

CHEST[®]

Official publication of the American College of Chest Physicians



Cough: Occupational and Environmental Considerations: ACCP Evidence-Based Clinical Practice Guidelines

Susan M. Tarlo

Chest 2006;129:186-196
DOI 10.1378/chest.129.1_suppl.186S

The online version of this article, along with updated information and services can be found online on the World Wide Web at:
http://chestjournal.org/cgi/content/abstract/129/1_suppl/186S

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder (<http://www.chestjournal.org/misc/reprints.shtml>). ISSN: 0012-3692.

A M E R I C A N C O L L E G E O F
 C H E S T
P H Y S I C I A N S[®]

Cough: Occupational and Environmental Considerations

ACCP Evidence-Based Clinical Practice Guidelines

Susan M. Tarlo, MBBS, FCCP

Objectives: This section of the guideline aims to review the role of occupational and environmental factors in causing and contributing to cough. It also aims to indicate when such causes should be considered in a clinical setting, and a general approach to assessment and management.

Methods: A review was performed of published data between 1985 and 2004 using PubMed. The search terms used included “air pollution,” “sick building syndrome,” “occupational asthma,” “occupational lung disease,” “hypersensitivity pneumonitis” (HP), “cigarette smoke,” and “asthma.” Selected articles were chosen when meeting the objectives, but the extent of articles available and the limited space for this section does not permit a fully comprehensive review of all of these areas, for which the reader is referred to other sections of this clinical practice guideline, the published literature, textbooks of occupational lung disease, or more specific review articles.

Results/conclusions: Almost any patient presenting with cough may have an occupational or environmental cause of or contribution to their cough. The importance of this is that recognition and intervention may result in full or partial improvement of the cough, may limit the need for medication/symptomatic treatment, and may improve the long-term prognosis. Nonoccupational environmental contributing factors for upper and lower airway causes of cough include indoor irritant and allergenic agents such as cigarette smoke, cooking fumes, animals, dust mites, fungi, and cockroaches. Causes of HP indoors include birds and fungal antigens. Outdoor pollutants and allergens also contribute to upper and lower airway causes of cough. Occupational exposures can cause hypersensitivity responses leading to rhinitis and upper airway cough syndrome, previously referred to as *postnasal drip syndrome*, as well as asthma, HP, chronic beryllium disease, and hard metal disease, as well as irritant or toxic responses. The diagnosis is only reached by initially considering possible occupational and environmental factors, and by obtaining an appropriate medical history to determine relevant exposures, followed by objective investigations. This may require referral to a center of expertise. (CHEST 2006; 129:186S–196S)

Key words: air pollution; asthma; cough; occupational asthma; occupational lung disease; occupational rhinitis

Abbreviations: HP = hypersensitivity pneumonitis; OA = occupational asthma; RADS = reactive airways dysfunction syndrome

Occupational and environmental factors may be a cause of cough or may exacerbate cough that has been initially caused by other mechanisms. Therefore, occupation and environment should be considered as possible factors when evaluating every pa-

tient with cough. A detailed discussion of the pathogenesis, diagnosis, and management of every occupational and environmental contributing factor/disease is beyond the scope of this section, and more detail, when needed, can be obtained from other relevant sections of this clinical practice guideline or from more specialized publications. A review was performed of data published between 1985 and 2004 using PubMed. The search terms used included “air pollution,” “sick building syndrome,” “occupational asthma” (OA), “occupational lung disease,” “hyper-

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians (www.chestjournal.org/misc/reprints.shtml).

Correspondence to: Susan M. Tarlo, MBBS, FCCP, Toronto Western Hospital, EW7-449, 399 Bathurst St, Toronto, ON, M5T 2S8 Canada; e-mail susan.tarlo@utoronto.ca

sensitivity pneumonitis" (HP), "cigarette smoke," and "asthma." Selected articles were chosen when meeting the objectives.

NONOCCUPATIONAL ENVIRONMENTAL CAUSES OF COUGH

Pathogenesis

Exposures to allergens (*eg*, dust mite, animal or cockroach allergens, fungi and pollen) in the home, at school, in other indoor environments, or outdoors can trigger upper or lower respiratory causes of cough (*eg*, allergic rhinitis, asthma, allergic bronchopulmonary mycoses, or HP). Exposures to respiratory irritant agents, other than cigarette smoke, are generally less than would be encountered in occupational settings. Exposure to tobacco smoke should be assessed in all children and adults with cough. There is evidence of increased risk and severity of asthma in the children of parents who smoke,¹⁻⁴ and the airway irritant effects of tobacco smoke will aggravate asthma, both from personal smoking and the inhalation of second-hand smoke, in addition to the other risks associated with tobacco smoking⁵⁻⁷ such as chronic obstructive lung disease, lung cancer, and cardiac disease, all of which commonly include cough as a symptom. Exposure to indoor biomass, which is widely used for cooking or heating in several developing countries, has also been shown to be a significant risk factor for childhood and adult asthma.^{8,9} The term *hut lung* has been used for the finding of increased respiratory symptoms and lung disease associated with indoor particulate pollution from biomass combustion.¹⁰⁻¹³

Exposures to water-damaged homes or buildings have been reported to be associated with increased respiratory symptoms, including cough, which potentially may relate to dampness itself, or to associated exposures to dust mites, endotoxin, or fungal components such as glucans,¹⁴⁻¹⁶ and the reduction of bioaerosols such as by ultraviolet germicidal lights in buildings may reduce some symptoms.¹⁷ Mechanisms include IgE antibody-mediated responses to dust mites or fungi causing allergic rhinitis and upper airway cough syndrome, which has previously been referred to as *postnasal drip syndrome*, or asthma. Alternatively, there may be mucous membrane irritation from endotoxin or fungal glucans causing cough.^{18,19} Air pollutants such as nitrogen oxides from gas cooking stoves or outdoor traffic have been linked in some studies, particularly in children,²⁰ to increased respiratory symptoms including cough and increased symptoms from respiratory viral infections.^{21,22} Outdoor air pollutants such as ozone can cause cough and can increase airway inflammation,

triggering asthma symptoms.²³⁻²⁷ The response varies with individual susceptibility, but symptomatic effects are likely to be greater in those persons who have preexisting poorly controlled asthma. There is also the suggestion that chronic, relatively high outdoor ozone exposure may be associated with the increased development of childhood asthma.²³ Other outdoor pollutants may also be associated with increases in cough as a component of asthma or chronic bronchitis; an increase in the number of emergency department visits and hospital admissions, and in mortality rates for respiratory diseases have been associated with increases in acid aerosols and particulate air pollutants measured as the coefficient of haze, particulate air pollution of mass mean aerodynamic diameter $\leq 10 \mu\text{m}$, and particulate air pollution of mass mean aerodynamic diameter $\leq 2.5 \mu\text{m}$.^{25,28,29}

