Title

Indoor Humidity and Human Health--Part I: Literature Review of Health Effects of Humidity-Influenced Indoor Pollutants

Authors

Baughman, A.
Arens, Edward A

Publication Date

1996

Peer reviewed

Indoor Humidity and Human Health—Part I: Literature Review of Health Effects of Humidity-Influenced Indoor Pollutants

Anne V. Baughman
Student Member ASHRAE

Edward A. Arens, Ph.D.
Member ASHRAE

ABSTRACT

Standards for indoor thermal conditions and ventilation include upper limits for relative humidity (RH) that typically are in the range of 60% to 80% RH. Although the reasons for the limits are often not explicitly stated, it is generally known that they were set out of concern for the health effects that might occur should the humidity become too high. The primary health effects of high humidity are caused by the growth and spread of biotic agents, although humidity interactions with nonbiotic pollutants, such as formaldehyde, may also cause adverse effects. This literature review identifies the most important health issues associated with high humidities and presents humidity requirements, typical contamination sites within buildings, and remediation measures for each pollutant. Part two of the paper addresses the physical causes of moisture-related problems in buildings.

INTRODUCTION

Standards for indoor thermal conditions and for ventilation have traditionally put upper limits on the amount of humidity permissible in interior spaces because of concern for the health effects that might occur should the humidity become too high. Such limits are found in past versions of ASHRAE Standards 55-1992 (ASHRAE 1992) and 62-1989 (ASHRAE 1989) and in most international standards. The values set for the upper limits have typically ranged from 60% to 80% RH, although boundaries of absolute humidity have also been used. To date, the relationship of high humidity to the full spectrum of air quality issues and to the relevant characteristics of building envelopes and conditioning systems has not yet been addressed in a comprehensive manner. This situation affects our ability to set rational standards and building specifications.

Human health is not affected by high levels of humidity *per se*. Known health effects related to high humidity are primarily caused by the growth and spread of biotic agents under elevated humidities, although humidity interactions with nonbiotic pollutants, such as formaldehyde, may also cause adverse effects. Existing limits appear to be based on engineering experience with such humidity problems in buildings.

The position of any upper humidity limit has great economic significance, particularly in hot and arid parts of the country, where evaporative cooling is an energy-conserving option. In the West, it affects the need for billions of dollars of new peak electrical generating capacity that could be offset by noncompressor-based cooling. It also directly affects a substantial fraction of the cooling load in hot, humid climates. Under such economic imperatives, it is desirable to carefully examine the position of any upper humidity limit. Ideally, one would be able to assess the health risks against the economic benefits for any given humidity limit. At present, there is not enough information on this subject to even begin such an analysis.

This review of the literature identifies a number of health-related agents that are affected by indoor humidity. All of them affect human health primarily through their inhalation from the air, although some of them have lesser effects through the skin. Biological agents require appropriate conditions in the building for their germination, growth, release to the air, and transport to the human host. Airborne levels of nonbiological pollutants, such as formaldehyde and ozone, may also be affected by humidity through influences on offgassing and surface reaction rates. Finally, the occupants' susceptibility to these agents may also be a function of humidity, although this appears to be a problem primarily at low humidities, when respiratory ailments result from dry mucous membranes (Green 1985). The health implications of low humidities are not addressed in this paper. Part two of this paper addresses the relationships of the environments within buildings and conditioning systems to the growth of biological pollutants.

Anne V. Baughman is a graduate student researcher and **Edward A. Arens** is a professor in the Department of Architecture at the University of California, Berkeley.

OVERVIEW OF HUMIDITY-RELATED HEALTH CONCERNS

The primary influences of humidity on health are through biological pollutants. The following outline describes the health issues most commonly associated with biological pollutants.

Infectious disease (pathogens)
- bacteria (e.g., *Streptococcus, Legionella*)
- viruses (e.g., common cold, flu)
- fungi (e.g., *Aspergillus fumigatus*)

Allergic reactions (e.g., asthma, rhinitis)
- dust mites (dried body parts and fecal excreta)
- fungi

Nonallergic immunologic reactions (e.g., hypersensitivity pneumonitis)
- fungi
- bacteria

Myctoxicosis
- fungi

Infectious disease can occur when viable pathogenic organisms enter (usually through inhalation) and colonize in the body of a susceptible host. The most commonly found pathogens are bacteria or viruses, although fungal pathogens, such as *Aspergillus fumigatus,* also exist (Flannigan 1992). Most pathogens are transmitted through human-to-human contact when droplet nuclei form as a result of sneezing or coughing and are subsequently inhaled by a human receptor. A few pathogens, most notably the bacterium *Legionella*, can colonize abundantly within moist environments outside the human body and become airborne given proper conditions.

Noninfectious health conditions related to biological pollutants include allergic, immunologic, and toxic responses. The primary sources of these adverse health effects are the byproducts of organisms rather than the viable organism itself. The term *allergy* is used specifically to refer to illnesses that take place as a result of the formation of IgE antibodies in affected persons. All human beings have some IgE antibodies, but only a fraction of the population responds readily to allergen exposure and produces enough IgE antibodies to cause an allergic reaction. Once the antibodies form, the person becomes sensitized and re-exposure to the allergen can then trigger larger immune reactions resulting in allergic symptoms. This IgE-mediated reaction develops in 20% to 30% of the people in the United States (Seltzer 1995). The allergic diseases with well-documented links to indoor air quality (IAQ) include allergic rhinitis (rhino conjunctivitis), primarily affecting the nasal area, and allergic asthma and bronchopulmonary aspergillosis (ABPA), both of which affect the lower airways and alveoli. The majority of patients suffering from asthma are allergic to dust mites, mold, and/or animal dander. The estimated overall prevalence of asthma and rhinitis may be as high as 20% of the population (Berglund et al. 1992) and the American Lung Association estimates that the number of people with reported asthma in the U.S. has greatly increased in recent years, with a 49% increase since 1982.

Nonallergic immunologic responses, characterized by recurrent flu-like symptoms (e.g., hypersensitivity pneumonitis, farmer's lung, and humidifier fever), seem to be unrelated to the IgE antibody. They occur as a result of repeated pollutant exposures that trigger other antibody-dependent mechanisms as well as cellular immune responses. Although there seems to be no genetic predisposition, only a fraction of those exposed develop overt symptoms (Burge 1988).

Mycotoxins are produced by fungi and can lead to respiratory irritation, interference with pulmonary macrophage cells, and/or higher risks of cancer (Flannigan and Miller n.d.). Many fungi also produce volatile organic compounds (VOCs) that may be respiratory irritants and have been suggested as a contributing factor to sick-building-type symptoms in microbially contaminated buildings (Bjurman 1993; Sorenson 1989).

