

Correspondence

Prevalence of allergic diseases in patients with schistosomiasis mansoni

To the Editor:

More than 200 million persons living in tropical regions are parasitized with helminths, and in Brazil schistosomiasis mansoni accounts for about 8 million. Contradictory results on the prevalence of allergic diseases in parasitized populations have been reported in several studies,^{1,2} most of them with a low prevalence, contrasting with an intense IgE immune participation against the parasites, particularly in patients with chronic schistosomiasis mansoni (CSM).

We studied 150 patients with CSM (average age, 43 years; 54% female) living most of the time in endemic rural areas. These individuals were evaluated by direct questionnaire to establish their clinical history of allergy, taking into account their generally low level of education. Physical examination was performed when the personal history of the patients suggested the diagnosis of allergic rhinitis, asthma, or urticaria/angioedema. These allergic patients were asked about the influence of allergens on symptoms according to the conventional procedure. Seventy-five individuals from non-endemic areas (average age, 25 years; 40% female) comprised a control group and answered the same questions related to allergic diseases and the possible influence of allergens. All interviews were carried out by physicians trained by allergists. Those with more than one allergic disease were scored for each different group. Allergy tests were not done. Chi square test was performed to compare the control group with rhinitis, urticaria/angioedema, and asthma groups.

The prevalence of allergic diseases in patients with CSM did not differ from that of the control group (Table I). Although patients with CSM showed a lower prevalence of asthma, this was not statistically significant. The prevalence of asthma in the control group was similar to that of the Brazilian population, which is estimated to be about 10%. The prevalence was similar in patients living most of the time in rural areas, compared with those living in urban regions. Also, the clinical manifestations of allergic diseases presented by patients with CSM were related to common allergens such as inhalants, foods, and drugs, apparently without any clinical evidence of relationship to the *Schistosoma* infection. These observations are in agreement with recent studies on eosinophils bearing IgE receptors,³ which participate in the immune defense against schistosomes, in addition to their role in allergic manifestations. These findings could explain a normal prevalence of allergic diseases coexisting with an active IgE immune response against *Schistosoma* antigens.

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TABLE I. Prevalence of allergic diseases in patients with CSM and control subjects

Group	Allergic diseases		
	Rhinitis	Asthma	Urticaria/ angioedema
Schistosomiasis (<i>n</i> = 150)	12 (8%)	10 (6.7%)	13 (8.7%)
Control (<i>n</i> = 75)	8 (10.7%)	9 (12%)	6 (8%)
Chi square	0.44*	1.84*	0.03*

*Not significant.

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Allergy syndrome after ingestion of salami

To the Editor:

Gennaro Liccardi et al. (*J Allergy Clin Immunol* 1996;98:850-2) present an interesting case report regarding oral allergy syndrome after ingestion of salami in a subject with monosensitization to mite allergens.

In 1994 we reported three cases of occupational allergy caused by manipulation of "chorizo" (typical Spanish salami), cheese, and salty ham.¹ An IgE-mediated sensitization was demonstrated by skin tests, RAST, and positive challenge test responses to *Acarus siro* and *Blomia kulagini* in the cheese rind, *Euroglyphus maynei* in the salami, and *Tyrophagus putrescentiae* in the salty ham. A microscopic study of samples of recently brushed salami rind showed the presence of different fungal colonies and mite groups. Sensitization to other allergens and problems derived from additives used in the manufacture of these foods were excluded. We have also published studies of patients in which the manipulation of contaminated food sources with mites (wheat flour, garlic dust) could be a cause of allergic disease.^{2,3} In the study by Liccardi et al., prick-prick testing was done with the same fresh food eaten by the patient. She previously demonstrated a sensitization to *Dermatophagoides* species. Was a mite contamination of salami ruled out?

Because of the extensive consumption of salami and its possible implication in allergic reactions, we think it would be interesting to rule out eventual contaminant allergens. In our opinion it is interesting to point out that the microscopic study

of the rind of food makes the diagnosis easier, without presenting important difficulties.

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Reply

To the Editor:

We read with interest the comments by Armentia and Vega about our article published in the October 1996 issue of *The Journal*.