Unusual outdoor environmental exposures may trigger cough. "Epidemics" of emergency department visits for asthma have resulted from allergic responses to soya bean dusts from the unloading of soya beans in the harbors in Spain,^{30,31} to grass pollen fragments during thunderstorms in Britain,³² and to fungal spores in the midwest of North America.³³ Fungi can also potentially trigger cough through the induction of HP, as in Japanese summer-type HP, which has been linked to indoor exposure to *trichosporon cutaneum*.³⁴

Diagnosis

The identification of nonoccupational environmental causes or triggers of cough depends on a detailed exposure history. As with other causes of cough, the anatomic source of the cough needs to be identified by medical history, physical examination, and appropriate investigations as indicated from the medical history and physical examination findings. If an allergic mechanism is suspected, then skin-prick testing or *in vitro* tests for specific IgE antibodies, including allergen extracts that are suggested from the exposure history, can be helpful. Unfortunately, these tests are of limited value for some allergens, such as several fungal allergens, due to a degree of cross-reactivity between some species and poorly sensitive or specific skin test extracts, since there is variability in expressed allergens under different conditions of fungal growth and different substrates. Objective exposure assessment is also limited for fungi due to a lack of correlation between bulk sampling results and airborne exposures, and the lack of a standard interpretation of air-sampling results. When cough is diagnosed on the basis of an allergic respiratory response that can be objectively documented by abnormalities in pulmonary function

or chest radiography, then improvements that occur in these tests after removal from a specific environment or exposure may be helpful in further linking the respiratory disease to the environmental agent. Similarly, short-term deterioration in the results of these tests after specific exposure challenges to these agents either in the environmental setting or in specific controlled challenge tests would provide further diagnostic information. However, such objective confirmation is difficult to obtain in clinical practice for upper respiratory causes of cough and for cough triggered by respiratory irritants, and often the approach taken is to empirically suggest a trial of avoidance of the suspected agent/environment, if feasible. If the exposure is in the outdoor environment, such as during the unloading of soya beans,³⁵ then appropriate public health environmental changes may be needed. In the case of the soya bean unloading in Barcelona, the installation of appropriate filters prevented significant outdoor exposure to the soya bean dust. If avoidance is not practical, or if symptoms are considered to be due to mild upper or lower respiratory irritation, then pharmacologic management is an alternative approach as with non-environmentally triggered diseases.

OCCUPATIONAL CAUSES OF COUGH

Pathogenesis

Almost every nonoccupational respiratory disease has an occupational equivalent, which may not be identified unless a good occupational history is taken. Many patients with occupational lung diseases may present with a cough. Relatively common examples include OA (with a differential diagnosis including non-OA and work-related aggravation of asthma), HP, hard metal disease (*ie*, giant cell interstitial pneumonitis from cobalt) or asbestosis (with differential diagnoses including idiopathic pulmonary fibrosis), chronic beryllium lung disease (with a differential diagnosis including sarcoidosis), occupational bronchitis (with a differential diagnosis including nonoccupational bronchitis), and occupational lung cancer (with a differential diagnosis including nonoccupational causes of lung cancer). Relatively low workplace respiratory irritant exposures may induce cough on the basis of mucous membrane irritation as a presumed mechanism of cough associated with sick building syndrome (possibly related to components of bioaerosols such as endotoxins or fungal glucans) or from upper airway irritation secondary to chemicals such as solvents. In addition, workplace factors can induce or aggravate rhinitis and upper airway cough syndrome. The suggestion has also been made that some high-level occupa-

tional irritant exposures might also trigger gastroesophageal reflux as a cause of cough.³⁶ Finally, in some patients cough may follow an irritant exposure without any objective explanation; findings in one study³⁷ have suggested that there may be increased capsaicin sensitivity and cough without airway hyperresponsiveness in some cases after exposures that may be expected to irritate the airways.

OCCUPATIONAL RHINITIS

Pathogenesis and Diagnosis

Occupational rhinitis causing cough as a result of an upper airway cough syndrome is most commonly recognized as allergic occupational rhinitis^{38–40} with or without conjunctivitis, caused by an IgE antibody-mediated response to a workplace sensitizer, and often preceding OA.^{38,41,42} Rhinitis may also accompany OA due to low-molecular-weight chemical sensitizers at work, but it is difficult to diagnose in individual patients since the immunologic mechanism is less commonly IgE-mediated and specific tests to confirm causation are usually not possible to perform clinically. Common work exposures associated with occupational rhinitis include laboratory animal workers with allergic rhinitis from laboratory animal allergy,⁴³ or nonallergic rhinitis associated with endotoxin exposure; bakers with allergic rhinitis to wheat, egg, enzymes, or other high-molecular-weight allergens in the bakery;^{44,45} health-care workers exposed to natural rubber latex from powdered gloves,⁴⁶ enzyme workers,³⁹ and workers exposed to acid anhydrides,⁴⁷ acrylic compounds,⁴⁸ and diisocyanates.

OCCUPATIONAL AND WORK-RELATED ASTHMA

Prevalence and Pathogenesis

OA is now the most common chronic occupational lung disease in most developed areas and has been estimated to account for approximately 10% of all cases of adult-onset asthma from cross-sectional studies,⁴⁹ with up to 29% and 17%, respectively, attributable fraction among men and women in a Finnish population incidence study.⁵⁰ Cough is a common presenting symptom in OA, either alone or in combination with wheeze, chest tightness, and shortness of breath, as is the case with non-OA. OA is most commonly induced by sensitization to a workplace substance, either through an IgE antibody-mediated response or other presumed immunologic response, or, less commonly, by an acute high-level irritant exposure (*ie*, irritant-induced asthma), of which the most clear-cut example is reactive airways dysfunction syndrome (RADS).

