OCCUPATIONAL REACTIONS TO FOODS

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SUMMARY

The spectrum of occupational diseases most commonly seen in the food industry includes: occupational asthma, rhinitis, conjunctivitis, dermatitis and hypersensitivity pneumonitis. Occupational asthma represents between 3% and 20% of all asthma cases and is the most common form of occupational lung disease. Occupational skin diseases may represent between 10% and 15% of all occupational diseases and have significant economic impact. Hypersensitivity pneumonitis affects the food industry, with farmers' lung representing a common form of the disease. Each of these diseases can have serious and potentially irreversible effects on the health of farmers, food processors, or food preparers even after removal of the offending exposure.

INTRODUCTION

The food industry is one of the largest employers of workers exposed to numerous allergens that are capable of inducing immunological reactions leading to occupational diseases. Such reactions can occur at every level of the industry, from growing/harvesting of crops or animals, storage of grains, to processing and cooking of food substances. Making the diagnosis of an occupational disease can have a significant social and economic impact on both the individual and society as a whole. Diagnosing an occupational disease requires confirmation of the causal relationship between exposure at work and disease. Although most patients present with new-onset disease, this is not exclusive, e.g. the history of previous asthma does not exclude occupational asthma. In addition, an agent known to cause the occupational disease must be identified and isolated from the worksite. Most sensitising materials are food-derived protein allergens, such as flour and shellfish, but non-food agents may also induce allergic or immunological diseases, e.g. grain storage mites, antibiotics, and even rubber boots. The routes of exposure for food allergens are primarily inhalation and skin contact and vary depending on agents and industries. The ensuing diseases include occupational asthma, rhinitis, hypersensitivity pneumonitis, and occupational dermatitis. Each of these types of reactions will be briefly summarised below. Two more complete reviews have been published recently.[1,2]

OCCUPATIONAL ASTHMA

Occupational asthma (OA) is the most common form of occupational lung disease, with asthma in workers representing 3 - 20% of all asthma patients. A generally agreed upon definition of occupational asthma is variable airflow limitation and airway hyperresponsiveness due to causes and conditions that are attributable to a particular occupational environment and not to stimuli encountered outside the workplace.[3] Another pulmonary disease with significance in occupational lung disorders is reactive airways dysfunction syndrome (RADS).[4] RADS occurs after a single high-intensity exposure to irritant materials, while occupational asthma results from previous sensitisation to a substance and therefore has a latent period, which is highly variable depending on the specific agent, amount and duration of exposure.

Sensitising materials that cause OA are often viewed as high-molecular weight (HMW) or low molecular weight (LMW) agents. HMW (greater than 2 kD) allergens frequently cause OA through an IgE-dependent mechanism. LMW (less than 1 kD) allergens act as haptens that must be conjugated to a carrier protein to be allergenic. LMW allergens cause disease through largely unknown mechanisms, though non-IgE-mediated and cell-dependent immunological mechanisms appear to be important. A variety of materials known to cause OA or rhinitis in food or food-related industries are summarised in Table I. A more extensive list of airway-sensitising agents can be found in a recent review.[5]

Approximately 5% of workers exposed to sensitising agents develop OA. Therefore, individual host factors are likely to affect an individual's development of OA. Atopy and smoking are important risk factors for reactivity to HMW allergens but are not significant risk factors for non-IgE-mediated OA. The epidemiology of RADS has not been studied extensively but atopy does not appear to be a significant risk factor in RADS.[6] The amount of irritant, duration of exposure and proximity to the irritant are significant risk factors for RADS.

