the pet was introduced into his home.

We prepared extracts from sugar glider urine and feces. Urine was collected by lining the bottom of the cage with paper towels. The bedding material in the bottom of the cage was collected and the fecal pellets removed. Protein was extracted from the fecal pellets and urine-soaked paper towels in 0.125 mol/L NH_4HCO_3 at 4°C overnight. Extracts were then centrifuged and the supernatant passed through a graded series of filters. The filtrate was dialyzed against distilled water overnight at 4°C in 3500-D dialysis tubing, lyophilized, then stored at 4°C.

Extracts were sterilized by filtering through a 0.45-μm filter. Our patient was skin-tested with the fecal and urine extracts. Skin testing was inconclusive because the patient was dermatographic on both his arm and back. Informed consent was obtained, and the process was reviewed by our Institutional Review Board.

Extract protein concentrations were determined by a modification of the technique of Lowry et al.[3] The urine

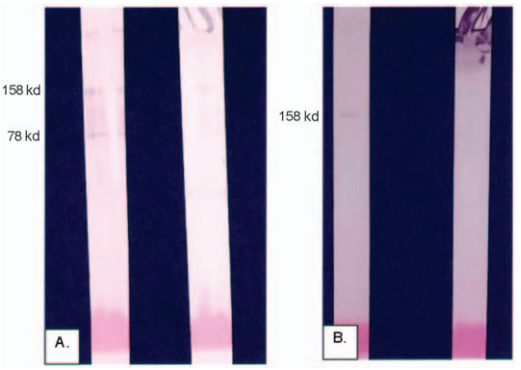

Fig 1. IgE immunoblots. A, Sugar glider feces in *lane 1*, cord blood in *lane 2*. B, Sugar glider urine in *lane 1*, cord blood in *lane 2*.

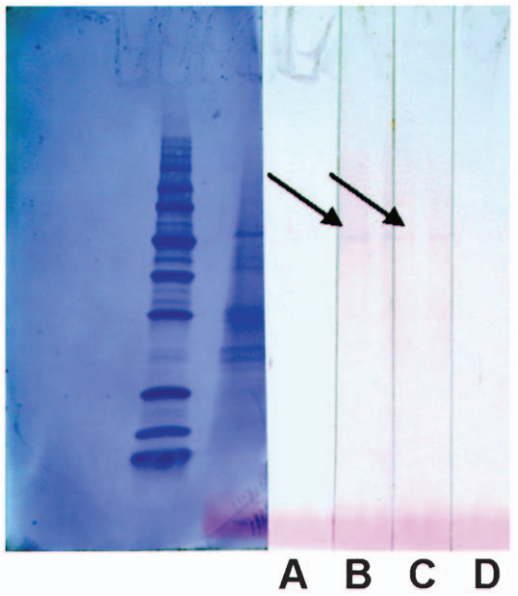

Fig 2. Sugar glider feces IgE immunoblot inhibited with cat pelt extract. A, Cord blood (negative control). B, Uninhibited. C, Inhibited with cat pelt extract. D, Inhibited with sugar glider feces.

extract contained 171 μg protein per milligram lyophilized extract, while fecal extract contained 480 μg protein per milligram lyophilized extract.

An SDS-PAGE of the fecal and urine extracts was accomplished as described previously.[4] Gels were stained with 0.025% Coomassie blue and revealed multiple protein bands. IgE immunoblots were accomplished as previously described.[4] Briefly, this required transferring the proteins from the SDS-PAGE gels (for both urine and fecal extracts) to 0.45-μm nitrocellulose membranes. After transfer, the molecular weight standards were cut from the membrane and stained with Coomassie blue. The urine and fecal protein nitrocellulose membranes were washed, blocked with 20% FBS, and incubated overnight with the patient's serum in 10% FBS, and pooled cord serum was used as a negative control. A 1:10 dilution of patient's serum and cord serum were used with the fecal extract, and full-strength patient serum and pooled cord serum were used with the urine extract. Membranes were washed and incubated for 6 hours at room temperature in mouse anti-human IgE mAb diluted 1:5000 in 10% FBS. Membranes were then incubated overnight in alkaline phosphatase–conjugated goat antimouse IgG diluted 1:2000 in 10% FBS. The membranes were developed, and the molecular weight of each protein was determined from the molecular weight standards.

Inhibition immunoblots were accomplished as previously described.[4] The procedure was performed with the fecal protein extract. Inhibition immunoblots were not performed with the urine extract because of an insufficient amount of source material. This procedure duplicated that of the immunoblots, as described, with preincubation of 3 mL patient serum with 4.6 mg cat pelt extract (3 mg protein) before the immunoblot procedure. Self-inhibition was performed with a separate serum aliquot preincubated with 6.3 mg fecal extract (3 mg protein) to serve as a control. Cat pelt extract was chosen because our patient was allergic to cat, and previous work in our laboratory discovered that patients with IgE to ferret were cross-reactive to cat at a 66-kd protein thought to be cat albumin.[5]

IgE binding to the fecal extract was seen at a 78-kd protein and a 158-kd protein. IgE binding to the urine extract was seen at a 158-kd protein. IgE immunoblots with molecular weight standards are shown in Fig 1. The cord blood showed no IgE binding.