Diagnosis

OA cannot be diagnosed without obtaining a thorough medical history. A key component of the history is a full occupational history, with special detail of the occupational exposures at the time of symptom onset, not only substances used by the patient in the workplace, but also substances used by coworkers that may become airborne. Details of the introduction of new materials shortly before the onset of symptoms, and a history of any accidental high-level exposures such as spills, may lead to the suspicion of OA. Other aspects of the medical history that lead to an increased suspicion of OA are a history of improved symptoms when away from work, such as weekends or holidays with recurrence or worsening on a return to the workplace. These historical features, while sensitive, are not specific for OA, however, and need to be assessed with objective investigations to confirm the diagnosis of asthma and its relationship to work.^{51,52} The initial investigations aim to confirm whether asthma is the cause of cough, as in cases of suspected asthma from nonoccupational causes, by means of spirometry before and after bronchodilator therapy, and if the spirometry findings are normal or there is no significant bronchodilator responsiveness, then histamine or methacholine challenge testing is helpful to identify the associated airway hyperresponsiveness. These tests need to be performed within 24 h of symptoms or within 24 h of the suspected causative job exposure, because they may become normal with longer periods away from exposure to a relevant workplace sensitizer and may result in a missed diagnosis in that event. If a diagnosis of asthma is confirmed (from the clinical history and pulmonary function responses), then it is necessary to objectively demonstrate whether a work relationship is present.

OA INDUCED BY SPECIFIC SENSITIZERS

Diagnosis

As has been detailed in some guidelines and reviews^{51,52} for OA caused by a specific sensitizer, the relationship of asthma to work may be determined by assessing the variability in airflow limitation in relationship to work by patient self-recorded serial peak expiratory flows or spirometric data (as well as respiratory medication use and symptom scores), which can then be interpreted in relation to workplace exposures. These are best recorded at least four times a day in triplicate, using either inexpensive peak flow meters, electronic hand-held peak flow devices, or spirometers. Recordings should be performed during periods of work weeks as well

as periods away from the suspected exposure for comparison, preferably at least 10 days away from the workplace exposure area (ideally when off work). The addition of a measure of airway responsiveness such as methacholine challenge tests performed near the end of a working exposure week (within 24 h of exposure),⁵¹ and for comparison at the end of a period away from exposure (ideally 2 to 3 weeks away from work) adds a further objective measure to assess airway changes related to work. A threefold improvement in the provocative concentration of methacholine causing a 20% fall in FEV₁ when away from the work exposure supports a diagnosis of OA if the results have not been confounded by other factors such as intercurrent upper or lower respiratory infection, or nonoccupational allergen exposure. If these tests cannot be performed or cannot be clearly interpreted, then specific laboratory exposure challenge tests can be helpful, although they are available in relatively few centers.⁵³ Alternatively, it is sometimes possible to perform a closely monitored "workplace challenge" when a technician supervises hourly spirometry during separate days, with the patient performing work in the suspected occupational setting and in an unexposed environment.⁵⁴ Immunologic tests, such as skin tests when the exposure at work has included high-molecular-weight allergens such as animal proteins or natural rubber latex, can provide further assistance with the diagnosis; however, these tests, although sensitive, are relatively nonspecific for OA, and the results can be positive in exposed workers who do not have a history of respiratory allergy up to the time of testing.^{38,55} For low-molecular-weight occupational sensitizers, skin testing is only useful to assess sensitization to a few agents such as complex platinum salts and, to a lesser extent, salts of other metals such as nickel and cobalt.⁵⁶⁻⁵⁹ Similarly, *in vitro* immunologic tests can be used to demonstrate the presence of specific IgE antibodies to a workplace sensitizer,⁶⁰⁻⁶³ but the use of such tests for low-molecular-weight sensitizers is limited by their reliable availability for relatively few agents, generally in research laboratories,⁶⁴⁻⁶⁹ and by antibody presence in only a minority of patients with OA as a result of exposure to the more common low-molecular-weight sensitizers such as diisocyanates (used in polyurethane products and spray paints)⁷⁰ and plicatic acid (in Western red cedar),⁷¹ in whom other immunologic mechanisms may also be important.⁷²⁻⁷⁵

Newer investigations, which have been reported in research studies, include the use of induced sputum to assess changes in sputum eosinophilic inflammation during periods at work vs periods off work. While this appears to add diagnostic accuracy to other investigations such as serial peak flow moni-

toring,⁷⁶ some cases of OA have been associated with neutrophilic inflammatory airway markers rather than eosinophilic inflammation.⁷⁷ Consequently, further investigation will be needed to clarify the interpretation of findings from induced sputum samples in the assessment of OA. The role of other newer investigations such as exhaled nitric oxide measurements and breath condensate analyses also needs further investigation to determine their possible roles in clinical evaluation of OA.^{78,79}

Management

Once a diagnosis is reached, the management of OA that is caused by a specific work sensitizer includes the avoidance of further exposure to that sensitizer and medical management of the asthma. Often, the patient will need to move to a different work environment or change jobs. Outcome is best with early diagnosis and early removal from exposure.

IRRITANT-INDUCED OA

Diagnosis

A diagnosis of OA related to an irritant exposure relies mainly on the documentation of an accidental high exposure to a respiratory irritant agent with the onset of asthma symptoms, persisting for at least 3 months and starting shortly after the irritant exposure, in a patient with no preceding evidence of asthma. An objective diagnosis of asthma after the exposure is reached by evidence of a significant airway bronchodilator response or positive methacholine challenge result. Patients who meet the relatively stringent criteria for RADS, as described by Brooks et al,⁸⁰ most certainly will have a diagnosis of irritant-induced asthma. Other reports^{81–83} have modified these criteria to include less massive exposures, less persistent symptoms, or greater delays in the onset of symptoms after exposures. Such an expansion of the criteria of Brook et al⁸⁰ may represent true irritant-induced asthma, and increases the frequency of diagnosis but decreases the certainty of a true diagnosis.⁸³

The irritant exposure leading to RADS or irritant-induced asthma has most frequently been reported indoors in enclosed spaces with high fume exposures. However, high outdoor exposures to alkaline dust (largely related to calcium oxide) from the collapse of the World Trade Center were associated with occupational airway irritant responses, leading to “World Trade Center cough” in firefighters and other occupationally exposed groups.³⁶ There was an increase in airway responsiveness associated with this

exposure,⁸⁴ which was reported to have a later onset after exposure than the 24 h used in the criteria Brooks et al.⁸⁰ The findings of cough and increased airway responsiveness were likely based on the presence of irritant-induced asthma (or RADS), but also in some exposed workers cough was associated with increases in symptoms of rhinitis and gastroesophageal reflux, so the causes of cough may have been multifactorial. Although the New York firefighters underwent preemployment medical assessments and periodic medical surveillance to exclude asthma among the active workers, there were also reports of increased symptoms among asthmatic lower Manhattan residents following the collapse of the World Trade Center that may have resulted from an irritant aggravation of underlying asthma, although, as with the firefighters, other mechanisms may also have played a role.

