American Journal of Industrial Medicine 12:205–218 (1987)

Occupational Asthma From Inhaled Egg Protein

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We evaluated workers and performed an industrial hygiene assessment at a plant where raw eggs are processed into powdered egg yolk and whole egg. Egg dust levels in the packaging room straddled the American Conference of Governmental Industrial Hygienists' (ACGIH) exposure guideline of 10 mg/m^3 for nuisance dust. We obtained medical histories from 25 workers, and performed physical examinations, spirometry, and serial determinations of peak expiratory flow rate (PEFR) by portable meter every 3 hrs (while awake) for 7 days. We defined symptomatic bronchial lability to be a decrement in PEFR on any one day of 20% or more of the day's maximum, with concurrent symptoms. Skin-prick tests and serum assays for specific IgE by the radioallergosorbent (RAST) method were performed to assess sensitivity to commercial egg proteins, egg protein fractions, and freshly prepared extracts of whole egg powder and yolk. We classified participants as definite cases of asthma if both the examining physician diagnosed asthma and symptomatic bronchial lability was demonstrated by serial PEFR determinations. Definite noncases of asthma were those participants in whom the physician did not diagnose asthma and in whom symptomatic bronchial lability was not demonstrated by PEFR. All five definite cases, compared to three of 16 definite noncases of asthma, had one or more positive skin-prick tests to egg proteins. Four of five cases, compared to 0 of 14 noncases, who had serum determinations, had an elevated RAST to one or more of the egg proteins. This study demonstrates that occupational asthma associated with IgE-mediated allergy to egg proteins occurs among workers exposed to inhaled egg proteins.

Key words: allergy, conalbumin, ovalbumin, ovomucoid, lysozyme

INTRODUCTION

Chicken egg white is a common food allergen, ingestion of which may cause immediate symptoms of urticaria, angioedema, bronchial asthma, or systemic anaphylaxis in sensitized individuals [Langeland, 1983]. Inhaled egg protein has also been

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Accepted for publication March 13, 1987.

shown in one study to produce respiratory symptoms suggestive of asthma [Edwards et al, 1983]. However, in that study only one of eight workers so affected had pulmonary function changes diagnostic of reversible airways obstruction.

In 1984, employees of a factory where powdered eggs are produced asked us to investigate respiratory symptoms suggestive of asthma, which they believed were related to their work. We conducted a medical and industrial hygiene assessment to characterize the pathophysiology underlying the workers' symptoms and define its relation to workplace exposures [Smith et al, 1986]. We will describe in detail elsewhere the immunologic studies of five workers with occupational asthma and IgE-mediated allergy to egg protein (D.I. Bernstein et al, in preparation). In this report, we describe the industrial hygiene survey, case reports, and supporting medical study, from which we determined that at least 5%, and possibly 11%, of (then) currently employed workers had developed occupational asthma because of workplace exposure to egg proteins.

METHODS

Plant Description

Each working day the plant processes approximately 1.5 million eggs into powdered whole egg, powdered egg yolk, and liquid egg white. In the transfer room, eggs are loaded onto a conveyor and travel through one of six washing machines, after which they are sprayed with a sanitizer that contains 1.75% titratable iodine, and are then candled (inspected). The washed, sanitized, and candled eggs pass into the adjacent breaking room, where they fall onto one of six continuous chains that grip the eggs, break the shells, and drop the contents into a separating cup. Operators judge the thoroughness of each separation of yolk and white. If the separation is not good or the yolk is broken, an operator tips the cup, sending the whole egg into a separate collection system. Otherwise, separated egg yolk and white pass by and flow into their respective collection systems. Liquid egg white is pumped to refrigerated storage, to await loading into tank trucks for transport to another plant. Liquid whole egg and egg yolk are pasteurized and refrigerated before they are sent to the drying room.

Additives such as sugar, powdered milk, and corn syrup are mixed into the product stream. The liquid product is pumped through four high-pressure spray nozzles into a large drying oven. Water is evaporated, and the dried product falls to the oven floor, where it is mechanically scraped to one side, picked up by vacuum, and transported overhead to a cyclone separator. The product is removed by the cyclone and passed down to a sifter in the packaging room, where it is weighed, packed, and sealed prior to shipment.

