

Biological contaminants

James M. Seltzer, MD San Diego, California

"The potential economic impact of indoor air pollution is quite high and is estimated in the tens of billions of dollars per year. Such impacts includes direct medical costs and lost earnings due to major illness as well as increased employee sick days and lost productivity." The health consequences of biological contamination range from uncomfortable odors to end-stage lung disease to fatality. What factors predispose to biologic contamination? Which contaminants cause adverse health effects? What illnesses are associated with which contaminants, and how do these occur? This discussion will help answer these questions, leaving the discussion of environmental sampling, interpretation of sampling results, and remediation to other speakers who presented this information. A few interesting ideas for consideration regarding the implications of biological contamination will also be presented. (J ALLERGY CLIN IMMUNOL 1994;94:318-26.)

Key words: Biological contaminants, fungus, bacteria, virus, allergen, hypersensitivity pneumonitis, asthma, allergic rhinitis, humidifier fever, mycotoxin

The U.S. Environmental Protection Agency in its 1989 Report to Congress on Indoor Air Quality stated that "biological contaminants are an important dimension of indoor air quality, can be the principal problem in some buildings, and can result in death, as in Legionnaire's disease, or serious infectious or allergic diseases."¹ Consequently, minimizing the risk of indoor environmentally induced illness should be as simple as identifying existing contaminants and removing them. Unfortunately, the task of detection, accurate identification, linking specific contaminants to human diseases, effectively removing them, and then preventing their recurrence is anything but simple and straightforward.

Temperature, relative humidity, sources of nutrients, and air movement affect the growth and dissemination of biological contaminants. Many organisms grow independent of other living organisms, including the great majority of bacteria and fungi. Others, such as viruses, which are obligate parasites, use other living organisms synergistically to further their own growth. At least one viable organism must be available to contaminate and colonize the source of the indoor environmental contaminant.

Biological agents and their byproducts are usu-

ally microscopic in size. Even large pollens and molds cannot be identified without careful microscopic examination or by culturing the molds when they are viable. Biological contaminants include bacteria, viruses, fungi, protozoa, insects (fleas and cockroaches), and arachnids (dust mites). Fungi can be further subdivided into yeasts (unicellular organisms that reproduce by budding), molds (multicellular organisms composed of long chains of cells called hyphae that produce spores), and other higher level fungi (e.g., mushrooms).

Biological contaminants primarily cause upper and lower airway diseases by inducing immediate hypersensitivity (IgE) reactions, other types of immunologic reactions (i.e., Gell and Coombs classification types II through IV), or infection. These same contaminants are potential irritants and even toxins. They can also injure other organ systems, such as contact or irritant dermatitis, mycotoxin-induced flulike symptoms, diarrhea, and cancer (generally from ingestion). Biological contaminants can contaminate the indoor air in various situations: when living, dead, or debris from dead organisms are distributed by ventilation systems; when the contaminant is physically disturbed, such as with construction or renovation; when a solid component of the organism dissolves in water and then becomes aerosolized; and when noxious gases from contaminants are released into the indoor environment. "Bioaerosols are airborne particles, large molecules, or volatile compounds that are living or were released from living organisms."² They range in size from less than 0.1 μm to more than 100 μm in diameter. Table I lists characteristics, sources,

From the University of California, San Diego School of Medicine, and Graduate School of Public Health, San Diego State University.

Reprint requests: James M. Seltzer, MD, Asthma and Allergy Prevention and Treatment Center, 9855 Erma Rd., Suite 105, San Diego, CA 92131.