MANAGEMENT OF IRRITANT-INDUCED ASTHMA

Those with Irritant-induced asthma may be able to stay in the same workplace with appropriate asthma pharmacotherapy and environmental control measures if provisions are made to prevent further high-level irritant exposures.

WORK-RELATED AGGRAVATION OF ASTHMA

In addition to the induction of new-onset asthma, occupational exposures can aggravate preexisting asthma, particularly workplace exposures to dusts, fumes, and sprays, which trigger bronchoconstriction in workers with hyperreactive airways. A similar response can occur in asthmatic patients who at work are exposed to cold dry air or put forth strenuous exertion, and these effects are likely to be greatest in those with poor pharmacologic control of their asthma or in workers with severe airway hyperresponsiveness. A diagnosis of true OA needs to be excluded in such patients by the means discussed above, and management of the condition consists of optimizing environmental control measures and pharmacotherapy, as well as controlling personal workplace exposures to respiratory irritants and triggers (*eg*, with the use of respiratory protection for short-term potential irritant exposures, and the institution of appropriate ventilation and containment measures for respiratory irritants at work).

OCCUPATIONAL EOSINOPHILIC BRONCHITIS

Occupationally induced eosinophilic airway inflammation can result in cough with or without

sputum production but without other physiologic changes of asthma (*ie*, an absence of airflow limitation and bronchial hyperresponsiveness). This can be induced by the same workplace sensitizers, which can cause OA, and is a presumed immunologic response. Diagnosis relies on the demonstration of significant eosinophilia in sputum samples, usually obtained by sputum induction, and the presence of $\geq 3\%$ eosinophils in sputum samples obtained at the end of a working week, with reduction in the percentage of eosinophils in sputum during periods off work, has been suggested as the criterion for diagnosis.⁸⁵ The same management regimen as that for OA from a sensitizer includes the avoidance of exposure to the causative work agent and the use of therapy with inhaled steroids.

HP

Cough is a common symptom of HP in addition to dyspnea, chills, and fever. HP can be caused by a hypersensitivity response to medications, to inhaled environmental high-molecular-weight antigens, or to certain chemicals. The range of inhaled agents is large, including antigens from fungi,^{86–88} mycobacteria,⁸⁹ thermophilic actinomycetes (bacteria),⁹⁰ parasites, and birds.⁹¹ Nonoccupational antigenic causes, as covered in a separate section of this guideline, include indoor fungi for Japanese summer HP³⁴ and indoor microbial contamination, including contamination of humidifiers and vaporizers,⁹² hot tubs (including mycobacterial contamination),⁸⁹ and swimming pool areas,⁹³ in office settings or homes.

Occupational causes of HP include organic dust exposures, as in farmers exposed to “moldy” hay or to chickens, turkeys, or other birds, or in lifeguards or office workers exposed to water contaminated by microorganisms. Chemical exposures that can trigger HP include diisocyanates, most commonly diphenylmethane-diisocyanate.⁹⁴ Metal-working fluid (coolant) contaminated by microorganisms is also a relatively commonly reported cause of HP in the industrialized workplace.⁹⁵ Key aspects of the diagnosis, as with the diagnosis of OA, include an initial suspicion of an extrinsic cause for any patient with interstitial lung disease, careful history-taking for exposure to birds, areas that may generate fungal or other bioaerosol exposures (*eg*, barns, contaminated homes, offices, hot tubs, swimming pool areas, or other workplace exposures), and potential work exposure to chemicals that can cause this response. Suspicion is further increased if respiratory findings are intermittent in relation to these exposures and clear up without pharmacologic intervention after a few days away from a particular area. BAL findings

of predominant lymphocytosis in a patient with apparent interstitial lung disease and findings of giant cell granulomas on lung biopsy specimens also raise suspicion of an extrinsic cause. The demonstration of specific serum IgG antibodies to the suspected agent further supports the diagnosis, although the standardization of antigen extracts is difficult in some cases. Pulmonary function tests can show restrictive changes with reduced diffusing capacity apparent especially on exercise, but chronic airflow limitation may predominate in patients with chronic HP with bronchiolitis. In patients with the acute form of HP, chest radiograph findings may mimic pneumonia, and in patients with the chronic form of HP the findings may resemble idiopathic pulmonary fibrosis. A high-resolution CT scan showing a ground-glass appearance may be helpful in distinguishing the chronic form of HP.^{96,97} Occasionally, a specific challenge may be needed for medico-legal purposes to confirm the diagnosis, especially if the findings of radiographic and other investigations are nonspecific.

OTHER OCCUPATIONAL CAUSES OF INTERSTITIAL LUNG DISEASE

Patients with hard metal disease (*ie*, giant cell interstitial pneumonitis from cobalt) or asbestosis (with differential diagnoses including idiopathic pulmonary fibrosis) also may initially present with a dry cough. As with the previous occupational diseases, the work history is a key component in making the diagnosis, but the patient may not be aware of all occupational exposures, and the physician needs to be aware of the types of work in which such exposures may occur and obtain details from a review of material safety data sheets and any occupational hygiene reports that may be obtained from the workplace. Early identification and removal of the patient from further exposure are key components of treatment.

CHRONIC BERYLLIUM LUNG DISEASE

Beryllium is being used more widely than in the traditional uses in the aerospace industries and nuclear power plant facilities. Current uses include the manufacture of materials from alloys containing beryllium for the production of pen clips, golf clubs, and other products. The presenting symptoms can include cough and dyspnea with chest radiographic findings that are identical to those for sarcoidosis.⁹⁸ Thus, chronic beryllium lung disease should be included in the differential diagnosis of sarcoidosis,

and an overt or covert exposure to beryllium should be carefully assessed from a medical history supplemented, where appropriate, by beryllium lymphocyte proliferation tests.⁹⁹

OTHER OCCUPATIONAL LUNG DISEASES WITH COUGH

Other occupational lung diseases in patients who may present with cough include occupational bronchitis (with a differential diagnosis including nonoccupational bronchitis) and occupational lung cancer (with a differential diagnosis including nonoccupational causes of lung cancer). The workplace has been estimated to account for approximately 15% of the burden of chronic obstructive lung disease (a mean estimate from several studies).¹⁰⁰ Exposure assessment is necessary in addition to information as to other known contributing factors to assist in estimating the probability of occupational contributions to these diseases.

