

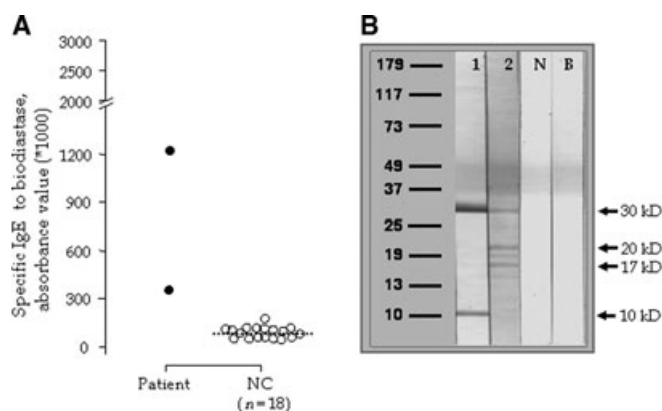


Figure 1. (A) Specific binding of IgE antibodies to budiastase by ELISA in sera of two patients (●) and unexposed healthy controls (○). (B) IgE immunoblot analysis. N, nonatopic controls; B, buffer control.

specific IgE by ELISA and IgE immunoblotting. Twelve nonatopic healthy subjects with no history of exposure and negative SPT results for budiastase were enrolled as controls. The positive cutoff value for a specific IgE antibody was the mean $\pm$ 3 SD of its absorbance value. Two rhinitis subjects suffering from work-related symptoms had strong positive responses to budiastase by SPT, and showed high levels of specific IgE to budiastase by ELISA (Fig. 1A). Both patients were nonatopics, and one had a highserum level of specific IgG4 to budiastase. IgE immunoblot analysis demonstrated the presence of four IgE-binding components (30, 20, 17, and 10 kDa), and the 30-kDa component was common to both patients (Fig. 1B). The major pathogenic mechanism of occupational allergies induced by high-molecular-weight drugs is an IgE-mediated response (6). In this study, we confirmed the involvement of an IgE-mediated mechanism, as both subjects showed positive responses by SPT, high serum levels of specific IgE antibodies to budiastase, and four IgE-binding components were noted by IgE immunoblot analysis. Furthermore, a high serum level of specific IgE to fungal α -amylase was noted by ELISA in both patients, suggesting that the fungal α -amylase included in this digestive powder is the major allergenic component. The role of specific serum IgG4 antibodies in occupational allergy patients seems to be variable according to the causative agent. In this study, one patient had a high serum level of specific IgG4, while the other did not. Further

investigation will be needed to evaluate the clinical significance of specific IgG4 antibodies in patients with occupational rhinitis.

In conclusion, we determined that inhalation of budiastase power induced IgE-mediated occupational rhinitis in hospital and pharmaceutical workers, and identified IgE-binding components.

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The changing pattern of ragweed allergy in the area of Milan, Italy

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Key words: pollen; ragweed; respiratory allergy; sensitization

Ragweed allergy has become a serious problem in northern Italy during the last two decades. After the first, sparse cases reported at the end of the '80s (1, 2) ragweed now represents the second more frequent cause of respiratory allergy in this area after grass pollen (3), and in several instances, it causes the most severe airborne allergies. In a previous study looking at the pattern of *de novo* ragweed sensitization in this area, most patients did not have a familiar history of atopic diseases and, interestingly, were much older (mean age at onset about 35 year) than patients becoming sensitized to other airborne allergens (4).

Ragweed allergy trends in Milan, Italy.

Table 1. Patients monosensitization to ragweed

Year of onset of allergy	No. of patients	Mean age (range)	Median age
1990–1996	42	39.0 (20–68)	37
1997	17	36.0 (24–65)	34
1998	32	36.2 (13–63)	36
1999	43	36.5 (5–67)	37
2000	55	36.7 (5–72)	34
2001	70	36.7 (10–71)	34,5
2002	90	35.0 (6–69)	33
2003	97	36.2 (8–65)	37
2004	107	35.1 (8–80)	33
2005	63	35.5 (5–68)	35
2006	49	35.7 (5–66)	37

