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Occupational rhinitis

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Patients who present with symptoms of rhinitis frequently are encountered in physicians' offices. Clinicians should survey the workplace as a source of these complaints. Occupational allergic diseases are common, and their occurrence seems to be increasing, with an estimated prevalence of 5% to 15% [1]. People spend more time at work and have greater exposure to many new, potentially irritative and immunogenic substances, such as chemicals (soaps, detergents, dyes, paints) and biologic products (live animals, plants, fungi, pollen). The incidence of these diseases is underestimated because of a failure to diagnose and a reluctance of the worker to complain for fear of losing his or her job. A new appreciation of the workplace as an arena for exposure to allergens is increasing. Traditionally, the workplace has been overlooked as a source of the problem, because there may be a long latency period between exposure and development of symptoms. There has been a general feeling that low-molecular-weight substances that commonly are encountered in the working environment are not of sufficient size to elicit an immunologic response. Although many of these substances are not immunogenic in and of themselves, they are excellent haptens. They can combine with tissue proteins, such as human serum albumin, in the respiratory tract to form a hapten–protein conjugate that serves as a potent immunogen. By concentrating on the patient's workplace, the clinician has an opportunity to practice preventive medicine: to recognize substances in the patient's micro- and macroenvironment that are causing the problems and then to intervene by altering the environment or removing the patient from the environment.

Asthma has overshadowed rhinitis, perhaps because of its potentially more serious nature and associated disability. A review of several classic occupational medicine texts reveals several paragraphs dedicated to Q fever and cheese workers' lung and warts, but there is scant mention of occupational rhinitis [2,3]. Conversely, over the past 10 years, a growing number of case reports have

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documented the diagnosis of occupational rhinitis and have described the offending agents. The Occupational Safety and Health Administration (OSHA) documents more than 500,000 potentially hazardous chemicals in the workplace, of which 200 are implicated as causes of allergic respiratory diseases [4]. The existence of occupational rhinitis and its resulting symptoms are significant enough to require inquiry and management of this disorder [5].

Definition of occupational rhinitis and its relationship to occupational asthma

Occupational rhinitis is defined as the episodic work-related occurrence of sneezing, nasal discharge, and nasal obstruction [6]. Exposure to known respiratory irritants at the workplace can result in increased nasal hyperreactivity and can predispose the worker to occupational rhinitis [4]. This disease can have a profound effect on the worker, resulting in performance deficits, reduced productivity, and psychosocial problems. Occupational rhinitis often coexists with occupational asthma. Although it commonly is accepted that allergic rhinitis may precede asthma [7], reports of occupational rhinitis developing into asthma are rare.

In one report from Finland, investigators examined cases of occupational rhinitis and asthma as reported to the Finnish Register of Occupational Diseases. Most patients with occupational asthma presented at an older age than did patients with rhinitis, suggesting that rhinitis precedes asthma [8]. The progression from occupational rhinitis to occupational asthma was reported in a laboratory technician who worked in a forensic laboratory. She had developed immunoglobulin E (IgE)-mediated allergic rhinitis to ninhydrin, a chemical used in fingerprint detection. Despite handling ninhydrin only in a flow cabinet, symptoms of dyspnea developed after 6 months of exposure. Peak flow varied by 20% during work days. In a specific inhalation challenge, slight exposure to ninhydrin produced an asthmatic response, and the titre of ninhydrin-specific IgE increased [9]. Additional evidence supporting the trend of rhinitis to asthma has been documented in glutaraldehyde exposure (a cold sterilization agent) and in incidents resulting from the handling of laboratory animals [10,11]. In a survey of laboratory workers with allergic symptoms, 100% of those affected had rhinitis with conjunctivitis, whereas only 71% had asthma [12].

Nasal physiology

To best understand the clinical entity of occupational rhinitis, aspects of basic nasal physiology should be reviewed. The nasal mucous membrane responds to stimuli in four ways. Congestion results from vasodilation, vascular permeability, and sinusoidal pooling. Pruritus and sneezing are caused by sensory nerve stimulation. Secretions are the result of glandular stimulation and increased vascular permeability.