In principle, we cannot rule out the possibility of a mite contamination of salami eaten by our patient, because different mites (such as those suggested by Armentia and Vega) are commonly found in sausages and other foods. However, various considerations can be produced to demonstrate that, in our case report, the role of mite allergens in inducing oral symptoms is unlikely compared with that of pork allergens.

First of all, ingestion of salami produced by the food industry, which contains a smaller quantity of pork but the same potential amount of mite allergens, did not induce significant oral and/or systemic symptoms in our patient.

In addition, the ingestion of other potentially infested foods, such as cheeses or sausages made with different meats, did not induce the appearance of any symptom.

Moreover, our patient's habit of storing salami in the refrigerator at a temperature of about 3° to 4° C for some time surely inhibits the growth of mites.

Epidemiologic considerations could be also adduced to demonstrate the unusual occurrence of allergic symptoms after ingestion of potentially mite-infested foods in the large population of mite-sensitized individuals.

On the basis of these data, we suggest that a large amount of mite allergens in foods is necessary to induce appreciable oral and/or systemic symptoms after ingestion. This point of view is also confirmed by recent observations.^{1,2}

However, we agree with Armentia and Vega's suggestion of performing a microscopic study of the rind of potentially moldy and/or mite-infested foods before they are eaten by patients at high risk for mite sensitivity.

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Atopy and NSAID sensitivity

To the Editor:

Quiralte et al. recently reported (*J Allergy Clin Immunol* 1996;98:678-85) the existence of an increased prevalence of atopy in patients intolerant to nonsteroidal antiinflammatory drugs (NSAIDs), particularly those with periorbital angioedema as their main clinical manifestation. We agree with these authors on the fact that the published data on the incidence of atopic disease in patients with NSAID sensitivity are conflicting, and we would like to contribute our point of view on this issue.

A few years ago, while studying additive and aspirin sensitivity in patients with chronic urticaria and angioedema by means of oral challenges, we became intrigued by the presence of a statistically significant increase in NSAID sensitivity in atopic subjects when they were compared with nonatopic individuals.¹

More recently, we have reported our observations on 30 atopic patients who experienced systemic anaphylaxis after the ingestion of foods containing high amounts of mites. Interestingly, 66.6% of them had a personal history of NSAID intolerance, manifested as urticaria and/or angioedema, and positive prick test responses to inhalant allergens.² Coincidentally, the same group of investigators from the Canary Islands found an 87% prevalence of NSAID intolerance in patients with severe reactions to ingested mites.³ In our experience, a history of NSAID sensitivity is present in 23% of adults with atopic disease (asthma, rhinosinusitis, or eczema plus positive prick test responses to inhalant allergens), whereas within several family members who have eaten the same mite-contaminated foods, aspirin-tolerant individuals never had severe reactions but only mild to moderate gastrointestinal symptoms (abdominal pain, vomiting). We have proposed a new aspirin sensitivity triad consisting of sensitivity to acetylsalicylic acid, atopic disease, and severe allergic reactions to ingested aeroallergens.⁴

Because no pathogenetic relationship has been established between NSAID sensitivity and allergic IgE-mediated reactions, the association between atopy and NSAID sensitivity remains unexplained. Do these patients have an increased predisposition for urticaria, angioedema, and anaphylaxis after exposure to multiple triggering factors of a diverse nature? Are there cross-reactions between mite components and NSAIDs? Do mites contain some chemical constituent able to inhibit prostaglandin synthase? All these questions should be addressed in the future.

In the meantime, Quiralte et al. have proposed that the presence of an atopic disease might predispose patients to certain clinical manifestations of NSAID intolerance, and Hoigne and Szczeklik⁵ have proposed that severe reactions to aspirin occur more often in atopic subjects than in nonatopic subjects. Then it is also possible that aspirin sensitivity may predispose the atopic patient to the development of severe allergic reactions to the ingestion of mite allergens. The exacerbation of chronic urticaria or angioedema by NSAID ingestion in up to one third of the patients is a widely accepted observation. NSAID intolerance could also be postulated as a marker for increased severity of allergic disease in atopic patients when they are exposed through particular routes or to very high allergen doses.