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Immunotherapy Decreases Seasonal Rise in Serum-Soluble CD23 in Seasonal Allergic Rhinitis

[Independent Papers]

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Abstract

There is increasing in vitro evidence that soluble CD23 (sCD23) is capable of potentiating IgE synthesis, but the in vivo physiologic significance remains to be established. This study investigated the seasonal changes in sCD23 in patients with seasonal allergic

rhinitis. It included 112 adult patients with seasonal allergic rhinitis due to Japanese cedar pollens and 20 nonatopic healthy volunteers. The 64 patients of the pharmacotherapy group were treated with nonsedating antihistamine tablets alone throughout the pollen season and the remaining 48 patients of the immunotherapy group continued to be treated with immunotherapy. Serum concentrations of sCD23 were measured in each patient, before and during the pollen season of 1996, by a sandwich enzyme-linked immunosorbent assay. The serum levels of sCD23 in the pharmacotherapy group before the pollen season were significantly higher than those in the nonatopic group ($P = .0130$) and those in the immunotherapy group ($P = .0316$). Seasonal increase in sCD23 was significant in the pharmacotherapy group, irrespective of the clinical response ($P < .0001$). By contrast, sCD23 was not significantly increased in the good responders to immunotherapy ($P = .1826$), but was significantly increased in the poor responders to immunotherapy ($P = .0052$). A significant correlation between seasonal increase in rate in specific IgE and seasonal increase in rate in sCD23 was confirmed in both the pharmacotherapy group ($r_s = 0.321$, $P = .0107$) and the immunotherapy group ($r_s = 0.474$, $P = .0012$). In conclusion, seasonal rise in sCD23 is associated with and is probably involved in seasonal rise in specific IgE in patients with seasonal allergic rhinitis, and successful immunotherapy is capable of blunting seasonal increase in sCD23, thus resulting in attenuation of seasonal increase in specific IgE and clinical benefits during the pollen season.

INTRODUCTION

CD23 (Fc[small element of]RII) is the low-affinity receptor for IgE that is found on multiple cells including lymphocytes, monocytes, and eosinophils.^{1,2} An increasing number of observations suggest that CD23 may be involved in B-cell activation and antigen presentations to T cells,² although the function of CD23 on cells is likely to depend on which cell type expressed it. An augmentation of the number of CD23-bearing cells and of the cellular density of CD23 has been documented in patients with allergic ^{3,4} and parasitic diseases ⁵ as well as other hyper-IgE conditions.⁶ Soluble CD23 (sCD23), which is released after a series of proteolytic steps, is also known as IgE-binding factor.^{2,7,8} There is increasing in vitro evidence that sCD23 is capable of potentiating the synthesis of human IgE ⁹⁻¹³ and is likely involved in the expression of allergic diseases. Anti-CD23 antibodies can inhibit both ongoing and interleukin-4-stimulated IgE synthesis,¹⁰⁻¹² and the supernatant from the RPMI 1886 cell line, which contains relatively large amounts of CD23 materials, can enhance IgE production from lymphocytes of atopic individuals.¹³

All these activities of sCD23 have been observed in vitro, but the in vivo physiologic significance of sCD23 is not fully understood.

Elevated serum levels of sCD23 have been reported in atopic patients,[14-17](#) although the correlation was weak [15-17](#) or not significant [14](#) between total IgE and sCD23 in sera. The specific IgE level is more important in allergic diseases than the total IgE level. However, a possible correlation between the relevant or specific IgE and sCD23 in patients with allergic diseases has yet to be discussed. Seasonal rises in specific IgE in pollen-sensitive patients have been well established.[18,19](#) A seasonal rise in sCD23 might be associated with seasonal rise in specific IgE in seasonal allergic rhinitis. In addition, this possible seasonal increase in sCD23 in seasonal allergic rhinitis might be modulated by an active treatment such as allergen-specific immunotherapy, because the seasonal increase in specific IgE is indeed modulated by immunotherapy.[18,19](#) However, none of these hypotheses has much evidential support. Our study is aimed at investigating the seasonal changes in sCD23 in patients with seasonal allergic rhinitis due to Japanese cedar (*Cryptomeria japonica*) pollens, with special reference to the possible modulation by allergen-specific immunotherapy.

