

and phosphorylated p38 MAPK to constitute a novel read-out (1).

The authors would like to thank Ms Christel Mertens for her skilful technical assistance and Hans-Jörg Bühring (Department of Internal Medicine, Division of Haematology, Immunology and Oncology, University of Tübingen, Germany) for kindly providing the CD203c antibodies.

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Accepted for publication 8 February 2008
Allergy 2008; 63:941–942

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DOI: 10.1111/j.1398-9995.2008.01700.x

References

1. Ebo DG, Dombrecht EJ, Bridts CH, Aerts NE, de Clerck LS, Stevens WJ. Combined analysis of intracellular signalling and immunophenotype of human peripheral blood basophils by flow cytometry: a proof of concept. *Clin Exp Allergy* 2007;**37**: 1668–1675.
2. Ebo DG, Stevens WJ, Bridts CH, de Clerck LS. Latex-specific IgE, skin testing, and lymphocyte transformation to latex in latex allergy. *J Allergy Clin Immunol* 1997;**100**:618–623.
3. Soul-Lawton J, Seaber E, On N, Wootton R, Rolan P, Posner J. Absolute bioavailability and metabolic disposition of valaciclovir, the L-valyl ester of acyclovir, following oral administration to humans. *Antimicrob Agents Chemother* 1995;**39**:2759–2764.
4. Acosta EP, Fletcher CV. Valaciclovir. *Ann Pharmacother* 1997;**31**:185–191.
5. Brantley JS, Hicks L, Sra K, Tying SK. Valaciclovir for the treatment of genital herpes. *Expert Rev Anti Infect Ther* 2006;**4**:367–376.
6. Henry RE, Wegmann JA, Hartle JE, Christopher GW. Successful oral acyclovir desensitization. *Ann Allergy* 1993;**70**:386–388.
7. Lammintausta K, Makela L, Kalimo K. Rapid systemic valaciclovir reaction subsequent to aciclovir contact allergy. *Contact Dermatitis* 2001;**45**:181.
8. Vernassiere C, Barbaud A, Trechot PH, Weber-Muller F, Schmutz JL. Systemic acyclovir reaction subsequent to acyclovir contact allergy: which systemic antiviral drug should then be used? *Contact Dermatitis* 2003;**49**:155–157.
9. Bayrou O, Gaouar H, Leynadier F. Famciclovir as a possible alternative treatment in some cases of allergy to acyclovir. *Contact Dermatitis* 2000;**42**:42.
10. Khunda A, Kawsar M, Parkin JM, Forster GE. Successful use of valciclovir in a case of recurrent urticaria associated with genital herpes. *Sex Transm Infect* 2002;**78**:468.
11. Ebo DG, Sainte-Laudy J, Bridts CH, Mertens CH, Hagendorens MM, Schuerwegh AJ et al. Flow-assisted allergy diagnosis: current applications and future perspectives. *Allergy* 2006;**61**:1028–1039.

The secretory tailpiece isoform of IgE is not associated with Allergy

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Key words: atopy; hayfever; IgE; IgE tailpiece.

Atopy, an exaggerated IgE-mediated immune response, is a common feature in many inflammatory diseases, including bronchial asthma and allergic rhinitis (hayfever). Type-I hypersensitivity reactions underlying all atopic conditions and indicators, such as positive skin prick tests to common environmental allergens, correlate well with increased IgE levels in serum or plasma (1, 2). Tailpiece (IgE-tp) is a novel secreted isoform of IgE, formed by alternative splicing of the ϵ gene locus, whose precise role in inflammation is unknown (3, 4). Our preliminary study herein has for the first time attempted to

Levels of IgE tailpiece isoform are unchanged in atopy and may not be a major determinant of allergic inflammation.

address the role of IgE-tp in the inflammatory process. Using indirect sandwich ELISAs specific for either IgE-tp or total IgE, we have investigated their levels in plasma from atopic and non-atopic subjects.