Nasal challenge has provided an excellent model for quantifying responses and for obtaining insights into nasal physiology. The nose can be challenged with allergens, mediators, and irritants. The response can be quantified by symptom scores, by nasal airway resistance, and by study of secretions, including amount, composition, regulatory molecules, proteins, enzymes, and cells. Components of nasal secretions that have been studied include prostaglandin D_2 (PGD_2), toxoid–antitoxoid mixture esterase (TAMe), kinins, albumin, immunoglobulins, lactoferrin, tryptase, and major basic protein.

A brisk cellular influx occurs in nasal lavage fluid several hours after nasal challenge with antigen. This influx includes a significant increase in total cells, epithelial cells, eosinophils, neutrophils, and mononuclear cells. An early response is seen universally in atopic individuals after nasal challenge with an appropriate antigen. In addition to sneezing, nasal congestion, and an increase in secretions, nasal lavage fluid within 11 hours shows increased levels of histamine, TAME esterase, PGD_2, and kinins [13]. The early reaction frequently is followed by a late response 4 to 6 hours later that is similar to that seen in the lung after bronchial challenge. The late response is associated with the cellular influx and by increased levels of histamine, TAME esterase, and kinins. Levels of PGD_2 are not increased. Because PGD_2 is found in mast cells and not basophils, this is indirect evidence that basophils are involved in the late response. As in the lung, the late reaction in the nose is associated with a heightened response to antigen and histamine [14].

Causes of occupational rhinitis

Bardana has suggested the following classification for occupational rhinitis [15]:

Annoyance: occurs in individuals with a heightened olfactory awareness to such substances as perfumes and detergents. The likelihood of developing annoyance reactions is increased by nasal polyposis, sinusitis, tobacco abuse, and overuse of over-the-counter nasal decongestants or illicit drugs [4].

Irritational: nonspecific inflammation of the nose that has no immunologic or allergic basis. Exposure to substances, such as cigarette smoke, formalin, and capsaicin, results in the release of substance P, a sensory transmitter that triggers a neurogenic inflammatory response. This type of occupational rhinitis is seen in patients who work in an enclosed environment and who are exposed to such materials as paints, talc, and coal dust.

Corrosive: exposure to a high concentration of irritating and soluble chemical gases, such as chlorine or ammonia, that results in such a degree of nasal inflammation that the mucosa burns and ulceration may occur, leading to permanent changes in physiologic functions of the olfactory system.

Immunologic: allergic responses are mediated by IgE, with resultant early and late nasal reactions. High-molecular-weight allergens or low-molecular-weight allergens bound to proteins can elicit an allergic response.

Causative agents and typical exposures in occupational rhinitis are reviewed (Table 1). Psyllium, a major component of popular bulk laxatives, may cause rhinitis in persons who work in laxative-producing factories or in nurses who dispense the medication [16]. Guar gum commonly is reported as a cause of occupational rhinitis and asthma, especially in Europe. This gelling and thickening agent affects workers in the food industry. Carpet workers may become sensitive to guar gum through its use as a fixator of dye to fiber and as an insulator in rubber cables [17].

Laboratory workers most commonly are affected with occupational rhinitis and asthma; more than 30% of these workers become sensitized. The rat is the most commonly implicated animal, and, as stated earlier, rhinitis with conjunctivitis is the most common complaint among these workers [12].

In one of the earliest reports of sensitivity to western red cedar dust (plicatic acid), approximately half of the affected workers had associated rhinitis [5]. In another report, rhinitis caused by exposure to western red cedar preceded the onset of asthma [18].

Latex-induced occupational rhinitis is increasingly diagnosed. OSHA estimates that 8% to 12% of heathcare workers are sensitized to latex [19]. In one report, a nurse experienced immediate itching, redness, and irritation of the hands after direct skin contact with natural rubber latex gloves over a 6-year period [20]. With use of vinyl gloves, her symptoms improved. Co-workers, however, still used latex gloves, and the nurse began noticing symptoms of rhinitis at work because of the significant amount of airborne natural rubber latex antigen. Nasal provocation and skin test to latex were positive, and she had elevated serum levels of latex-specific IgE.