MATERIALS AND METHODS

Patients

This study included 112 adult patients (age range, 19 to 55 years) with seasonal allergic rhinitis due to Japanese cedar (*C japonica*) pollens who gave informed consent to participation in the study. All the patients were selected from our outpatients and completely satisfied the following conditions before treatment: 1. well-documented history of typical Japanese cedar pollinosis but no history of asthma and no nasal symptoms out of the cedar pollen season, 2. positive skin test to Japanese cedar pollens, 3. positive nasal provocation to the pollens, and 4. eosinophilia in nasal smears during the cedar pollen season. The patients were divided into the pharmacotherapy group and the immunotherapy group according to their treatment background. None of the patients of the pharmacotherapy group had previously been treated with immunotherapy or was receiving steroid treatment. The patients of the immunotherapy group had variable periods (mean, 5.0 ± 4.4 years; range, 1 to 15 years) of immunotherapy using the Japanese cedar pollen antigen (Torii Co., Ltd., Tokyo, Japan). The details of the immunotherapy used in this study were described elsewhere.[19](#)

To serve as control subjects, 20 nonallergic healthy volunteers (age range, 25 to 64 years, nonatopic group) were chosen on the basis of the following criteria: 1. a negative history for allergic diseases, 2. no physical findings consistent with atopic diseases, and 3. negative serum IgE antibodies specific to the major allergens in Japan, such as house dust mite, Japanese cedar pollen, ragweed, Japanese cypress pollen, and mold.

Study Design

The period for pollination of Japanese cedar in 1996 was from the first week of February to the second week of April. The 64 patients of the pharmacotherapy group were treated with nonsedating antihistamine tablets alone from the last week of January to the end of April. The 48 patients of the immunotherapy group continued to receive scheduled maintenance dose subcutaneous injection. The patients of the immunotherapy group were allowed to take rescue antihistamine tablets during the pollen season, if necessary.

Venous blood was collected twice from each patient, before the cedar pollen season of 1996 (from December 1995 to January 1996) and during the cedar pollen season of 1996 (first week of April 1996), to determine cedar pollen-specific IgE and sCD23 in sera. Venous blood was also collected out of the pollen season from each person in the nonatopic group to determine cedar pollen-specific IgE and sCD23 in sera. All the serum samples were separated and stored at -80°C and were simultaneously used for the following examinations.

Determination of Specific IgE and Soluble CD23

Specific IgE antibodies and sCD23 in sera were determined at the same time. Specific IgE to Japanese cedar pollens was measured by the CAP radioallergosorbent test. Serum concentrations of sCD23 were measured by a sandwich enzyme-linked immunosorbent assay (ELISA), with the use of two monoclonal antibodies that recognize different epitopes of CD23. The method employed in this

study was a modification of the method as previously described by Yanagihara et al.²⁰ The methodology and justification of the method and monoclonal antibodies employed in this study have been described elsewhere.²⁰ Minimum sensitivity for this assay was 40 U/mL of sCD23. The seasonal increase in rate in specific IgE and sCD23 was calculated using the following formula. [Equation 1](#)

Seasonal increase in rate (%)	=	$\frac{\text{values during pollen season} - \text{values before pollen season}}{\text{values before pollen season}} \times 100$
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Equation 1

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Clinical Evaluation

Clinical evaluation was performed for each patient, based on our criteria using daily symptom diary cards, during the first week of April in 1996. The details of the method employed in this study have been described elsewhere.^{18,21} In brief, all the patients were asked to complete daily symptom diary cards in the 7 days before clinical assessment. The daily symptom card includes the number of sneezing attacks, the number of nose blowings, and the degree of nasal obstruction. A careful survey of the diary card graded the three nasal symptoms (sneeze, rhinorrhea, and obstruction) on a scale of 0 to 3, depending on severity. The average daily scores of sum total of the three nasal symptom scores were used as total symptom scores (maximum: 9). According to their total symptom scores, the patients of the pharmacotherapy group were divided into the good responders whose total symptom scores were 0 to 2 and the poor responders whose total symptom scores were 3 to 9. The patients of the immunotherapy group were also divided into the good responders and the poor responders. Those whose symptom scores were 2 or less without any rescue medication were judged as the good responders, and the remainders were judged as the poor responders.

Statistical Analysis

For comparison of data between groups, Mann-Whitney's *U* test was used. For comparison of the paired values within a group, Wilcoxon's signed-rank test was used. Significant differences were accepted in each analysis when the *P* value was less than .05. Spearman's correlation coefficient (*r_s*) was calculated to analyze possible correlation between two different parameters, and significant correlation was accepted when *P* was less than .05 according to the Spearman's rank correlation analysis.