COUGH DUE TO MUCOUS MEMBRANE IRRITATION BY LOW-LEVEL RESPIRATORY IRRITANTS

Cough and other asthma-like symptoms are a relatively common component of sick building syndrome, which has been reported most often in sealed office buildings.^{14–16} Associated factors have included psychosocial stress, poor building maintenance, complaints as to temperature or humidity control, and volatile organic compound exposures. Several studies have shown an association with airborne endotoxin or fungal contamination in buildings, and one study¹⁷ showed a significant improvement in symptoms with blinded use of biocidal ultraviolet radiation in a crossover study. A similar syndrome, darkroom disease, occurring in radiograph technologists, has also been associated with greater self-reporting of work conditions that would be expected to be associated with low-level irritant exposures, as well as with psychosocial stressors.¹⁰¹

COUGH DUE TO ORGANIC DUST TOXIC SYNDROME

This syndrome has been described mainly in agricultural settings and may be confused initially with HP because the symptoms are similar. Cough may be a prominent feature, with chest tightness, fever, and malaise starting 4 to 8 h after exposure to contaminated grain dust and usually lasting 36 to 48 h before clearing. Although some exposed workers can have airway hyperresponsiveness, and more

typical features of asthma or chronic bronchitis, the acute symptoms are more similar to humidifier fever, polymer fume fever, and metal fume fever. Unlike HP, the chest radiograph and pulmonary function test findings are usually normal, and BAL fluid samples show mainly neutrophils. The cause and mechanisms are not fully understood, but in some cases it has been suggested to relate to the contamination of grain or other organic dust by fungi or endotoxins, triggering neutrophil activation and the release of cytokines. More chronic airway inflammatory effects can also occur from these exposures, with chronic cough and sputum, as can more typical findings of asthma.^{102–104}

FINDINGS FROM HISTORY THAT MAY LEAD TO SUSPICION OF AN OCCUPATIONAL OR ENVIRONMENTAL CAUSE OF COUGH

As noted from the above review, almost any cause of cough may have an occupational or environmental cause or contribution. Therefore, a review of the patient's occupational and environmental exposures is needed in all patients with cough. The failure to identify and correct an occupational or environmental cause or contribution to cough will lead to an increased need for the pharmacologic management of disease, and may lead to progressive disease despite the use of medications to treat the condition. The most common potentially modifiable nonoccupational environmental causes/triggers are tobacco smoke for children and adults, and indoor allergens, such as from cats or other animals, and dust mites. Exposure to these and an assessment of their relevance in cough should be specifically determined for all patients with rhinitis, asthma, and COPD, while other antigenic triggers such as birds and contaminated humidifiers should be considered for all who may have HP.

Occupational exposures should be considered specifically for all patients with rhinitis, asthma, or HP whose symptoms start during their working life. For those with potential diseases of longer latency, such as chronic beryllium disease and hard metal disease or chronic HP, the medical history should specifically include all previous occupational exposures.

RESEARCH NEEDS

The outcome of many occupational causes of cough improves with an early diagnosis and a change in occupational exposure. Research is needed to determine effective strategies to enable the early recognition of occupational causes and contributions

to cough. The role of submassive respiratory irritant exposures needs to be further understood and criteria developed for a diagnosis of these effects. Interactions between occupational sensitizers and irritants also need to be better understood to minimize morbidity from such exposures. Host susceptibility factors need clarification and may eventually permit modification. Methods for identifying new potential respiratory sensitizers prior to their use in the workplace need to be developed and alternative nonsensitizing materials need to be developed. Better exposure assessment is also needed to understand details of the exposures leading to many of the occupational and environmental causes of cough.

SUMMARY OF RECOMMENDATIONS

1. In every patient with cough, when taking a medical history, ask about occupational and environmental causes. Level of evidence, expert opinion; benefit, substantial; grade of recommendations, E/A

2. In every patient with cough who has potentially significant exposures to suspicious environmental or occupational causes, determine the relationship of these occupational and environmental factors to confirm or refute their role in cough and to modify or eliminate exposure to the relevant agents. Level of evidence, expert opinion; benefit, substantial; grade of recommendations, E/A

3. Because outdoor environmental pollution and occupational exposures can be important factors in causing cough, physicians should play a role in developing and supporting enforceable standards for safe workplace and outdoor air pollution exposure limits. Level of evidence, expert opinion; benefit, substantial; grade of recommendations, E/A

4. In patients with a high suspicion of cough due to environmental or occupational exposures, consider referring the patient to a specialist in this area or consult evidence-based guidelines. Level of evidence, expert opinion; net benefit, substantial; grade of recommendation, E/A

REFERENCES

1 King ME, Mannino DM, Holguin F. Risk factors for asthma incidence: a review of recent prospective evidence. *Panminerva Med* 2004; 46:97-110

