

# Correspondence

## Erratum

### To the Editor:

We, three of the co-authors (M. Luo, H. Ni, and A. H. Sehon) of the abstract, "Identification of a novel highly crossreactive allergen in Kentucky Bluegrass pollen" (*J Allergy Clin Immunol* 1994;93:231), wish to be disassociated from its authorship because its conclusions are misleading. Two of us (Luo and Ni) performed the experiments and established that only 10 of 48 sera, that is, less than 20% (and not as stated 60% of 44 sera) of individuals allergic to grass pollen had IgE antibodies cross-reacting with the recombinant KBG51 allergens. The ELISA tests were performed with the fusion protein, consisting of the KBG51 allergens and glutathione S-transferase (GST), and not with KBG51 alone.

The results of further experiments were reported, under the title "Identification of a New Highly Cross-Reactive Allergen in Plant Pollens," in Abstract 260 of the combined XV International Congress of Allergology and Immunology and the Annual Meeting of the European Academy of Allergology and Clinical Immunology 1994, held in Stockholm, June 26 to July 1, 1994. This abstract under the co-authorship of S. S. Mohapatra, M. Luo, and H. Ni was submitted and presented by S. S. Mohapatra without the knowledge of M. Luo and H. Ni. The claim was made that about 40% of 1000 sera of individuals allergic to pollens possessed IgE antibodies specific to rCRAL 51, as opposed to 51% of sera, which possessed IgE antibodies specific to KBG pollen extract. Because of this apparent, high cross-reactivity, the former was designated as cross-reactive allergen CRAL51.

The latter experiments were performed once, solely by H. Ni, who established that only 242 of 979 sera (i.e., 24%) produced positive reactions with the fusion protein and that most of the sera proved to be positive also to the GST moiety alone. GST is not a KBG allergen but a translation product of the expression vector, PGEX-2T-1. Hence, even the observations that only 20% to 24% of the sera produced reactions with the fusion protein represent at best an upper limit of cross-reactivity, because the positive reactions would have also included reactions with GST.

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## Hereditary angioedema with ascites

### To the Editor:

In the August 1995 issue of the Journal, Shah et al. (*J Allergy Clin Immunol* 1995;96:259-61) described a case

of hereditary angioedema (HAE) in which recurrent abdominal pain with ascites was demonstrated by repeated ultrasonography of the abdomen. The ascites resolved on day 3, at the time of clinical remission. The authors stated that: "ascites has not been previously described with intestinal involvement in HAE."

We have previously reported, following Burghardt and Wernze,<sup>1</sup> the diagnostic value of ultrasonography in HAE when fluid accumulation developed in the peritoneal cavity.<sup>2</sup>

In this rare disease the diagnosis of HAE was established nearly 20 years after the first acute episode in the first recognized member of a family with HAE.<sup>3</sup> Thus recurrent abdominal pain may often be unexplained in such patients. However, when the diagnosis of HAE was made, the abdominal pain could be considered as HAE with gastrointestinal involvement, even in the absence of vomiting. When clinical findings are present, we usually perform ultrasonography to detect accumulation of fluid in the peritoneal cavity. Thus we recently observed another case of HAE with ascites when gastrointestinal involvement occurred in spite of long-term treatment with danazol (100 mg/day) in a 17-year-old woman. Indeed, ultrasonography appears useful in the treatment of patients with HAE who have abdominal pain.

Such peritoneal fluid accumulation is not confined to HAE. We have also observed it in acute idiopathic nephrotic syndrome.<sup>2</sup> Moreover, it has been recently described with submucosal edema induced by treatment with angiotensin-converting enzyme inhibitors; thus, similarities to HAE occur in gastrointestinal attacks.<sup>4</sup> In both cases, ultrasonography can easily detect ascites.

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