

Correspondence

β_2 -ADRENOCEPTOR GENOTYPES AND DIPTYPES AT POSITIONS 16 AND 27 IN ALLERGIC RHINITIS AND ASTHMA

To the Editor:

The distribution of β_2 -adrenoceptor genotypes and diplotypes at positions 16 and 27 in allergic rhinitis is currently unknown. The concept of the unified airway has gained much acceptance with the recognition of a strong pathophysiological link between allergic rhinitis and asthma.^{1,2} An analysis was performed to determine whether a particular genotype or diplotype was more prevalent in a population of 535 patients with allergic rhinitis alone, asthma alone, and allergic rhinitis with concomitant asthma. Patients were identified on the basis of their medical history in keeping with their medical therapy. None of the patients with allergic rhinitis alone or asthma alone were receiving inhaled or intranasal corticosteroids, respectively, whereas patients with allergic rhinitis with concomitant asthma were receiving both corticosteroids. Data are shown in Table 1. There were no significant differences comparing all groups for either genotypes or diplotypes at positions 16 and 27. The absence of the Arg-Arg/Glu-Glu diplotype is due to linkage disequilibrium between genotypes at positions 16 and 27.³ In summary, there was no evidence of a particular genotype or diplotype being more prevalent with equal distribution observed in patients with allergic rhinitis alone, asthma alone, and allergic rhinitis with concomitant asthma.

Table 1. Data for Patients with Allergic Rhinitis Alone (AR), Asthma Alone (A), and Allergic Rhinitis with Concomitant Asthma (ARA)

	AR	A	ARA
Men/women, No.	40/43	102/106	89/155
Age, y	41	45	43
Genotype at position 16, %			
Arg-Arg	14	10	15
Gly-Gly	38	42	37
Arg-Gly	48	48	48
Genotype at position 27, %			
Gln-Gln	31	29	32
Glu-Glu	22	23	16
Gln-Glu	47	48	52
Diplotype at positions 16 and 27, %			
Arg-Arg/Gln-Gln	15	10	13
Arg-Arg/Glu-Glu	0	0	0
Arg-Arg/Gln-Glu	0	1	2
Gly-Gly/Gln-Gln	7	4	4
Gly-Gly/Glu-Glu	20	23	15
Gly-Gly/Gln-Glu	10	14	18
Arg-Gly/Gln-Gln	10	15	15
Arg-Gly/Glu-Glu	1	0	1
Arg-Gly/Gln-Glu	37	33	32

DANIEL K. C. LEE, MBBCh
Department of Respiratory Medicine
Ipswich Hospital
Suffolk, England

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ORAL TOLERANCE OF CARBOXYMETHYLCELLULOSE IN PATIENTS WITH ANAPHYLAXIS TO PARENTERAL CARBOXYMETHYLCELLULOSE

To the Editor:

We read with interest the article by Karsh and Yang¹ and the letter by Garcia-Ortega et al² in recent issues of the *Annals*. We agree with Garcia-Ortega et al that the culprit agent in anaphylaxis to some corticosteroid preparations may be carboxymethylcellulose. In this regard, we would like to report our own observations, which may add some important information.

We have previously described 3 patients with anaphylaxis to parenteral corticosteroid preparations.³ In all 3 individuals, the results of intradermal skin tests with carboxymethylcellulose were positive, and the test results for pure corticosteroids were negative. We were not able to demonstrate specific IgE to carboxymethylcellulose with an immunodot technique. However, the results of the sulfidoleukotriene stimulation test were positive in 2 patients.

Recently, a 52-year-old man was observed who had experienced an anaphylactic reaction with nausea, sweating, thoracic oppression, pruritus, generalized urticaria, and hypotension (60/30 mm Hg) after having received paravertebral infiltration of lidocaine and Kenacort A10. He had negative prick/puncture test results to several corticosteroids, including the triamcinolone preparation. Intradermal test results were positive with carboxymethylcellulose (7.5 mg/mL, 1:10) and 2 corticosteroid preparations containing carboxymethylcellulose but negative to lidocaine and 4 other corticosteroids (betamethasone, methylprednisolone, hydrocortisone), including Kenacort A80 (triamcinolone), which does not contain carboxymethylcellulose. The results of a subcutaneous provocation with lidocaine (1.5 mL) were negative.

Carboxymethylcellulose is a viscous carbohydrate used as a suspending agent in injectable hormone preparations such as corticosteroids, luteinizing hormone-releasing hormone, and somatostatin. It is also present in tablets as a binding, gliding, and antiadherent substance, as well as in oral contrast media and as the additive E 466 in food. It is the active ingredient in bulk laxatives and hydrocolloid wound dressings. Ingestion of a large volume of barium sulfate containing carboxymethylcellulose has resulted in anaphylaxis.⁴

Because carboxymethylcellulose is widely used in food and tablets and complete eviction may be impossible, we have performed oral provocation tests after obtaining informed consent. All 4 patients tolerated 136 to 250 mg of carboxymethylcellulose without adverse event. This indicates that typical oral doses are tolerated, whereas small amounts of parenteral carboxymethylcellulose may cause life-threatening allergic reactions. However, caution is required with large doses as present in oral contrast media.

ANDREAS J. BIRCHER, MD
JAN IZAKOVIC, MD

PAUL L. BIGLIARDI, MD
Allergy Unit
Department of Dermatology
University Hospital and
Dermatology, Kantonsspital
Schaffhausen
Basel, Switzerland

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Erratum

In the rostrum article by Bobby Lanier, MD, and Gailen D. Marshall, MD, PhD, “Unanswered questions on omalizumab (Xolair) patient selection and follow-up” (*Ann Allergy Asthma Immunol.* 2004;92:198–200), there was an error in the first paragraph of the *Discussion* section. The sentence “Studies to date suggest that safety should pose major concerns for most patients, at least in the short term” should read “Studies to date suggest that safety should **not** pose major concerns for most patients, at least in the short term.” The authors apologize for this oversight.