- 2 Mannino DM, Homa DM, Redd SC. Involuntary smoking and asthma severity in children: data from the Third National Health and Nutrition Examination Survey. *Chest* 2002; 122:409-415
- 3 Mannino DM, Moorman JE, Kingsley B, et al. Health effects related to environmental tobacco smoke exposure in children in the United States: data from the Third National Health and Nutrition Examination Survey. *Arch Pediatr Adolesc Med* 2001; 155:36-41
- 4 Wilson SR, Yamada EG, Sudhakar R, et al. A controlled trial of an environmental tobacco smoke reduction intervention in low-income children with asthma. *Chest* 2001; 120:1709-1722
- 5 Mannino DM, Ford E, Giovino GA, et al. Lung cancer mortality rates in birth cohorts in the United States from 1960 to 1994. *Lung Cancer* 2001; 31:91-99
- 6 Mannino DM. Chronic obstructive pulmonary disease: definition and epidemiology. *Respir Care* 2003; 48:1185-1191
- 7 Mannino DM. COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. *Chest* 2002; 121: 121S-126S
- 8 Mishra V. Effect of indoor air pollution from biomass combustion on prevalence of asthma in the elderly. *Environ Health Perspect* 2003; 111:71-78
- 9 Schei MA, Hessen JO, Smith KR, et al. Childhood asthma and indoor woodsmoke from cooking in Guatemala. *J Expo Anal Environ Epidemiol* 2004; 14(suppl):S110-S117
- 10 Gold JA, Jagirdar J, Hay JG, et al. Hut lung: a domestically acquired particulate lung disease. *Medicine* 2000; 79:310-317
- 11 Kara M, Bulut S, Tas F, et al. Evaluation of pulmonary changes due to biomass fuels using high-resolution computed tomography. *Eur Radiol* 2003; 10:2372-2377
- 12 Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991; 100:385-388
- 13 Mishra V. Indoor air pollution from biomass combustion and acute respiratory illness in preschool age children in Zimbabwe. *Int J Epidemiol* 2003; 32:847-853
- 14 Weltermann BM, Hodgson M, Storey E, et al. Hypersensitivity pneumonitis: a sentinel event investigation in a wet building. *Am J Ind Med* 1998; 34:499-505
- 15 Jarvis JQ, Morey PR. Allergic respiratory disease and fungal remediation in a building in a subtropical climate. *Appl Occup Environ Hyg* 2001; 16:380-388
- 16 Redlich CA, Sparer J, Cullen MR. Sick-building syndrome. *Lancet* 1997; 349:1013-1016
- 17 Menzies D, Popa J, Hanley JA, et al. Effect of ultraviolet germicidal lights installed in office ventilation systems on workers' health and wellbeing: double-blind multiple crossover trial 1. *Lancet* 2003; 362:1785-1791
- 18 Teeuw KB, Vandenbroucke-Grauls CM, Verhoef J. Airborne gram-negative bacteria and endotoxin in sick building syndrome: a study in Dutch governmental office buildings. *Arch Intern Med* 1994; 154:2339-2345
- 19 Wan GH, Li CS. Indoor endotoxin and glucan in association with airway inflammation and systemic symptoms. *Arch Environ Health* 1999; 54:172-179
- 20 Lin M, Chen Y, Villeneuve PJ, et al. Gaseous air pollutants and asthma hospitalization of children with low household income in Vancouver, British Columbia, Canada. *Am J Epidemiol* 2004; 159:294-303
- 21 Barnes PJ. Air pollution and asthma. *Postgrad Med* 1994; 70:319-325
- 22 Chauhan AJ, Inskip HM, Linaker CH, et al. Personal exposure to nitrogen dioxide (NO₂) and the severity of

- virus-induced asthma in children. *Lancet* 2003; 361:1939–1944
- 23 McConnell R, Berhane K, Gilliland F, et al. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 2002; 359:386–391
 - 24 Linn WS, Gong H Jr. The 21st century environment and air quality influences on asthma. *Curr Opin Pulm Med* 1999; 5:21–26
 - 25 Peden DB. Pollutants and asthma: role of air toxics. *Environ Health Perspect* 2002; 110(suppl):565–568
 - 26 Romieu I, Sienra-Monge JJ, Ramirez-Aguilar M, et al. Genetic polymorphism of GSTM1 and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City. *Thorax* 2004; 59:8–10
 - 27 Schwartz J. Air pollution and children's health. *Pediatrics* 2004; 113:1037–1043
 - 28 Verones B, Oortgiesen M. Neurogenic inflammation and particulate matter (PM) air pollutants. *Neurotoxicology* 2001; 22:795–810
 - 29 Ward DJ, Ayres JG. Particulate air pollution and panel studies in children: a systematic review. *Occup Environ Med* 2004; 61:e13
 - 30 Anto JM, Sunyer J, Rodriguez-Roisin R, et al. Community outbreaks of asthma associated with inhalation of soybean dust: Toxicoepidemiological Committee. *N Engl J Med* 1989; 320:1097–1102
 - 31 Anto JM, Sunyer J, Reed CE, et al. Preventing asthma epidemics due to soybeans by dust-control measures. *N Engl J Med* 1993; 329:1760–1763
 - 32 Davidson AC, Emberlin J, Cook AD, et al. A major outbreak of asthma associated with a thunderstorm: experience of accident and emergency departments and patients' characteristics; Thames Regions Accident and Emergency Trainees Association. *BMJ* 1996; 312:601–604
 - 33 O'Hollaren MT, Yunginger JW, Offord KP, et al. Exposure to an aeroallergen as a possible precipitating factor in respiratory arrest in young patients with asthma. *N Engl J Med* 1991; 324:359–363
 - 34 Yoshida K, Ando M, Sakata T, et al. Prevention of summer-type hypersensitivity pneumonitis: effect of elimination of *Trichosporon cutaneum* from the patients' homes. *Arch Environ Health* 1989; 44:317–322
 - 35 Anto JM, Sunyer J, Newman Taylor AJ. Comparison of soybean epidemic asthma and occupational asthma. *Thorax* 1996; 51:743–749
 - 36 Prezant DJ, Weiden M, Banauch GI, et al. Cough and bronchial responsiveness in firefighters at the World Trade Center site. *N Engl J Med* 2002; 347:806–815
 - 37 Gordon SB, Curran AD, Turley A, et al. Glass bottle workers exposed to low-dose irritant fumes cough but do not wheeze. *Am J Respir Crit Care Med* 1997; 156:206–210
 - 38 Archambault S, Malo JL, Infante-Rivard C, et al. Incidence of sensitization, symptoms, and probable occupational rhinoconjunctivitis and asthma in apprentices starting exposure to latex. *J Allergy Clin Immunol* 2001; 107:921–923
 - 39 Sarlo K, Kirchner DB. Occupational asthma and allergy in the detergent industry: new developments. *Curr Opin Allergy Clin Immunol* 2002; 2:97–101
 - 40 Platts-Mills TA, Longbottom J, Edwards J, et al. Asthma and rhinitis related to laboratory rats: use of a purified rat urinary allergen to study exposure in laboratories and the human immune response. *N Engl Reg Allergy Proc* 1987; 8:245–251
 - 41 Malo JL, Lemiere C, Desjardins A, et al. Prevalence and intensity of rhinoconjunctivitis in subjects with occupational asthma. *Eur Respir J* 1997; 10:1513–1515
 - 42 Karjalainen A, Martikainen R, Klaukka T, et al. Risk of asthma among Finnish patients with occupational rhinitis. *Chest* 2003; 123:283–288
 - 43 Draper A, Newman TA, Cullinan P. Estimating the incidence of occupational asthma and rhinitis from laboratory animal allergens in the UK, 1999–2000. *Occup Environ Med* 2003; 60:604–605
 - 44 Smith TA, Parker G, Hussain T. Respiratory symptoms and wheat flour exposure: a study of flour millers. *Occup Med (Lond)* 2000; 50:25–29
 - 45 Brisman J, Belin L. Clinical and immunological responses to occupational exposure to alpha-amylase in the baking industry. *Br J Ind Med* 1991; 48:604–608
 - 46 Tarlo SM, Sussman GL, Holness DL. Latex sensitivity in dental students and staff: a cross-sectional study. *J Allergy Clin Immunol* 1997; 99:396–401
 - 47 Grammer LC, Ditto AM, Tripathi A, et al. Prevalence and onset of rhinitis and conjunctivitis in subjects with occupational asthma caused by trimellitic anhydride (TMA). *J Occup Environ Med* 2002; 44:1179–1181
 - 48 Lindstrom M, Alanko K, Keskinen H, et al. Dentist's occupational asthma, rhinoconjunctivitis, and allergic contact dermatitis from methacrylates. *Allergy* 2002; 57:543–545
 - 49 Blanc PD, Toren K. How much adult asthma can be attributed to occupational factors? *Am J Med* 1999; 107:580–587
 - 50 Karjalainen A, Kurppa K, Martikainen R, et al. Work is related to a substantial portion of adult-onset asthma incidence in the Finnish population. *Am J Respir Crit Care Med* 2001; 164:565–568
 - 51 Tarlo SM, Boulet LP, Cartier A, et al. Canadian Thoracic Society guidelines for occupational asthma. *Can Respir J* 1998; 5:289–300
 - 52 Chan-Yeung M, Malo JL, Tarlo SM, et al. Proceedings of the first Jack Pepys Occupational Asthma Symposium. *Am J Respir Crit Care Med* 2003; 167:450–471
 - 53 Ortega HG, Weissman DN, Carter DL, et al. Use of specific inhalation challenge in the evaluation of workers at risk for occupational asthma: a survey of pulmonary, allergy, and occupational medicine residency training programs in the United States and Canada. *Chest* 2002; 121:1323–1328
 - 54 Chan-Yeung M, McMurren T, Catonio-Begley F, et al. Occupational asthma in a technologist exposed to glutaraldehyde. *J Allergy Clin Immunol* 1993; 91:974–978
 - 55 Tarlo SM, Wong L, Roos J, et al. Occupational asthma caused by latex in a surgical glove manufacturing plant. *J Allergy Clin Immunol* 1990; 85:626–631
 - 56 Merget R, Schultze-Werninghaus G, Bode F, et al. Quantitative skin prick and bronchial provocation tests with platinum salt. *Br J Ind Med* 1991; 48:830–837
 - 57 Merget R, Caspari C, Dierkes-Globisch A, et al. Effectiveness of a medical surveillance program for the prevention of occupational asthma caused by platinum salts: a nested case-control study. *J Allergy Clin Immunol* 2001; 107:707–712
 - 58 Dolovich J, Evans SL, Nieboer E. Occupational asthma from nickel sensitivity: I. Human serum albumin in the antigenic determinant. *Br J Ind Med* 1984; 41:51–55
 - 59 Kusaka Y, Yokoyama K, Sera Y, et al. Respiratory diseases in hard metal workers: an occupational hygiene study in a factory. *Br J Ind Med* 1986; 43:474–485
 - 60 Platts-Mills TA, Longbottom J, Edwards J, et al. Occupational asthma and rhinitis related to laboratory rats: serum IgG and IgE antibodies to the rat urinary allergen. *J Allergy Clin Immunol* 1987; 79:505–515
 - 61 Kim YK, Oh SY, Jung JW, et al. IgE binding components in