Workers were exposed primarily to aerosolized liquid egg products in the transfer and breaking rooms, powdered whole egg and egg yolk dust in the drying area and in the sifting and packaging room, and iodide in the transfer room. (A chlorine sanitizer previously had been used. This had been thought to be the source of employees' respiratory complaints, and consequently was changed to the iodine-containing compound.)

Other than to maintain comfort, no ventilation was provided, except in the egg breaking and in the packaging rooms. There, filtered air was supplied to maintain positive pressure in accordance with U.S. Department of Agriculture guidelines.

Environmental Assessment

Two personal total dust, two area total dust, and two area respirable dust air samples were collected in the sifter and packaging room during the day shift on 2 days. The samples were collected on tared filters, and the quantity of dust present was determined gravimetrically [NIOSH, 1984a,b]. One bulk dust sample was analyzed for total protein [AOAC, 1984] and for an amino acid profile. Five high-volume samples were collected for analysis of amino acid profile [Spackman et al, 1958].

Chloride and iodide ions were measured on 14 day shift area samples in the transfer and the breaking rooms and on one personal sample collected from a machine operator in the breaking room. These were collected on midget impingers containing $NaHCO_3$. Separate aliquots were analyzed, using a Dionex Model 2010i ion chromatograph, with AS-4 and AS-5 anion separators, respectively, for the chloride and the iodide analyses.

Medical Survey

We developed a questionnaire that we believed would be sensitive enough to identify employees with occupational asthma. We suspected a respondent might have occupational asthma if he or she reported experiencing within the preceding month; wheezy or whistling respiration; episodes of shortness of breath or chest tightness; *and* the symptom(s) occurred following specific activities or exposures at work; *and*, on days away from work and on vacation, symptom(s) occurred less frequently or not at all. (The questionnaire is reproduced in its entirety in a technical report by London et al [1986] and is available from the first author (A.B.S.) or from the National Technical Information Service.) This questionnaire was administered to all current employees who agreed to be interviewed. To identify the underlying pathophysiology of positive responses, we invited to participate in a set of medical examinations all respondents who reported having had any episode of wheezing, shortness of breath, or chest tightness within the month preceding the questionnaire (regardless of apparent temporal relationship to work) and an equal number of asymptomatic workers, frequency matched to the symptomatic on age and sex. Six months elapsed between the initial screen and the follow-up, allowing time to contract for equipment and laboratory services and to receive administrative approvals. The follow-up medical examinations consisted of the following.

1. To diagnose asthma clinically, one of us (S.K., who is a physician trained in internal medicine and board certified in occupational medicine) examined each participant. He was blinded to the questionnaire responses and the results of all examinations. We specified no diagnostic criteria for asthma prior to his assessment. We asked him to render an opinion, based upon his clinical judgment and experience, whether the examinee had asthma and, if so, whether the asthma was occupational or nonoccupational.

2. To identify airways obstruction, we measured forced vital capacity (FVC) and 1-sec forced expiratory volume (FEV_1), using an Ohio Medical Model 822 dry rolling seal spirometer attached to a Spirotech 220 B dedicated computer. All spirometries were performed at least 2 hr after the beginning of the workshift, but were scheduled without specific attention to time of day or workplace exposures immediately preceding the measurements. We required as evidence of pulmonary obstruction an FEV_1 less than 80% of predicted, an FEV_1/FVC ratio less than 0.70, and FVC greater than 80% of predicted [Morris et al, 1973].

3. To identify airways obstruction variability over time, we obtained serial determinations of peak expiratory flow rate (PEFR), using Wright's portable mini-peak flow meters for 1 week every 3 hr while awake and during the night if the participant was awakened for any reason. Three exhalations were recorded each time, and the maximum of the three was accepted as the PEFR determination. Any wheezing, shortness of breath, chest tightness, or cough experienced concurrently with each PEFR determination was also recorded. We diagnosed a participant to have significant bronchial lability if the difference between the minimum and maximum PEFR on at least 1 day exceeded 20% of the day's maximum PEFR [Hetzel and Clark, 1980].