Nonbiological pollutants, such as formaldehyde, ozone, oxides of nitrogen, and sulfur, affect humans primarily through chemical irritation of the mucous membranes. Formaldehyde is released into the indoor air from building materials in ways that are dependent on atmospheric humidity. Surface reactions, and consequently the amount and toxicity of ozone and nitrogen oxides (NO_X) and sulfur oxides (SO_X) in the air, may be influenced by humidity levels. The extent to which humidity increases or decreases the health impacts of these pollutants, however, is relatively small compared to other environmental factors, such as air change rates and outdoor pollutant levels. For example, the use of direct evaporative cooling leads to a rise in indoor humidity levels, which may reduce ozone by increasing surface reactions. However, this effect is relatively insignificant compared to the increased influx of outdoor air, which tends to increase indoor ozone concentrations to levels near those of the outdoor air (Stock et al. 1993).

DUST MITES

Introduction

Mites are considered one of the most important allergens in house dust, particularly in regions with high humidities and temperate climates. The most common genus of mites found in house dust in North America and Europe is *Dermatophagoides,* of which there are two species, *D. pteronyssinus* and *D. farinae.* It is estimated that 10% of the population in the U.S. is allergic to house dust and 70% of these people are specifically allergic to mite allergens (Bates et al. 1993). The actual allergen is not the mites themselves, which are approximately 1/3mm in length at maturity, but the dried fragments of their body parts and fecal excreta. These by-products are initially 10 to 50 μm in diameter but break down into smaller fragments that become airborne when dust is disturbed. According to one study, more than half of the weight of mite allergens within a home were found to be less than 5 μm in length (Reed et al. 1986). These particles are the primary health concern since they can be inhaled into the lower airways of the lungs and, if quantities are significant, IgE antibodies can form, leading to allergic reactions in the susceptible portion of the population.

A number of studies have demonstrated a high prevalence of sensitization to mite allergens among patients with asthma and nonspecific respiratory symptoms (Voorhorst et al. 1964; Korsgaard 1983b; Platts-Mills et al. 1989; Smith et al. 1985; Arlian et al. 1992). In most of these studies the patients were referred to the researchers by clinics and compared with control subjects randomly selected from the same or a similar population base. Sensitization to mite allergens was demonstrated by a positive skin prick test. The study of Danish homes by Voorhorst et al. (1964) was the first to establish a definitive link between the presence of mite allergens and respiratory symptoms. Korsgaard (1983a, 1983b) confirmed this finding when he found significantly higher concentrations of dust mites in the homes of 25 asthmatic patients compared to 75 randomly selected homes. Arlian et al. (1992) conducted a five-year study of 252 homes inhabited by dust-mite-sensitive people in eight different regions of the U.S. They found that 83% of the homes had average mite densities greater than the estimated sensitivity threshold of 100 mites/gm of dust.

Studies in the literature also provide evidence to support a connection between damp housing and sensitivity to dust mites and childhood respiratory symptoms. For example, Murray et al. (1985) studied 774 homes inhabited by children with respiratory symptoms in British Columbia and found that more than 90% of these children lived in areas defined as "humid" (i.e., indoor humidity estimated to be 50% or greater for four or more months out of the year). In addition, there was a significant difference in the number of mite-sensitive children in the "humid" areas (skin prick test positive for *D. farinae* in 31% and *D. pteronyssinus* in 40%) as compared to those living in areas defined as "dry," with an indoor RH of 50% or higher for no more than two months per year (skin prick test positive for *D. farinae* and *D. pteronyssinus* in 3% and 2%, respectively). Verhoeff et al. (1995) conducted a study that included 259 children with chronic respiratory symptoms and 257 control children. There were more cases of mite and mold sensitization in the children with respiratory symptoms. These children were also slightly more likely than the controls to have been living in homes where mold or damp was reported or observed.

Along with respiratory symptoms, high levels of dust mite allergens have also been correlated with atopic dermatitis (AD), characterized by itchy, irritated skin (Harving et al. 1990; August 1984). In general, these studies suggest that those susceptible to mites (i.e., those likely to form IgE antibodies) are also likely to develop skin sensitization if exposed to high concentrations of mite allergens. For example, Colloff (1992) examined the density of dust mite populations in mattresses in the homes of 23 people with AD who were mite-sensitive and found that counts were significantly higher than in the mattresses of the nonatopic control group. Colloff also cites a number of references that link atopic dermatitis to high dust mite exposure, including IgE antibody responses to mite allergens, among patients with AD and marked clinical improvement following intensive eradication of mite allergens.

The regional diversity of mite studies in the literature suggests that mites occur indoors all over the world, from arctic Greenland to tropic Africa (Anderson and Korsgaard 1986). The study by Arlian et al. (1992) included eight different geographic regions within the U.S. They found that *D. pteronyssinus* and *D. farinae* were by far the most common species, with *D. pteronyssinus* predominating in humid regions with moderate climates and *D. farinae* predominating in areas with prolonged periods of dry weather. This finding was supported by Lang et al. (1977), who examined mite populations in four different climatic zones of southern California and found significant numbers of *D. pteronyssinus* and *D. farinae* in 14 of 15 of the coastal homes and in 9 of 15 of the inland valley homes. *D. pteronyssinus* was the predominant species in the coastal region, while *D. farinae* predominated in the inland regions.

Mites are relatively sparse in regions with low outdoor humidity, such as at high elevations and in desert areas (Brundrett 1990; Murray et al. 1985; Lang et al. 1977). However, if the indoor humidity is allowed to rise due to internal sources such as direct evaporative cooling, mite populations, particularly *D. farinae*, can become significant even when outdoor humidities are low. For example, O'Rourke et al. (1993) evaluated 190 evaporatively cooled homes in Tucson, Arizona, and detected mites in more than half of the homes, with *D. farinae* being the overwhelmingly predominant species (greater than 98% of all mites recovered).

Environmental Requirements

Mites contain about 70% to 75% water by weight and must maintain this in order to reproduce (Arlian 1992). Their primary source of water is ambient water vapor, which they are able to extract directly from unsaturated air by means of a hygroscopic salt solution in the supracoxal gland (Fernandez-Caldas et al. 1994). The amount of water gained through ingestion of moist food is relatively small. Laboratory studies of *D. pteronyssinus* suggest that optimal conditions for growth and development occur between 70% and 80% RH at 25°C, with acceptable ranges of 55% to 80% RH and 17°C to 32°C (Anderson and Korsgaard 1986). The upper humidity limit is constrained by the possibility of mold growth, particularly above 88%, which can inhibit mite development (Brundrett 1990).

Arlian (1992) performed laboratory studies of both *D. farinae* and *D. pteronyssinus* and found that the critical equilibrium humidity (CEH) for fasting mites, defined as the lowest RH at which mites are able to maintain their water balance, was 73% and 70% RH at 25°C for *D. pteronyssinus* and *D farinae,* respectively. The CEH was found to be influenced by temperature and ranged from 55% at 15°C to 75% at 35°C for *D. farinae*. According to Arlian, this temperature relationship, together with the fact that feeding mites do gain small amounts of water from food, may explain why significant populations of mites are found in environments with relative humidities below 70%. Survival of mites for prolonged periods at lower humidi-

ties may also be explained in part by the crystallization of salts within the supracoxal gland, which may slow down the rate of dehydration (Fernandez-Caldes et al. 1994).