Diagnostic evaluation

In order to make a definitive diagnosis of OA, it is crucial to establish a temporal relationship between exposure to an occupational sensitising agent and clinical symptoms of asthma. Demonstration of antibodies against a particular sensitiser by skin-prick test (Fig. 1)

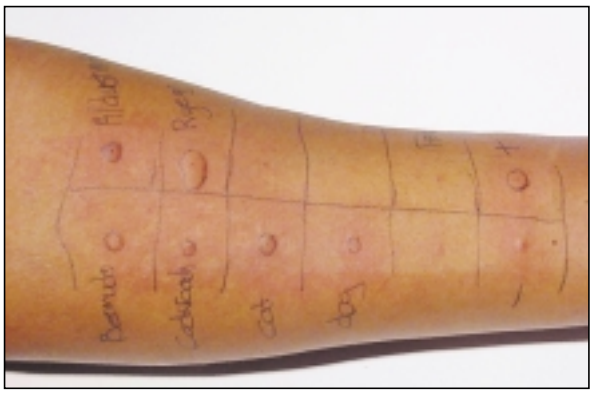

Fig. 1. Skin-prick tests can be used to detect allergic sensitisation to common inhalant allergens and specific occupational allergens.

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Table I. Materials used in food-related industries that induce occupational asthma or rhinitis

Agents	Occupational exposure
Animal products	
Sea animals	
Prawn, crab, king crab, snow crab, lobster, oyster, clams	Seafood processing
Shrimp meal	Aquaculture
Fish meal, fish flour	Factory workers
Mother of pearl	Button factory workers
Sea squirt	Oyster shuckers
Seashells	Shell grinders
Trout	Fish-processing workers
Farm products	
Cows	Dairy farmers
Hogs, swine food	Hog farmers
Poultry	Poultry workers
Pheasants, quail, doves, eggs	Breeders
Egg lysozyme	Egg processors, bakery workers
Insects	
Poultry mites *(Ornithonyssus sylviarum)*	Poultry workers
Grain storage mites *(Glycyphagus destructor)*	Grain workers
Honey bees	Beekeepers, honey processors
Bee-moth	Fish-bait breeders
Rice flower beetle	Rice flower workers
Enzymes	
Pepsin, trypsin, pancreatic enzymes	Pharmaceutical workers
Miscellaneous	
Spiramycin	Chick breeders
Pyrolysis products of polyvinyl chloride or label adhesives	Meat wrappers
Plants/fungi	
Grains/flours	
Flour (wheat, rye)	Bakers, millers
Buckwheat, carob bean flour	Food workers
Rice	Rice millers
Soybeans, soybean lecithin	Agricultural workers
Grain dust	Grain handlers
Spices/herbs	
Garlic	Factory workers, farmers
Coriander, mace, ginger, paprika	Factory workers
Cinnamon	Spice workers
Paprika plants	Greenhouse workers
Vegetables	
Green beans, okra	Homemaker
Enzymes	
Bromelain, papain	Factory workers
Miscellaneous	
Coffee	Coffee factory workers
Castor	Factory workers, dock workers
Tea, herbal tea	Tea factory workers, tea garden workers
Pollen	Sugar beet workers, grape workers
Pectin	Candy or jam makers
Alkaline hydrolysis derivative of gluten	Bakers
Alternaria/Aspergillus, colophony	Poultry vendors
Hops	Brewery chemists
Devil's tongue *(Amorphophallus konjac)*	Food workers
Mushrooms	Soup manufacturers, growers
Fungal amylase	Bakers
Verticillium albo-atrum	Greenhouse workers

or radio-allergosorbent test (RAST) does not imply causality, it merely suggests that there was exposure. Asthma symptoms or objective data such as peak flows that improve when outside the workplace are also suggestive of OA. Peak flows should be monitored serially several times a day along with a log of symptoms and use of medications. They should also be followed up for 1 - 2 weeks after the patient is removed from the workplace. Because of possible secondary gain issues and other factors, it is important to realise that peak flow is extremely effort dependant.[7]

Repeatable spirometric or bronchial hyperresponsiveness (e.g. methacholine challenge) data that shows improvement after removal from the workplace has good positive predictive value. However, its negative predictive value is not as good, since airway hyperresponsiveness can have a long lag time for improvement. Therefore, the absence of improvement does not rule out OA as a diagnosis. If a definitive diagnosis still remains elusive, an additional method of evaluation is to expose the patient to the agent in question in a controlled environment. Challenge testing in a chamber should be performed at doses that are not irritating and in a setting that is prepared to deal with a potential asthmatic episode.

