

Prevalence of occupational asthma and immunologic sensitization to guar gum among employees at a carpet-manufacturing plant

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Guar gum is a high-molecular-weight agent that can cause occupational rhinitis and asthma. We surveyed the employees at a carpet-manufacturing plant in which guar gum is used to adhere the dye to the fiber; 162/177 of the employees (92%) participated in the first part of the survey that included a questionnaire and skin prick tests with common allergens and guar gum (1 mg/ml). IgE and IgG antibodies to guar gum were also measured in those subjects (133/162 or 82%) who agreed to blood tests. Thirty-seven subjects (23%) had a history suggestive of occupational asthma and 59 (36%), of occupational rhinitis. Eight subjects (5%) demonstrated immediate skin reactivity to guar gum. Eleven subjects (8.3%) had serum IgE antibodies to guar gum. All subjects, except one subject who had a history suggestive of occupational asthma (n = 37) or positive skin tests (n = 4), participated in the second part of the study. A methacholine-inhalation test was performed during a workshift or in the 3 to 4 hours after the workshift. Five subjects had a concentration of methacholine causing a 20% fall in FEV₁ of <16 mg/ml (significant bronchial hyperresponsiveness) and positive skin reactions to guar gum. Four of these subjects underwent specific inhalation challenges. The remaining subject had a history of severe bronchospastic reaction on exposure to guar gum, and his FEV₁ of 1.6 L made specific challenges impossible. Two subjects had typical isolated immediate reactions. We conclude that the prevalence of IgE sensitization to guar gum is between 5% (8/162) (as assessed by skin tests) and 8.3% (11/133) (as assessed by measurement of serum IgE antibodies). Occupational asthma was confirmed in two subjects and highly probable in a third subject (prevalence of occupational asthma, 2%). (J ALLERGY CLIN IMMUNOL 1990;86:562-9.)

Vegetable gums are high-molecular-weight agents that can cause occupational asthma.^{1,2} Guar gum is derived from a vegetable, *Cyamopsis tetragonolobus*, that grows in India. It is incorporated into numerous foods and pharmaceutical products and is used as a fixing agent for colors. It is also used in carpet manufacturing to adhere the dye to fiber. Guar gum has recently been linked to occupational rhinitis.³

In a recent article, we described three cases of oc-

Abbreviations used

PBS: Phosphate-buffered saline
PC₂₀: Concentration of methacholine causing a 20% fall in FEV₁

cupational asthma caused by guar gum and demonstrated that it is mediated through an IgE mechanism.⁴ Two of the three subjects worked in carpet-manufacturing plants. The aim of this study was to assess the prevalence of immunologic sensitization and occupational asthma in a carpet-manufacturing plant in which one of the three cases originated.

MATERIAL AND METHODS

Carpet manufacturing

The carpets in the plant are produced in the following way: Knitted socks are first made from spools of nylon. The socks are then colored with several dyes mixed with guar

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gum in large reservoirs. An employee working in a mezzanine pours a bucket of guar gum into the reservoir. Because guar gum is easily aerosolized, some of the guar gum is dispersed into the air in the process. The socks are later unraveled, and the threads are dried and wound onto spools. Guar gum can be dispersed into the air during this process as well. The threads are then tufted and shaved. The resulting carpet is washed, dried, and pasted onto canvas. After being inspected, it is rolled and kept in shipping or storage.

We believed that all the employees should be investigated because of a suspicion that the ventilation system was carrying guar gum into every department, including the offices. Furthermore, with the exception of the office workers, all the employees had, at one time or another, performed various duties. The company acknowledged that guar gum has always been used in the manufacturing process.

Environmental study

An environmental study was done at 11 different work stations. Several samples of dust were gathered with different brushes to avoid contamination. The dust was found on the machinery and on shelves above floor level. No assessment was made of the offices. The dust was put into plastic bags and sent for circularly polarized light analysis. This method can detect the presence or absence of guar gum but cannot measure the level of airborne guar gum.

Survey

The physicians in charge of the survey first met with the local Health and Safety Committee. The protocol was accepted by a local ethics committee, and all employees signed a consent form for each of the two parts of the survey.