- Tetranychus urticae* and *Panonychus ulmi*-derived crude extracts and their cross-reactivity with domestic mites. *Clin Exp Allergy* 2001; 31:1457–1463
- 62 Desjardins A, Malo JL, L'Archeveque J, et al. Occupational IgE-mediated sensitization and asthma caused by clam and shrimp. *J Allergy Clin Immunol* 1995; 96:608–617
 - 63 Bardy JD, Malo JL, Seguin P, et al. Occupational asthma and IgE sensitization in a pharmaceutical company processing psyllium. *Am Rev Respir Dis* 1987; 135:1033–1038
 - 64 Shimoda T. Detection of IgE antibodies specific to isonicotinic acid hydrazide and its metabolite by enzyme-linked immunosorbent assay and the mechanism of sensitization by inhalation or ingestion of this compound. *Arerugi* 1990; 39:567–576
 - 65 Park JW, Kang DB, Choi SY, et al. Heterogeneity of IgE epitopes of vinyl sulphone reactive dye: human serum albumin that react with IgE. *Clin Exp Allergy* 2001; 31:1779–1786
 - 66 Park JW, Kim CW, Kim KS, et al. Role of skin prick test and serological measurement of specific IgE in the diagnosis of occupational asthma resulting from exposure to vinyl sulphone reactive dyes. *Occup Environ Med* 2001; 58:411–416
 - 67 Kramps JA, van Toorenebergen AW, Vooren PH, et al. Occupational asthma due to inhalation of chloramine-T: II. Demonstration of specific IgE antibodies. *Int Arch Allergy Appl Immunol* 1981; 64:428–438
 - 68 Howe W, Venables KM, Topping MD, et al. Tetrachlorophthalic anhydride asthma: evidence for specific IgE antibody. *J Allergy Clin Immunol* 1983; 71:5–11
 - 69 Bernstein DI, Zeiss CR, Wolkonsky P, et al. The relationship of total serum IgE and blocking antibody in trimellitic anhydride-induced occupational asthma. *J Allergy Clin Immunol* 1983; 72:714–719
 - 70 Son M, Lee M, Kim YT, et al. Heterogeneity of IgE response to TDI-HSA conjugates by ELISA in toluene diisocyanate (TDI)-induced occupational asthma (OA) patients. *J Korean Med Sci* 1998; 13:147–152
 - 71 Tse KS, Chan H, Chan-Yeung M. Specific IgE antibodies in workers with occupational asthma due to western red cedar. *Clin Allergy* 1982; 12:249–258
 - 72 Frew A, Chang JH, Chan H, et al. T-lymphocyte responses to plicatic acid-human serum albumin conjugate in occupational asthma caused by western red cedar. *J Allergy Clin Immunol* 1998; 101:841–847
 - 73 Frew A, Chan H, Salari H, et al. Is tyrosine kinase activation involved in basophil histamine release in asthma due to western red cedar? *Allergy* 1998; 53:139–143
 - 74 Chan-Yeung M. Mechanism of occupational asthma due to western red cedar (*Thuja plicata*). *Am J Ind Med* 1994; 25:13–18
 - 75 Bernstein DI, Cartier A, Cote J, et al. Diisocyanate antigen-stimulated monocyte chemoattractant protein-1 synthesis has greater test efficiency than specific antibodies for identification of diisocyanate asthma 2. *Am J Respir Crit Care Med* 2002; 166:445–450
 - 76 Girard F, Côté J, Boulet LP, et al. An effective strategy for diagnosing occupational asthma: use of induced sputum. *Am J Respir Crit Care Med* 2004; 170:845–850
 - 77 Magni K, Lemiere C, Ghezzi H, et al. Airway inflammation after cessation of exposure to agents causing occupational asthma. *Am J Respir Crit Care Med* 2004; 169:367–372
 - 78 Lemiere C. Non-invasive monitoring of airway inflammation in occupational lung diseases. *Curr Opin Allergy Clin Immunol* 2002; 2:109–114
 - 79 Rahman I, Kelly F. Biomarkers in breath condensate: a promising new non-invasive technique in free radical research. *Free Radic Res* 2003; 37:1253–1266
 - 80 Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposures. *Chest* 1985; 88:376–384
 - 81 Tarlo SM, Broder I. Irritant-induced occupational asthma. *Chest* 1989; 96:297–300
 - 82 Brooks SM, Hammad Y, Richards I, et al. The spectrum of irritant-induced asthma: sudden and not-so-sudden onset and the role of allergy. *Chest* 1998; 113:42–49
 - 83 Tarlo SM. Workplace irritant exposures: do they produce true occupational asthma? *Ann Allergy Asthma Immunol* 2003; 90:19–23
 - 84 Banauch GI, Alleyne D, Sanchez R, et al. Persistent hyper-reactivity and reactive airway dysfunction in firefighters at the World Trade Center. *Am J Respir Crit Care Med* 2003; 168:54–62
 - 85 Quirce S. Eosinophilic bronchitis in the workplace. *Curr Opin Allergy Clin Immunol* 2004; 4:87–91
 - 86 Moreno-Ancillo A, Padial MA, Lopez-Serrano MC, et al. Hypersensitivity pneumonitis due to inhalation of fungi-contaminated esparto dust in a plaster worker. *Allergy Asthma Proc* 1997; 18:355–357
 - 87 Woodard ED, Friedlander B, Leshner RJ, et al. Outbreak of hypersensitivity pneumonitis in an industrial setting. *JAMA* 1988; 259:1965–1969
 - 88 Cormier Y, Israel-Assayag E, Bedard G, et al. Hypersensitivity pneumonitis in peat moss processing plant workers. *Am J Respir Crit Care Med* 1998; 158:412–417
 - 89 Rickman OB, Ryu JH, Fidler ME, et al. Hypersensitivity pneumonitis associated with *Mycobacterium avium* complex and hot tub use. *Mayo Clin Proc* 2002; 77:1233–1237
 - 90 Duchaine C, Meriaux A, Brochu G, et al. *Saccharopolyspora rectivirgula* from Quebec dairy barns: application of simplified criteria for the identification of an agent responsible for farmer's lung disease. *J Med Microbiol* 1999; 48:173–180
 - 91 Zacharisen MC, Schlueter DP, Kurup VP, et al. The long-term outcome in acute, subacute, and chronic forms of pigeon breeder's disease hypersensitivity pneumonitis. *Ann Allergy Asthma Immunol* 2002; 88:175–182
 - 92 Ganier M, Lieberman P, Fink J, et al. Humidifier lung: an outbreak in office workers. *Chest* 1980; 77:183–187
 - 93 Moreno-Ancillo A, Vicente J, Gomez L, et al. Hypersensitivity pneumonitis related to a covered and heated swimming pool environment. *Int Arch Allergy Immunol* 1997; 114:205–206
 - 94 Baur X. Hypersensitivity pneumonitis (extrinsic allergic alveolitis) induced by isocyanates. *J Allergy Clin Immunol* 1995; 95:1004–1010
 - 95 Fox J, Anderson H, Moen T, et al. Metal working fluid-associated hypersensitivity pneumonitis: an outbreak investigation and case-control study. *Am J Ind Med* 1999; 35:58–67
 - 96 Lacasse Y, Selman M, Costabel U, et al. Clinical diagnosis of hypersensitivity pneumonitis. *Am J Respir Crit Care Med* 2003; 168:952–958
 - 97 Glazer CS, Rose CS, Lynch DA. Clinical and radiologic manifestations of hypersensitivity pneumonitis. *J Thorac Imaging* 2002; 17:261–272
 - 98 Newman LS, Lloyd J, Daniloff E. The natural history of beryllium sensitization and chronic beryllium disease. *Environ Health Perspect* 1996; 104(suppl):937–943
 - 99 Newman LS, Mroz MM, Maier LA, et al. Efficacy of serial medical surveillance for chronic beryllium disease in a beryllium machining plant. *J Occup Environ Med* 2001; 43:231–237
 - 100 American Thoracic Society Statement. Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003; 167:787–797

