S-141

# Occupational Reactions in the Food Industry

Food industry employees are exposed to a wide variety of substances, some of which can induce allergic reactions. Coffee bean hypersensitivity is one example

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□ AT LEAST 21 million people in the United States work in some aspect of the food industry (USDC, 1988). Although 2.1 million individuals are employed in the farm sector, the majority (18.9 million) work in the nonfarm portion, including food processing, manufacturing, transport, trade, retail, and restaurants.

Employees in the food industry are exposed to a wide variety of substances, a number of which can induce allergic disease (O'Neil and Lehrer, 1992). Most food-derived or food-associated sensitizing materials, such as those present in green coffee beans, flour, and shellfish, are protein allergens. In addition, nonfood agents, such as honeybees, grain storage mites, antibiotics, thermophilic actinomycetes, and even rubber boots, have also been shown to induce occupational disease in food workers. Exposure to allergens can occur by inhalation and/or contact, depending on the agents and the specific food industry; thus, routes are the same as for other occupational allergens (Emmett, 1983).

Occupational diseases associated with workplace exposures are occupational asthma, hypersensitivity pneumonitis (also called extrinsic allergic alveolitis), and dermatitis. *Occupational asthma* is a reversible obstruction of the airways induced by inhaling agents encountered in the workplace. *Hypersensitivity pneumonitis* is a disease spectrum characterized by diffuse, predominantly mononuclear inflammation of the lung parenchyma, particularly of the interstitium and alveoli. In some instances, hypersensitivity pneumonitis progresses, and granulomas and/or fibrosis may occur. This disease is associated with intense and often prolonged exposure to organic dust. *Dermatitis* describes any rash of the skin. *Contact dermatitis* describes any rash resulting from a substance touching the skin. *Allergic contact dermatitis* is characterized by an eczematous skin reaction appearing 48 hr after exposure to the antigen, following an acquired sensitivity to a given substance. It is caused by a cell-mediated immune response.

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The prevalences/incidences of occupational disease, particularly in the food industries, are difficult to estimate accurately because of an underreporting of health problems by both employees and physicians, and a lack of epidemiologic data on agricultural workers or food handlers.

In food-related industries where prevalences of occupational asthma are available, they are not significantly different from rates in nonfood industries. For example, occuptional asthma occurs in 3–10% of individuals exposed to green coffee beans (Jones et al., 1982; Kaye and Freedman, 1961), 15% of crab processing workers (Cartier et al., 1984), and 10–30% of bakers (Herxheimer, 1967).

The prevalence of hypersensitivity pneumonitis is more difficult to determine because of its generally low occurrence, problems with diagnosis, and the lack of prospective epidemiologic studies (O'Neil and Lehrer, 1992). Incidence of hypersensitivity pneumonitis, as well as of other occupational diseases, also depends on exposure levels of the offending antigen and may vary widely in different areas or plants.

Most studies of dermatologic reactions in food industry workers have included only subjects already diagnosed as having occupational skin disease. Thus, although distinction can be made among types of skin reactions and most important etiologic agents can usually be identified, incidence is difficult to determine.

A number of factors influence the manifestation of allergic disease; these include dose, duration, and route of exposure; physical/chemical properties of the occupational agents; allergenic po-

*Table 1*—Materials Used in Food Industries Known *to induce occupational asthma/rhinitis*

| Agent | Example | Occupational exposure |
|---|---|---|
| **Animal products** | | |
| Sea animals | Prawn, crabs, oysters, shrimp, fish | Seafood processing |
| Farm products | Cows, hogs, poultry, eggs | Farmers, poultry workers, bakery workers |
| Enzymes | Pepsin, trypsin, pancreatic enzymes | Pharmaceutical workers |
| **Plant products** | | |
| Grains/flours | Flour, buckwheat, rice, soybeans, grain dust | Bakers, millers, food workers, agricultural workers, grain handlers |
| Spices/herbs | Garlic, coriander, ginger, cinnamon, paprika | Factory, farm, spice workers |
| Enzymes | Bromelain, papain | Factory workers |
| Miscellaneous | Coffee, tea, pollen | Coffee and tea workers, sugarbeet and sunflower workers |
| **Fungal materials** | | |
| *Alternaria/Aspergillus* | — | Poultry vendors |
| Colophony | — | Poultry vendors |
| Hops | — | Brewery chemists |
| Mushrooms | — | Growers, soup manufacturers |
| Fungal amylase | — | Bakers |