4. To identify allergy to workplace exposures, we administered skin-prick tests and measured serum-specific IgE by the radioallergosorbent (RAST) method to a panel of egg allergens. The egg allergen panel included commercial egg white, yolk, and whole egg reagents (prick tests: Hollister-Steir, Spokane, WA; RASTs: Pharmacia, Piscataway, NJ); extracts prepared from factory-powdered whole egg and powdered egg yolk; and the egg fractions conalbumin, ovalbumin, lysozyme, and ovomucoid (Sigma Co., St. Louis, MO). Results were expressed as counts per minute of ^{125}I-labeled anti-IgE bound to allergen-coated discs, and they were considered positive if the test sera binding was more than 4 SD above the mean of nonexposed laboratory controls. We also measured total serum IgE levels by radioimmunoassay (normal range, 10–125 IU/ml; IU = 2.3 ng).

5. To diagnose personal atopy, we administered skin-prick tests to a panel of common airborne allergens, including ragweed, mixed trees, mixed grasses, cat, and house dust mites (Hollister-Steir). Negative and positive control skin tests included phosphate-buffered saline and histamine, respectively. A prick test was considered positive if the wheal measured 3 mm greater than the saline control.

RESULTS

Environmental Assessment

Personal total dust exposures during the 2 days' sampling in the sifting and packaging room were 12.8 and 7.3 mg/m^3, which straddled the American Conference of Governmental Industrial Hygienists' (ACGIH) guideline of 10 mg/m^3. Area sampling results were 1.2 and 0.94 mg/m^3 for total dust and 0.07 and 0.03 mg/m^3 for respirable dust. The area total and respirable dust levels were well below the ACGIH guidelines of 10 and 5 mg/m^3, respectively [ACGIH, 1986].

The bulk dust sample was determined to be 50.5% protein. The amino acid analysis of three of five high-volume filter samples resembled both the profile of the bulk dust sample and the reference standards for egg yolk protein [USDA, 1976]. The dust content of the other two high-volume samples was insufficient for analysis.

Area and personal samples for chloride ranged from detected but not quantified to 18.8 μg/m^3; samples for iodide ranged from nondetected to 45.4 μg/m^3.

Medical Assessment

In the approximately 4 years of the plant's operation that preceded our initial investigation, 340 persons had been hired. One hundred seven were currently employed. Of the 233 employees who had terminated prior to our initial survey, 58 (24%) had left within 2 weeks of hire, and an additional 58 had left within 2 months.

Ninety-four of 107 current employees (88%) responded to the questionnaire. Twenty-three stated that within the preceding month, they had had at least one episode of wheezing, shortness of breath, or chest tightness. Seventy-one denied such symptoms. In the 6 months that elapsed between the questionnaire and the medical follow-up, five of 13 nonrespondents (38%), six of 23 symptomatic respondents (26%), and 13 of 71 asymptomatic respondents (18%) left employment. Thirty-one workers participated in the follow-up: 13 of the 17 remaining symptomatic respondents plus 18 volunteers from the asymptomatic group. Not every one of the 31 participants underwent the full set of examinations. Data analyses are limited to the 25 who were examined by the physician, gave us serial peak flow rate determinations, and underwent spirometry and skin-prick testing. Nineteen of these 25 also gave blood for determination of total and specific IgE levels.

Without knowledge of the results of any other procedure, the examining physician diagnosed possible asthma, either occupational or nonoccupational, in nine of 25 participants. Peak expiratory flow rate evidence of bronchial lability was found in five of these nine. All five reported wheezing or shortness of breath at the times their PEFR dropped by 20% or more of the day's maximum. One participant, diagnosed as normal by the physician, also had bronchial lability, but was asymptomatic throughout the week. The clinical histories of the five physician-diagnosed asthmatics with symptomatic bronchial lability are exemplified in the following case history. Details for the four others are given in Table I. Peak expiratory flow rates over 7 days are depicted in Figures 1 through 5 for all five cases.

Case History

Case No. 1 had worked at the plant approximately 1.5 years in the transfer room as an egg-loader, where he was exposed to broken raw eggs. He had no personal or family history of allergy or asthma. He had smoked approximately one-half pack of cigarettes per day for 7 years.

Approximately 4 months after beginning employment, he developed wheezing, shortness of breath, and chest tightness, which generally began within 1 hour of beginning his job, and lasted until 1.5 hours after leaving the plant. His symptoms occurred less frequently on days off work, and not at all on vacations. Within the preceding 6 months, he also had asthmatic symptoms after eating eggs. He used an over-the-counter preparation of epinephrine by inhalation one or two times per day during the workweek.