Copyright 1994 by Mosby-Year Book, Inc.
0091-6749/94 \$3.00 + 0 1/0/56011

TABLE I. Characteristics and sources of common bioaerosol components

Living source	Airborne unit	Examples of sources	Primary human effects	Lifestyle	Principal indoor sources
Bacteria	Organisms	<i>Legionella</i>	Pneumonia	Facultative parasites	Cooling towers
	Spores	<i>Thermoactinomyces</i>	Hypersensitivity pneumonitis	Saprophytes	Hot water sources, hot damp surfaces
	Products	Endotoxin	Fever, chills	—	Stagnant water reservoirs
Fungi	Organisms	Proteases	Asthma	—	Industrial processes
		<i>Sporobolomyces</i>	Hypersensitivity pneumonitis	Saprophytes	Damp environmental surfaces
	Spores	<i>Alternaria</i>	Asthma, rhinitis	Saprophytes	Outdoor air, damp surfaces
	Spores	<i>Histoplasma</i>	Systemic infection	Facultative parasites	Bird droppings
	Antigens Toxins Volatiles	Glycoproteins Aflatoxins Aldehydes	Asthma, rhinitis Cancer Headaches, mucous membrane irritants	— — —	Outdoor air Damp surfaces Damp surfaces
Protozoa	Organisms	<i>Naegleria</i>	Infection	Facultative parasites	Contaminated water reservoirs
	Antigens	<i>Acanthamoeba</i>	Hypersensitivity pneumonitis	—	Contaminated water reservoirs
Viruses	Organisms	Influenza	Respiratory infection	Obligate parasites	Human hosts
Algae	Organisms	<i>Chlorococcus</i>	Asthma, rhinitis	Autotrophic*	Outdoor air
Green Plants	Pollen	<i>Ambrosia</i> (ragweed)	Asthma, rhinitis	Autotrophic*	Outdoor air
Arthropods	Feces	<i>Dermatophagoides</i> (mites)	Asthma, rhinitis	Phagotrophic†	
Mammals	Skin scales	Horses	Asthma, rhinitis	Phagotrophic†	Horses
	Saliva	Cats	Asthma, rhinitis	Phagotrophic†	Cats

Source: Guidelines for the Assessment of Bioaerosols in the Indoor Environment, American Conference of Governmental Industrial Hygienists, Cincinnati, 1989.

*Autotrophic: synthesizes carbohydrates.

†Phagotrophic: ingests food.

and potential adverse effects caused by common bioaerosols.

Properly evaluating an indoor environment for possible biological contaminants poses several challenges. First, the most appropriate sampling test(s) must be selected. Second, knowing how, where, and what to sample is important. Third, the test results must be interpreted correctly with respect to any potential or reported adverse health effects. Consequently, an accurate analysis of the indoor environment also requires sufficient knowledge of the nature and extent of the health complaints, the characteristics of disease states that can be caused by biological contaminants, locations of illnesses within the building and where sources of contaminants may exist, and an understanding of the limitations of the assays used to measure such contaminants.

One of the most difficult aspects of the evaluation process is relating the environmental sampling data to health complaints allegedly caused by the building. Biological contaminants are ubiquitous and may be present even in higher than expected concentrations without inducing human illness or discomfort. Human illnesses share many common features, such as cough or fever. Some individuals who are ill in contaminated environments may be adversely affected by biological contaminants, while others sharing the same environment suffer from health problems unrelated to these contaminants. The diagnostician must exercise great care in attributing illness to biological contaminants just because they are present.

Once found, the source of contamination must be identified, the contaminant removed or contained to minimize the health risk to the build-

ing's occupants, and measures to prevent recontamination taken. This can be challenging when the source is hidden or distant from the site or when multiple sources of contamination are present. The complexity of the task of (1) detecting and characterizing biological contaminants, (2) determining which, if any, are responsible for human illness, and (3) how to deal effectively with the contaminants should be readily apparent. This review provides an in-depth understanding of biocontaminants: their physical properties, the development of indoor environmental contamination, and the adverse health consequences associated with biocontaminants.

CONDITIONS PROMOTING BIOLOGICAL CONTAMINATION

Ambient conditions

Very low temperatures tend to inhibit the growth of many organisms that readily replicate at room temperature. However, the yeast *Sporobolomyces* and the mold *Aureobasidium pullulans* (*Pullularia*) grow and sporulate well in cool environments. Some microbes thrive at higher temperatures. While the fungal genus *Aspergillus* can grow between 12° and 57° C, the optimal temperature range for growth is between 37° and 43° C (body temperature). The bacterium *Legionella pneumophila* and the Actinomycetes *Faeni rectivirgula* (formerly *Micropolyspora faeni*) and *Thermoactinomyces vulgaris* grow best at temperatures in excess of 50° C. *L. pneumophila* can grow in water temperatures of up to 140° F and even reside and survive within protozoa. Higher temperatures also increase the respiratory rate of organisms and thereby increase the load of contaminants from metabolism such as water vapor and CO₂.

When the temperature indoors exceeds the outdoor temperature, hot air inside the building rises in a column, creating a positive pressure above. This positive pressure forces the air through available openings (leaks) in the top of the structure, reducing air pressure at the bottom of the building, and thereby increasing the infiltration of outdoor air to equalize the outdoor/indoor pressure gradient. If the air immediately outside the building is contaminated by biological contaminants, the net result of this "stack effect" is to increase indoor air contamination.

High relative humidity promotes the growth of many molds, bacteria, and the arachnid dust mite by providing an abundance of one required nutrient, water. In general, maintaining an indoor relative humidity between 35% and 50% will

minimize condensation and indoor dampness, reduce the growth of fungi, dust mites, and bacteria, and provide a reasonable comfort level for the building's occupants.