Treatment and prevention

Once sensitised, workers can react to exquisitely small amounts of the agent. Therefore, it is imperative that treatment begins with removal of the worker from the work environment. Pharmacological treatment for these patients should follow the NHLBI asthma management guidelines.[8,9] Even after removal from further exposure, workers may not fully recover. The most relevant factors responsible for duration of symptoms after work withdrawal seem to be the duration of exposure after onset of symptoms, the total duration of exposure, and the severity of asthma at the time of diagnosis.[10-12] Although some patients may continue to suffer from occupational asthma after removal from the work environment, the best prognosis results when there is early diagnosis and early removal from the exposure environment.[13]

OCCUPATIONAL RHINITIS

Occupational rhinitis (OR) has been defined as the episodic work-related occurrence of sneezing, nasal discharge, pruritus, and congestion which contribute to distress, discomfort and work inefficiency.[14] OR occurs 2 - 3 times more frequently than OA. It often coexists with OA and the rhinitis symptoms frequently precede the development of asthma in the work environment.[15]

OR has been classified by Bardana as immunological or as annoyance, irritational, or corrosive (all non-immunological).[13] Annoyance reactions occur from exposure to mild workplace irritants such as perfumes, air fresheners, and cooking odours. Irritational rhinitis is caused by inhalation of high concentrations of airborne chemicals. This reaction is often associated with a burning sensation. Corrosive rhinitis occurs after exposure to high concentrations of chemical gases, like ammonia, chlorine, and organophosphides. Signs/symptoms of systemic intoxication may also be present. Immunological or allergic OR can result from HMW or LMW allergens and is usually IgE-mediated, although the exact mechanism is unkown for most LMW agents. The majority of allergens in the food industry are of HMW, e.g. flour, soybean dust, vegetable gums, and animal proteins.

The food industry accounts for the largest number of cases of occupational rhinitis.[16] The prevalence of OR has been reported to be between 3% and 60% depending on the exposure environment. Its prevalence in patients with OA is 76 - 92%.[15,17]

Diagnostic evaluation

As in OA, the history of workplace exposure is essential. A medication history is equally important because symptoms may be masked by the use of certain medications. Symptoms initially felt to be related to the work environment may become prolonged or worsen after removal from the culprit environment with the overuse of certain medications. For example, rhinitis medicamentosa may develop as a result of chronic topical decongestant use for the treatment of OR. Physical findings in OR are non-specific and similar to findings in rhinitis from non-occupational causes. Atopy and the development of OR have not been linked. In making the diagnosis of OR, allergen-specific IgE should be measured if the test is available (skin test or RAST). The presence of allergen-specific IgE helps support the diagnosis of OR when the history is suggestive, but positive skin tests or RAST are not themselves diagnostic of disease as they merely indicate exposure and sensitisation. Nasal challenge can help confirm the diagnosis of OR, but is not being widely used. Nasal challenges are time-consuming and not standardised.

Treatment and prevention

Like other forms of occupational disease, OR symptoms typically improve with removal from the work environment. Simple treatment with H_1-antagonists or inhaled corticosteroids may be enough to control the symptoms and allow the worker to continue his/her job, preferably in a much lower exposure environment. The prognosis of occupational rhinitis has not been well studied.

HYPERSENSITIVITY PNEUMONITIS

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, is an immune response to an environmental antigen resulting in an inflammatory parenchymal lung disease. The mechanisms involved in HP appear to involve Gell-Coombs type III and type IV hypersensitivity reactions. The clinical presentation of HP is often classified as acute, subacute or chronic. In the acute presentation, flu-like symptoms including fever, chills and cough often result in its confusion with a bacterial or viral respiratory infection. Patchy or diffuse infiltrates may be seen on chest X-ray. The symptoms usually begin 4 - 12 hours after work exposure and may resolve within 24 hours of antigen removal. The subacute form may have a more prolonged course of shortness of breath, weight loss and fatigue. In chronic disease, if the antigen exposure is not interrupted, the subject may develop pulmonary granulomas and even go on to develop irreversible pulmonary fibrosis.