Under optimal conditions, mites live for three months with three different larval stages. The survival of active adult mites (both male and female) is limited to 4 to 11 days at humidities below 50% RH at 25°C (Arlian et al. 1982). The protonymph, however, which is one of the dormant larval forms, can survive for months at low humidities and then evolve to the more active forms when optimal conditions return (Arlian 1992). This observation is supported by the field study by Lang et al. (1978) in which the different stages of mite development were quantified. In this study a higher number of protonymphs were found when the RH fell below critical levels (50% to 65% RH). These protonymphs are particularly difficult to remove with normal vacuuming since they can bury themselves within surfaces (Arlian 1992).

As one might expect, most mite allergens are formed by adults during their active phase. Thus, for a given number of mites, the highest levels of allergens found in the environment usually correspond to optimal humidity conditions. Arlian (1992) examined the effect of RH on mite metabolism for a range of relative humidities between 22% and 95% and observed that feeding rates, and consequently the amount of fecal matter produced, increased with increasing RH. The effect was particularly significant between 75% and 85% RH, for which there was a fivefold increase in the weight of food consumed for both *D. pteronyssinus* and *D farinae*. Below the CEH, Arlian found that mites fed sparingly and produced little fecal matter. These results suggest that significant reductions in the level of mite allergens, which consist primarily of metabolic by-products, may occur if RH is reduced below the CEH. (For more detailed information on the mite life-cycle and metabolism, see Arlian [1992].)

Laboratory studies suggest that temperatures within the range typically found in occupied spaces have little direct effect on the length of the mite's life-cycle and that mites are able to survive extreme temperature conditions for limited periods. For example, under laboratory conditions, more than half of *D. pteronyssinus* survive after 12 days when continuously exposed to 34°C and 75% RH, while at 2°C and 75% RH approximately 64% of *D. farinae* adults survive after 72 hours (Lang et al. 1977). Mites are also able to reproduce at temperatures as low as 17°C, albeit more slowly than at 25°C (Murray et al. 1979).

Mites subsist primarily on shed human and animal skin scales. It is believed that mites cannot digest lipids within the skin scales themselves and require the aid of xerophilic fungi, of the genus *Aspergillus,* to dissolve the lipids for them (Flannigan 1992; Hart and Whitehead 1990; Platts-Mills et al. 1989). This suggests that conditions for mite survival must also be suitable for these fungi.

Absolute vs. Relative Humidity

Some researchers have suggested that absolute humidity rather than relative humidity is the limiting factor controlling mite metabolism (Korsgaard 1983a, 1983b; Platts-Mills et al. 1987b). However, the predominant evidence from laboratory studies and field work suggests that relative humidity is the controlling factor. Mites have a high surface-to-volume ratio and are poikilothermal (i.e., their body temperature is identical to that of the surrounding environment) (Anderson and Korsgaard 1986). Since there is no temperature gradient between the mite and the surrounding environment, the relative difference between the air vapor pressure and the mite's internal saturation vapor pressure is proportional to the relative humidity rather than the absolute humidity. Arlian (1992) has demonstrated that the driving force in the uptake of water from unsaturated air is the number of water molecules impinging on the mite's uptake surface. Arlian also performed laboratory studies that suggest that mites are able to maintain a water balance at 20°C and 79% RH but die at 27°C and 56% RH (i.e., the same absolute humidity).

Field Studies: Residential

Field studies within homes generally support the laboratory findings that indoor relative humidity is the most significant environmental condition associated with high mite populations and allergen concentrations. However, it is not clear from these studies what specific level of humidity is critical. Significant concentrations of mites and allergens have been found at indoor humidities as low as 40% RH (O'Rourke et al. 1993) but more often at indoor humidities above 50% RH. For example, in a study of homes in Vancouver, Murray et al. (1979) detected significant numbers of mites only when the RH was greater than 50% for at least part of every day during the month of collection. Smith et al. (1985) also found a direct correlation between mite population and indoor RH in a study of 20 homes of mite-sensitive children, with mite populations peaking at RH of 50% or greater. Hart and Whitehead (1990) evaluated 30 homes in the United Kingdom and found that mite populations were most strongly correlated with indoor RH and that bedrooms with humidities above 64% RH contained significantly more mites in mattresses than those with humidities below this level.

Regional studies suggest that dust samples from different homes within the same region can exhibit wide differences in mite concentration due to differences in indoor humidity alone. For example, Lintner et al. (1993) evaluated 424 homes across the U.S. and found that the greatest variations in mite population occurred as a result of differences in the indoor relative humidity among homes rather than regional climatic differences. Korsgaard (1983a, 1983b) conducted a four-season study of 50 Danish apartments, all within the same region, and found that seasonal variation in dust mite populations in mattresses correlated with the indoor humidity, while homes with the lowest indoor humidities did not contain detectable levels of mite populations. Ellingson et al. (1995) studied the

effect of direct evaporative cooling on the prevalence of mite allergen in Colorado homes. Their results show that during the peak cooling season 48 of 95 samples from homes with evaporative coolers (average interior RH of 51% or greater) had levels of Der p 1 and Der f 1 of 2 μm/gm dust or greater, but only 5 of 95 control samples (average interior RH of less than 45%) had levels of 2 μm/gm dust or greater.

Long-term studies suggest that seasonal trends in mite populations correlate with seasonal variations in indoor humidity. For example, in a 19-month study of six homes in southern California, Lang et al. (1978) found seasonal differences in species composition, with *D. pteronyssinus* more abundant from July through November and *D. farinae* more abundant from August through December. Both species were found to be less prevalent from late spring to July. Although monthly population fluctuations correlated with indoor relative humidity, the population increases lagged one to two months behind the time that conditions first became favorable, while population declines correlated directly to the time that relative humidity levels fell below critical levels (47% to 50% RH for *D. farinae* and 60% to 65% RH for *D. pteronyssinus*). Arlian et al. (1982) also found significant seasonal fluctuations in the two-year study of 19 homes in Ohio, with highest densities of mites occurring during the humid summer months and lowest densities occurring during the drier late heating season. In the study by Korsgaard (1983a, 1983b), those apartments that had low absolute indoor humidities in the winter did not contain noticeable concentrations of mites in the summer and autumn despite the fact that the humidity conditions increased to levels that were high enough to support peak populations. This suggests that, in this case, winter conditions may have been severe and long enough to kill off even the dormant protonymph, assuming other eradication steps were not taken.