tency; and industrial hygiene and engineering practices. Host factors may play an important role in disease development, since only a limited number of presumably uniformly exposed workers develop occupational reactions. Host factors include atopy, HLA type, cigarette smoking, and possibly nonspecific bronchial hyperreactivity.

## Agents Associated with Occupational Reactions

At least 50 agents encountered in food-related industries have been reported to induce occupational asthma. In some industries, such as in coffee factories, occupational asthma is a well-recognized problem, while in others only individual cases have been reported. Agents of animal, plant, and fungal origin encountered in food-related businesses which are known to induce occupational asthma are listed in Table 1.

Several examples of organic dusts inducing hypersensitivity pneumonitis are illustrated in Table 2. It is noteworthy that many of these are of microbial origin.

A number of foods, additives, and flavorings, in addition to materials used in food preparation, are known to cause occupational skin disease. Etiologic agents and associated diagnosis are summarized in Table 3.

*Table 2*—Etiology of Hypersensitivity Pneumonitis *occurring in food and related industries*

| Agent | Source | Disorder |
|---|---|---|
| **Thermophilic actinomycetes** | | |
| *Micropolyspora faeni* and *Thermoactinomyces vulgaris* | Moldy hay<br>Moldy compost | Farmer's lung<br>Mushroom worker's lung |
| *Thermoactinomyces sacchari* | Moldy sugarcane | Bagassosis |
| *Thermoactinomyces viridis* | Vineyards | Vineyard sprayer's lung |
| **Fungi** | | |
| *Aspergillus clavatus* | Moldy barley/malt<br>Moldy cheese | Malt worker's lung<br>Cheese worker's lung |
| *Aspergillus flavus* | Moldy corn | Farmer's lung |
| *Aspergillus fumigatus* | Vegetable compost | — |
| *Cladosporium* | Moldy hay | Farmer's lung |
| *Mucor stolonifer* | Moldy paprika pods | Paprika slicer's disease |
| *Penicillium* sp. | Moldy hay | Farmer's lung |
| *Penicillium casei* | Cheese | Cheese worker's lung |
| *Penicillium roqueforti* | Cheese | Cheese worker's lung |
| *Botrytis cinerea* | Moldy grapes | Wine grower's lung |
| **Plant products** | | |
| Mushrooms | Spores | Mushroom worker's disease |
| **Insects** | | |
| Grain weevil (*Sitophilus grainarius*) | Infested wheat | Miller's lung |
| Cheese-mites (*Acarus siro*) | Cheese | Cheese worker's lung |
| **Animal products** | | |
| Duck proteins | Feathers | Duckfever |
| Chicken proteins | Chicken products | Feather plucker's disease |
| Turkey proteins | Turkey products | Turkey handler's disease |
| Goose proteins | Feathers | — |
| Fish meal | Fish meal | — |

## Diagnosis

Workers with suspected occupational asthma usually present with episodic dyspnea (shortness of breath), chest tightness, and/or wheezing. When an individual is evaluated for occupational asthma, the diagnosis of asthma must be confirmed by physical examination and objective evidence of reversible airways disease. It is essential to obtain a complete history, including an occupational one, when occupational asthma or any other occupational illness is suspected.

For diagnosis of occupational asthma, it is important to know when symptoms began—whether shortly after beginning a new job or subsequent to a change within the workplace; if agents with known asthma-inducing potential are being used in the work site; if new materials or processes are being introduced into the work environment; and if other employees are similarly affected.