Initial assessment. In the first part of the survey, a questionnaire and skin tests with various inhalants, as well as guar gum, were administered by a trained technician and a nurse. A blood sample was also requested.

The questionnaire had been used in previous surveys of occupational asthma^{5,6} and was derived from a questionnaire on asthma and occupational asthma developed by Walter W. Holland (personal communication). Asthma was suspected when there was at least one respiratory symptom (wheezing, shortness of breath, tightness in the chest, or cough) in normal life and/or at least two respiratory symptoms after exercise and/or exposure to irritants. Occupational asthma was suspected when there were at least two reports of the following: cough, wheezing, chest tightness, difficult breathing, or awakenings at night because of chest symptoms on working days or after handling guar gum directly. Nonoccupational rhinoconjunctivitis was suspected when there were two reports of daily blocked nose, sneezing, or runny nose. Occupational rhinoconjunctivitis was suspected when there was runny or blocked nose, sneezing, or running or itchy eyes on working days or after handling guar gum. Information was also obtained on length of time on the job and smoking habits.

Skin prick tests were performed with 11 common inhalants (tree, grass, and ragweed pollens, *Alternaria*, *Aspergillus*, *Cladosporium*, and feathers from Omega, Montreal, Canada; cat and dog furs, house dust, and *Dermatophaga*

goides farinae from Hollister-Stier Laboratories, Mississauga, Ontario), as well as with a diluent as a negative control and histamine phosphate (1 mg/ml) as a positive control. Skin tests were also performed with a guar-gum extract. To prepare the antigen solution, 1 mg of guar gum from a commercial source used by the workers was added to 1 ml of PBS and mixed. The Lowry method with human serum albumin, 1 mg/ml as a standard, was used to determine the protein concentration of the guar-gum solution. The protein concentration of the 1 mg/ml PBS of guar gum was 0.12 mg/ml. Reactions were considered to be positive when the wheal was ≥ 3 mm as compared with the negative control reaction wheal to the diluent and the positive reaction wheal to histamine phosphate 10 minutes after the introduction of the allergen. Three subjects refused skin testing, and one subject had dermatographism. Atopy was defined as at least one immediate skin reaction in the presence of a reaction to histamine phosphate and the absence of reaction to the diluent.

Blood was drawn and the serum kept at -20°C for measurement of IgE and IgG antibodies to guar gum.

Specific IgE assessment. The IgE antibodies were measured with a guar-gum RAST as described elsewhere⁴ and expressed as a percent of bound IgE. The sera of six subjects with high levels of serum IgE antibodies to ragweed served as control sera. The mean $\pm$ SD specific IgE value was $2.15\% \pm 1.7\%$ bound. Any value $>3.85\%$ bound (upper limit of 1 SD of the mean) was considered a positive test. RAST-inhibition studies were performed by adding 50 μl of the subject's serum to 50 μl of guar gum (1 mg and 5 mg of guar gum per milliliter of buffer saline) or ragweed-pollen extract. Serum, incubated in PBS, served as the noninhibited reference control (buffer control).⁷

Specific IgG assessment. Serum IgG antibodies to guar gum were determined by a micro-ELISA method.⁴ All results are the mean of two determinations. Each IgG ELISA assay included the sera of serum nonexposed normal control subjects that served as reference sera. These data are expressed as IgG ratio, consisting of test result/mean result for the seven control sera. Test sera came from 133 workers exposed to guar gum and 49 subjects with ragweed hay fever who had had no known occupational exposure. Values of IgG ratios >2 were considered positive.

For blocking of the antibody, test serum was diluted with an equal volume of the guar gum solution or buffer. The mixture was incubated overnight at 4°C , prepared in a final serum dilution of 1:100, and tested. The percent blocking was determined by the following calculation with ELISA readings: $[(\text{TS}-\text{NCS})-(\text{BS}-\text{NCS})]/(\text{TS}-\text{NCS})$, where TS is the test serum, NCS is mean of the seven normal control sera, and BS is blocked serum (i.e., serum to which guar gum had been added).

For serial dilutions of serum, test serum was diluted 1:2, 1:4, and 1:8 in the serum of a subject with no known exposure to guar gum. The dilutions were then tested. Tests on 38 sera were performed on 2 separate days to determine reproducibility. By analysis of variance, the calculated intraclass correlation coefficient was 0.91.

