

Correspondence

Sting anaphylaxis and urticaria pigmentosa

To the Editor:

Fricker et al. (J Allergy Clin Immunol 1997;100:11-15) recently reported that anaphylactic symptoms after Hymenoptera sting in patients with urticaria pigmentosa are most often IgE mediated. They do indicate that occasionally reactions can be observed in the absence of IgE sensitization to venom allergens. They offer several hypotheses regarding mechanisms by which Hymenoptera venoms could induce anaphylaxis. On the other hand, they discussed the possibility that circulating IgE in patients with urticaria pigmentosa may be largely absorbed by the abundant tissue mast cells. This is probably what is occurring.

A few years ago my colleagues and I became intrigued by the presence of anaphylactic sensitivity to fire ant venom in a 4-year-old girl with urticaria pigmentosa. Specific antibody levels to *Solenopsis invecta* were clearly positive at 25 ng/ml. More importantly, IgE-mediated sensitivity to fire ant venom in this patient was confirmed by basophil histamine release after exposure of the patient's cells to several concentrations of purified fire ant venom. This young girl had the first reported case of anaphylaxis to Hymenoptera venom confirmed by basophil histamine release and not the presence of tryptase. The findings of Fricker et al. are consistent with our findings.

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REFERENCE

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Maternal atopy and childhood bronchial asthma

To the editor:

A recent article showed that the level of association of asthma or wheezing in parents and their children suggests that a susceptibility is important in the etiology of bronchial asthma.¹ Unpublished data from 215 Greek children with asthma who were 4 to 12 years of age showed that maternal atopies were more frequent (13.5% of children) than paternal atopies (3.5% of children) ($p < 0.01$). Familial aggregation of the

phenotypes associated with asthma and other atopic conditions has been reported in recent articles.² Our data agree with those of other authors who found significant sharing of maternal chromosome 11q13 in alleles from sibling-pairs with atopic IgE responsiveness. These findings suggest that the inheritance of atopy at this locus has occurred through the maternal line.³ Our results, in combination with previously published evidence, reinforce the hypothesis that a genetic susceptibility to allergic disorders may exist in patients with asthma.¹ Maternal atopy especially seems to increase the risk of asthma in children.

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2. Wilkinson J, Holgate ST. Candidate gene loci in asthmatic and allergic inflammation. *Thorax* 1996;51:3-8.
3. Cookson WOCM, Young RP, Sandford AJ, Moffatt MF, Shirakawa T, Sharp PA, et al. Maternal inheritance of atopic IgE responsiveness on chromosome 11q. *Lancet* 1992;340:381-4.

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RANTES

To the Editor:

For some reason, when the acronym RANTES is used in papers published in *The Journal of Allergy and Clinical Immunology* and elsewhere, it is not spelled out in the abbreviation list or in the text. I only recently discovered its meaning. In case anyone else out there is wondering, it stands for "Regulated upon Activation Normal T cell Expressed and Secreted."

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