- 101 Tarlo SM, Liss GM, Greene JM, et al. Work-attributed symptom clusters (darkroom disease) among radiographers versus physiotherapists: associations between self-reported exposures and psychosocial stressors. *Am J Ind Med* 2004; 45:513–521
- 102 Von Essen S, Fryzek J, Nowakowski B, et al. Respiratory symptoms and farming practices in farmers associated with an acute febrile illness after organic dust exposure. *Chest* 1999; 116:1452–1458
- 103 Monso E, Magarolas R, Radon K, et al. Respiratory symptoms of obstructive lung disease in European crop farmers. *Am J Respir Crit Care Med* 2000; 162:1246–1250
- 104 Spurzem JR, Romberger DJ, Von Essen SG. Agricultural lung diseases. *Clin Chest Med* 2002; 23:795–810

**Cough: Occupational and Environmental Considerations: ACCP
Evidence-Based Clinical Practice Guidelines**

Susan M. Tarlo

Chest 2006;129;186-196

DOI 10.1378/chest.129.1_suppl.186S

This information is current as of February 18, 2008

Updated Information & Services	Updated information and services, including high-resolution figures, can be found at: http://chestjournal.org/cgi/content/full/129/1_suppl/186S
References	This article cites 100 articles, 40 of which you can access for free at: http://chestjournal.org/cgi/content/full/129/1_suppl/186S#BIBL
Citations	This article has been cited by 2 HighWire-hosted articles: http://chestjournal.org/cgi/content/full/129/1_suppl/186S
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://chestjournal.org/misc/reprints.shtml
Reprints	Information about ordering reprints can be found online: http://chestjournal.org/misc/reprints.shtml
Email alerting service	Receive free email alerts when new articles cite this article sign up in the box at the top right corner of the online article.
Images in PowerPoint format	Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S[®]