Subjects with a history suggestive of occupational asthma (at least two symptoms suggestive of occupational asthma

TABLE I. Summary of results for the first step of the study (questionnaire and immunologic sensitization)

No. of workers	177
No. participating	162 (91.5%)
Sex M/F	156/6
Age (mean $\pm$ SD yr)	35.2 $\pm$ 9.2
Length of time at job (mean $\pm$ SD yr)	9.2 $\pm$ 6.1
Job location	
Office	21
Departments with detectable guar gum	132
Departments without detectable guar gum	6
Smoking habits	
Smokers	74
Exsmokers	40
Nonsmokers	48
Chest symptoms	
Wheezing and/or chest tightness and/or dyspnea and/or cough (one, two, three or all)	35/29/18/3
Provoked chest symptoms	
Wheezing and/or chest tightness and/or difficulty in breathing on exercise (one, two, three or all)	24/19/14/8
Wheezing and/or chest tightness and/or cough and/or difficulty in breathing on exposure to irritants (one, two, three or all)	35/13/9/5
Symptoms at work	
Wheezing and/or chest tightness and/or cough and/or difficulty in breathing and/or awakenings at night because of symptoms	17/14/4/9/4
Runny or blocked nose and/or sneezing and/or runny or itchy eyes.	32/18/19
Redness or itching of the skin	17
Skin testing	
Atopy (dermographism)	39 (1)
Reactivity to guar gum	8
Specific antibodies to guar gum	
No. participating	133
No. with IgE $\geq$ 3.85% bound	11
No. with IgG ratio $\geq$ twofold the mean of the reference sera	19

or one symptom suggestive of occupational asthma coupled with two symptoms suggestive of occupational rhinoconjunctivitis) and/or immediate skin reactivity to guar gum were asked to participate in the second part of the study.

Second assessment. Spirometry and assessment of bronchial responsiveness to methacholine were performed during a regular working day at the time of the workshift or in the following 3 to 4 hours. Spirometry was obtained on a Vitalograph apparatus, model R (Vitalograph Ltd., Buckingham, England) according to the American Thoracic Society standards.⁸ Reference values for FEV₁ and FEV₁/FVC were derived from Knudson et al.⁹ Bronchial responsiveness to methacholine was assessed with a Wright nebulizer (output, 0.14 ml/min) at tidal volume breathing for 2 minutes, according to a standardized methodology,¹⁰ with methacholine instead of histamine. Dose-response curves were drawn on a semilogarithmic noncumulative scale, and the PC₂₀ was

interpolated on the individual dose-response curve. Any PC₂₀ methacholine value $\leq$ 16 mg/ml was considered to reflect significant bronchial hyperresponsiveness.¹¹

Specific inhalation challenges. Specific inhalation tests were performed on subjects with a history suggestive of occupational asthma, a positive skin reaction to guar gum, and a PC₂₀ $<$ 16 mg/ml. Tests were done with a new aerosolization device that produces steady concentration of dust during exposure.¹² FEV₁ was recorded before exposure, every 10 minutes for 1 hour after the challenge, every 30 minutes for 1 hour, and hourly for up to 7 or 8 hours. PC₂₀ was obtained at the end of the day. On the second day, guar-gum dust was used instead of lactose for progressive intervals of 5, 15, 30, or 45 seconds, 1 minute, 2 minutes, 15 minutes, and up to a maximum of 1 hour with the apparatus, as well as for 1 hour by asking the subject to tip guar gum from one tray to another as suggested by other

TABLE II. Summary of results for the second step of the study (spirometry and bronchial responsiveness)

Characteristics	
No. of workers	41
No. participating	40
Sex (M/F)	38/2
Age (mean $\pm$ SD yr)	34.7 $\pm$ 9.2
Length of time at job (mean $\pm$ SD yr)	10.3 $\pm$ 6.1
FEV ₁	
% Pred (mean $\pm$ SD)	100.2 $\pm$ 14.7
No. <80% pred	3
FEV ₁ /FVC	
% Pred (mean $\pm$ SD)	98.7 $\pm$ 7.5
No. <85% pred	4
PC ₂₀ (mg/ml)	
No. <2 mg/ml	2
No. 2-16 mg/ml	9
No. >16 mg/ml	29

investigators.¹³ The reason for having a shorter period of exposure with the latter approach is that this method results in much higher concentrations of particles.¹² If FEV₁ at the end of the day was within $\pm$ 10% of the baseline methacholine assessment on the control day of exposure to lactose, PC₂₀ was obtained. In the case of negative challenges, PC₂₀ values had to remain unchanged, i.e., <3.2-fold difference in PC₂₀¹⁴ at the end of the active day of exposure to guar gum.