RESULTS
Background of Subjects

There were no significant differences of sex between the 112 patients and the 20 nonatopic healthy volunteers (Table I). The ages of the 112 patients were not significantly different from those of the 20 nonatopic healthy volunteers (*P* = .3839). Neither the ages nor the duration of the disease was significantly different between the pharmacotherapy group and the immunotherapy group (age: *P* = .2558; duration of the disease: *P* = .2154).

Group	Sex		Age (yr)				Duration of disease (yr)				Duration of immunotherapy (yr)			
	Female	Male	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max
Nonatopic	11	9	26.4	6.8	17	39	—	—	—	—	—	—	—	—
Pharmacotherapy	58	54	26.4	6.8	17	39	8.5	4.8	0.5	17.5	—	—	—	—
Immunotherapy	25	15	26.2	6.4	17	39	8.3	4.2	0.5	17.5	5.2	4.4	0.5	17.5

TABLE I. Background of the Subjects.

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Specific IgE and Soluble CD23 Before the Pollen Season

Specific IgE to the cedar pollen was not detected in any individual of the nonatopic group. The serum levels of specific IgE were not significantly different between the pharmacotherapy group and the immunotherapy group (*P* = .3246, Table II). The serum levels of sCD23 in the patient group were not significantly different from those in the nonatopic group (*P* = .0983). The serum levels of sCD23

in the immunotherapy group were not significantly different from those in the nonatopic group ($P = .7466$). However, the serum levels of sCD23 in the pharmacotherapy group were significantly higher than those in the nonatopic group ($P = .0130$) and those in the immunotherapy group ($P = .0316$, [Table III](#)).

	Pharmacotherapy group (n = 10)	Immunotherapy group (n = 10)	Nonatopic group (n = 10)
Specific IgE (Mean ± SD)	22.88 ± 23.94	23.47 ± 20.85	17.8 ± 10.2
Pre-treatment (n = 10)	24.17 ± 24.88	27.88 ± 27.58	28.5 ± 18.8
Post-treatment (n = 10)	17.87 ± 7.18	20.88 ± 12.88	20.7 ± 11.2
Immunotherapy group (n = 10)	24.88 ± 21.88	20.88 ± 11.88	17.1 ± 10.2
Pre-treatment (n = 10)	24.88 ± 24.88	20.88 ± 10.88	18.8 ± 17.2
Post-treatment (n = 10)	18.88 ± 10.17	18.88 ± 11.88	18.8 ± 10.2

TABLE II. Specific IgE (Mean ± SD).

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	Pharmacotherapy group (n = 10)	Immunotherapy group (n = 10)	Nonatopic group (n = 10)
Soluble CD23 (Mean ± SD)	300.2 ± 174.2	285.4 ± 127.8	17.2 ± 10.2
Pre-treatment (n = 10)	300.2 ± 174.2	285.4 ± 127.8	18.8 ± 10.2
Post-treatment (n = 10)	24.8 ± 14.2	24.2 ± 10.2	18.8 ± 10.2
Immunotherapy group (n = 10)	300.2 ± 174.2	285.4 ± 127.8	18.8 ± 10.2
Pre-treatment (n = 10)	300.2 ± 174.2	285.4 ± 127.8	18.8 ± 10.2
Post-treatment (n = 10)	24.8 ± 14.2	24.2 ± 10.2	18.8 ± 10.2

TABLE III. Soluble CD23 (Mean ± SD).

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Specific IgE and Soluble CD23 During the Pollen Season📈

The serum levels of specific IgE were not significantly different between the pharmacotherapy group and the immunotherapy group ($P = .6234$) ([Tables II and III](#)). The serum levels of sCD23 in the immunotherapy group were not significantly different from those in the nonatopic group ($P = .4798$). However, the serum levels of sCD23 in the pharmacotherapy group were significantly higher than those in the nonatopic group ($P = .0004$) and those in the immunotherapy group ($P = .0018$).

Seasonal Changes in Specific IgE and Soluble CD23📈

Both specific IgE and sCD23 were significantly increased during the pollen season in the pharmacotherapy group (specific IgE: $P < .0001$; sCD23: $P < .0001$) (Tables II and III). In the immunotherapy group, specific IgE was significantly increased during the pollen season ($P = .0052$), but sCD23 was not significantly increased during the season ($P = .2079$). A seasonal increase in rates in both specific IgE and sCD23 was significantly smaller in the immunotherapy group than the pharmacotherapy group (specific IgE: $P < .0001$; sCD23: $P = .0243$).