Outdoor wind currents can transport contaminants from long distances and also stir up and aerosolize many biological contaminants that inhabit ground structures and soil. Kozak et al.³ found that the Santa Ana winds (from the eastern desert) in Southern California increased the outdoor viable mold spore count in the coastal areas from a usual baseline of 1000 to 1500 spores/m³ to 43,946 spores/m³. Yardwork, such as disturbing compost piles or mowing the lawn, can increase outdoor mold counts more than 1000-fold and potentially increase the pollen concentration in the outdoor air of those plants that are disturbed while pollinating.³ The wind can also increase the pressure gradient between the outdoor and indoor environment, driving indoors the contaminants in the outdoor air.

Because the outdoor air is a rich source of biological contaminants, the availability of ports of entry into the building becomes important, especially if a building is tight (little air leaks in or out). Most commercial buildings constructed in the past 20 years have been built "tight" to conserve energy and depend on mechanical ventilation to supply fresh air and exhaust stale indoor air. If the HVAC (heating, ventilating, and air-conditioning) system does not provide adequate fresh air, air filtration, and exhausting of contaminants, it may actually concentrate outdoor and indoor contaminants within the building. Also, larger and denser bioaerosols tend to settle out of the air more readily than smaller, less dense particles, which tend to remain suspended longer. As a result, indoor environments with better ventilation generally are more effective clearing the air of small respirable bioaerosols.

The degree and type of light can affect biological contaminant growth. Ultraviolet light inhibits the growth of many bacteria and some molds. Kozak et al.³ found that marked shading proximate to a house increased the indoor mold spore counts fivefold. Yet a total absence of light will inhibit the sporulation of some molds, such as *Alternaria* and *Drechslera*.

Nutrient sources

Generally, the most limiting and significant nutrient source is the presence of dead organic matter, even though some molds, many bacteria, and all viruses grow and replicate on or within

TABLE II. Water or moisture reservoirs in or around buildings

Outdoors		
Cooling towers	Swamp coolers	Drainage ditches
Small lakes	Agricultural storage (e.g., grains)	Compost piles
Fountains	Air conditioners	Wooden structures
Poorly kept landscaping	Wood piles	Water softeners
Industrial HVAC exhaust	Under house crawlspaces	
Wells or potable water storage containers		
Sewer drains		
Indoors		
Humidifiers	Air conditioners	Crawlspaces
Leaky appliances	Leaky plumbing	Refrigerator pans
Subterranean rooms	Ground-level cement slabs and walls	
Leaky roofs	Improperly placed vapor barriers	
Water-damaged carpet, ceiling tiles, walls, and furniture		
Bathroom showers, tubs, carpet, wallpaper, and window coverings		
Condensation on windows, cold water pipes, ventilation ducts, and insulation		

living substrates. Molds and bacteria typically seed and grow on organic debris found in soil, compost, and dung heaps, wood piles, hay, animal feed, and dead plants or leaves. They can also grow on building and finishing materials, especially if there has been wetting or water damage. Paint, wallpaper, carpeting, especially those with jute (a plant fiber) backing, foam rubber carpet pads, drapery, upholstery fabric and filler, soap scum on bathroom tile and the bathtub, baseboards, hardwood floors, ceiling tiles, cement, exterior and interior wood beams, and framing and roofing material can provide nutrients sufficient to grow bacteria and fungi. Exposed sound and thermal insulation within walls and ventilation ducts provides an organic substrate for microorganism growth. Also, dust and dirt accumulation within insulation material or within ventilation ducts and other HVAC components add further substrate. Disrupting or disturbing contaminated structural components, such as occurs with renovation or repair, can increase the concentration of indoor air contaminants 1000-fold.⁴

Stagnant water from wells and hot water heaters, humidifiers, vaporizers, and condensate pans of HVAC systems and cooling units (e.g., water towers, air-conditioners, and evaporative coolers) can harbor bacteria, fungi, algae, and protozoa. Showers, water particle emitters (e.g., vaporizers and humidifiers), and ventilation systems can aerosolize water particles adsorbed with live organisms or pathogenic portions of the organisms. Table II provides a comprehensive list of poten-

TABLE III. Potential sources of indoor air biological contaminants

Carpets	Bacteria, fungi, and protozoa
Plants, animals, birds, and humans	Standing water
Pillows, bedding, and house dust	Humidifiers, and evaporative coolers
Wet or damp material	Hot water tank

Source: Introduction to Indoor Air Quality—a self-paced learning module, Environmental Protection Agency, 1991.

tial water or moisture reservoirs that can be found in or around buildings. These microbes may be living within the water source or on surfaces over which the water or air currents highly saturated with water vapor droplets move. Alternatively, the water or air currents may dislodge nearby biological contaminants and sweep them up, disseminating them in the air or water stream (including potable water) a long distance from their nutrient source. Even in dry environments, focal sources of moisture or water can result in significant biocontaminant growth. Locating nutrient sources and recognizing the ambient climatic conditions can suggest the type of biocontaminants that may be present.