Analysis of results

Logistic regression was applied to subjects included in the first part of the study with the results of specific IgE to guar gum (>3.85 or ≤ 3.85) as the independent variables, and the following dependent variables: age, length of time on the job, the presence or absence of atopy, the presence or absence of skin reactivity to guar gum, smoking habits, FEV₁ (percent predicted), FEV₁/FVC (percent predicted), and bronchial responsiveness (PC₂₀ $\leq$ 16 mg/ml or >16 mg/ml). This test was performed with the SYSTAT 4.0 statistical package (SYSTAT, Inc., Evanston, Ill.).

RESULTS

Environmental study

With polarized microscopy, it was demonstrated that there was guar gum in most departments except shipping and storage.

First step of the study

The subject in whom occupational asthma had first been confirmed⁴ is included in the following results.

The summary of results for the first step of the study is presented in Table I. The participation rate was 91.5%. Most subjects worked in departments in which guar gum could be detected. Most employees

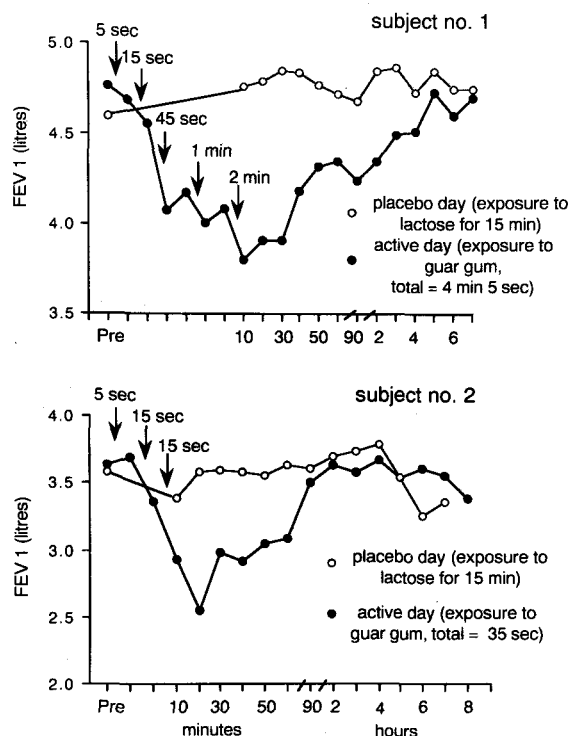


FIG. 1. Specific inhalation challenges for subjects 1 and 2 (see Table III) who experienced isolated immediate bronchospastic reactions. The duration of exposure with a recently developed aerosolization device¹² is presented. Progressive bronchoconstriction was demonstrated by increasing the duration of exposure. Each tick between the preexposure value of FEV₁ (*pre*) and the 10-minute mark represents the 10-minute period needed for assessment of spirometry between each exposure.

(N = 99) had worked at the plant for more than 10 years. Atopy was present in 39/162 (24%) of subjects. Eight subjects (162 or 5%) had skin reactivity to guar gum. Positive tests for serum IgE antibodies to guar gum were found in 11/133 subjects (8.3%), and 19/133 subjects (14.3%) had positive tests for IgG antibodies. Only two subjects had both IgE and IgG antibodies.

Inhibition of the IgE binding in the RAST was assessed in six workers with significantly increased specific IgE levels. Three subjects' sera demonstrated inhibition, blocking $>50\%$ with a 1 mg/ml of guar-gum concentration (74%, 84%, and 89%, respectively). Inhibition of specific IgG binding was assayed in seven ragweed-sensitive subjects with positive guar-gum IgG ELISA; four subjects' sera demonstrated inhibition, that is, blocking $>50\%$ (61%, 62%, 63%, and 71%, respectively).

