

Eicosanoids derived from arachidonic acid are important mediators of allergic inflammation. Furthermore, in the mechanisms of AIU, a shift of arachidonic acid metabolism in favor of leukotrienes by aspirin has been suggested.¹ Therefore, our results of altered arachidonic acid levels in patients with AIU during aspirin provocation may support previous findings of altered arachidonic acid metabolism in patients with AIU. Aspirin-induced arachidonic acid may affect the formation of leukotrienes because the formation of leukotrienes depends on the deacylation of arachidonic acid. A relationship between AIU and arachidonic acid is suggested in this study, but further investigation into the relationship between plasma FA composition and leukotriene production is needed. In conclusion, our results suggest that aspirin ingestion may affect a specific pathway in FA metabolism and that arachidonic acid levels may be related to aspirin intolerance in patients with AIU.

SEUNG-HYUN KIM, PhD*

MAN-JEONG PAIK, PhD†

GWANG LEE, PhD‡

HAE-SIM PARK, PhD‡

* Department of Allergy and Rheumatology

† Institute for Neuroregeneration and Stem Cell Research

‡ Department of Allergy and Rheumatology

Ajou University School of Medicine

Suwon, Korea

hspark@ajou.ac.kr

- Grattan CE. Aspirin sensitivity and urticaria. *Clin Exp Dermatol*. 2003;28:123–127.
- Doeglas HM. Reactions to aspirin and food additives in patients with chronic urticaria, including the physical urticarias. *Br J Dermatol*. 1975;93:135–144.
- Aihara M, Miyazawa M, Osuna H, et al. Food-dependent exercise-induced anaphylaxis: influence of concurrent aspirin administration on skin testing. *Br J Dermatol*. 2002;146:466–472.
- Gollapudi RR, Teirstein PS, Stevenson DD, Simon RA. Aspirin sensitivity: implications for patients with coronary artery disease. *JAMA*. 2004;292:3017–3023.
- Paik MJ, Kim KR. Sequential ethoxycarbonylation, methoximation and tert-butyldimethylsilylation for simultaneous determination of amino acids and carboxylic acids by dual column gas chromatography. *J Chromatogr A*. 2004;1034:13–23.
- Mantzioris E, James MJ, Gibson RA, Cleland LG. Differences exist in the relationships between dietary linoleic and alpha-linolenic acids and their respective long-chain metabolites. *Am J Clin Nutr*. 1995;61:320–324. 96–206.

UPPER RESPIRATORY TRACT INFECTIONS AND SUBLINGUAL IMMUNOTHERAPY: PRELIMINARY EVIDENCE

Allergic rhinitis (AR) is characterized by T_H2 polarization with consequent defective interferon γ (IFN- γ) production.¹ Because IFN- γ is recruited to fight infections, it has been suggested that allergic patients may be more susceptible to upper respiratory tract infections (URIs) than nonallergic individuals.² URIs are frequent in the general population and constitute a demanding challenge for physicians.³ Recently, it has been evidenced that allergic children have more numerous and severe respiratory infections than nonallergic children.⁴ It has also been confirmed that adult patients with AR have more numerous and prolonged URIs than nonallergic adults.⁵

AR management includes, when appropriate, allergen-specific immunotherapy.⁶ The sublingual immunotherapy (SLIT) rationale is based on evidence that oral administration of high-dose allergens reduces and prevents IgE responses, achieving clinical tolerance to

the causal allergen. SLIT induces the appearance of allergen-specific tolerance, associated with restored allergen-specific IFN- γ production.⁷ However, no studies have evaluated the possible effect of SLIT on URIs. Therefore, this preliminary study was designed to evaluate whether a 2-year preseasonal SLIT might affect the number and the duration of URIs in AR patients.

Forty consecutive monosensitized patients (18 men and 22 women; mean age, 36.6 years) with AR due to pollen allergy were treated with SLIT. Forty consecutive well-matched patients (19 men and 21 women; mean age, 36.1 years) with pollen AR (16 of them were allergic only to *Betula alba*, 16 only to *Parietaria judaica*, and 8 only to Gramineae) were included in the study as controls because they refused SLIT. AR was diagnosed according to validated criteria provided by international guidelines.^{2,8} In addition, 40 healthy individuals (25 men and 15 women; mean age, 35.2 years; all of whom were US Navy soldiers) were enrolled as controls on the basis of a history negative for AR, clinical examination, nasal endoscopy, and skin prick test results.