Diisocyanates are highly reactive chemicals that are encountered by foundry workers, polyurethane foam makers, and spray painters. It is the most common

Table 1
Causative agents and typical exposures in occupational rhinitis

Cause	Agent	Exposure
Annoyance	Detergents	Supermarket
	Perfumes	Department store
	Cooking	Kitchen
Irritational	Air pollutants	Outdoor or inner-city workplace
	Cigarette smoke	Any workplace
	Cold air	Meat packer
	Hairspray	Department store
	Talc	Cosmetics industry
Immunologic	Natural guar gum	Carpet worker
	Latex	Nurse
	Psyllium	Nursing home
	Rats	Laboratory researcher
	Synthetic acid anhydride	Epoxy worker
	Platinum	Jeweler
	Toluene diisocyanate	Spray painter

class of reagents that cause occupational asthma. Diisocyanates may cause a nonspecific irritant rhinitis, and in a small number of workers who have an IgE sensitivity to diisocyanates, allergic rhinitis may result. The risk for occupational rhinitis or occupational asthma is related directly to the exposure level; atopy and smoking do not seem to be major risk factors [4].

In the study of the Finnish Register of Occupational Diseases in which 1244 new cases of occupational rhinitis were reported during 1986 to 1991, animal dander, flowers, wood dusts, textiles, phthalic acid anhydrides, and storage mites were found to be important causes of occupational rhinitis [8]. Furriers, bakers, and livestock breeders had the highest relative risk of occupational rhinitis.

Diagnosis of occupational rhinitis

The following four steps are taken in the diagnosis of occupational rhinitis:

1. History and physical examination
2. Site visit
3. Skin tests or RAST
4. Nasal challenge

The association of nasal congestion, sneezing, and increased nasal secretions with exposure to the workplace provides the first clue to the diagnosis. Occupational rhinitis may be associated with an isolated late reaction; therefore, symptoms might not appear until several hours after leaving the workplace. Evidence leading to the suspicion of the workplace as a source of occupational rhinitis comes from a comprehensive medical history as shown in the following list from Puchner [4]:

Onset of symptoms at work
Improvement on weekends or vacations
Recurrence of symptoms on reexposure
Exposure to:
  Cold air
  Ozone
  Pollution
  Tobacco smoke
  Animals
  Dust
Similar symptoms in co-workers
Duration working at current job
Adequacy of ventilation
Compliance with wearing gloves, masks, protective clothing
History of accidents or spills
  Monitoring

Review of material safety data sheets (MSDSs)
Personal or family history of atopy
Coexisting symptoms
  Bronchial
  Dermatologic
  Conjunctival
  Anosmia
Smoking history
Hobbies
Previous employment
Intranasal medication or drugs

The primary symptom of a late reaction is nasal congestion rather than sneezing, itching, or rhinorrhea. Symptoms usually abate when the patient is away from the workplace on weekends or holidays, but chronic exposure may require several days away from work before the symptoms remit. Physical examination during the time of active rhinitis reveals swollen nasal turbinates and evidence of increased secretions. Nasal findings may be minimal in patients with annoyance causes of occupational rhinitis, whereas corrosive causes reveal ulcerated and hemorrhagic mucosa [4].

Infectious paranasal sinusitis should be excluded, because it is such a common occurrence. Occupational rhinitis may predispose to sinusitis. The mechanism is believed to be inflammatory swelling of the nasal mucosa, which leads to blockage of the ostia and decreased aeration and drainage from the sinuses. Symptoms of sinusitis include persistent nasal blockage and purulent postnasal drainage.

A site visit to the workplace is frequently helpful in terms of eliciting the exact cause of occupational rhinitis. The patient may be unaware of the ingredients to which he or she is exposed and may forget certain routine acts in relating the occupational history. A site visit enables the physician to see the patient in action and to obtain a more accurate picture of the types of exposure. MSDSs must be available by law at the workplace. These data give detailed information about the substances to which the worker is exposed and the health effects of each substance.

If the occupational rhinitis is caused by an IgE mechanism, appropriate skin tests or in vitro radioimmunoassay testing may be performed. Skin testing is useful in allergic occupation rhinitis if the disease is caused by animal, dust, latex, or mold exposure. Low-molecular-weight substances may need to be conjugated with serum albumin to elicit a response [4].