Based on a number of field studies it is also apparent that allergen levels correlate with seasonal variations and that changes in allergen levels lag behind both increases and decreases in indoor RH. Lintner and Brame (1993) studied 424 homes across the U.S. and found a distinct seasonal fluctuation of mite allergens for both *D. pteronyssinus* and *D. farinae* species, with the *D. pteronyssinus* allergens peaking in July and the *D. farinae* allergens peaking in September. In a study by Friedman et al. (1992) of homes in the upper Connecticut River Valley, there was a marked seasonal increase in total *D. pteronyssinus* allergens from June to September. In a one-year study of 12 homes in central Virginia, Platts-Mills et al. (1987a) found that increases in both mite populations and allergen levels lagged approximately one month behind increases in indoor humidity and that several months passed before a fall in allergen levels was detected after a drop in indoor humidity.

In all of the field studies cited, the highest concentrations of mites were found in mattresses, thick carpeting, and/or heavily used fabric-upholstered furniture. This suggests that mites thrive best within microenvironments that contain a source of food (shed skin scales) and have a relatively high and consistent moisture level. For example, bedding is a common site for mites because there is an ample supply of food and the humidity within an occupied bed is higher than that of the air of the surrounding space. This is also true of furniture upholstered with permeable fabric that can absorb and retain the moisture given off by an occupant. Field studies have found less variation over time in the number of mites within bedding as compared to other sites (Smith et al. 1985). Murray et al. (1979) also found significant numbers of mites in mattress dust during the winter even when the indoor RH fell below 50%.

Carpeting can also be a localized site of increased humidity and consequently may be an important reservoir for allergens in both homes and schools. Studies conducted in schools have demonstrated that carpets contain high levels of a variety of allergens including pollen, cat and dog dander, and mite and mold allergens (Fernandez-Caldas et al. 1994). This may be the primary source of exposure for young children, who generally live closer to the floor and do not have high exposures in bedding since they usually sleep on plastic-covered mattresses. Arlian (1992) studied the microenvironment of a carpeted floor and found that the relative humidity within the carpeting was 9.6% higher than that of the ambient air 1 to 2 meters above the floor. This was attributed to the decreased temperature of the floor (3.7°C lower on average than the ambient air), which drove the relative humidity up. In this study, Arlian also found that long-pile carpeting contained significantly more mites than short-pile carpets and tile or wooden floors (i.e., short-pile carpets did not contain significantly more mites than floors without carpets). This finding is also supported by a study by O'Rourke et al. (1993) in which house mites were found four times more frequently in homes with wall-to-wall carpets than in homes with other floor types.

Field Studies: Office Buildings

The few studies of mite populations that have been conducted within commercial buildings have shown that mite levels are generally low (Menzies et al. 1992). In a study of office buildings in the mid-Atlantic states, Hung et al. (1992) found moderate to high levels of mite allergens within carpeting and chairs of one of the five buildings studied. In a study of buildings in the New England area by Friendman et al. (1992), very low population levels of dust mites were found within the carpets of workplaces. The observation of low mite levels within commercial buildings may not be surprising since these buildings tend to be less humid than residences due to the frequent use of air conditioning and fewer internal sources of moisture (e.g., cooking, showering, etc.). In addition, commercial buildings do not usually contain bedding and thick carpeting, the most common sites for mites in residences. It has also been suggested that mites tend to be more common on ground floors than on upper stories and are rare in hotels (Reed et al. 1986).

Remediation

The strong correlation between indoor relative humidity and dust mite population has led to recommendations to reduce indoor humidity. However, exactly where the upper limit should lie is not obvious. Most of the field studies suggest that when indoor humidity is kept below 50% RH, mite populations do not grow to significant levels. Laboratory studies, on the other hand, in which the microenvironment of the mite is in equilibrium with the surrounding air, suggest that mite population growth and metabolism (related to the amount of allergen produced) can be significantly reduced if relative humidity is kept below 70% RH at 25°C (Arlian 1992).

One reason for the discrepancy between field and laboratory studies may be the difference in relative humidity between the mite's microhabitat, which can be considered to be within a few millimeters of the horizontal surfaces on which they lie, and that of the surrounding air due to differences in temperature as well as the ability of certain types of surfaces to retain moisture. The humidity measurements for the field studies cited were taken from the air within the core of the room, which may not correlate with the RH within the microenvironments from which the mite samples were taken. The time frame of the RH measurements was also not indicated for most of these field studies—instantaneous measurements taken at the time of sampling may not be representative of long-term conditions. In addition, seasonal changes in indoor humidity have a significant effect on mite populations and allergen levels, as suggested by the long-term field studies; however, the specific time constraints have not yet been resolved.

There are a number of effective remedial methods directed at reducing allergens and mites within their microhabitat in addition to control of relative humidity. These include specialized vacuuming procedures, removal of long-pile carpeting and heavily used upholstered furniture, regular hot-water cleaning of bedding, encasement of mattresses and pillows, and application of acaricides (Htut 1994). For example, in a study of laundry procedures, McDonald and Tovey (1992) found that all mites were killed by water at 55°C or higher. In a study by Platts-Mills et al. (1989), a tenfold or greater reduction in mite allergen levels was achieved in many houses by hot-washing all bedding at least every 10 days and removing carpets and upholstered furniture. Wickman et al. (1994a) found a significant decrease in mite allergen levels on mattress surfaces six months after they had been encased with a semipermeable polyurethane cover. Based on the observed seasonal effects for temperate climates, it seems that late winter and early spring are the best times to clean mattresses and carpets aggressively to kill the few mites that survived the winter. Theoretically, this should reduce the chances of having a large infestation in the summer months.

Vacuuming is effective only if central vacuuming systems, HEPA filters, or systems that entrain dust in a liquid medium are used. Conventional vacuuming does not help to reduce mite populations and allergens within carpets and can actually aggravate the problem. Allergen particles in the size range of the greatest health significance (< 2 μm) easily pass through the filter bags of conventional vacuums, causing a significant increase in the concentration of airborne allergens during and shortly after vacuuming (Kalra et al. 1990). In a two-year study of mites in 19 homes in Ohio, Arlian et al. (1982) found no significant correlation between the number of mites and the frequency or thoroughness of cleaning, amount of dust, or age of furnishings or dwelling. Arlian (1992) suggested that the ineffectiveness of cleaning may also be related to the difficulty in removing larval forms of mites adhering to surfaces.

Acaricides are now available that have been specially designed to eliminate mites from carpeting. One such product uses benzyl benzoate as the active ingredient. It is formulated as a moist powder with a wax to bind mite fragments and excrement so that they can be vacuumed. It is designed to be reapplied every six to eight months. Results from a number of studies suggest that this product has been successful in reducing mite populations (Htut 1994). Benzyl benzoate was initially marketed in Europe and has been approved for use in all states in the U.S. except California (CL 1994). Fungicides such as natamycin kill the fungi required by mites to digest lipids in the skin scales and have also been used with some success (Flannigan 1992; Platts-Mills et al. 1987b; Htut 1994); however, in one study in which a double-blind, placebo-controlled method was used, no significant improvement was observed (Reiser et al. 1990). Other surface treatments that have been used include liquid nitrogen, benzyl benzoate in combination with tannic acid, and benzoic acid (Htut 1994).