Microbial sources

Table III lists some potential sources of biological contaminants. Pet excreta and urine provide nutrient sources for bacteria and fungi. They also contain proteins that can cause immunologic

diseases; for example, pigeon and parakeet droppings can produce hypersensitivity pneumonitis (pigeon breeder's disease). Cat excreta may contain *Toxoplasma gondii* and bird excreta *Cryptococcus neoformans* and *Histoplasma capsulatum*. Pets' saliva, hair, dander, feathers, or urine are composed of animal proteins that can be allergenic and may persist for years after removal of the pet. Fecal material from dust mites and cockroaches contain very potent allergens, and *Legionella* may grow within the cysts of amoebae that protect them from the germicidal effects of chlorination of the water source. Kozak et al.⁵ found that the most prevalent fungal spores within homes in sites evaluated in Southern California, in descending order, were *Cladosporium* (100% of homes), *Penicillium* (91%), nonsporulating mycelia (90%), and *Alternaria* (87%).

INDUCTION OF HUMAN ILLNESS BY BIOLOGIC CONTAMINANTS

The ability of airborne particles to reach different parts of our respiratory tract depends on their size. Large particles in the size range of 30 to 60 μm usually consist of organic and inorganic dirt, fibers, and the larger pollens and mold spores. These are filtered out by the nasal vibrissae. Many pollens, mold spores, hyphal fragments, and smaller inert particles (some containing adsorbed biological contaminants) in the range of 5 to 20 μm will impact on the nasal mucosa or penetrate further down into the major lower airways, that is, primary and secondary bronchi. Bacteria, smaller fungal spores, and droplet nuclei from talking, coughing, and sneezing make up the group of truly respirable particles, that is, those reaching the terminal airways and ranging in size from 1 to 5 μm . Particles smaller than 1 μm are generally expelled from the human respiratory tract with exhalation. Although viral particles fall into this category, they usually enter the respiratory tract adsorbed to droplet nuclei that may remain there, eventually penetrating the respiratory tract epithelial cell in cases of successful infection.

Biological contaminants most often disseminate in the indoor environment through air currents or water aerosols. The great majority of biological contaminants cause human illness or discomfort by three mechanisms: (1) infection, (2) intoxication, and (3) immunologic responses. Infectious bacteria, viruses, and some fungi enter the human host through the mucous membranes of the respiratory tract or eye, through disrupted areas of our skin (e.g., atypical mycobacteria), and some-

times through ingestion (e.g., *Salmonella*). Occasionally infection is iatrogenic from use of contaminated parenteral products such as blood. The microbial load (concentration) is an important determinant of the probability and eventual severity of infection. Some of these infectious agents are saprophytes, which are usually only hazardous for the immunocompromised or debilitated host. *Aspergillus*, *Candida*, *Geotrichium*, *Scedosporium*, *Paecilomyces*, and *Scopulariopsis* are examples of saprophytic mold spores that are small enough to enter the lower airways and grow well at 37° C. However, under certain conditions, they can become pathogenic, resulting in infection or hypersensitivity reactions. The desquamated skin of occupants of a building is the predominant source of bacteria normally found indoors e.g., *Micrococcus* and *Staphylococcus*. Our respiratory tract provides an additional source of ambient bacteria and sometimes viruses.⁶

Nosocomial organisms can spread indoors and cause human diseases, such as the anthrax bacillus contracted from the indoor processing of animal products from infected animals and Q-fever, a rickettsial organism usually contracted from infected laboratory animals or in buildings nearby areas where animals are housed. Two episodes in which tuberculosis bacillus dissemination has been linked to ventilation systems^{7, 8} are of great concern given the recent epidemic of tuberculosis in this country. Viruses may be spread by unconventional routes and cause infection: a measles epidemic within an elementary school spread by its HVAC system⁹ and rabies contracted by inhalation of rabies virus in a bat cave harboring infected bats.¹⁰ An unusual situation arose in England where *Acanthamoeba* infections of the eye occurring in persons wearing contact lenses were linked to the use of contaminated tap water to make saline from salt tablets.¹¹