HP is primarily an occupational disease; however, individuals can be sensitised to culprit agents from heating/air conditioning systems or from their home environment. Table II lists the specific agents that have been implicated in HP and the diseases they cause in food-related industries.

The incidence of HP is difficult to determine as a result of its generally low occurrence, problems with differential diagnosis, and the lack of prospective epidemiological studies. Studies have noted incidence rates of between 2.5 and 153.1 per 1 000 farmers. Atopic subjects do not have a higher incidence of HP. Interestingly, smoking seems to have a protective effect against the development of HP, but once HP has started, smoking does not appear to be protective.[18]

Diagnostic evaluation

There is no single test that is pathognomonic for HP.

Table II. Aetiology of hypersensitivity pneumonitis occurring in food and food-related industries

Agent	Source	Disorders
Thermophilic actinomycetes		
Faenia rectivirgula	Mouldy hay	Farmers' lung
Micropolyspora faeni	Mouldy compost	Mushroom workers' lung
Thermoatinomyces sacchari	Mouldy sugar cane	Bagassosis
T. vulgaris	Mouldy compost	Mushroom workers' lung
	Mouldy hay	Farmers' lung
T. viridis	Vineyards	Vineyard sprayers' lung
Fungi		
Aspergillus clavatus	Mouldy barley/malt	Malt workers' lung
	Mouldy cheese	Cheese workers' lung
A. flavus	Mouldy corn	Farmers' lung
A. fumigatus	Vegetable compost	
A. oryzae	Soy sauce brewer	Hypersensitivity pneumonitis
Cladosporium	Mouldy hay	Farmers' lung
Mucor stolonifer	Mouldy paprika pods	Paprika slicers' disease
Penicillium sp.	Mouldy hay	Farmers' lung
P. caseii, P. roqueforti	Cheese	Cheese workers' lung
Botrytis cinerea	Mouldy grapes	Wine growers' lung
Insects		
Grain weevil *(Sitophilus grainarius)*	Infested wheat	Millers' lung
Cheese mites *(Acarus siro)*	Cheese	Cheese workers' lung
Animal products		
Duck proteins	Feathers	Duck fever
Chicken proteins	Chicken products	Feather pluckers' disease
	Hen litter	
Turkey proteins	Turkey products	Turkey handlers' disease
Goose proteins, bird proteins	Feathers	
Plant products		
Miscellaneous		
Mushrooms	Spores	Mushroom workers' disease
Erwina herbicoa (Enterobacter Agglomerans)	Contaminated grain	Grain workers' lung
Tea plants		Tea growers' lung
Oyster shells	Oyster shell dust	Hypersensitivity pneumonitis

For occupational HP, a history of workplace exposure is essential along with the appropriate clinical symptoms. Elevated IgG levels are seen and IgM and IgA may also be elevated. The workup should include testing for specific IgG antibody to the putative agent by immunoprecipitation. Finding precipitating antibody is highly suggestive of disease, but it should be noted that 3 - 50% of asymptomatic subjects may also have precipitants (evidence of exposure but not causality). IgE levels are usually not elevated and skin testing for immediate hypersensitivity is of no value in making a diagnosis of HP. Routine workup should include chest X-rays and pulmonary function testing (PFT) (Fig. 2). HP subjects classically have a restrictive pattern of PFTs but a mixed obstructive/restrictive pattern may also be seen. Chest X-rays and PFTs may be normal between attacks in HP, until chronic disease develops. A high-resolution computed tomography (CT) scan is more sensitive than chest X-ray or traditional CT and may reveal abnormalities when the chest X-ray is normal. HP has a characteristic bronchioalveolar lavage (BAL) that shows neutrophilia within the first 48 hours and then a lymphocytosis. When clinical history and laboratory data are not sufficient to make a diagnosis, a lung biopsy may be needed.