Direct nasal challenge can be performed naturally at the work place or under controlled circumstances in the office or laboratory. Responses may be quantified by symptom score or rhinomanometry. A simple technique that is used widely in Europe is to impregnate a piece of cotton wool with the offending agent, such as guar gum, and to apply this wool directly to the nasal mucosa [21]. An aqueous solution also can be blown into the nose from a syringe. One hour later, the nose is examined, and the patient's symptoms are noted. Nasal challenge is performed

best when the patient is not taking antihistamines and when symptoms are minimal. The technique of rhinomanometry, which measures changes in nasal airway resistance, is the preferred technique to objectively measure the response to nasal challenge [22]. It has been suggested that skin prick tests combined with specific IgE tests for latex are highly specific and sensitive for latex allergy and that nasal provocation should be performed only in symptomatic glove users who have borderline results of skin and humoral tests for latex [23].

Rhinomanometry

In anterior rhinomanometry, nasal airway resistance is derived from the relationship between flow rate and pressure. Commercially available anterior rhinomanometers measure airflow through one nares by means of a closely fitting face mask, and the other nares is sealed with a small tube to measure nasopharyngeal pressure. The technique is easy to perform, requires little patient cooperation, and is convenient for nasal provocation. Its disadvantages are that the probes distort the nasal valves, tactile sensation may result in reflex changes, and the technique is affected by nasal cycling [22]. The results of tests that use this method should be interpreted using other criteria, such as the number of sneezes and the amount of nasal secretions. [4].

In posterior rhinomanometry, a face mask that is fitted with a pneumotachometer monitors airflow through both nasal cavities simultaneously and monitors anterior nasal pressure. Although posterior rhinomanometry avoids the disadvantages of artifacts from nasal instrumentation and nasal cycling, the equipment is expensive, and the procedure is difficult to perform for some patients. About 20% of normal individuals do not have sufficient palatal control to allow pressure-flow recordings that are consistently free of artifacts [22].

The results of a positive nasal challenge in a nurse who experienced rhinitis when distributing psyllium, a commonly used bulk laxative, are shown (Table 2). The challenge was performed by pouring the powdered laxative from one container into another. In 2 minutes, significant increase in nasal airway resistance is seen, and the peak obstruction was noted 5 minutes after the challenge began.

Table 2
Airway response to psyllium

Time	NAR	Baseline NAR valve (%)	FEV_1	Baseline FEV_1 valve (%)
Baseline	1.58	100	3.33	100
After 2 min	6.40	405	3.24	97
After 5 min	9.17	580	3.24	97
After 15 min	6.40	405	3.24	97
After 30 min	5.39	341	3.24	97

Abbreviations: FEV_1, forced expiratory volume in 1 second; NAR, nasal airway resistance.
Data from Schwartz HJ, Arnold JL, Strohl KP. Occupational allergic rhinitis reaction to psyllium. J Occup Med 1989;31:624–6.

The response decreased, but at 30 minutes evidence of nasal obstruction persisted. There was little or no change in forced expiratory volume in 1 second [16].

Nasal challenge can provide insights into the physical characteristics of materials that may cause occupational rhinitis. Blainey et al reported the case of a carpenter whose job involved using a circular saw and planing wood [18]. The carpenter noted that his nose became blocked within 15 minutes of cutting western red cedar, but he had no symptoms if he did not use the circular saw. Nasal challenge with coarse dust derived from sawing the cedar wood resulted in no response, but challenge with the fine dust that was obtained from planing or sanding the wood caused a significant isolated late increase in nasal airway resistance (6.5 hours after the challenge began). Acoustic rhinometry, a simple, easily reproducible, and less invasive procedure has been reported to be useful in diagnosing occupational rhinitis [24].

Disease prevention

Occupational rhinitis affords the physician an opportunity to practice preventive medicine, the most definitive approach to managing disease processes. One frequently suggested method is to screen applicants for jobs in which there is a possibility of workplace sensitization. By means of history and skin tests, it is possible to isolate potential workers who are basically atopic.