Biological contaminants rarely cause intoxication. A few molds synthesize mycotoxins as secondary metabolites. Mycotoxins are highly variable complex polypeptides that are generally not volatile and remain associated with fungal structures, including spores, or dissolved within the substrate on which the mold is growing. Many genera of fungi can produce mycotoxins, but this toxigenic potential is species specific. For example, *Aspergillus flavus*, *Penicillium viridicatum*, and *Stachybotrys atra* are frequent mycotoxin producers, whereas *Aspergillus fumigatus* and *Penicillium chrysogenum* are not. The environmental conditions also help determine whether a toxi-

genic fungus will actually produce mycotoxin. Mycotoxins, when ingested, can produce central nervous system effects (anorexia, nausea, and fatigue), immunosuppression, gastrointestinal lesions, hematopoietic suppression, and suppression of reproductive function.¹² Some, such as the aflatoxin produced by *A. flavus*, are potent carcinogens.² Because mycotoxins are present in high concentrations in some mold spores, many health experts share the opinion of the U.S. Environmental Protection Agency that "it is reasonable to assume that these toxins have a systemic effect when inhaled, since inhalation more effectively allows entry for dissolved substances."¹² However, to date despite several reports linking inhaled mycotoxins to human illness, few well-documented cases of inhalation-induced human mycotoxicosis exist. One such case involved a house heavily infested with *S. atra*.¹³

In addition to toxins produced by fungi, many gram-negative bacteria produce endotoxin, a potent lipopolysaccharide moiety of the cell wall. This toxin produces fever, malaise, respiratory distress, peripheral blood leukocytosis with a shift to the left, and shock, which can be fatal. However, well-defined reports of human intoxication from inhaled endotoxin have been limited to experimentally induced air flow obstruction.^{14, 15}

Immunogenic substances that are integral parts of the structures of various plants, animals, and microorganisms or that are released by them into the environment can cause indoor environmentally induced human illness. These immunogenic epitopes can produce immunologically induced inflammation by all four Gell and Coombs pathologic mechanisms: (1) immediate (IgE-mediated) hypersensitivity or allergy, (2) cytotoxic antibodies against homologous tissue antigens (e.g., group B hemolytic streptococcus and rheumatic fever), (3) antibody-antigen complex disease (e.g., precipitins in hypersensitivity pneumonitis), and (4) delayed-type T cell-mediated immunity (e.g., *Mycobacterium tuberculosis*).

A lack of understanding of several different aspects of these immunologic responses can lead to confusion or misunderstanding regarding the relevance of environmental biocontaminants to human illness. Our ability to distinguish different mechanisms of inflammation is blurred somewhat because often more than one mechanism may contribute to the pathologic findings. For example, immediate hypersensitivity involves both cell-mediated and humoral immune activity. Also, antibody responses, including IgE, can be mea-

sured against antigens to infectious agents such as viruses that have traditionally been thought of as evoking cell-mediated immunologic responses. The exact role of antiviral (e.g., respiratory syncytial virus) IgE in the pathogenesis of asthma remains unclear. Microbial antigens from fungi, bacteria, and protozoa can induce specific precipitating antibodies (IgG, IgM, and IgA) in susceptible persons. A role for local immune complexes in the pulmonary parenchyma or airways has been suggested in the pathogenesis of hypersensitivity pneumonitis¹⁶⁻¹⁸ and allergic bronchopulmonary aspergillosis.¹⁹ Cytotoxic antibodies may also contribute to the pulmonary parenchyma damage seen in hypersensitivity pneumonitis.²⁰

Hypersensitivity responses to biological components are limited to those with the genetic ability to respond to specific allergens, such as an IgE response to the reproductive progeny of plants (pollen) and fungi (spores), the excreta of insects and arachnids (cockroach and dust mite), structural components and byproducts of animals and insects (danders, saliva, urine, feathers, and venom), and products produced from some plants (pyrethrins used in insecticides, cotton lintens, kapok, and gums). Bacteria, such as *Bacillus subtilis*, can synthesize enzymes that stimulate specific IgE synthesis in a small minority of genetically predisposed people who become sensitized to these allergens. There have been several reports of occupational asthma in detergent manufacturing workers and in other occupations involving frequent large exposures to this and other enzymes. Prior exposure and the nature of this exposure, that is, high or low dose, continuous or intermittent, may play a determining role in whether illness develops and what form it will take in a given individual.