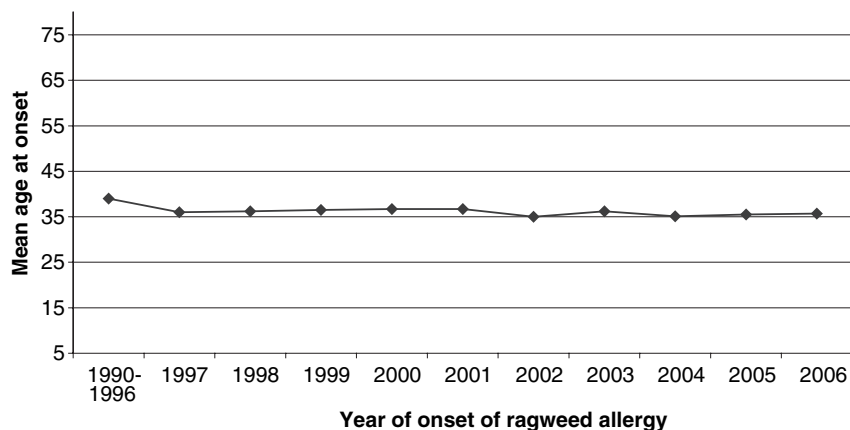


Figure 1. Mean age at onset of ragweed allergy in monosensitized patients 1990–2006.

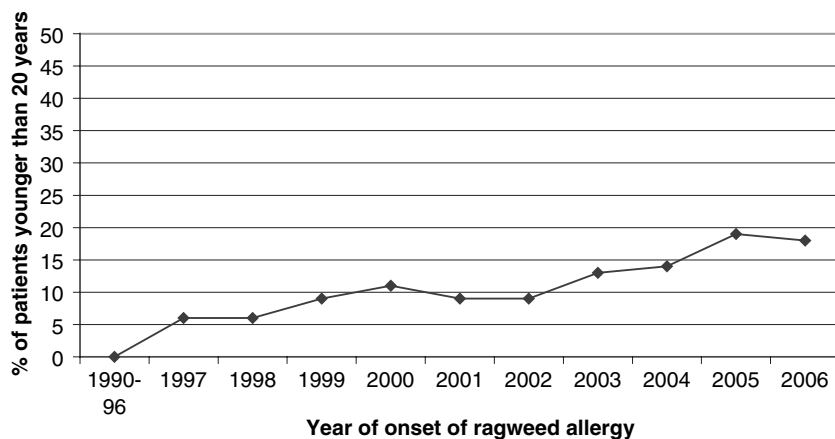


Figure 2. Proportion of young ragweed allergies by year of onset of clinical symptoms.

One suggested explanation for this difference was that the sudden exposure of the whole 'naïve' population to high

levels of a widespread airborne allergen could result in the contemporary sensitization of all those individuals who were

genetically prone to become allergic to that particular allergen, irrespective of their age (4). If this were the case, a gradual drop in the mean age of patients becoming ragweed allergic, at least in the long run, should be expected. The present study examined ragweed allergic patients over a period of 15 year.

All patients monosensitized to ragweed seen at this allergy centre between 1 January 2003 and 31 December 2006 were included. Monosensitization was defined as the presence of both typical seasonal respiratory symptoms (rhinoconjunctivitis with or without asthma) from mid-August to the end of September associated with a clear-cut positive skin prick test with commercial ragweed pollen extract (Allergopharma, Reinbeck, Germany), in the absence of any skin reactivity to other seasonal allergens, including grass, mugwort, pellitory, plantain, birch, olive, and cypress. Based on a thorough clinical interview, and on previous clinical recordings where available, the year of onset of the first ragweed allergy symptoms was ascertained.

Results are summarized in Table 1. A total of 665 patients monosensitized to ragweed were studied. Although the mean age at the onset of clinical ragweed allergy remained virtually unchanged during the study period, ranging from 39 year in 1990–96 to about 36 year in 1997–2006 (Fig. 1), a regular increase in the proportion of patients aged < 20 year becoming allergic to ragweed was observed throughout the study period; this subset of patients was 0% 1990–96 and grew up to 18% during the last 10 year (Fig. 2).

In areas where ragweed has been an allergen source for a long time, such as the USA, sensitization to this pollen is frequently observed at a very young age (5); *de novo* sensitization to aeroallergens in old individuals seems infrequent (6). It is possible that in the area north of Milan, with the gradual exhaustion of the first phase of massive allergen exposure characterized by sensitization of genetically predisposed patients irrespective of their age, a phase of a progressive reduction in the mean age of sensitization to ragweed seems to have begun. The next years will tell us whether this view is correct.

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