The inhibition blots showed self-inhibition at the 78-kd band as expected, but no inhibition with cat pelt extract. This proved that this protein in the sugar glider feces was not shared by cat pelt extract. The 158-kd band was not found on the inhibition blot and was thought to be a dimer of the 78-kd band that was seen on the first immunoblot. Inhibition blots are shown in Fig 2.

Sugar gliders are becoming popular exotic pets in the United States, but there are no published reports on IgE-mediated allergies to sugar glider. Many sugar glider pet owners report a contact dermatitis from their pet, thought by some to be a result of subcutaneous introduction of bacteria from their sharp claws.[1] Our patient, however, described immediate, distinctly urticarial lesions. Although the patient was dermatographic, the lesions inflicted by the sugar glider were more pronounced and pruritic by description than those elicited by skin testing. The urticarial nature of the lesions, along with his worsening asthma symptoms, led us to pursue an IgE-mediated etiology. Other sugar glider owners have reported conjunctivitis, rhinitis, and asthma symptoms to their sugar gliders.[6]

In conclusion, we report a patient with allergic symptoms to his pet sugar glider who demonstrated serum specific IgE to proteins found in both sugar glider feces and urine. These proteins are distinct from those found in cat pelt extract. This is the first known report of an IgE-mediated reaction to a sugar glider.

Melinda M. Rathkopf, MD
Bonnie A. Whisman, MS
Larry L. Hagan, MD
Department of Allergy/Immunology
Wilford Hall Medical Center
759 MDOS/MMIA
2200 Bergquist Drive, Ste 1
Lackland Air Force Base, TX 78236

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Effect of topical pimecrolimus on epicutaneous skin testing

To the Editor:

The majority of patients with atopic dermatitis have both seasonal and perennial allergies to environmental elements. In addition, as many as 40% of the patients with severe atopic dermatitis have food allergies.[1] An essential part of the diagnostic work-up for patients not responding to standard treatment regimens is the use of skin prick tests. Pimecrolimus cream 1% is a safe and effective treatment for atopic dermatitis.[2,3] In addition, pimecrolimus has been shown to inhibit the release of proinflammatory mediators from mast cells *in vitro*.[4] If pimecrolimus can prevent the release of histamine from mast cells, there is a possibility of false-negative skin tests, affecting the diagnosis of patients. In this scenario, patients would have to refrain from using this treatment, before skin testing, as with antihistamines.

We studied 12 adult participants (>18 years old) with a known history of atopic dermatitis and/or seasonal allergies and asthma. On obtaining written informed consent, we performed a randomized, double-blind, within-patient comparison of pimecrolimus cream 1% versus vehicle in reactivity of skin prick tests. During the first visit, each participant underwent standard skin prick testing to common inhalant allergens: Der p 1, Der f 1, cat, dog, ragweed, maple, birch, and bluegrass (Greer Laboratories, Lenior, NC). The skin was pricked with sterile prick lancets, and reactions were recorded after 20 minutes by measuring the maximal longitudinal diameter of the wheal and flare and the diameter orthogonal to it. Wheals more than 3 mm larger in diameter than those produced after saline pricks were regarded as positive reactions.

The trial was approved by the Institutional Review Board of the Children's Hospital of Philadelphia. Each participant was instructed on the application of study medications, pimecrolimus cream 1% and vehicle cream. Participants applied each cream twice daily to each forearm for 2 weeks. The tubes were weighed at the beginning and the end of the study to ensure equal use of creams. On completion of the 2-week treatment, participants returned for follow-up skin testing. Skin testing was performed for previously positive allergens, histamine,

TABLE I. Effect of pimecrolimus on skin testing*

	Pimecrolimus (no. positive skin tests, 3.63 ± 2.5)		Difference (pimecrolimus-treated arm – placebo-treated arm)	Placebo (no. positive skin tests, 3.63 ± 2.3)	
Allergen	**Wheal, diameter (mm)**	**Flare, diameter (mm)**	**Wheal, diameter (mm)**	**Wheal, diameter (mm)**	**Flare, diameter (mm)**
Histamine	5.16 ± 1.7	17.4 ± 6.5	0.33 ± 1.4	4.83 ± 1.1	17.3 ± 6.8
Der p 1	3.27 ± 3.0	8.14 ± 9.2	−0.5 ± 3.5	3.00 ± 3.5	8.88 ± 8.8
Der f 1	3.25 ± 3.2	7.38 ± 7.8	−0.25 ± 3.6	3.50 ± 3.9	9.25 ±10.4
Cat	3.17 ± 4.5	5.5 ± 7.9	0.17 ± 3.8	3.00 ± 3.6	7.67 ± 8.1
Dog	1.20 ± 0.7	2.20 ± 1.5	0.0 ± 1.2	1.20 ± 1.6	3.20 ± 4.6
Ragweed	7.34 ± 5.4	18.25 ± 10.7	0.38 ± 2.3	7.00 ± 5.6	15.88 ± 9.1
Maple	3.63 ± 3.0	10.75 ± 9.2	−0.13 ± 3.1	3.75 ± 3.5	9.38 ± 8.1
Birch	5.70 ± 6.2	12.60 ± 12.4	−0.5 ± 6.4	6.2 ± 6.9	12.40 ± 12.3
Bluegrass	7.10 ± 5.2	19.11 ± 10.3	−0.4 ± 5.5	7.50 ± 6.0	19.11 ± 11.6

*Mean ± SD.