Second step of the study

All subjects, except one subject with a history suggestive of occupational asthma (N = 37) and the

TABLE III. Characteristics of subjects with immunologic sensitization to guar gum

No.	Sex	Age (yr)	Length of time at work (yr)	Atopy	Skin test guar gum (diameter)	Symptoms			
						A	RC	OA	ORC
Occupational asthma									
1	M	32	13	+	3	—	—	+	+
2	M	46	15	+	3	—	—	+	+
3	M	56	15	+	6.5	—	—	+	+
Skin reactivity, bronchial hyperresponsiveness, no occupational asthma									
4	M	21	0.5	+	3	—	—	—	+
5	M	31	14	+	3	+	—	—	+
Skin reactivity, no bronchial hyperresponsiveness									
6	M	29	12	—	4	+	—	+	+
7	M	22	2	+	3	—	+	—	—
8	M	34	10	+	3	—	—	—	—
Increased specific IgE, no skin reactivity									
9	M	32	12	+	—	—	+	—	—
10	M	30	12	+	—	+	+	+	+
11	M	50	15	—	—	—	+	—	+
12	M	39	14	+	—	—	—	—	—
13	M	31	13	+	—	—	—	—	—
14	M	20	1.5	+	—	—	+	—	—

ND, Not done; A, asthma; RC, rhinoconjunctivitis; OA, occupational asthma; ORC, occupational rhinoconjunctivitis; B₂, inhaled β_2 -adrenergic agent; T, theophylline; Be, inhaled beclomethasone; prn, only if needed (not regularly).

*Values >3.85% bound (for specific IgE) and >2 (for the IgG ratio) are positive.

†Specific challenges; the temporal type of reaction is presented; for subjects with negative reactions, the total exposure time is presented; for subject No. 3, challenges could not be performed because the FEV₁ was too low.

four subjects with immediate skin reactivity to guar gum but with a history that did not suggest occupational asthma, participated in the second part of the study (Table II). Baseline characteristics of these subjects (age and length of time on the job) were not significantly different from subjects participating in the first part of the study. Three subjects had significant reduction in FEV₁, and 11 subjects had significant bronchial hyperresponsiveness.

Specific inhalation challenges

Five subjects had skin reactivity to guar gum and bronchial hyperresponsiveness (Nos. 1 to 5 in Table III). All these subjects had specific inhalation challenges with the exception of No. 3 whose FEV₁ of only 1.6 L contraindicated specific inhalation challenges. This subject had a history strongly suggestive of occupational asthma to guar gum. After minute exposure to guar gum, he reported sneezing and shortness of breath. Two subjects had positive specific inhalation challenges (Nos. 1 and 2, Table III). Both subjects experienced isolated immediate reactions (Fig. 1). The remaining two subjects (Nos. 4 and 5,

Table III) did not demonstrate significant bronchial obstruction after exposure to guar gum for 2 hours. Of the three subjects with skin reactivity to guar gum but no significant bronchial hyperresponsiveness, two subjects asked that specific inhalation challenges be performed. Neither subject demonstrated significant bronchial obstruction. Of the six subjects with increased specific IgE levels but no immediate skin reactivity to guar gum (Nos. 9 to 14, Table III), only one subject (No. 10) had a history suggestive of occupational asthma. However, the PC₂₀ was >16 mg/ml. The positive predictive value of atopy in relation to the presence of occupational asthma was low, only three of 39 atopic subjects (7.7%) having occupational asthma. Seven subjects not listed in Table III had a history suggestive of occupational asthma but had negative skin testing and no IgE antibodies to guar gum. Since dyes were also handled in the plant, we monitored peak expiratory flow rates for 2 weeks in five of the seven individuals who accepted and who were at work. None of the subjects demonstrated changes suggestive of work-related asthma.