In general, there is a latent period between the first exposure and the onset of symptoms during which sensitization occurs. Following sensitization, a temporal relationship often exists for the occurrence of symptoms after exposure. In immediate type asthma, this relationship is readily apparent; however, in subjects with late, dual, or recurrent nocturnal asthma, a workplace connection may be less apparent.

Hypersensitivity pneumonitis is characterized by dyspnea, chills, fever, cough, and malaise, beginning 4–6 hr after initial exposure. When exposure is continuous and less intense, chills and fever may be absent, while cough, exertional dyspnea, fatigue, and weight loss are present. In general, symptoms subside 18–24 hr after presentation, although they may recur upon subsequent exposure. Since no laboratory tests are available for the diagnosis of hypersensitivity pneumonitis, physical examination and history are extremely important.

A physical examination is also very important in evaluating individuals with possible occupational skin diseases. The appearance of the dermatitis is helpful in determining whether the disease is endogenous, contact, or a combination of the two. Approximately 90% of occupational dermatitis involves hands, usually the palm and the back of the wrist. When occupational skin disease is suspected, location of the dermatitis and exposure source must be matched; reenactment of actual or simulated job practices may be required.

Identification of the etiologic agent may eventually aid in the induction of workplace modifications that will lead to reduction of disease. When putative agents are antigenic, laboratory tests may be useful in establishing a diagnosis. Many of these tests can be performed at the job site; however, others are only possible in the laboratory.

Some antigens encountered in the workplace are suitable for skin prick testing, a technique useful, in combination with other information, to monitor the sensitization of work populations or to establish a diagnosis of occupational allergy. Results must be interpreted with caution, since skin prick tests are not diagnostic by themselves. Specific IgE levels can also be measured using the radioallergosorbent test (RAST). Although less sensitive than skin tests, RAST can be more convenient for industrial populations: serum may be taken during the worker's regular plant physical, removal of the employee from the production line is not necessary, and a physician need not be present. RAST can also be used for retrospective studies.

Double diffusion in gel is frequently used to demonstrate exposure to relevant antigens in subjects with suspected hypersensitivity pneumonitis. However, the presence of antibodies directed against a suspected offending antigen simply confirms exposure and cannot be used for diagnosis.

The patch test is the only practical assay to demonstrate allergic contact dermatitis. In the classic patch test, the

putative agent is applied to a piece of cloth or paper, which is placed on intact skin and covered with an impermeable substance which is taped to the skin. After 24–48 hr, the patch is removed and the underlying skin is examined.

Provocative inhalation challenges to simulate industrial exposures can be performed at the workplace or in a controlled laboratory environment. In the workplace challenge, the employee's lung function is monitored throughout the workday as well as during periods away from the workplace. Workplace challenges demonstrate a decline in lung function over the work period and cannot be used to identify putative agents or definitively establish a temporal relationship to workplace exposure. Workplace challenges have the advantages of minimal risk, since the individual has presumably encountered this level of exposure previously, and low cost. Disadvantages include lack of close supervision, the possiblility that results reflect submaximal effort, inability of the test to be blinded (making adequate controls difficult), and inability to definitively identify the causative agent in all cases.

Laboratory challenge is the method of choice for diagnosis of occupational asthma and identification of the etiologic agent (O'Neil and Lehrer, 1992). This type of testing is indicated when evaluating materials not previously known to induce asthma or when precise identification of the etiologic agent is necessary, such as for litigation. No other test can provide such definitive results. However, laboratory challenge can be dangerous and expensive, and should only be performed in specialized clinics.

## Treatment of Occupational Asthma

The best treatment for allergic occupational disease is prevention. Reduction of allergen levels in some industries may reduce the incidence of respiratory symptoms in exposed employees (O'Neil and Lehrer, 1992). However, once an individual has been sensitized, some occupational responses, notably asthma, can occur at minute levels of exposure—usually lower than an industrial plant can maintain.