Plasma was obtained from three cohorts ($n = 80, 33$ and 34 , respectively). These subjects were originally recruited as families with two siblings with doctor-diagnosed asthma (5). In the current analyses, cohort 1 represents the parents (mean age = 42.7 ± 10.1), cohort 2 asthma sibling A (mean age = 15.3 ± 9.2) and cohort 3 asthma sibling B (mean age = 13.7 ± 9.6). For quantifying IgE-tp and total IgE, ELISA plates were coated with a tp-specific mAb (367) and an anti- $\epsilon\epsilon 2$ mAb (4.15), respectively. Wells were then incubated with plasma preceded by incubation with a polyclonal anti-human IgE-Fc ϵ antibody (Serotec)-conjugated to HRP. IgE levels were compared to atopy data (as assessed by subjects suffering from hayfever (questionnaire) or with positive skin prick tests to common allergens (e.g. Derp1, Feld1). Total IgE levels were significantly increased in atopic subjects when compared with control subjects for all the three cohorts (Fig. 1A and C). This was not the case with IgE-tp, with similar levels observed in all subjects (Fig. 1B and D). Measurements of lung function [including forced expiratory volume in 1 s (FEV₁), FEV₁/forced vital capacity (FVC) ratio and bronchial hyper-responsiveness (BHR)] were also compared with the IgE levels for cohorts 2 and 3; however, no significant correlations were observed with either isoform (data not shown). Lung function data were not available for cohort 1.

Our preliminary studies have confirmed the previous observations that total IgE in plasma or serum samples from atopic subjects is elevated (1, 2). This was not, however, accompanied with an increase in IgE-tp, suggesting that the Th2-type inflammatory responses observed in allergy occur through other isoforms of IgE (3, 4). Though lung function data did not correlate with total IgE levels, there were trends for differences in bronchial hyper-responsiveness in cohorts 2 and 3. Higher numbers of subjects in each group may well elicit a better relationship between