Logistic regression demonstrated that atopy only

Medication	FEV ₁ (% pred)	FEV ₁ /FVC (% pred)	PC ₂₀ (mg/ml)	Specific IgE* (% bound)	Specific IgG* (IgG ratio)	Specific challenge†
β ₂ prn	90	81	4.6	47.3	0.5	+ (immediate)
β ₂ prn	90	84	0.23	10.1	3.0	+ (immediate)
β ₂ , T, Be	63	83	1.2	22.3	1.1	ND
Nil	113	108	11.2	27.7	1.0	Negative (2 hr)
Nil	106	95	5.5	1.9	2.6	Negative (2 hr)
Nil	87	104	118	4.4	0.7	ND
Nil	86	91	50	1.9	0.5	Negative (2 hr)
Nil	104	102	>128	ND	ND	Negative (2 hr)
Nil	ND	ND	ND	6	0.8	ND
Nil	95	108	21	7.6	1.3	ND
Nil	ND	ND	ND	4.2	1.6	ND
Nil	ND	ND	ND	5.4	0.8	ND
Nil	ND	ND	ND	5.1	3.0	ND
Nil	ND	ND	ND	6.3	0.7	ND

was a predictor of increased specific IgE levels to guar gum ($t = 4.14$; $p < 0.001$; odd ratio, 3.3).

DISCUSSION

Some vegetable gums, including acacia used by printers and karaya and tragacanth used by hairdressers, are known to cause occupational asthma.^{1,2} Immunologic sensitization has been identified. Gums are derived from plants and contain carbohydrates that, when they react with water, produce mucilages. They originate either from the exudate (acacia or arabic gum, *Acacia senegal*, a vegetable grown in Africa; tragacanth, *Astragalus gummifer*, a vegetable grown in Asia; and karaya, *Sterculia Urens*, which grows in India) or from the seeds (carob, which grows in the Mediterranean region, and guar gum, *Cyamopsis tetragonolobus*, a vegetable grown in India).¹⁵ Vegetable gums are used in numerous pharmaceutical products, as well as in salad bars, soups, ice cream, preserved foods, cheese, baby food, emulsions, lotions, and creams.

Although occupational rhinitis caused by guar gum has been documented,³ asthmatic reactions to guar gum have not, to the best of our knowledge, until a recent study by our department.⁴ As two of the three subjects confirmed to have occupational asthma worked in carpet-manufacturing plants,⁴ we believed it would be interesting to document the frequency of

immunologic sensitization and occupational asthma caused by guar gum in this kind of enterprise. A confidential report of a study at a chemical plant that imports and processes guar gum has been made available by courtesy to the authors of this study. This study demonstrated that 32/70 (46%) subjects had respiratory symptoms, 13 (19%) of whom complained of symptoms compatible with occupational asthma. Eighteen subjects (26%) demonstrated evidence of immunologic sensitization with either immediate skin reactivity and/or elevated specific IgE levels to guar gum. The diagnosis of occupational asthma was highly probable in 10 subjects (14%) who had respiratory symptoms and the highest IgE levels. However, specific inhalation challenges were not used to confirm the diagnosis of occupational asthma.

Our three-step study design was justified for the following reasons: In the first step, we believed we had to establish the proportion of subjects with symptoms and skin reactivity to guar gum. Since occupational asthma and rhinitis caused by guar gum have been demonstrated by us⁴ and other investigators³ to be IgE dependent, we argued that subjects had to develop immunologic sensitization before getting the disease in the target organ, that is, occupational asthma. In the second step, we wanted to determine the proportion of subjects with respiratory symptoms who also had significant bronchial responsiveness,

that is, asthma or occupational asthma. We believed that subjects with both skin reactivity to guar gum and significant bronchial hyperresponsiveness were likely to have occupational asthma. Assessment of bronchial responsiveness was performed when guar gum was being processed. Three of the eight subjects with skin reactivity to guar gum, including one subject who had symptoms suggestive of occupational asthma, did not have bronchial hyperresponsiveness. Of the five subjects with skin reactivity and bronchial hyperresponsiveness, all but one subject underwent specific inhalation challenges in the last step of the study to confirm the diagnosis of occupational asthma. We believed that these challenges were essential. Cockcroft et al.^{16, 17} have demonstrated that subjects with skin reactivity to an inhalant and a clinical history of sensitization on the one hand, and significant bronchial responsiveness on the other hand, all demonstrated immediate bronchoconstriction on exposure to the products. However, these data had not been confirmed for occupational allergens. Two of the four subjects who underwent specific inhalation challenges to guar gum did not experience bronchoconstriction after 2 hours of exposure. They had not been removed from exposure to guar gum before the tests. It is therefore unlikely that they had lost their sensitivity to the product. The subjects were exposed for 1 hour with a recently described aerosolization device that produces steady and below threshold limit value-short-term exposure level concentrations of particles.¹² Subjects were also exposed in the previously proposed way¹³ that produces higher concentrations of particles. A false negative reaction therefore appears unlikely. It was impossible to perform specific inhalation challenges on one subject who was assumed to have occupational asthma. He had significant bronchial obstruction (FEV_1 , < 2L) and a history of severe bronchospastic reactions after handling guar gum, which precluded specific inhalation challenges. However, the fact that he had the strongest skin reactivity to guar gum of all the subjects, as well as high IgE levels, make the diagnosis very probable.