Pre-employment screening and/or periodic monitoring has been suggested to prevent development of allergic respiratory disease; the main question is, which tests are appropriate? Although skin prick testing with specific allergens may be useful as a monitoring test for occupational asthma or rhinitis, positive responses do not necessarily correlate with disease. It has been demonstrated that, in industries where atopy is thought to be a predisposing factor for the development of asthma, exclusion of an atopic individual with positive skin tests from the workplace does not eliminate the problem from occurring in the remaining work force.

Once occupational respiratory disease has been diagnosed, the individual should be removed from further exposure, especially since continued exposure after onset of symptoms may adversely affect prognosis. In those instances where workers cannot be

*Table 3*—Dermatitis in Food Processing/Service Workers

| Industry | Exposure | Diagnosis |
|---|---|---|
| **Agriculture** | | |
| Milk industry | Bronopol, Kathon CG chrome, dichromate | Dermatitis, allergic contact dermatitis |
| Celery harvesters | Celery fungus *(Sclerotinia sclerotiorum)* | Phototoxic dermatitis |
| Apple packers | Apples sprayed with ethoxyquin | Allergic contact dermatitis |
| **Food Preparation** | | |
| Fish factory | Fish, mustard | Dermatitis, contact urticaria |
| Cook | Mustard, rape, garlic/onions | Eczema |
| Salad maker | Mustard | Dermatitis |
| Food worker | Cashew nuts (cardol) | Dermatitis |
| Sandwich makers | Codfish, plaice, chicken onion, garlic | Dermatitis |
| Food worker | Lettuce, chicory, endive | Dermatitis, contact dermatitis |
| Bakers | Sodium metabisulfite, persulfate, cinnamon, sorbic acid, propyl gallate, dodecyl gallate, chromium flour mite, sugar mite, karaya gum, flour | Dermatitis, allergic contact dermatitis, eczema |
| **Butchers/poultry processors** | | |
| Butcher, slaughtermen, poultry workers | Rubber boots, knife handle, blood (cow and pig), gut casings, calf's liver | Allergic contact dermatitis, dermatitis, contact urticaria, eczema, urticaria |
| **Seafood** | | |
| Fishmarket worker | Shrimp | Allergic contact, urticaria |
| Caterer | Prawns | Contact urticaria |
| Seafood processors | Oysters | Dermatitis |
| Oyster shuckers | Mussels | |
| Mussel processors | Fish and shellfish | |
| Food handlers | Bryozoa, rubber boots | Contact dermatitis, dermatoses, eczema |
| Fishermen | Fishnets | |
| **Miscellaneous** | | |
| Snackbar meat products | Penicillin residues | Dermatitis |
| Spice workers | Tumeric | Allergic contact dermatitis |
| Margarine manufacturers | Octyl gallate | Eczema, dermatitis |
| Peanut butter manufacturers | Octyl gallate | Dermatitis |
| Food workers | Sesame oil, artichokes | Contact sensitivity, eczema |
| Confectioners | Cardamom | Allergic contact dermatitis |
| Cookie worker | "Thin mint" cookie | Eczema |
| Beekeeper | Propolis | Dermatitis |
| Beekeeper | Beeswax (poplar resin) | Dermatitis |

removed from the offending environment, prophylactic bronchodilator or cromolyn therapy may be attempted; however, their efficacy when used on a regular basis in the workplace has not yet been established.

More-extreme treatment measures, such as immunotherapy, have been attempted on a very limited scale. Preliminary studies indicate some success (O'Neil and Lehrer, 1992).