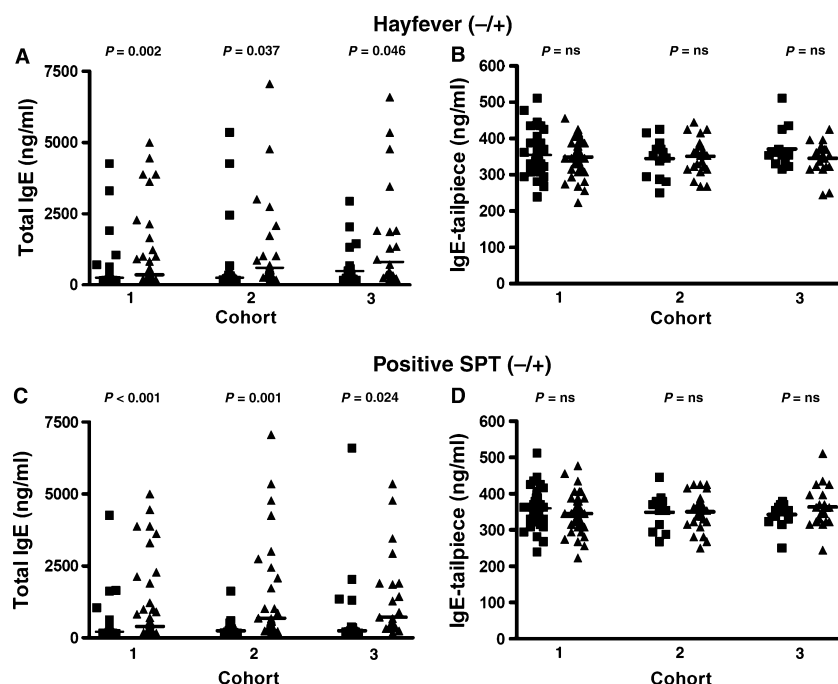


Figure 1. Plasma total IgE and IgE-tailpiece levels in the presence and absence of atopy. Total IgE (A, C) and IgE-tailpiece (B, D) levels were investigated in plasma taken from families with multiple-asthma children. These families were divided into non-related individuals, cohort 1 parents ($n = 80$), cohort 2 asthma sibling A ($n = 33$) and cohort 3 asthma sibling B ($n = 34$). Atopy was defined by the presence of hayfever (questionnaire) or positive skin prick test (SPT) to common allergens. Total IgE data were not normally distributed (histogram not shown); therefore, medians were compared using the Mann–Whitney U -test. IgE-tailpiece data were normally distributed (histogram not shown) and therefore means were compared using Student's t -test. Data represent all data points in the absence (squares) or presence (triangles) of atopy with median or mean values shown for total IgE and IgE tailpiece, respectively.

levels of IgE and lung function. The task ahead will be to investigate IgE-tp levels in other conditions, such as helminth infection, where total IgE levels are far higher than that for allergy and the Th2 immune response is more prominent (6).

We thank Professor Ian Hall for providing access to the Nottingham Family samples. We would also like to thank Professor Sherie Morrison (University of California, Los Angeles) and Dr Ke Zhang (University of California, Los Angeles) for their kind gifts of the recombinant human IgE-Tp and anti-IgE-Tp specific monoclonal Ab 367, respectively.

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Accepted for publication 13 February 2008
Allergy 2008; 63:942–943
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Munksgaard
DOI: 10.1111/j.1398-9995.2008.01708.x

References

1. Pekkarinen PT, von Hertzen L, Laatikainen T, Mäkelä MJ, Jousilahti P, Kosunen TU et al. A disparity in the association of asthma, rhinitis, and eczema with allergen-specific IgE between Finnish and Russian Karelia. *Allergy* 2007;**62**:281–287.
2. Ballardini N, Nilsson C, Nilsson M, Lilja G. ImmunoCAP Phadiatop infant – a new blood test for detecting IgE sensitisation in children at 2 years of age. *Allergy* 2006;**61**:337–343.
3. Chan LA, Lyczak JB, Zhang K, Morrison SL, Saxon A. The novel human IgE epsilon heavy chain, epsilon tailpiece, is present in plasma as part of a covalent complex. *Mol Immunol* 2000;**37**:241–252.

4. Aslam A, Quinn P, McIntosh RS, Shi J, Ghumra A, McKerrrow JH et al. Proteases from *Schistosoma mansoni* cercariae cleave IgE at solvent exposed interdomain regions. *Mol Immunol* 2008;**45**:567–574.
5. Wheatley AP, Bolland DJ, Hewitt JE, Dewar JC, Hall IP. Identification of the autoantigen SART-1 as a candidate gene for the development of atopy. *Hum Mol Genet* 2002;**11**:2143–2146.
6. Yazdanbakhsh M, Kremsner PG, van Ree R. Allergy, parasites, and the hygiene hypothesis. *Science* 2002;**296**:490–494.

Occupational asthma due to tampico fiber from agave leaves

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Key words: exhaled nitric oxide; induced sputum; occupational asthma; specific inhalation challenge; tampico fiber.

Tampico fiber is extracted from the leaves of *Agave lechugilla* which grows mainly in Mexico. The

material is used

extensively for making yard brooms, deck brushes and bath brushes. Agave-induced irritant contact dermatitis has been previously reported among workers

We surveyed the five employees (two supervisors and three machine operators) at a brush making-plant. The two supervisors (patients 1 and 2), who were brothers and had been exposed to tampico for 13 and 10 years, respectively, reported work-related rhinitis and asthma symptoms. Patient 1 also experienced localized contact urticaria with tampico fiber. Patient 1 had experienced

Two brush-making workers who developed asthma and rhinitis symptoms following occupational exposure to tampico fiber are reported.