

b). In IgE immunoblotting, a single band was shown at about 85 kDa in both patients (Fig. 1, lanes c and d, respectively), whereas no band was demonstrated in the patient with nonatopic asthma (Fig. 1, lane e). These bands were not found in patient sera which had been preincubated with banana extract (data not shown).

■ In our patients, immediate-type hypersensitivity to banana seemed to be the most likely mechanism of the systemic symptoms, in view of the presence of specific IgE antibody against banana. However, we could not confirm this with oral provocation tests with banana because of the possibility of severe systemic reactions after challenge. In both of our patients, systemic reactions were apparent approximately 1 h after banana ingestion. We cannot explain the time difference between the appearance of the symptoms in each patient after eating banana. A difference in the time to digest banana and absorb it into the body might be the cause.

Patient 1 may have been sensitized to banana, because she had been given banana twice before the episode. In contrast, patient 2 had eaten banana for the first time. Sensitization may have occurred by ingestion of breast milk, because his mother had eaten banana frequently. Another possibility is that IgE antibody against banana was produced through cross-allergenicity with other common allergens such as egg. However, cross-allergenicity between egg white and banana was not observed (data not shown).

In sera from adult patients showing cross-allergenicity between banana and latex, prominent IgE binding was shown at less than 50 kDa (5-7). We found no specific IgE antibodies against either latex or several kinds of fruits, and prominent IgE binding was demonstrated at around 85 kDa, but not less than 50 kDa, with banana extract. Thus, our results may confirm previous findings concerning the cross-allergenicity of banana allergens.

Key words: anaphylaxis; cross-allergenicity; infancy; urticaria.

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Changed allergy pattern in outpatients

THE PREVALENCE rates of asthma and allergic rhinitis in population samples have been broadly reported (1, 2). Numerous studies have reported an increasing prevalence of these disorders in different parts of the world (1, 2). We previously reported that there is an increasing prevalence of asthma and allergic rhinitis in Ligurian (Italy) conscripts (3).

Moreover, we reported the allergy pattern in 215 outpatients, visited during 1981 for respiratory allergy, considering the distribution of asthma and allergic rhinitis, their association, and allergen sensitizations (4). In 1995, we considered 219 outpatients examined in our service: all of them had the same diagnosis, made on the basis of the same criteria.

Lifetime allergic asthma has been diagnosed on the basis of the patient's history when the patient reported repeated attacks of heavy and/or short breathing and/or wheezing that could not be explained by other conditions, there was a positive skin prick test in agreement with history, and the disorder was con-

There has been an increasing trend of association of asthma and rhinitis during the last 15 years.

firmed by clinical examination (3, 4). Allergic rhinitis has been diagnosed on the basis of patient history, when patients reported recurrent symptoms of rhinitis for at least two seasons or years, and

Table 1. Rate of asthma, allergic rhinitis, their association, and sensitization in outpatients during 1981 and 1995

	1981	1995	
Asthma	64	35	$P < 0.05$
Allergic rhinitis	82	70	NS
Asthma and allergic rhinitis	69	114	$P < 0.05$
Polysensitization	117	158	$P < 0.05$

positive skin prick test in agreement with history and clinical local examination (3, 4).

We implemented our studies with the same criteria to establish the diagnosis of asthma and allergic rhinitis as well as to perform the skin prick test.

The skin prick test was performed on all subjects, according to methodology and criteria proposed by the Italian Society of Allergy and Clinical Immunology (5), and with a common panel of allergens (3, 4). Sensitization was considered when there was a relationship between history and skin prick test positivity (i.e., wheal diameter ≥ 3 mm). Subjects with just one positive skin prick test were considered monosensitized; on the other hand, subjects with two or more positive skin prick tests were considered polysensitized. Statistical analysis was performed by the chi-square test, confidence limits of the relative risk being calculated at 95%.

The distribution of lifetime asthma and allergic rhinitis rates, and their association in 1981 and 1995, respectively, are shown in Table 1. The prevalence of asthma was significantly decreased in the second observation ($P < 0.05$), while the prevalence of its association with rhinitis was increased ($P < 0.05$).

Polysensitized subjects significantly increased in 1995 in comparison with 1981 ($P < 0.05$).

Mite allergen appeared to be the most important causal allergen: mite cutaneous positivity increased ($P < 0.05$); there was an increase in polysensitized subjects ($P < 0.001$).

The pollen sensitization decreased in total ($P = 0.001$) and for monosensitized ($P < 0.009$) subjects.

Pet sensitization showed an increasing trend ($P < 0.05$); only one patient was monosensitized.

The most important finding of this study was the increasing trend of the association of asthma with allergic rhinitis over the period studied. This finding confirms the trend of a growing worsening of allergic diseases, and it is similar to that of our previous survey on conscripts (3).

Other findings of the present study show that indoor allergens are predominant in the present context and there is an increasing trend of polysensitizations. These findings are also consistent with the conscript observations (3).

In other words, it would seem that the epidemiologic role of allergic diseases is growing both as prevalence and spectrum of clinical features and

sensitizations. A possible explanation of these findings is the increasing pathogenetic role played by environmental factors (1). In fact, it is well known that allergic diseases are genetically influenced (1), but they appear clinically when aggressive factors break the immunologic equilibrium, allowing sensitization to occur (2). Among these factors, air pollution, viral infections, and changes in the indoor microenvironment (such as heating methods, thermal proofing devices, the introduction of pets, etc.) may play a critical role. In fact, this study suggests that indoor allergens are the major cause of allergic respiratory diseases, probably because they are perennial. In conclusion, it clearly appears that asthma and allergic rhinitis are two pathologic conditions causing increasing economic costs in terms of health care.

Key words: allergic rhinitis; asthma; epidemiology; polysensitization.

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Autoimmune vasculitis and aortic stenosis in aspirin-induced asthma (AIA)

ASPIRIN-induced asthma (AIA), a distinct clinical syndrome, affects about 10% of adult asthmatics. Attacks of asthma precipitated by aspirin are related to the inhibition of cyclooxygenase in the lungs of sensitive patients, and not

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