The examining physician noted mild expiratory wheezing and diagnosed occupational asthma. The FEV_1 was 77% of predicted, FVC 111% of predicted, and the FEV_1/FVC ratio was 0.57. Serum total IgE level was normal; skin-prick tests were positive to conalbumin and lysozyme, and RAST tests were positive to ovomucoid, ovalbumin, factory whole egg, and commercial (Pharmacia) egg whites.

The PEFR record (Fig. 1) showed immediate diminution of PEFR on exposure to egg products, with a progressive fall throughout the workday, and dramatic improvement to baseline soon after leaving work. There was no late bronchospastic reaction. During the time that PEFR fell toward its minimum, he reported symptoms of wheezing, shortness of breath, and chest tightness. The daily PEFR variation was from 6 to 12% on days off work and 24 to 49% during workdays.

TABLE I. Clinical Histories of Physician-Diagnosed Asthmatics With Symptomatic Bronchial Lability

Case No.	Length of employment (months)	Workplace exposures	Time to onset of symptoms (months)	Hx. of atopy	Smoking (pk-years)	% Predicted FEV_1	% Predicted FVC	FEV_1/FVC	Daily PEFR variability (%)	Total IgE	Positive skin tests	Positive RAST
1[a]	18	Broken raw eggs	4	No	3.5	77	111	0.57	6–49	Normal	Conalbumin Lysozyme	Whole egg (factory) Egg white (Pharmacia) Ovalbumin Ovomucoid
2	38	Broken raw eggs	34	Yes	0	94	116	0.64	10–31	Normal	Whole egg (factory) Lysozyme Ovalbumin Ovomucoid Ragweed	Whole egg (factory) Egg white (Pharmacia) Conalbumin Ovalbumin Ovomucoid
3	13	Broken raw eggs; dried powdered eggs	5	No	8	59	72	0.67	18–52	Elevated	Lysozyme Ovomucoid	Whole egg (factory) Egg white (Pharmacia) Conalbumin Lysozyme Ovalbumin Ovomucoid
4	26	Broken raw eggs; dried powdered eggs	2	No	0	89	99	0.76	11–25	Normal	Whole egg (H-S)[b] Egg yolk (H-S) Conalbumin Lysozyme Ovomucoid	Whole egg (factory) Egg white (Pharmacia) Conalbumin Lysozyme Ovalbumin Ovomucoid
5	37	Broken raw eggs	18	No	0	90	98	0.75	6–21	Normal	Conalbumin Ovalbumin	None

[a]Also described in the text.
[b]H-S, Hollister-Steir.

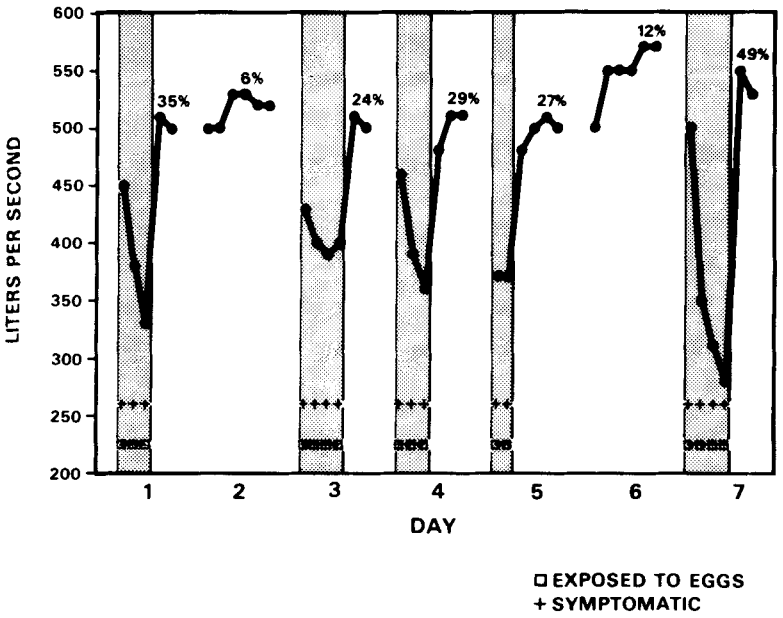

Fig. 1. Case 1.

Figs. 1–5. Peak expiratory flow rate (PEFR) over 7 days. The percentage of daily variation in PEFR is given at the peak of each day's PEFR. Exposure to egg products at work is indicated by vertical shaded bands.