Indoor air may not be the vehicle by which certain pathogenic biocontaminants contact those who become ill. Plant resins from poison oak, ivy, and sumac can be spread in the indoor environment from contaminated clothing or human contact to the skin of family members. Individuals sensitive to these allergens can develop contact dermatitis to these resins and to other natural plant products (some of which may later become airborne, e.g., ragweed pollen) that can mimic photosensitivity dermatitis or atopic eczema.^{21, 22} Once the inflammation becomes established, the initial immunogens may no longer be required to perpetuate the inflammatory response, which then becomes nonspecific and may persist for months to years.

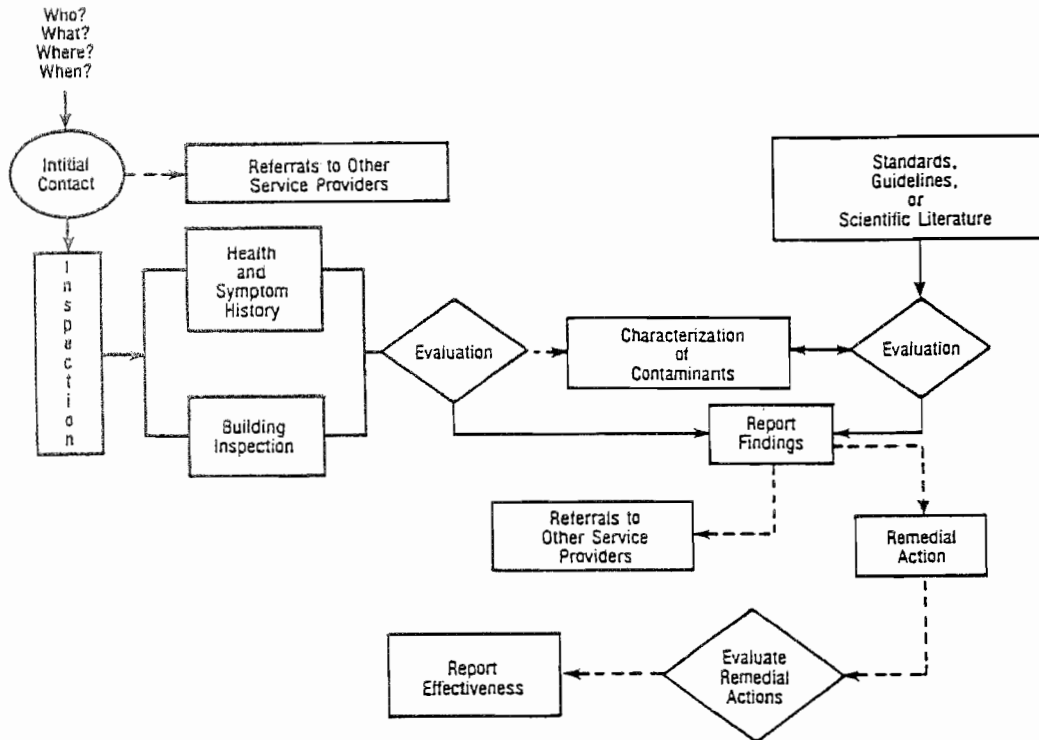


FIG. 1. Flowchart of an investigation strategy for indoor air quality problems (From Introduction to Indoor Air Quality—a self-paced learning module. Environmental Protection Agency, 1991.)

Some molds²³ and indoor house plants²⁴ synthesize and release volatile organic compounds into the environment, which can result in a characteristic and sometimes irritating odor, such as the musty odor from mold overgrowth. Whether volatile organic compounds of organic origin pose significant human health hazards other than the discomfort caused by their odor remains as yet undetermined.

RELATING SAMPLING INFORMATION TO HUMAN ILLNESS

Many issues prevent simple interpretation of environmental testing results for biological contaminants:

1. Were the sampling assays selected appropriate for the environmental conditions and the adverse health effects in question?
2. Was the sampling done properly and with the proper controls?
3. Did the technician and laboratory process the samples correctly?
4. What is the sensitivity of the assay?
5. What is the specificity of the assay?