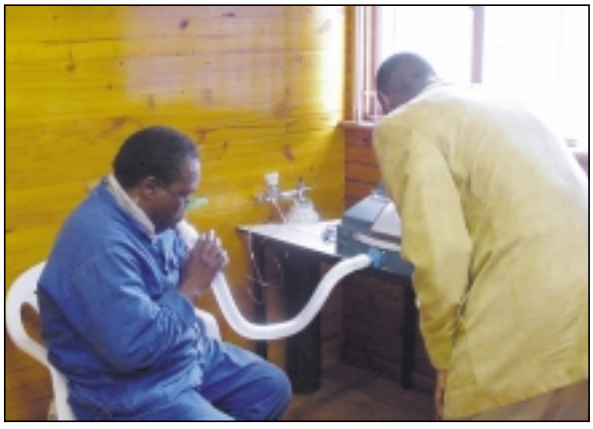

Fig. 2. Lung function testing can be used to detect airway obstruction and confirm the presence of asthma and bronchial hyperresponsiveness.

Table III. Dermatitis in food-processing and food-service workers

Industry	Exposure	Diagnosis
Agriculture		
Milk controllers, milk recorders, milkers	Bronopol, Kathon CG	Dermatitis
Milk testers	Chrome, dichromate	
Milk analyzers	Dichromate	Allergic contact dermatitis
Ewe milker		Dermatitis
Celery harvesters	Celery fungus *(Sclerotinia sclerotiorum)*	Phototoxic dermatitis
Apple packers	Apples sprayed with ethoxyquin	Allergic contact dermatitis
Orange pickers	Omite-CR	Dermatitis
Grocery workers	Celery (furocoumarins)	
Food preparation		
Fish factory workers	Fish	Dermatitis, contact urticaria
Cooks	Mustard, rape	Eczema
	Garlic, onions	Eczema
	Paprika, curry	Contact dermatitis
Salad makers	Mustard	Dermatitis
Food workers	Cashew (cardol)	Dermatitis
	Lettuce	Dermatitis, contact dermatitis
	Chicory, endive	Contact dermatitis
Sandwich makers	Codfish, plaice, chicken, onion, garlic	Dermatitis
Bakers	Sodium metabisulphite	Dermatitis
	Persulphate, cinnamon	Dermatitis
	Sorbic acid	Allergic contact dermatitis
	Propyl gallate	Eczema
	Dodecyl gallate	Eczema
	Chromium	Dermatitis
	Flour mite, sugar mite	Dermatitis
	Karaya gum, flour	Contact urticaria
Butchers/poultry processors		
Butchers	Rubber boots	Allergic contact dermatitis
	Knife handle	Dermatitis
	Povidone-iodine	Allergic contact dermatitis
	Calf liver, pig gut, beef	Urticaria
Slaughterhouse workers	Blood, gut casings	Contact urticaria, eczema
Poultry workers	Various	Irritant allergic dermatitis, eczema
Chicken vaccinators	Antibiotics	Contact dermatitis
Seafood		
Fish market workers	Shrimp	Allergic contact urticaria
Caterers	Shrimp	Contact urticaria
	Fish	Dermatitis
Seafood processors	Prawns, crabs	Asthma, dermatitis
Oyster shuckers	Oysters	Dermatitis
Mussel processors	Mussels	Dermatitis
Food handlers	Fish, shellfish, cuttlefish	Contact dermatitis
Fishermen	Fish	Skin diseases
	Rubber boots	Dermatitis
Fish workers, cooks	Fish	Contact urticaria
Trawlermen	Bryozoa	Dermatitis, eczema
Fishnet repairers	Fishnets	Dermatitis
Miscellaneous		
Snack bar meat products	Penicillin residues	Dermatitis
Spice workers	Turmeric, cinnamon, cinnamic aldehyde	Allergic contact dermatitis
Margarine manufacturers, workers	Octyl gallate	Eczema, dermatitis
Peanut butter manufacturers	Octyl gallate	Dermatitis
Food workers	Sesame oil	Contact sensitivity
	Artichokes	Eczema
Confectioners	Cardamom	Allergic contact dermatitis
Cookie workers	'Thin mint' cookies	Eczema
Beekeepers	Propolis	Dermatitis
	Beeswax (poplar resin)	Dermatitis
Coconut climber	Coconut trees/coconuts	Dermatitis
Bartender	Citrus peel, geraniol citral	Allergic contact dermatitis