Clinical Effect in the Pharmacotherapy Group

The number of the good responders and the poor responders to antiallergic pharmacotherapy was 33 and 31, respectively (Table II and III). Neither specific IgE nor sCD23 before the pollen season was significantly different between the good responders and the poor responders of the pharmacotherapy group (specific IgE: $P = .7067$; sCD23: $P = .4319$). Both specific IgE and sCD23 before the pollen season were significantly increased during the pollen season in both the good responders (specific IgE: $P < .0001$; sCD23: $P = .0022$) and the poor responders (specific IgE: $P = .0002$; sCD23: $P = .0013$; Fig. 1). Neither the seasonal increase in rate in specific IgE nor the seasonal increase in rate in sCD23 was significantly different between the good responders and the poor responders (specific IgE: $P = .4600$; sCD23: $P = .3465$).

Clinical Effect in the Immunotherapy Group

The number of the good responders and the poor responders to immunotherapy was 32 and 16, respectively (Tables II and III). Neither the age, the duration of the disease, the duration of immunotherapy, nor specific IgE or sCD23 before the pollen season was significantly different between the good responders and the poor responders (age: $P = .5037$; duration of the disease: $P = .1124$; duration of immunotherapy: $P = .2093$; specific IgE: $P = .0860$; sCD23: $P = .3640$). Neither specific IgE nor sCD23 was significantly increased during the pollen season in the good responders (specific IgE: $P = .3631$; sCD23: $P = .1826$; Fig. 1). By contrast, both

specific IgE and sCD23 were significantly increased during the pollen season in the poor responders (specific IgE: $P = .019$; sCD23: $P = .0052$; Fig. 1). Both the seasonal increase in rate in specific IgE and the seasonal increase in rate in sCD23 were significantly smaller in the good responders than in the poor responders (specific IgE: $P = .0024$; sCD23: $P = .0001$).

Correlation Analysis in the Pharmacotherapy Group

A significant correlation was observed between the levels of specific IgE and the levels of sCD23 not only before the pollen season ($r_s = 0.555$, $P < .0001$) but also during the pollen season ($r_s = 0.473$, $P = .0002$). Additionally, the seasonal increase in rate in specific IgE was significantly correlated with the seasonal increase in rate in sCD23 ($r_s = 0.321$, $P = .0107$).

Correlation Analysis in the Immunotherapy Group

The levels of specific IgE before the pollen season were not significantly correlated with the levels of sCD23 before the pollen season ($r_s = 0.224$, $P = .1259$). On the other hand, the levels of specific IgE during the season were significantly correlated with the levels of sCD23 during the season ($r_s = 0.382$, $P = .0088$). The seasonal increase in rate in specific IgE was significantly correlated with the seasonal increase in rate in sCD23 ($r_s = 0.474$, $P = .0012$). The duration of immunotherapy was inversely correlated with the seasonal increase in rate in specific IgE ($r_s = -0.474$, $P = .0006$) and the seasonal increase in rate in sCD23 ($r_s = -0.425$, $P = .0021$).

DISCUSSION

The first purpose of this study was to examine whether serum levels of sCD23 in patients with seasonal allergic rhinitis were higher than those in nonatopic controls. The serum level of sCD23 shows a marked age-dependent variation; it is highest in infants and then decreases gradually with age.¹⁷ In this study, all the subjects enrolled were 19 or more years old, and there was no significant difference in ages among the nonatopic group, the pharmacotherapy group, and the immunotherapy group. The serum levels of

sCD23 in the pharmacotherapy group before the pollen season were significantly higher than those in the nonatopic group. This may suggest that relevant cells to the potential source of sCD23 are primed in patients with seasonal allergic rhinitis, even out of the pollen season when the allergen does not naturally exist. In the pharmacotherapy group, the level of sCD23 before the pollen season was significantly correlated with the level of specific IgE before the pollen season. Elevated serum levels of sCD23 have been reported in atopic patients,¹⁴⁻¹⁷ although correlation between total IgE and sCD23 was weak ¹⁵⁻¹⁷ or not significant.¹⁴ This is the first documentation of the significant correlation between specific IgE and sCD23 in patients with allergic diseases.