Other possible methods of reducing mite levels include the use of electric blankets, which can reduce the local humidity within bedding (Hart and Whitehead 1990), and dehumidification/air conditioning (Lintner et al. 1993). According to a study by de Boer and van der Geest (1990), a reduction in dust mite populations of 19% to 84% can be achieved by heating the mattresses with electric blankets when the beds are not in use. In a field study by Cabrera et al. (1995), dust mite allergens were reduced by more than 50% with the use of a dehumidifier.

Improved ventilation systems within homes can also help reduce mite levels by counteracting internal sources of humidity, such as cooking and showering, in climates where outdoor humidity is not the major source of moisture. Wickman et al. (1991) suggest that house dust mite infestation used to be rare in Stockholm; however, mite-sensitive children are now frequently observed, which may indicate an increased infestation rate. The authors attribute this to a reduction in the ventilation rate resulting from the energy conservation programs. In a follow-up study, Wickman et al. (1994b) looked at the concentration of dust mites in 70 homes in Stockholm belonging to two major house types—those with crawlspace basements and those with concrete floor slabs—and determined that the highest risk factors for allergen concentration exceeding the median were unimproved natural ventilation (i.e., no mechanical exhaust), concrete floor slabs, and condensation on windows. In a study of Danish homes, Harving et al. (1994) found that decreases in indoor humidity levels through

the use of supply-and-exhaust ventilation systems significantly reduced dust mite levels.

FUNGI

Introduction

Fungi (via airborne fungal spores, fragments of hyphal mat, and metabolic by-products) have been linked to a number of adverse health effects, including allergic reactions, hypersensitivity pneumonitis, mycotoxicosis, and pathogenic disease. In general, however, fewer people are allergic to fungi than to dust mites and animal dander (Flannigan et al. 1991). Beaumont et al. (1985) demonstrated that many more respiratory patients with suspected allergies react to animal dander (34%) and house dust (44%) than to molds (3%). The most common genera known to cause asthma and rhinitis include *Alternaria, Aspergillus, Cladosporium,* and *Penicillium* (Flannigan and Miller n.d.). A few genera, such as *Aspergillus* (*A. niger* and *A. fumigatus*), *Histoplasma, and Cryptococcus,* are pathogenic and can infect the lungs, ears, or eyes in susceptible hosts; however, reported cases are relatively rare (Gravesen 1979; Flannigan 1992; Miller 1992). Metabolic gases produced by fungi contain volatile organic compounds (VOCs) that are responsible for the mildew odor. These VOCs may be a contributing factor to sick-building-type symptoms, including eye, nose, and throat irritation; headache; and fatigue (Bjurman 1993). In a study of microbially contaminated buildings, VOCs of the type commonly associated with indoor man-made materials were actually found to be the metabolic by-products of fungi growing in the buildings (Bayer et al. 1993). High indoor spore levels of fungi such as *Cladosporium* and the dry rot fungus *Sepula* have been associated with cases of hypersensitivity pneumonitis. Fungal spores and vegetative mycelium are also known to contain toxic substances (mycotoxins) that can lead to respiratory symptoms unrelated to allergic mechanisms (Flannigan and Miller n.d.). Flannigan et al. (1991) list a number of toxigenic species isolated from the indoor air of houses.

Most fungi originate outdoors and are saprophytic (i.e., grow on substrates of dead or dying plant and animal matter). Outdoor concentrations vary with the season, the time of day, local weather conditions, and whether the site is rural or urban. For example, phylloplane (leaf-loving) fungi, which include *Cladosporium* and *Alternaria*, are more common in rural areas and show a strong seasonal activity with peak concentrations in the summer. *Penicillium* and *Aspergillus* are the most common soil fungi found in urban environments, and airborne spore concentrations of these species remain relatively constant throughout the year (Brundrett 1990). Fungal spores are typically in the range of 3 to 30 μm in diameter and, once they are released into the air, can travel intercontinental distances. Airborne spores enter buildings through ventilation equipment and can set up colonies on surfaces where moisture and nutrient conditions are favorable.

Buildings with no internal sources of fungi have nearly the same proportion of fungal species as outdoor air, with total counts reduced due to settling and filtration within air-conditioning equipment. In a study of Canadian office buildings, Miller (1992) found that those buildings not associated with microbial problems had micofloral counts that were qualitatively similar and quantitatively lower than those of outdoor air, while contaminated buildings tended to have a higher proportion of nonphylloplane fungi, particularly *Penicillium and Aspergillus.* In a study of fungal concentrations within daycare centers and dwellings, Hyvarinen et al. (1993) found that the total concentration of airborne fungal spores was higher in moldy buildings. In addition, the concentrations of *Aspergillus* and *Oidoidendron* in the fall and *Aspergillus* and *Penicillium* in the winter were higher in the buildings with mold problems than in the reference buildings. The presence of wet-habitat fungi, such as *Phoma, Stachybotrys, Trichoderma,* and *Ulocladium,* in significant quantities suggests the existence of either rotting vegetation near the air intake or an extremely damp amplification site within the building (Flannigan 1992). Xerophilic species, including the toxigenic species *Penicillium auranteogriseum* and *Aspergillus versicolor,* can form an appreciable percentage of the population within indoor dust samples. These had not been widely detected until the recent use of new sampling methods designed for detection of xerophilic species (Miller 1992).

Environmental Requirements

Fungi need water, carbon, and nitrogen for growth, as well as minute amounts of other nutrients normally present in natural environments. Typical construction materials containing nutrients used by fungi include wood, cellulose, wallpaper, organic insulation materials, textiles (especially natural fibers), and glues and paints containing carbohydrates or proteins. Although materials such as metal, concrete, plastics, fiberglass, and other synthetic products cannot be used directly by fungi, they can collect organic debris that serve as a nutrient source for fungi. For example, despite air filtration, some dust containing living microorganisms passes through air-handling units and settles on porous insulation within ducts. If this insulation material then becomes wet (e.g., due to condensation), fungi will grow and release spores into the ventilation air (Morey et al. 1991; Pasanen et al. 1993).

Fungi acquire most of their nutrients through a solvent process (Griffin, 1981). Thus the moisture on and within a substrate is the important factor determining fungal growth rather than the moisture of the ambient air (Block 1953). Laboratory studies support this observation. For example, Pasanen et al. (1991) measured colony diameters for both *Penicillium* sp. and *Aspergillus fumigatus* as a function of RH in the range of 11% to 92% and found that fungal colonies grew on wet substrates even at low levels of atmospheric humidity. The authors conclude that growth is dependent on substrate moisture and is not directly affected by atmospheric moisture. Systems containing water, such as the water reservoirs of humidifiers, favor the growth of bacteria, algae, protozoa, and certain types of fungi, especially yeasts. Most fungi, however,

prefer surfaces of moist materials to liquid water (Pasanen et al. 1992). Thus, since nutrients and airborne fungal spores are abundant within buildings, the availability of moisture on and within surfaces appears to be the limiting factor for growth.