This was a prospective, longitudinal, observational study conducted during a 3-year period. It was initiated after obtaining approval from the local ethics committee and written informed consent from all participants. SLIT-treated patients were evaluated the year before SLIT and during the 2-year preseasonal SLIT. All study participants, both allergic patients and healthy controls, were visited every 6 months for 3 years during the same period. In addition, a diary card was provided to each participant. At each visit, study participants had to report respiratory symptoms on a weekly basis.

Participants undergoing SLIT received either a *B alba* pollen allergen extract (Anallergo, Florence, Italy; 17 cases, cumulative dose was 60 μ g), *P judaica* (15 cases, cumulative dose was 24 μ g), or a mix of 3 Gramineae (7 cases, cumulative dose was 24 μ g) for at least 3 months before the 2 pollen seasons, as preseasonal immunotherapy, for 2 consecutive years. The dose during the maintenance phase was 5 drops 3 times per week. The diagnosis of URI was made according to previously validated studies.^{4,5} The primary end point was the comparison of the number of URI episodes recorded per year among groups during the study period. The secondary outcome was the comparison of the total duration of URI episodes (days per year).

Only SLIT-treated allergic patients had a significant reduction in incidence rate (IR) during the 3-year period ($P < .001$, Friedman test), whereas both untreated allergic patients and healthy controls did not show significant IR variation ($P = .60$ and $P = .90$, respectively) as reported in Figure 1A. With the group of untreated allergic patients as a reference, the IR for the treated group was 0.98 (95% confidence interval [CI], 0.73 to 1.33; $P = .94$) during the year before treatment, 0.81 (95% CI, 0.60 to 1.11; $P = .18$) during the first year of treatment, and 0.66 (95% CI, 0.47 to 0.90; $P = .01$) during the second year; this result indicates a significant decrease in the URI episode rate after 2 years of treatment.

Also regarding the mean duration of URI episodes, only SLIT-treated allergic patients had a significant reduction in IR during the 3-year period ($P < .001$, Friedman test), whereas no heterogeneities were found in the other 2 groups of patients ($P = .50$ and $P = .60$, respectively, Fig 1B). SLIT-treated patients had a mean difference in duration of -0.1 day compared with untreated patients during the year preceding treatment (95% CI, -2.1 to 1.92 ; $P = .92$), -3.5 days during the first year (95% CI, -5.1 to -1.7 ; $P < .001$), and -5.7 (95% CI, -7.1 to -4.2 ; $P < .001$) during the second year.

Disclosures: Authors have nothing to disclose.

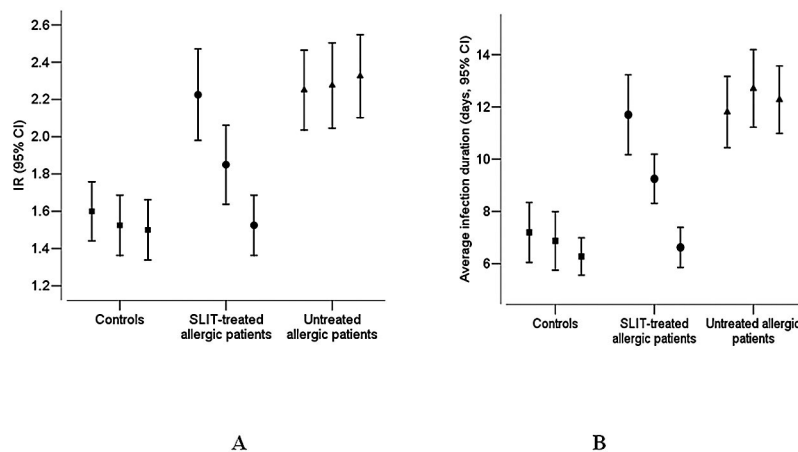


Figure 1. Incidence rates (IRs) and average durations of upper respiratory tract infections (URIs). A, IRs (95% confidence intervals [CIs]) of URI episodes for the 3 groups of study participants (healthy controls are represented by squares, sublingual immunotherapy (SLIT)-treated allergic patients by circles, and untreated allergic patients by triangles) during the year before treatment and during the first and the second years of treatment. B, Average duration of URI episodes for the 3 groups of study participants during the same period.