Treatment and prevention

As antigen exposure is the underlying promoter of disease, strict avoidance of the inciting agent is the first treatment measure. Corticosteroids are the main medical therapy in HP, except in the case of chronic disease where the fibrotic changes are irreversible. With early diagnosis of HP and strict avoidance of exposure to the aetiological agent, the outcomes are generally good with full recovery of the subject. Delays in diagnosis and treatment may lead to the chronic form of HP with irreversible damage.

OCCUPATIONAL DERMATITIS

Cutaneous reactions to foods generally result from handling or ingestion of the food products. Unlike occupational lung disease, there is a paucity of information on occupational dermatitis and there is significant discrepancy regarding the incidence of disease. Occupational dermatitis is divided into irritant contact dermatitis (ICD), allergic contact dermatitis (ACD), and occupational contact urticaria. Table III reviews the various industries, exposures and disease processes resulting in dermatitis in food-processing and food-service workers.

Occupational contact dermatitis presents clinically with erythema, pruritus, oedema and a papulovesiclar eruption. In chronic cases there is minimal pruritus; however, there is fissuring, scaling, desquamation, postinflammatory hyperpigmentation, and lichenification. Based on epidemiological studies, contact dermatitis represents 90 - 95% of all occupational dermatoses and occurs most commonly on the hands. ACD represents 20% of the cases and ICD represents 80%. ACD is a delayed hypersensitivity reaction (Gell-Coombs type IV) in which there is recruitment of previously sensitised, antigen-specific T lymphocytes into an exposed area of the skin.[19] ICD is generally a non-immunological reaction to a high concentration of agent that does not require previous exposure to sensitise the patient. The amount of the agent and the duration of exposure are important in ICD. Morphologically, the borders of an ACD reaction are limited to the area of contact whereas in ICD the borders are indistinct.

Occupational contact urticaria presents with typical erythematous, papular, pruritic hive lesions, but in this case it is associated with a specific occupational exposure. The mechanism is usually an IgE-mediated process. Significant risk factors include a history of atopy as well as anything that results in the breakdown of the skin barrier. Although there are no significant data on occupational contact urticaria, epidemiological data have shown that cooks, bakers, caterers, and food handlers are at increased risk. The potential allergens for these individuals include fruits, vegetables, fish, meat, dairy and grain products.

Important environmental factors that predispose individuals to occupational skin dermatitis include humidity or temperature extremes, fissured skin and occlusion of the skin. The hygiene requirements of the food industry lead to repeated hand washing by workers, which can potentially damage the skin.[20] Significant host factors include anatomical site, history of atopy, skin pigmentation, aged skin and immunosupression. Atopic dermatitis may predispose workers to develop ICD, however it does not appear to predispose to ACD. Skin breakdown resulting from ICD can be a predisposing factor to ACD. Therefore, a given worker may have both ICD and ACD.[21] Experimental studies have not shown any gender differences in the development of hand eczema.[22]

Diagnostic evaluation

As with all occupational disorders, a careful medical and occupational history must be performed in order to make a temporal association between exposure to a potential agent and onset of disease. Furthermore, non-occupational exposures such as leisure time activities should be excluded. This is particularly important in cases where the dermatitis does not improve with time away from work. There are no specific tests that can confirm the diagnosis of ICD. In ACD, patch testing can be performed for confirmation of a suspected agent. The sensitivity and specificity of patch testing is about 70%.[23] In occupational contact urticaria, specific reactivity to potential allergens identified in the history can be tested with skin-prick or RAST testing.