Fungi are able to withstand dry periods to some extent by becoming dormant or by utilizing metabolically generated water that they add to the substrate. For example, wood will not decay if the moisture content is less than 20% to 25% of its dry weight, except when it has been invaded by a dry-rot fungus such as *Merulius lacrymans*, which is able to translocate water (Moore-Landecker 1982). Fungi that inhabit soil and wood grow better in moderate rather than high moisture contents since soil aeration (and therefore oxygen supply) is limited when the moisture content is high (Moore-Landecker 1982).

In general, fungi can grow at temperatures between 0°C and 40°C. Below temperatures of 0°C the fungi may survive but will not continue to grow, and above temperatures of 40°C fungi cannot survive for long periods (TenWolde and Rose 1993). Temperature variations within the range found in most conditioned buildings do not appear to be a limiting factor but may affect growth rates. For example, the study by Pasanen et al. (1992) demonstrated that both *Penicillium* sp. and *Aspergillus fumigatus* grew at all temperatures from 10°C to 30°C, with *Aspergillus* growing fastest at 30°C and *Penicillium* growing fastest around 20°C.

The potential for fungal growth on a substrate has often been attributed to moisture content (MC), defined as the ratio of "free water" in the material to the material's dry weight after being dried in an oven (free water refers to water held in a porous material by van der Waals forces [i.e., hydrogen bonding] or capillary attraction, as opposed to water of hydration, which is chemically bound to the materials). For wood products, percent moisture content is defined as the weight of removed water divided by the weight of oven-dried wood. Wood consists of hygroscopic cell walls surrounding cellular spaces filled with water and/or air. Below fiber saturation, when the cell walls are fully hydrated yet have no water contained in the cellular spaces, fungal growth is inhibited since the fungi are not able to readily extract the water held by van der Waals forces (Wilcox 1995). The fiber saturation point for wood occurs at MCs of 25% to 30%.

Although moisture content is commonly mentioned, it is not the most appropriate measure of a substrate's potential to support fungus. This is because materials differ in how tightly they hold free water, and the measurement of moisture content may vary depending on the procedure used. For example, Block (1953) evaluated fungal growth on a number of different materials, including leather, wood, cheese, wool, and cotton, and found a common mold growth threshold value of about MC = 0.1, which has often been quoted in the literature. This is significantly below the fiber saturation point for wood. More recently, Foarde et al. (1993) demonstrated a critical MC of 0.055 to 0.065 for *Penicillium* growing in porous ceiling tiles.

It has been suggested that for biological purposes the more physically meaningful parameter is water activity, a_w, defined as the ratio of the water vapor pressure at the surface of the moist material to that of a pure liquid water surface at the same temperature and pressure (Ayerst 1969; Griffin 1981; Flannigan 1992). This is also referred to as the *equivalent relative humidity* (ERH) when written in the form of a percentage (i.e., an a_w of 0.80 is the same as an ERH of 80%). ERH is equal to RH at the surface of the material only when the system is confined to the extent that the atmosphere above a moist surface is at the same vapor pressure and temperature as that directly at the moist surface. In actual environments, however, there is usually a gradient of vapor pressure from the surface into the air above or vice versa. In this case, the relative humidity of the air has only an indirect influence through drying and moistening of the materials it contacts.

In general, favorable conditions for fungal growth depend on the species and the type of substrate on which it is growing. In addition, fungal growth does not occur in isolation but rather within a complex microbiological system that includes yeasts and bacteria as well as molds. The following excerpt from Flannigan (1992) presents his ecological classification of molds in terms of their moisture requirements, and describes the process by which different types of molds take hold and grow.

Although all molds growing on surfaces in buildings grow most rapidly in pure culture at a high a_w ..., individual species can be classified as:

- Primary colonizers, which are able to grow at an a_w of less than 0.80 and also are referred to as xerophilic fungi because they are able to grow at lower a_w than other molds, e.g., species in the *Aspergillus glaucus* group (*A. amstelodami, A. repens*, etc.), *A versicolor*, and *Penicillium brevicompactum.*
- Secondary colonizers, which are able to grow at an a_w of 0.80-0.90, e.g., *Cladosporium cladosporoides* and *C. sphaerospermum.*
- Tertiary colonizers, which are only able to grow at an a_w more than 0.90, e.g., *Alternaria alternata, Phoma herbarum, Ulocladium consortiale,* and *Stachybotrys atra.*

Where there is ingress of water over a restricted area, e.g., as a result of rain water penetrating via a structural fault in a wall, tertiary colonizers may be found at or near the site of ingress and primary colonizers, such as *A. versicolor* and *Penicillium*, at the less wet margins of the affected area (Grant et al. 1989). Pasanen et al. (1992) found that *Aspergilli* and *Penicillia* (primary colonizers) grew under all conditions when samples of timber, plywood, gypsum board, fiberboard, and wallpaper were incubated in atmospheres of 75-76% RH and above, but species of *Cladosporium* (secondary colonizers) and *Stachybotrys* and *Trichoderma* (tertiary colonizers) only developed at the highest RH, where the substrate a_w would be 0.96-0.98. The degree of dampness determines what species are able to grow and sporulate, and therefore strongly influences the composition of the spora in indoor air.

This statement agrees with the findings of Kalliokoski et al. (1993), who carried out a controlled-chamber study of fungal growth on a number of moisture-damaged building

materials. Based on this study, the authors suggest that fungal growth is dependent on temperature, composition, and hygroscopicity of materials and fungal species and is likely when the ERH exceeds 76% to 96%. The growth rate of a xerophilic fungi from a number of different ecological sites and contaminated materials was studied in the laboratory by Avari and Allsopp (1983). Optimum ranges for growth were found to be between a_w levels of 0.97 and 0.90. For all of the species studied, no growth was observed after one month at a_w levels of approximately 0.80. This study is in agreement with the recommendation by the International Energy Agency (Annex 14) that the monthly average water activity of a material surface should not exceed 0.8 (IEA 1991). This recommendation recognizes the importance of the surface microclimate and was developed in response to the request from building professionals for a simple criterion by which to judge the likelihood for mold growth.