We believe that missing cases with the type of design we used is unlikely. Indeed, both immunologic sensitization and increased bronchial responsiveness are involved in the pathophysiology of the condition. Every subject, except one subject with skin reactivity to guar gum and significant bronchial responsiveness, underwent specific inhalation challenges. However, the fact that the study was cross-sectional might have resulted in underestimating the prevalence of the disease because workers might have left the enterprise before the investigation was initiated.¹⁸

As in previous studies,^{5, 6} we found that the questionnaire overestimated the proportion of individuals with possible occupational asthma. Indeed, although 37 workers had symptoms suggestive of occupational asthma, only 11 had bronchial hyperresponsiveness, including five subjects who also had skin reactivity to guar gum. Since dyes, which have been reported as causing occupational asthma^{1, 2} were also present in this workplace, the six individuals with bronchial hyperresponsiveness but without skin reactivity to guar gum were proposed a monitoring of peak expiratory flow rates every 2 hours during waking hours for 2 weeks at work. Five subjects agreed for the monitoring, and direct visualization of the recording was not suggestive of occupational asthma.

Six subjects had increased specific IgE levels without having skin reactivity to guar gum. This result is not considered to reflect a lack of specificity of the guar-gum RAST. Negative results with this test with sera from most ragweed-allergic subjects have previously been reported.⁴ Discrepancies between skin test results and in vitro assessments of specific IgE have been demonstrated with standard allergens. However, only one of the six subjects had a history suggestive of occupational asthma without having significantly increased bronchial responsiveness. There was no evidence of involvement of IgG antibodies to guar gum in the pathogenesis of the asthma. Nineteen subjects had positive tests for IgG antibody, but only one subject had occupational asthma. Two subjects also demonstrated positive IgE RAST results. Although increased specific IgG antibodies appear to discriminate between subjects with and without occupational asthma to isocyanates,¹⁹ this finding does not appear to be the case in laboratory-animal workers for whom the mechanism is also IgE dependent.²⁰

We included an environmental monitoring to determine if guar gum could be detected in every section of the plant. The survey excluded the office and could not quantify the concentration of guar gum. The presence of guar gum was demonstrated by polarized microscopy to be almost everywhere except in shipping and storage, which employed very few subjects. All the subjects, with two exceptions who had occupational asthma or skin reactivity to guar gum, worked in departments in which guar gum was detected; the two exceptions had previously worked in departments where guar gum was present.

The frequency of immunologic sensitization (between 5%, i.e., 8/162, and 8%, i.e., 11/133, as confirmed by skin testing or serum IgE antibody measurement) and of occupational asthma (three workers or 2%) found in this study justifies either a regular medical surveillance of these workers with a ques-

tionnaire or immunologic assessment. Because it is known that occupational asthma can lead to permanent functional sequelae,^{1, 2} screening for occupational asthma in carpet manufacturing and pharmaceutical plants in which guar gum is used is recommended. We do not know if guar gum is an occupational hazard in the food industry in which it is also widely used. Based on the results of this study, we believe that skin testing with guar gum is the easiest way to screen on a regular basis. Subjects found to have skin reactivity could then be referred for additional tests.

One interesting finding of this study is that the three subjects with occupational asthma were atopic. However, the predictive value for the presence of occupational asthma in an atopic subject was only 7.7% (3/39). This does not justify routine screening to exclude atopic subjects from this type of work. A similar conclusion was reached by Slovak and Hill²¹ vis-à-vis laboratory-animal workers.

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