Treatment and prevention

Regardless of whether a worker suffers from allergic or irritant dermatitis, it is critical that the allergen or irritant be identified. Treatment begins with avoidance or substitution of the potential agent. The affected worker should also receive hands-on instructions concerning the use of protective equipment such as gloves and clothing. Educating patients is critical if they are going to practise proper avoidance. In addition, proper occupational hygiene must be emphasised. The general approach to treatment of ACD is to use topical steroids when less than 10% of the skin is involved and use oral steroids if there is greater skin involvement. Treatment should last 2 - 3 weeks since premature cessation can cause a rebound exacerbation of skin symptoms. In the case of occupational contact urticaria, pharmacological intervention should begin with use of second-generation non-sedating antihistamines, since the first-generation sedating antihistamines may involve occupational safety issues. Additionally, H_2-blockers or combination H_1- and H_2-blockers can be used. The most efficacious treatment for urticaria is oral corticosteroids such as prednisone. However, long-term oral steroid use has many well-known complications and is therefore not recommended.[24] If these efforts do not improve or resolve the dermatitis, the worker should be withdrawn from exposure to the allergen/irritant. The prognosis is excellent if exposure is eliminated.

CONCLUSION

The examples illustrated in this paper are but a few of the wide array of food-allergen-associated occupational reactions. An important aspect of making a diagnosis of occupational reaction is to take a careful and thorough history, looking for a temporal association between the reaction and the exposure. New causative agents are continually being reported and as new foods are developed it is possible that new occupational reactions may occur. Of particular interest is the development of genetically modified (GM) crops that may contain novel proteins to which there is no prior human exposure. Although reaction is unlikely because of the low levels of expression of such proteins, such a possibility should be considered whenever occupational reactions occur in industries growing or processing such foods. With globalisation of world markets and a continuing increase of individuals employed in the food industry, it is important for the practising clinician to continually keep abreast of these new reactions when approaching a new or unusual occupational reaction.

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CHAIRMAN'S REPORT

Another year has drawn to a close and it's time to reflect on the year's activity and to plan for the coming year. The ALLSA congress held in October 2002 was a huge success, with an excellent attendance and a stimulating scientific programme. The congress was held at Gallagher Estate in Gauteng, which proved to be an excellent venue. The planning for next year's congress is now advanced, and we invite you all to attend this historic joint congress between ALLSA, SATS and the Critical Care Society. This meeting will be held in Cape Town in August next year.

The exams for the Diploma in Allergology will be written for the first time in March 2003. We are preparing courses for prospective candidates. Details of these courses can be obtained from the ALLSA office. We have received numerous enquiries regarding the Diploma. It is now a reality and details can be obtained from the offices of the College of Medicine of South Africa.

Allergic diseases are increasing at a rapid pace in South Africa. Many studies have confirmed this. The teaching of allergology is sadly neglected in most medical schools. Most doctors therefore do not receive any teaching in allergology. This is further compounded by the fact that there is only one teaching institution in the entire country that offers postgraduate training in allergology. It is hoped that with the introduction of the diploma, more doctors will be attracted to allergology and establish departments at academic institutions.

ALLSA awards research grants on a yearly basis. We are once again grateful to GlaxoSmithKline and UCB Pharma for their generous research grants. These grants are much needed to support ongoing research in the field of allergology.

The next WAO meeting, which is held every 3 years, will be in Vancouver in September 2003. ALLSA is an affiliate of WAO and we encourage our members to attend this congress.

Finally, I would like to wish our readers well over the festive season.

Ahmed Manjra

ALLSA Chairman