Assuming all these issues were addressed ad-

equately and the test results are considered valid, the next step, maybe the most difficult one, is to relate the test results to the adverse health issues. The laboratory findings of biological contamination must support the characteristics of the illness profile to have positive significance, for example, the environmental presence of *F. rectivirgula* or *T. vulgaris* together with affected individuals with symptoms and signs of hypersensitivity pneumonitis having serum precipitins to *F. rectivirgula* or *T. vulgaris*. Similarly, findings of large amounts of dust mite antigen or mold spores to which one or more persons affected with asthma or rhinitis symptoms have positive skin test results strongly suggests but does not prove that their indoor environment is contributing to their illness. Yet finding small or even large amounts of *L. pneumophila* bacteria in a water reservoir or even in the air does not necessarily mean that persons with pneumonia are infected with this bacterium, because large concentrations of this organism are only one criterion required to establish the diagnosis of *Legionella*-induced human disease. However, if a rising serum antibody response to *L. pneumophila* or any bacteria-specific

antigen in the urine is found, then causality can be established. Another common error is to attribute infectious illnesses in the building to high levels of human skin bacterial counts found on sampling. These findings may simply indicate poor effective ventilation or overcrowding, which may or may not contribute to occupant complaints. Because of the complex interaction between (1) the nature and concentration of ubiquitous biological contaminants, (2) the ability of the indoor environment to amplify, disseminate, and sometimes concentrate these contaminants, and (3) host (building occupant) susceptibility, exposure, and physiologic or pathologic responses to these contaminants, drawing valid conclusions about cause and effect can be very problematic.

IMPLICATIONS OF BIOLOGIC CONTAMINATION

Because biological contaminants exist to varying degrees in every indoor environment, the potential to produce human illness is ever present. The conversion from a condition of the potential to induce illness to the actual production of illness depends on several factors:

1. The availability of a source of biological contamination—a given, considering that all indoor environments contain a number of different types of viable organisms.
2. The nature of the contaminants: their individual or collective abilities (considering synergy) to cause illness.
3. The nature of the indoor environment: does it provide sufficient nutrients and ambient conditions to amplify pathogens and does it have the mechanical or natural means to disseminate the organisms, their parts, or byproducts that can cause adverse health effects in the building occupants?
4. The genetic makeup, current health, and other relevant characteristics, such as age of the building occupants. Certain groups, such as the very young, the elderly, and the infirm, are often more susceptible to biological contaminants.

Valid scientific data provide a solid understanding of many of the infectious and immunologic complications caused by biological contaminants. Much still remains to be elucidated about the characteristics of human illness and mechanisms by which microbial products, such as endotoxins or mycotoxins, can adversely affect human health. Also lacking is essential knowledge

of the dose-response relationships between many biological contaminants and the human host that would permit establishment of indoor environmental standards for these agents. This void exists for most toxic, carcinogenic, and many immunologic reactions to inhaled biological contaminants.

Although some of this ignorance originates from the complexity of the problem, some derives from a lack of attention to well-understood non-medical aspects of the problem that have great impact: the presence or absence of adequate effective ventilation; the hygienic aspects of the structural, ventilation, and design (e.g., furniture, carpeting) components of the building; the cleanliness of indoor and outdoor air; ambient indoor and outdoor conditions; and the degree of effective execution of building maintenance plans designed to minimize biological contamination. These are only some examples of many building-related functions that impact on the health of occupants for which adequate knowledge exists to appropriately identify or correct existing or potential building problems, and yet they are often ignored. Finally, during the past 20 years funding for research to clarify these issues pales in comparison to that for outdoor air pollution research.²⁵

Do the fall-winter epidemics of respiratory viruses need to be as extensive as they always are? Couldn't their impact on human health be reduced by correcting deficiencies in effective ventilation and overcrowding in office buildings, schools, and hospitals? Couldn't reducing the indoor allergen load exposure to the 15%-25% of the population that has atopic disease improve their health and reduce their susceptibility to respiratory tract infections? If builders, architects, and designers considered the health impact of their choice of mechanical systems, building and finishing materials, and the sequence and methods of construction or cleaning (e.g., not renovating the floor above an occupied floor or working on the HVAC system while people work in the space ventilated by that system), wouldn't there be a much better chance of spending this 90% or more of our lives indoors in a healthier indoor environment?

The problem of biological contaminants can only be served well by a multidisciplinary team of health care professionals, engineers, building professionals, and often industrial hygienists and biologists who have broadened their scope of understanding about this problem. Fig. 1 provides a flowchart that can serve as a guide for conducting

a building investigation of environmental contamination. Input from experts in several fields is often necessary at each junction of the evaluation process to define the problem accurately and to correct identified or potential deficiencies. This strategy can be very effective in the short term. However, the major thrust of emphasis in construction, renovation, and building maintenance must shift from an almost exclusively reactive to a proactive approach if a significant impact is to be made on indoor biological contamination in both currently occupied and future buildings. Building homes and commercial buildings with a primary emphasis on providing a hygienic environment for the occupants is essential. Until building owners and building contractors change their current methods, some buildings will be constructed incorrectly and cause potentially preventable illnesses and health complaints for those persons who occupy them.