The Japanese cedar pollinates heavily during early spring, and there is no significant interference by other pollens. Therefore, Japanese cedar pollination provides a good in vivo model to investigate the effect of natural allergen provocation on the natural changes in immunologic parameters.¹⁸ The second aim of this study was to investigate seasonal changes in sCD23 in response to natural exposure to the pollens. Our study design failed to examine their seasonal changes in untreated patients because of ethical reasons. In the poor responders to pharmacotherapy as well as immunotherapy, sCD23 was significantly increased during the pollen season compared with levels before the season. Therefore, our data suggest that sCD23 in sera is elevated in response to natural allergen exposure during the pollen season. However, there is a report that contradicts our results. Yanagihara et al.¹⁵ monitored sCD23 levels for a year in six rye grass-sensitive hay fever subjects, and they demonstrated that the levels of sCD23 remained stable. However, they determined sCD23 before and after the pollen season, but not during the pollen season, which might be a reason for the discrepancy, together with a smaller number of subjects used in their study.

It is also of clinical interest whether seasonal increases in sCD23 and elevated levels of sCD23 could be modulated by medical treatments. Thus the third aim of this study was to investigate the effect of medical treatment on the seasonal increase in sCD23 and elevated levels of sCD23 before the pollen season. The levels of sCD23 before the pollen season in the immunotherapy group were significantly lower than those in the pharmacotherapy group, and were not significantly different from those in the nonatopic

group. This data may suggest possible modulation of sCD23 by immunotherapy. Possible effects of medical treatments on sCD23 in patients with allergic diseases were previously discussed by Yanagihara et al.,¹⁵ based on comparing sCD23 levels in patients groups under various treatments with the untreated patient group. The sCD23 levels in patients receiving immunotherapy or local steroids were not significantly different from those in untreated subjects, but subjects receiving systemic steroids had significantly lower sCD23 levels than the untreated subjects. Their data may suggest that sCD23 could be decreased by systemic steroids, but not by immunotherapy. However, such discussion based on group comparison may lead to confusing conclusions, because pretreatment values are considerably different in individual patients.¹⁴⁻¹⁷

Therefore, we examined the effect of immunotherapy and nonsedating antihistamines on seasonal increase in sCD23. The levels of sCD23 were significantly increased during the pollen season not only in the poor responders but also in the good responders to the pharmacotherapy. Besides, seasonal increase in rates in sCD23 was not different between them. It may not be conclusively determined because our study had no untreated patients, but it is probable that nonsedating antihistamines do not significantly modulate a seasonal rise in sCD23. Even though nonsedating antihistamines might somewhat modulate a seasonal rise in sCD23, this modulation by nonsedating antihistamines does not influence the magnitude of the clinical effect. By contrast, sCD23 was not significantly increased in the good responders to immunotherapy, but was significantly increased in the poor responders to immunotherapy. In addition, the seasonal increase in rates in sCD23 were significantly smaller in the good responders than the poor responders to immunotherapy. These data have therefore suggested that immunotherapy, unlike nonsedating antihistamines, could inhibit the seasonal increase in sCD23 seen in response to natural allergen exposure and that the magnitude of the modulation could affect the clinical effect.

Our previous study has demonstrated that immunotherapy is able to attenuate the usual rise in specific IgE in the pollen season and

additionally to switch the seasonal preferential IgE response to an IgG response.[18,19](#) In the present study, seasonal changes in specific IgE in response to the pollen exposure were also investigated. A seasonal increase in specific IgE was apparent in the good responders as well as poor responders to the pharmacotherapy, and no significant difference was seen between the seasonal increase in rate in the good responders and that in the poor responders to the pharmacotherapy. By contrast, specific IgE was not significantly increased in the good responders to immunotherapy, but was significantly increased in the poor responders to immunotherapy. In addition, the seasonal increase in rates in specific IgE was significantly smaller in the good responders than in the poor responders to immunotherapy. These results are in line with our previous report.[18,19](#) In the present study, a significant correlation between the seasonal increase in rate in specific IgE and the seasonal increase in rate in sCD23 was confirmed in both the pharmacotherapy group and the immunotherapy group. In conclusion, taking these lines of evidence together, a seasonal rise in sCD23 is associated with and is probably involved in a seasonal rise in specific IgE in patients with seasonal allergic rhinitis, and successful immunotherapy is capable of blunting a seasonal increase in sCD23, thus resulting in the attenuation of a seasonal increase in specific IgE and clinical benefits during the pollen season.

Our study has demonstrated that immunotherapy can decrease a seasonal increase in sCD23 in seasonal allergic rhinitis, but the effect of immunotherapy on sCD23 levels in patients allergic to Hymenoptera venom was recently reported by Forman et al.[22](#) In their study, sCD23 levels in 15 patients were determined before and at variable time courses during immunotherapy (mean duration, 54 months), and sCD23 levels were found to be unchanged over the course of venom immunotherapy. In addition, their patients had lower mean sCD23 levels when compared with nonatopic controls. The clinical role of sCD23 might be quite different between venom allergy and seasonal allergic rhinitis due to pollens.