In a study performed on laboratory workers, screening applicants in such a manner proved to be an inefficient method of reducing the incidence of allergy to laboratory animals (Table 3). To exclude the 12 workers who developed asthma and rhinitis, 71 workers with positive skin test results would have to be rejected. If the 41 workers with positive skin test results and a positive allergic history were rejected, only 44% of those who went on to develop asthma and rhinitis caused by rats and only 23% those who only developed rhinitis would be rejected. A significant number of individuals with rat allergies were not initially atopic, and most atopic individuals did not become sensitive to rats [25].

A second approach to preventive medicine is to institute environmental control measures, such as providing adequate ventilation. The offending allergen and its source should be recognized. Laboratory workers who become sensitive to rats largely react to rat urinary protein rather than epithelium. It may be possible for the sensitive worker to perform surgery on rats if the cage containing the rats,

Table 3
Laboratory workers who developed rhinitis, asthma, or both

Screening method	Rhinitis/asthma	Rhinitis alone	No symptoms	Total
Atopic by history and skin test	8/18 (44%)	3/13 (23%)	30/148 (20%)	41/179 (23%)
Atopic by skin test	12/18 (66%)	5/13 (13%)	54/148 (36%)	71/179 (40%)

Data from Platts-Mills TA, Longbottom J, Edwares J, Cockroft A, Wilkins S. Occupational asthma and rhinitis related to laboratory rats: serum IgG and IgE antibodies to the rat urinary allergen. J Allergy Clin Immunol 1987;79:505–15.

which usually is soaked with urine, is kept out of the laboratory. Remarkable results can be obtained by placing high-efficiency particulate air filters in areas of the laboratory where rat exposure is high.

A good example of the positive effect of environmental control measures can be seen in the detergent industry. A *Bacillus subtilis* enzyme that was added to household detergents caused a significant amount of upper and lower respiratory allergies in workers. By instituting a number of precautions in the plant, the incidence of occupational respiratory disease was decreased dramatically. These measures included enclosing previously open operations, automating hand operations, supplying space suits with air supply to workers with high exposure, providing disposable gloves and uniforms, providing masks with cartridge and filters to workers with low exposure, monitoring the air (ie, measuring enzyme concentration in the atmosphere), and performing spot checks on workers. These spot checks consisted of taking a history, physical examination, chest radiographs, pulmonary function testing, skin testing, and serologic testing [26].

Pharmacologic management

Pharmacotherapy of occupational rhinitis is similar to that of other types of rhinitis [27]. Antihistamines are a mainstay of managing allergic and nonallergic rhinitis. When the patient's symptoms are frequent or unpredictable, regular use of an over-the-counter or prescription antihistamine may be necessary. When symptoms are episodic and predictable, prophylactic use of antihistamines before exposure to irritants or allergens is generally sufficient.

The choice of the agent often is based on trial and error, according to frequency of side effects and efficacy. The sedative side effects of conventional antihistamines may be more bothersome than the rhinitis itself, especially when the goal of treatment is to increase alertness and productivity.

The second-generation nonsedating antihistamines loratadine (Claritin), fexofenadine (Allegra), and ceterizine (Zyrtec) are better for treating patients who need to be awake and alert at work. The duration of treatment is variable. Because antihistamines are only palliative, they should be continued until the patient can control symptoms by avoiding exposure to the offending agent. Leukotriene modifiers have been shown to decrease nasal congestion, sneezing, and rhinorrhea [4], but these agents work best in conjunction with antihistamines or nasal steroids.

Topical agents are effective for managing rhinoconjunctivitis. Ophthalmic solutions of antihistamines, decongestants, mast cell stabilizers, and nonsteroidal anti-inflammatory agents provide effective, as-needed prophylaxis or symptomatic treatment of rhinoconjunctivitis. Saline nasal spray is useful for nonallergic rhinitis. The addition of an anticholinergic agent, such as ipratropium bromide, benefits patients who have anterior or posterior drainage. Topical glucocorticoid nasal sprays are effective for allergic and nonallergic rhinitis. They often need to be taken on a regular daily schedule but can be effective on an as-needed basis. The same rule applies to antihistamine nasal sprays.

Allergen immunotherapy generally is not recommended for patients with occupational rhinitis. Immunotherapy has no role in the management of rhinitis that is caused by irritative substances, and its value in alleviating allergic occupational rhinitis is limited by the unavailability of occupational antigens.

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