Therefore, this preliminary study provides the first evidence that 2-year preseasonal pollen SLIT may be able to reduce the number and duration of URIs in AR patients. These findings are clinically relevant; a possible explanation for this progressive effect might be attributable to modulation of the T_H2 -polarized immune response in SLIT-treated patients as previously evidenced.⁷ However, this preliminary study is an open-label study and should be followed by a controlled trial with a much larger cohort on this issue.

GIORGIO CIPRANDI, MD*
MARIA PIA SORMANI, BS†
IGNAZIO CIRILLO, MD‡
MARIANGELA TOSCA, MD§

* Department of Internal Medicine
University of Genoa
Genoa, Italy
† Unit of Biostatistics
Department of Health Sciences
University of Genoa
Genoa, Italy
‡ Navy Medical Service
La Spezia, Italy
§ Allergy Center
IRCCS G. Gaslini
Genoa, Italy
gio.cip@libero.it

1. Bousquet J, Khaltaev N, Cruz AA, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA2LEN and AllerGen). *Allergy*. 2008;63(suppl 86):8-160.
2. Ciprandi G, Buscaglia S, Pesce GP, et al. Minimal persistent inflammation is present at mucosal level in asymptomatic rhinitic patients with allergy due to mites. *J Allergy Clin Immunol*. 1995;96:971-979.
3. Acute respiratory infections: the forgotten pandemic. *Bull World Health Organ*. 1998;76:101-103.
4. Ciprandi G, Tosca MA, Fasce L. Allergic children have more numerous and severe respiratory infections than non-allergic children. *Pediatr Allergy Immunol*. 2006;17:389-391.
5. Cirillo I, Marseglia GL, Klersy C, Ciprandi G. Allergic patients have more numerous and prolonged respiratory infections than non-allergic subjects. *Allergy*. 2007;62:1087-1090.

6. Passalacqua G, Durham SR. Allergic rhinitis and its impact on asthma update: allergen immunotherapy. *J Allergy Clin Immunol*. 2007;119:881-891.
7. Ciprandi G, Contini P, Fenoglio D, et al. Relationship between soluble HLA-G and HLA-A, -B, -C serum levels and IFN-gamma production after sublingual immunotherapy in patients with allergic rhinitis. *Hum Immunol*. 2008;69:409-413.
8. Wallace DV, Dykewicz MS. The diagnosis and management of rhinitis: an updated practice parameter. *J Allergy Clin Immunol*. 2008;122:S1-84.

CUTANEOUS ALLERGY TO COTTON

Traditionally, allergy to cotton has always been related to occupational asthma,¹ exposure to airborne cotton dust that could lead to byssinosis, or airway obstruction due to exposure to a mixture of materials derived from processing the cotton and natural contaminants (bacteria, endotoxins, and molds).² The occurrence of an immediate skin reaction to textiles is extremely rare. Most reported cases occurred after occupational exposure to chemicals.³ Cotton clothing occasionally causes itchy skin or erythema, but this is usually due to skin irritation.

A 24-year-old woman reported, during the last 3 years, itchy skin within minutes after wearing any cotton clothing and a flat erythematous eruption in the places where pure cotton brushed the skin; this reaction subsided within 20 minutes once the cotton was removed. She did not have any occupational exposure to cotton or contact with cotton-fabric workers. In addition, she reported a history of seasonal rhinoconjunctivitis due to pollens.

Skin prick tests (SPTs) to commercial common aeroallergens, to latex, and with cotton extract (60 μ g/mL; Immunotek Lab, Madrid, Spain) were performed. The findings included a positive result to grass pollens (wheal ≥ 3 mm) and negative results to dust mites, molds, danders, and latex. An SPT to cotton fiber extract showed a wheal of 4 $\times$ 4 mm with surrounding flare. The result of an SPT to cotton extract tested in 5 nonatopic patients was negative. A rub test with sanitary cotton on the skin of the volar surface of the arm was performed, prompting a well-defined flat erythematous pruritic eruption. To identify IgE-reactive protein components, in vitro assays were performed. The crude extract was separated by sodium

Disclosures: Authors have nothing to disclose.