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Harvard Medical School to Host Endoscopic and Image-Guided Sinus Surgery Course November 6-7, 1998

The Joint Center for Otolaryngology, Beth Israel Deaconess Medical Center and Brigham and Women's Hospital of Harvard Medical School is sponsoring a course "Advanced Endoscopic and Image-Guided Sinus Surgery: Beyond the Basics" on November 6th and 7th, 1998 at the Fairmont Copley Plaza Hotel in Boston, Massachusetts. This year's honored guest is Professor Heinz R.

This course provides state-of-the-art information and instruction to the practicing otolaryngologist regarding advanced techniques in endoscopic sinus surgery. The use of endoscopes has significantly decreased the need for more invasive techniques in the management of CSF leaks and frontal sinus disease. Real-time imaging techniques, including the use of CT and MRI scanners, may hold the key to safer revision and frontal sinus surgery.

Cadaver dissections with appropriate endoscopes and instruments will provide hands-on exposure to the various operations discussed. CT-guided sinus surgery devices and state-of-the-art endoscopic surgical simulators will also be available during the laboratory session.

For more information contact Harvard MEDCME, P.O. Box 825, Boston, MA 02117-0826 or call 617-432-1525.

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Equation 1

$$\text{Seasonal increase in rate (\%)} = \frac{\text{values during pollen season} - \text{values before pollen season}}{\text{values before pollen season}} \times 100.$$

Equation 1

From: Ohashi: Laryngoscope, Volume 108(5).May 1998.706-711

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TABLE I

Group	Sex		Age (y)				Duration of the Disease (y)				Duration of Immunotherapy (y)			
	Female	Male	Mean	SD	SE	Range	Mean	SD	SE	Range	Mean	SD	SE	Range
Nonatopic (n = 20)	12	8	35.4	9.8	2.2	25–64	—	—	—	—	—	—	—	—
Pharmacotherapy (n = 64)	38	26	32.4	9.6	1.2	19–55	8.3	4.8	0.6	2–19	—	—	—	—
Immunotherapy (n = 48)	29	19	34.0	8.4	1.2	19–55	9.3	4.2	0.6	3–18	5.0	4.4	0.6	1–15

TABLE I. Background of the Subjects.

From: Ohashi: Laryngoscope, Volume 108(5).May 1998.706-711

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TABLE II

	Before Pollen Season (U/mL)	During Pollen Season (U/mL)	Seasonal Increase Rate (%)
Pharmacotherapy group (n = 64)	23.88 ± 23.04	33.61 ± 26.83	77.8 ± 101.0
Good responders (n = 33)	25.77 ± 24.28	37.46 ± 27.54	98.6 ± 129.2
Poor responders (n = 31)	21.87 ± 21.85	29.52 ± 23.03	55.7 ± 51.5
Immunotherapy group (n = 48)	26.64 ± 21.63	28.86 ± 21.51	11.1 ± 22.3
Good responders (n = 32)	24.81 ± 24.06	24.60 ± 22.35	3.9 ± 17.1
Poor responders (n = 16)	30.28 ± 15.77	37.39 ± 17.35	25.3 ± 25.1

TABLE II. Specific IgE (Mean ± SD).

From: Ohashi: Laryngoscope, Volume 108(5).May 1998.706-711

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TABLE III

	Before Pollen Season (ng/mL)	During Pollen Season (ng/mL)	Seasonal Increase Rate (%)
Pharmacotherapy group (n = 64)	255.2 ± 114.1	290.4 ± 127.6	17.3 ± 25.8
Good responders (n = 33)	268.1 ± 131.8	307.1 ± 144.2	20.6 ± 28.1
Poor responders (n = 31)	241.6 ± 91.8	272.7 ± 106.7	13.8 ± 23.0
Immunotherapy group (n = 48)	205.8 ± 87.8	218.8 ± 97.4	9.3 ± 29.6
Good responders (n = 32)	196.7 ± 81.5	186.1 ± 73.2	-3.1 ± 14.2
Poor responders (n = 16)	223.9 ± 99.4	284.3 ± 108.5	34.1 ± 36.6
Nonatopic group (n = 20)	190.5 ± 49.9	—	—

TABLE III. Soluble CD23 (Mean ± SD).

From: Ohashi: Laryngoscope, Volume 108(5).May 1998.706-711

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