Field Studies

The most common cause of fungal spore contamination within residences is condensation on surfaces and reoccurring spills or leaks (Seltzer 1995). Besides superficial condensation, interstitial condensation within porous building materials (such as concrete, brick, and/or plaster) may provide a reservoir for fungal growth. Interior dampness problems are usually related to construction faults, such as inadequate insulation and thermal bridges, in combination with inadequate ventilation and/or the pattern of use within homes. For example, in a study of 86 newly built energy-efficient residences in the Pacific Northwest, Tsongas (1991) found that one-third had mold growing on indoor wall surfaces and one-third had mold on window frames and/or sills. Although homes were well insulated, condensation still occurred due to internal sources of humidity that were not properly ventilated. Nevalainen et al. (1991) studied residences in Finland, where microbial problems in buildings are relatively uncommon due to the heating and insulation requirements of a cold climate. They found that most of the houses with mold problems had improperly weatherproofed outside walls, which allowed rainfall into the insulation material, and/or inadequate drainage that allowed moisture to penetrate in through the floor. Becker (1984) conducted a post-occupancy evaluation of 200 homes in a coastal region of Israel, which has mild winters. He concluded that condensation was the main source of fungal growth and that the major factors contributing to mold problems were location and orientation of the dwelling (affecting wall surface temperatures), occupant density, cooking habits, and the type of paint or wall covering. Abe (1993) developed a biosensor method, using a xerophilic fungus, to study the potential for growth within different microenvironments of a Japanese apartment. A significant variation among and within rooms was evident, with lower potentials for growth observed in spaces with walls adjacent to internal spaces and highest potentials observed at cold corners of northern and eastern walls adjacent to the outside.

The most extensive fungal contamination problems occur in hot, humid climates; control of indoor humidity in these regions is an important factor. Bayer and Downing (1992) observed fungal contamination in schools in a climate where outdoor humidities ranged from 75% to 90% for most of the year. High indoor humidities resulted in visible mold growth on ceiling tiles, fan-coil units, papers, and books. In one case, carpeting adhesive was not able to cure in the highly humid conditions and provided a medium for microbial growth. Effective remediation procedures focused on removing and cleaning contaminated materials and controlling indoor humidity levels.

Within commercial buildings, microbiological contamination is frequently a result of the absence of or inadequate preventive maintenance of conditioning systems. Morey (1988) evaluated the occupied space and heating, ventilating, and air-conditioning (HVAC) systems of 21 commercial buildings for the presence of microbiological reservoirs and amplification sites. Microbiological growth was detected in 18 of the 21 buildings. In nine of the buildings, contamination was found in the porous duct insulation. Other significant sources included stagnant water in drain pans (10 buildings), excessive relative humidity (6 buildings), flood damage (6 buildings), and the location of outdoor air intakes near external bioaerosol sources (6 buildings). Ezeonu et al. (1994) demonstrated that the fiberglass duct liners and boards from eight buildings whose occupants complained of unacceptable or moldy odors were heavily colonized by fungi, particularly by *Aspergillus versicolor.*

Remediation

The most effective remediation procedures depend on the source of the contamination and regional climatic conditions. Control of indoor humidity is an important factor in hot, humid climates; however, for other climatic types other means of controlling moisture, such as insulation to keep interior surfaces above the dew point, proper placement of vapor barriers to control vapor and airflow between indoors and outdoors, and control of external rain and groundwater penetration into the building, may be more critical. Proper maintenance of HVAC equipment is also an important factor, particularly in commercial buildings. This may require design innovations focused on improving accessibility and maintenance procedures. These issues are discussed in greater detail in part two of this paper.

Once a material has become contaminated, it is almost impossible to completely eliminate the fungi and removal is often the only option. In a recent study of microbial growth in chipboard, Thogersen et al. (1993) found that water damage resulted in massive growth and that, even after drying, the material still contained spores. Use of biocides is usually discouraged, since most are toxic and continuous use may increase corrosion and encourage the development of resistant strains (Nevalainen 1993). Bleach treatment has been unsuccessful in cleaning contaminated duct liners (Morey 1988).

BACTERIA AND VIRUSES

Introduction

Most viral and bacterial respiratory infections are transmitted among human hosts. This may occur by touching an infected person or an object they have infected or by inhaling contaminated airborne droplets expelled from the nose and mouth during sneezing, coughing, and talking. Most of these airborne droplets are large enough to fall to the ground within a meter. Smaller droplets quickly shrink through desiccation to form "droplet nuclei," which are small enough (between 0.5 and 5 μm in diameter) to remain suspended in the air for long periods (LaForce 1986). Droplets within this range are of the greatest important when considering health effects since they are small enough to penetrate deep into the lungs. If these droplets contain viable infectious organisms and are in sufficient numbers, they will cause infection in susceptible hosts. Relative humidity can affect the desiccation process of droplets, which, in turn, affects droplet size and the viability and infectivity of the airborne pathogens (Burge et al. 1991). For more information concerning specific types of infectious disease, see Burge (1989, 1995).

Airborne Viability

Most of the information concerning the viability of airborne pathogens has been determined through *in vitro* studies. Evidence from these studies suggests that the humidity level has complex effects on the viability and virulence of airborne pathogens that vary from organism to organism, while the effect of temperature is not significant within the range of interest for conditioned environments. In many cases certain bacteria were found to have a window of relative humidity at which they died more quickly (Anderson et al. 1968; Cox 1966; Brundrett 1990). For example, Cox (1966) found that *Escherichia coli* strain jepp had minimal viability in the range of 70% to 80% RH and that the viability increased at RH values above and below this window. The results of a study by Hambleton et al. (1983) suggest that *Legionella pneumophila* has minimal viability at two humidity levels—between 50% and 60% RH and at 30% RH. Hambleton et al. also demonstrated that at the optimum humidity of 65% RH, only about 20% of the cells are viable after one hour. Experiments on the bacterium *Pneumoccus* suggest a sharp decrease in viability in a narrow band at 50% RH (Brundrett 1990). In a study of the survival of *Streptococcus*, Flynn et al. (1971) found that the change in the viable count was insignificant for the RH range of 0% to 92%.

In vitro studies of viruses suggest that particular strains, including mengovirus 37A, polio virus, foot-and-mouth disease virus, and encophalomyocarditis virus, are unstable as aerosols in atmospheres below about 70% RH (Cox 1989). In contrast to these viruses, other strains, including vesicular stomatitis, vaccinia, and influenza, are least stable at high RH. Cox suggests that this difference can be attributed to specific structural differences in the viruses. Mbithi et al. (1991) studied the survival of hepatitis A virus on surfaces at RH levels of 25%, 55%, 80%, and 95% and found that the survival of the virus was inversely proportional to the level of RH and temperature.

Expelled droplets and skin flakes that settle out may survive in dust and transmit disease if re-entrained when surfaces are disturbed. There is evidence that outbreaks of bacterial infections in hospitals have been associated with cleaning processes (Brundrett 1990). Studies of viability of bacteria in dust suggest that there is a trend of decreasing viability with increasing relative humidity (Brundrett 1990). In addition, dust is less likely to become airborne at higher humidities.

Field studies of airborne pathogen survival at high humidities are limited. Studies at lower humidities suggest a higher survival rate for airborne viruses at humidities below 30% (Green 1985). In general, considering the results from *in vitro* studies, there is little evidence to suggest that for humidities in the upper range (> 50% RH) one specific level is better than any other in reducing the viability or number of suspended microorganisms.