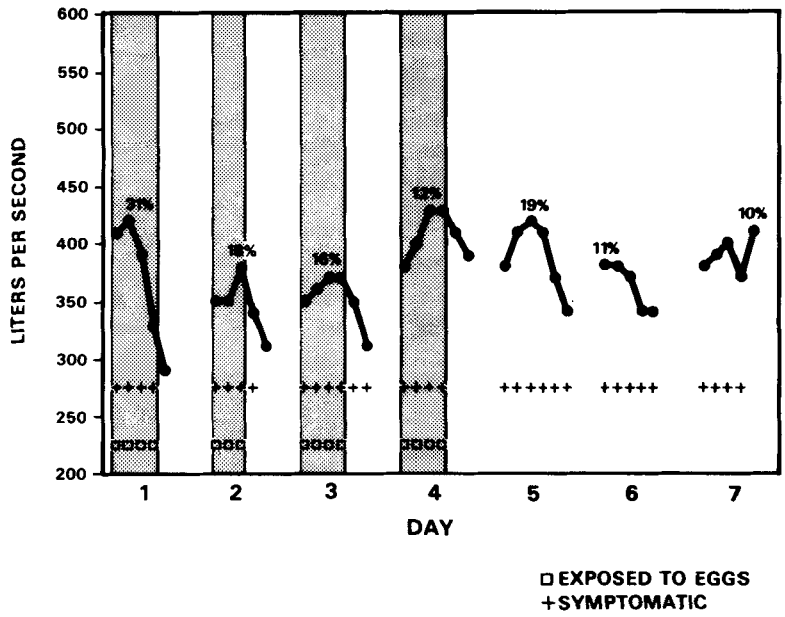

Fig 2. Case 2.

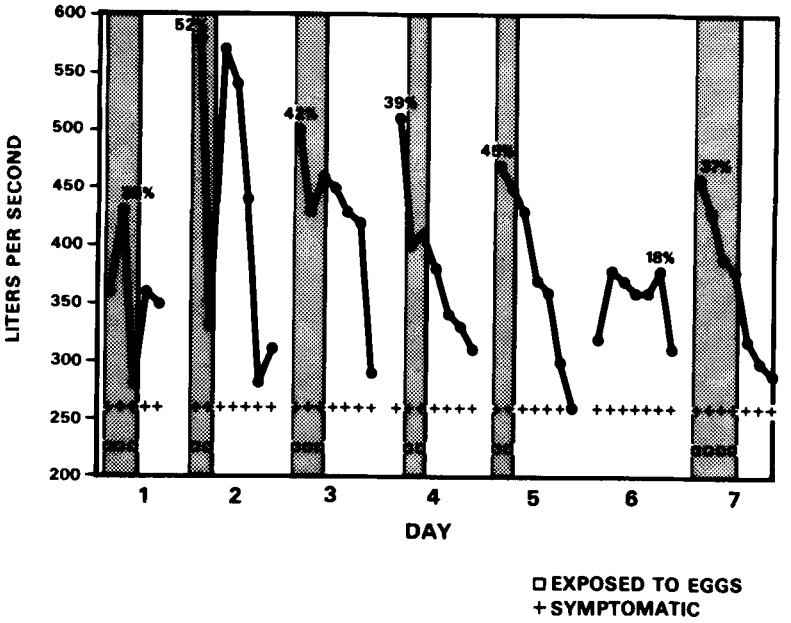

Fig 3. Case 3.