REFERENCES

1. US Environmental Protection Agency. Report to Congress on indoor air quality; executive summary and recommendations, EPA/400/1-89/001A. Washington, D.C.: US EPA, Office of Air and Radiation, 1989:chapter II.
2. Guidelines for the assessment of bioaerosols in the indoor environment, Cincinnati: American Conference of Governmental Industrial Hygienists, 1989.
3. Kozak PP Jr, Gallup J, Cummins LJ, Gillman SA. Endogenous mold exposure: environmental risk to atopic and nonatopic patients. In: Gammage RB, Kaye SV, eds. Indoor air and human health. Chelsea, Michigan: Lewis, 1985:149-70.
4. Morey PR. Microorganisms in buildings and HVAC systems: a summary of 21 environmental studies. In: Proceedings of the ASHRAE conference IAQ 88. Atlanta: American Society of Heating, Refrigeration, and Air-conditioning Engineers, 1988:10-21.
5. Kozak PP, Gallup J, Cummins LH, Gillman SA. Factors of importance in determining the prevalence of indoor molds. *Ann Allergy* 1979;43:88-94.
6. Spendlove JC, Fannin KF. Source, significance, and control of indoor microbial aerosols: human health aspects. *Public Health Rep* 1983;98:229-44.
7. Nolan CM, Elarth AM, Barr H, Saeed AM, Risser DR. An outbreak of tuberculosis in a shelter for homeless men: description of its evolution and control. *Am Rev Respir Dis* 1991;143:257-61.
8. DiStasio AJ II, Trump DH. The investigation of a tuberculosis outbreak in the closed environment of a US Navy ship, 1987. *Milit Med* 1990;155:347-51.
9. Riley EC, Murphy G, Riley RL. Airborne spread of measles in a suburban elementary school. *Am J Epidemiol* 1978;107:421-32.
10. Constantine DG. Rabies transmission by air in bat caves: National Center for Disease Control monograph. Atlanta: Center for Disease Control, 1967 (PHS Publication No. 1617).
11. Seal D, Stapleton F, Dart J. Possible environmental sources of *Acanthamoeba* spp in contact lense wearers. *Br J Ophthal* 1992;76:424-7.
12. US Environmental Protection Agency. Non-cancer health and discomfort effects of poor indoor air quality. In: Report to Congress on indoor air quality, vol II: assessment and control of indoor air pollution. Washington, D.C.: US EPA, Office of Air and Radiation 1989:chapter 3 (EPA 400/1/-89/001C).
13. Croft WA, Jarvis BB, Yatawara CS. Airborne outbreak of trichothecene toxicosis. *Atmos Environ* 1986;20:549-52.
14. Rylander R, Halind P, Lundholm M. Endotoxin in cotton dust and respiratory function decrement among cotton workers in an experimental cardroom. *Am Rev Respir Dis* 1985;131:209-13.
15. Rylander R, Haglund P. Airborne endotoxins and humidifier disease. *Clin Allergy* 1984;14:109-12.
16. Pepys J. Extrinsic allergic alveolitis. In: Hypersensitivity diseases of the lungs due to fungi and organic dusts. *Monogr Allergy* 1969;4:69-131.
17. Barrowcliff DF, Arbruster PG. Farmer's lung: a study of an early acute fatal case. *Thorax* 1968;23:490-500.
18. Spector WG, Heesom W. The production of granulomata by antigen antibody complexes. *J Pathol* 1969;98:31-9.
19. Geha RS. Circulating immune complexes and activation of the complement sequence in acute allergic bronchopulmonary aspergillosis. *J ALLERGY CLIN IMMUNOL* 1977;60:357-9.
20. Wenzel FJ, Emauel DA, Gary RL. Immunofluorescent studies in patients with farmer's lung. *J ALLERGY CLIN IMMUNOL* 1971;48:224-9.
21. Arlette J, Mitchel JC. Compositae dermatitis. *Contact Dermatitis* 1981;7:129-36.
22. Benezra C, Ducombs G, Sell Y, Fousserau J, eds. Plant contact dermatitis. St Louis: The CV Mosby Co, 1985.
23. Kaminski E, Stawicki S, Wasowicz E. Volatile flavor compounds produced by molds of *Aspergillus*, *Penicillium*, and *Fungi imperfecti*. *Appl Microbiol* 1974;27:1001-4.
24. Can house plants solve IAQ problems?. *Indoor Air Bulletin* 1992;2:1-5.
25. Kennedy JP. The indoor air quality act of 1991, extensions of remarks. *Congressional Record* Feb.22, 1991:E592.