## Coffee Bean Hypersensitivity

Bean hypersensitivity in coffee workers provides a good example of an occupational asthma in the food industry. It has been known for years that coffee industry workers develop occupational asthma, rhinitis, and dermatitis. Figley and Rawlings (1950) described seven cases of asthma in workers in a coffee processing plant. Their results suggested that castor bean, which contains a highly potent allergen, was an industrial hazard. Kaye and Freedman (1961) studied approximately 25% of the workers in a coffee manufacturing plant in Montreal. Skin testing revealed that more than one-third of those tested had positive reactions to coffee extracts. Most of these workers experienced symptoms of coffee allergy at the time of testing and had prior history of allergy.

During the past 20 years, there have been at least nine published accounts of occupational reactions in the coffee industry. These reactions occurred exclusively in workers employed in coffee manufacturing, rather than in coffee growers. Most of these reports cited green coffee beans, and in some cases castor beans, as the causative agent. Wheezing and shortness of breath were the major symptoms, although several reports suggested that rhinitis and conjunctivitis could occur.

In the past, individuals from coffee manufacturing plants in New Orleans with occupationally related symptoms were seen at the Tulane Clinic. To determine if green coffee dust caused their disease, increasing concentrations of green coffee bean and dust extracts were used to skin test two groups of coffee workers and one group of control subjects (Karr et al., 1978). The first group comprised six employees, who complained of shortness of breath and chest tightness immediately after occupational exposure to dust. The second group consisted of two coffee workers without typical symptoms of allergic disease but having periodic complaints of sore throat, and cough and sputum production unrelated to occupational exposure. The third group consisted of eight non-coffee-dust-exposed atopic laboratory personnel serving as controls.

Symptomatic employees had positive skin-test reactions to 0.01–1 μg/mL of green coffee bean extract and 0.01–10 μg/mL of coffee dust extract. The second group of coffee workers and the control group were skin-test negative at these concentrations. These results suggested that symptomatic workers reacted specifically to coffee allergens. All sera were tested by the RAST for IgE antibodies to coffee allergens. RAST results of sera from symptomatic workers ranged from 2.4 to 13 and were significantly elevated compared to those of the other two groups. Since previous reports in the literature suggested that coffee workers may also be sensitized to castor beans (Bernton, 1973; Figley and Rawlings, 1950), sera were tested for IgE antibodies to this allergen. Sera from the first group of symptomatic coffee workers had elevated castor RAST values ranging from 28 to 60, while the control sera had RAST values of approximately 1 (Figley and Rawlings, 1950). This suggested that symptomatic coffee workers had significantly elevated IgE antibody levels to both coffee and castor allergens. Symptomatic subjects also had positive skin-test reactions to both coffee and castor allergens.

The question of how coffee workers are exposed to castor allergens remains. Possibly, coffee and castor allergens cross-react. Another explanation, proposed by Figley and Rawlings (1950), is that green coffee beans may become contaminated from reuse of sacks previously employed in the transportation of castor beans. To determine whether the latter was true, different extracts of beans, dust, and sacks were analyzed by Karr et al. (1978) for cross-reactivity by their ability to inhibit the green coffee bean or castor bean RAST values. Extracts of coffee and castor beans, factory dust, green grass sack (which had contained Mexican coffee beans), burlap sack (which had contained Colombian coffee beans), and santo sacks (which had contained Brazilian coffee beans) were prepared.

The results indicated that green coffee bean and castor bean allergens did not cross-react. Factory dust contained both green coffee bean and castor bean allergenic activity. No other samples contained activity except the santo sack from Brazil, a country which also grows castor beans for export. Thus, contamination of green coffee beans with castor bean allergens probably occurs by reuse of sacks previously used in transportation of castor beans.

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Updated February 1992 from a paper presented during the IFT Toxicology and Safety Evaluation Division symposium, "Food Allergies and Related Diseases," at the Annual Meeting of the Institute of Food Technologists, Dallas, Tex., June 1-5, 1991.

The authors express their gratitude to Jeanette O'Rourke-Farrell for editorial assistance and gratefully acknowledge the support of the National Fisheries Foundation.

*—Edited by Neil H. Mermelstein, Senior Associate Editor*