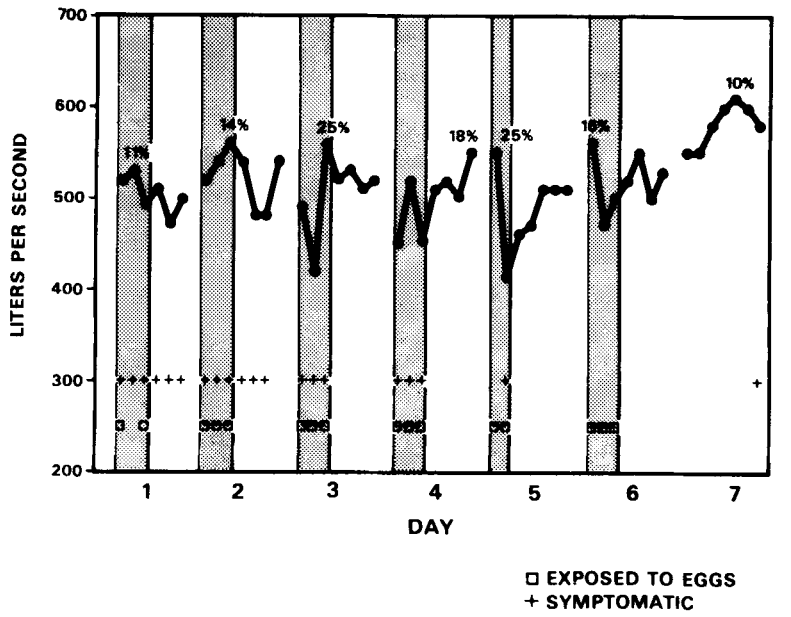

Fig 4. Case 4.

Medical Survey

Symptomatic bronchial lability was found in five of nine participants diagnosed by the physician to have asthma compared with none of 16 participants whom the physician did not diagnose to have asthma (Table II). There was good agreement between the physician and the PEFR determinations, as evidenced by a Kappa statistic of 0.615 [Fleiss, 1973].

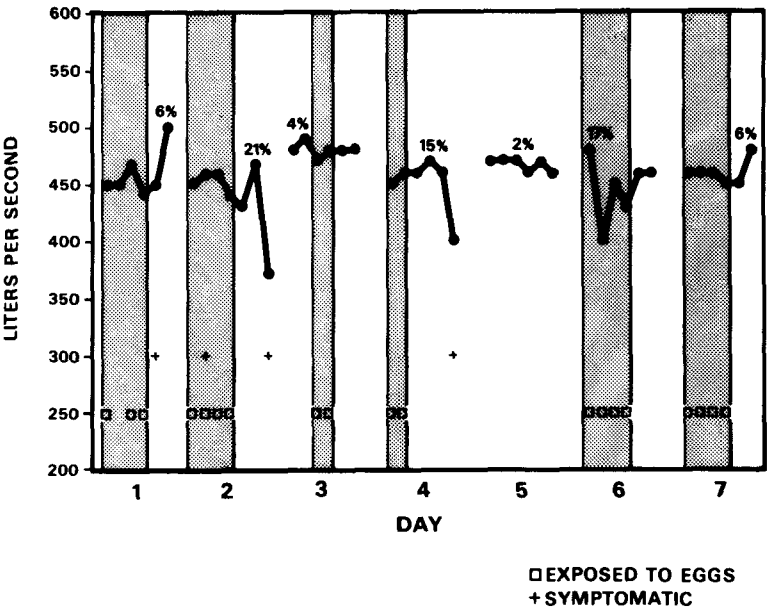

Fig 5. Case 5.

TABLE II. Agreement Between Symptomatic Bronchial Lability and the Physician's Diagnosis of Asthma

	Physician-diagnosed asthma		
	Yes	No	
Symptomatic bronchial lability			
Yes	5	0	5
No	4	16	20
Total	9	16	25

Kappa = 0.615; SE (Kappa) = 0.185; $p < 0.001$.

We were reasonably certain that the 21 individuals for whom there was diagnostic concordance by two independent, albeit imperfect, tests (physician diagnosis and PEFR determinations) were correctly classified as having or not having asthma [Marshall and Graham, 1984], regardless of whether it was etiologically related to the workplace. We therefore labeled these 21 participants, dually classified as asthmatic or nonasthmatic, as either "definite cases" or "noncases" of asthma. (The 16 noncases included five participants chosen for follow-up because they had reported respiratory symptoms, but who were neither diagnosed to have asthma by the physician nor demonstrated by serial PEFR determinations to have symptomatic bronchial lability. One was diagnosed to have coronary artery disease, one was diagnosed to have irritant respiratory symptoms, and three were diagnosed to be normal.)

Single spirometric measurements were not particularly useful in documenting airways changes compatible with asthma. Spirometric evidence of airways obstruction (reduced FEV_1 and FEV_1/FVC ratio with normal FVC) was found in only two of

five definite cases of asthma, both of them less than 5 pack-year smokers, and in one of 16 noncases, a 60 pack-year smoker with coronary artery disease.

Asthma was immunologically linked to the work environment. All five definite cases had at least one positive skin test to egg proteins compared with three of 16 noncases (Table III). Four of five cases had at least one positive RAST to egg protein compared with none of 14 noncases for whom RAST determinations were available (Table IV). Only one case (and zero noncases) had an elevated serum total IgE level.

One each of the five cases and 16 noncases had at least one positive skin-prick test to common airborne allergens. Although this might suggest that no association existed between personal atopy and the development of occupational asthma in the group of follow-up participants, because of biases inherent in cross-sectional studies, this conclusion is not warranted (see Discussion, below).

We concluded that the five definite cases indeed had IgE-mediated asthma that was immunologically related to the worksite. We reexamined the screening questionnaires to determine the concordance of questions that we believed a priori would identify occupational asthma, with this conclusion. All five cases had wheezing that followed specific activities or exposures at work, which on days away from work and on vacations occurred less frequently or not at all. In addition, four of five cases reported shortness of breath and chest tightness in the same temporal pattern. None of 16 noncases reported wheezing temporally related to work. One noncase reported shortness of breath only, and another reported chest tightness only, temporally related to work. Agreement between various possible combinations of questionnaire re-

TABLE III. Relation Between Asthma and Positive Skin-Prick Tests to Egg Protein

	Positive skin-prick test to egg protein		
	At least one	None	
Definite case of asthma			
Yes	5	0	5
No	3	13	16
Total	8	13	21

$p < 0.01$, Fisher's exact test, two-tailed.

TABLE IV. Relation Between Asthma and Positive RAST Tests to Egg Protein

	Positive RAST to egg protein		
	At least one	None	
Definite case of asthma			
Yes	4	1	5
No	0	14	14
Total	4	15	19

$p < 0.01$, Fisher exact test, two-tailed.

sponses and the case/noncase status obtained by dual physician/PEFR agreement from the medical follow-up is shown in Table V. Perfect agreement is noted for wheezing temporally related to work (ascertained from the questionnaire) and physician/PEFR diagnosed asthma (which was also immunologically associated with workplace exposures). Excellent agreement is also noted between wheezing, shortness of breath, *and* chest tightness, all three temporally related to work, and physician/PEFR-diagnosed asthma.

The question set that best identified physician/PEFR-diagnosed occupational asthma is noted a posteriori to be "wheezing temporally related to work." On reexamining the screening questionnaires of the 12 symptomatic workers who did not participate in the follow-up, or who participated but did not give us PEFR determinations, we found an additional five possible cases of occupational asthma. Three of these had quit employment prior to the follow-up. One had refused participation. One had participated and was diagnosed by the physician as having either early occupational asthmas or irritant respiratory symptoms, but had not given us PEFR determinations.

Overall, therefore, there were at this plant five definite cases of occupational asthma because of IgE-mediated allergy to egg protein plus an additional five possible cases among the nonparticipants in the medical follow-up, who were identified a posteriori from the questionnaire alone. The overall prevalence of occupational asthma among the original questionnaire respondents was at least 5% (5/94) and may have been as high as 11% (10/94).

DISCUSSION

Our results contrast with those of the only other report in the literature of asthma following exposure to inhaled egg protein [Edwards et al, 1983]. We identified five workers with occupational asthma associated with IgE-mediated allergy to egg protein, all of whom complained of wheezing temporally related to work. All five had bronchial lability demonstrated by serial PEFR determinations, and skin tests and

TABLE V. Agreement Between Possible Question Sets and Asthma (Dual MD/PEFR Agreement)

	Symptom(s)[a] present?	Asthma? (dual MD PEFR agreement) Yes	No	Kappa	p value
Wheezing	Yes	5	0	1.0	<0.001
	No	0	16		
Shortness of breath	Yes	4	1	0.738	<0.001
	No	1	15		
Chest tightness	Yes	4	1	0.738	<0.001
	No	1	15		
Wheezing, shortness of breath, and chest tightness	Yes	4	0	0.859	<0.001
	No	1	16		
Wheezing, shortness of breath, or chest tightness	Yes	5	2	0.769	<0.001
	No	0	14		

[a]All symptoms are temporally related to work.

RASTs clearly differentiated cases and noncases of asthma. In comparison, Edwards et al reported that eight of 13 bakery workers, who sprayed a 25% mixture of egg white and yolk in water on meat rolls, developed respiratory symptoms. Only four of eight complained of wheezing, and only one had pulmonary function changes suggestive of airways obstruction. Six of eight symptomatics were atopic. Skin tests and serologic reactivity did not differentiate the symptomatic and asymptomatic workers. Only one each of the symptomatic and asymptomatic had a positive skin test to egg protein, and both these workers had elevated total IgE.

Cross-sectional surveys suffer from a number of problems that must be recognized [Kelsey et al, 1986]. First, it may be difficult to identify cause and effect, since both exposure and outcome are ascertained simultaneously, and on occasion it may not be possible to determine which came first. In our study, the occupational asthmatics all reported their symptoms began after their hire date, so that exposure historically preceded outcome. Second, if characteristics of persons whose disease causes them to develop earlier or more severe disease are different from those of persons whose disease is milder or of longer duration, then the apparent association between exposure and disease observed in the cross-sectional study may not properly reflect the true association. For example, we observed no apparent association between atopy (as measured by positive skin-prick tests to common aeroallergens) and occupational asthma. If, however, atopics are more likely than nonatopics to develop earlier or more severe occupational asthma, and if earlier or more severe disease renders the individual more likely to leave work, then atopics preferentially will have left the study population, and nonatopics with no disease, later disease, or less severe disease will be overrepresented in the surviving population. To participate in our follow-up assessment, the employee must have been employed at the time of the initial questionnaire and continued employment during the 6 months that separated the initial screen and the follow-up. We observed that during the 6-month hiatus, 26% of symptomatic employees left compared with 18% of asymptomatic employees. We cannot rule out the possibility that the symptomatic individuals who left were atopic and had developed earlier or more severe disease than the nonatopics. While it is reasonable to use the data from the follow-up to examine associations between various methods for diagnosing occupational asthma, no conclusion reasonably can be drawn from our data as to the association of specific personal characteristic (such as atopy) and development of occupational asthma among workers exposed to egg proteins at this plant, based upon this study alone. Future research should be cognizant of this potential bias. To characterize the true risk of developing asthma as a consequence of egg protein exposures and relevant personal attributes (such as atopy), a prospective cohort study would be necessary at this or a sister plant.

Dust levels in the sifting and packaging room of this plant straddled the ACGIH guideline of 10 mg/m^3 for occupational exposures to nuisance dust. There is no occupational exposure standard specific for airborne levels of egg dust and no generic standard for airborne dust of organic orign. Consequently, for compliance purposes, the only workplace exposure standard applicable to dust levels measured at this plant is that for airborne nuisance dust, which by definition has little adverse effect on the lungs. As noted by the ACGIH, however, the nuisance dust guideline is not meant to "apply to those substances which may cause physiologic impairment at lower concentations, and for which threshold limits have not yet been recommended" [ACGIH, 1980]. Were we to argue by analogy, the ACGIH guideline for proteolytic enzymes

might illustrate the order of magnitude reduction in airborne egg dust necessary to protect the worker from respiratory sensitization. Respiratory impairment among enzyme dust-exposed workers is comparable in magnitude to that which we observed at the egg processing plant [Greenberg et al, 1970]. The ACGIH recommends a ceiling limit of 0.06 $\mu g/m^3$ for proteolytic enzymes of *Bacillus subtilis* [ACGIH, 1986]. It is likely that if conditions prevailing at this plant are typical of the industry as a whole, then significant reductions in aerosol and dust exposure levels may have to be achieved to prevent allergic sensitization of the unprotected worker.

We observed that three of the five definite asthmatics were on ineffective drug therapy for asthma. None of the five was taking sodium cromolyn, which specifically interdicts the IgE-mediated immune response. Although we do not recommend medication as the primary solution to the problem, if one is to be subjected to an agent that incites asthma on an ongoing basis, specific pharmacologic therapy would be beneficial while a permanent solution is sought, either through engineering or work practice controls.

Finally, egg-sensitive individuals should be wary of vaccines grown in eggs [Centers for Disease Control, 1985]. The vaccine most likely to be offered to an adult is influenza vaccine. Although healthy adult workers do not need routine influenza vaccination, the vaccine is recommended for those with pulmonary disease, including asthma, that is severe enough to require regular medical follow-up, which would include the five definite asthmatics at this plant.

ACKNOWLEDGMENTS

The authors thank David R. Moller, MD, for performing and interpreting the skin-prick tests on all study participants, and Gary Liss, MD, for assisting in the development of the questionnaire used in this study.

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