Building-Related Sources

A few microbes, pathogenic to humans, are able to flourish in nonhuman environments. These can be introduced into building systems from outside sources and proliferate if conditions are favorable. The most important example of such a contaminant is *Legionella,* which can lead to fatal pneumonia in susceptible hosts. Aside from Legionnaire's disease, no specific infections have been documented to be of great clinical importance in commercial buildings (Hodgson 1989).

Hypersensitivity pneumonitis has been directly associated with microorganisms (particularly thermophilic actinomycetes) cultured from poorly maintained humidification and air-conditioning systems (Hodges et al. 1974; Fink et al. 1976; Burge et al. 1980). Patients often report a relief of symptoms upon avoidance of the environment containing the offending contaminant (LaForce 1986). Outbreaks of bacterial infections in hospitals and flare-ups of asthma have also been associated with humidification systems (Covelli et al. 1973; Airoldi et al. 1972; Smith et al. 1977; Solomon 1974; Bencko et al. 1993). In all these cases the offending humidifiers have been of the spray or mist types that form aerosols in the airstream.

Use of direct evaporative cooling is a potential concern because poor maintenance, which is not uncommon in residential systems, can result in microbial growth within sump water (Macher and Girman 1990; Stetzenbach et al. 1990; Macher 1994). However, it is not apparent that this could lead to an outbreak of disease. One study of homes in the Las Vegas area cooled by direct evaporative systems traced the presence of gram-negative bacteria to a fouled sump in one of the homes, although none of the dwellers was infected (Stetzenbach et al. 1990). In a laboratory study using tracer organisms, Macher (1994) found "minimal transfer" from the fouled sump into the air under normal operating conditions. Conversely, direct evaporative cooling may help to reduce human-to-human spread of infectious disease because of the relatively high supply rate of

outside air required. Increased ventilation has been shown to lead to decreased rates of viral respiratory infections (Burge et al. 1991) and is often a recommended means of reducing indoor air contaminants (ASHRAE 1989).

NONBIOLOGICAL AIR POLLUTANTS

Formaldehyde

Formaldehyde is found in numerous building materials, including plywood, particleboard, and other pressed wood products; furnishings; carpets; and textiles. It is also a major component of urea-formaldehyde foam insulation, which is now banned in the U.S. Formaldehyde is highly water soluble and causes irritation of the mucous membranes within the eyes and upper respiratory tract at concentrations starting at 0.1 ppm but is most frequently reported at or above 1 ppm (Berglund et al. 1992). Formaldehyde is also classified by the U.S. Environmental Protection Agency (EPA) as a probable carcinogen. Both ASHRAE and the World Health Organization (WHO) have set maximum guidelines of 0.1 mg/m^3 to ensure sufficiently low formaldehyde concentrations in indoor air (Puhakka and Karkkainen 1993; Gammage 1990). The effect of these standards, along with the ban on urea-formaldehyde foam insulation, has been an overall decrease in indoor formaldehyde concentrations within the last decade (Marbury and Krieger 1991).

The rate of formaldehyde offgassing from pressed-wood products decreases exponentially with age and is sensitive to a number of factors, including the initial properties of the material, temperature, and humidity (Gammage 1990; Meyer 1986). Laboratory and field studies agree that temperature is the most significant environmental effect (van Netten et al. 1989; Godish and Rouch 1986; Arundel et al. 1992). In general, since formaldehyde within binding resins is water soluble, higher humidity levels also tend to increase the offgassing rate. In a controlled environment study within mobile homes, a decrease in humidity from 70% to 30% resulted in an approximate 40% reduction of formaldehyde levels (Godish and Rouch 1986). In a study of formaldehyde offgassing from chipboard within a controlled chamber, Anderson et al. (1975) found that the formaldehyde concentration within the air was directly proportional to temperature and air humidity. A change in relative humidity from 30% to 70% doubled the equilibrium formaldehyde concentration, while a 7°C rise in temperature in the range of 14°C to 35°C caused the formaldehyde concentration to double. In a study of 20 homes referred to by Arundel et al. (1992), a significant correlation was found between the indoor relative humidity and the formaldehyde concentration in the air.

Seasonal fluctuations have also been observed, with the highest rates of offgassing occurring in summer months, when marked increases in temperature, solar gains (which can cause localized increases in wall temperature), and humidity occur (Marbury and Krieger 1991). Puhakka et al. (1993) studied 46 apartments in Finland and found that the concentration of formaldehyde in the air increased from 0.08 mg/m^3 to 0.20 mg/m^3 when the relative humidity increased from 34% to 70% during a period of 24 hours. In addition, they found that short-term increases in relative humidity within a period of one to five hours, as occur when using a sauna or drying clothes, also caused increases in formaldehyde levels. The offgassing rate of chipboard within a chamber was also examined in this study and it was found that sealing chipboard on all sides with "reactive paint" significantly reduced offgassing rates.

Ozone

Ozone is well recognized as a respiratory irritant and a significant problem in urban areas of southern California, where the EPA standard for outdoor ozone concentration is often exceeded. Ozone is formed as a result of reactions between photochemically reactive hydrocarbons and oxides of nitrogen under the influence of sunlight. Because of its strongly oxidizing properties and low water solubility, ozone can penetrate deep into the alveoli of the lungs and affect lung function. It is also an irritant to the mucous membranes of the eyes.

The primary source of ozone indoors is infiltration/ventilation of polluted outdoor air. Indoor sources of ozone, such as photocopiers and air-cleaning equipment, exist; however, under normal living and working conditions there is no evidence that these would reach levels high enough to be of concern. Once indoors, ozone decomposes through heterogeneous reaction with indoor surfaces. Mueller et al. (1973) estimate the half-life of ozone in a typical bedroom to be less than 6 minutes, while Weschler et al. (1991) suggest a half-life estimate of 11.1 minutes for a typical office environment with a surface-to-volume ratio of 2.9 m^{-1}. Using this general knowledge, authorities have typically advised the public to remain indoors and reduce infiltration as much as possible during episodes of high outdoor ozone levels. However, this recommendation is not useful for those buildings that depend on natural ventilation or evaporative cooling as a means of cooling. This condition is made more problematic by the fact that ozone episodes often correspond with high outdoor temperatures.

In the study by Weschler et al. (1991), ozone concentrations were measured for three office buildings with different ventilation rates. The indoor values closely tracked those of the outdoor values and, depending on the ventilation rate, were 20% to 80% of those outdoors. Weschler et al. also cite a number of previous studies in which the concentration of ozone in the indoor environment was measured to be 10% to 80% of outdoor concentrations. Considering this information, along with the fact that people spend more than 90% of their time indoors, Weschler suggests that exposure (concentration × time) to ozone indoors is a more significant issue than outdoor exposures.

There is little information regarding the effect of humidity on ambient ozone concentrations within indoor environments. One controlled-chamber study found that the rate of ozone decay increased as either temperature or humidity was increased (Mueller et al. 1973). However, in the case of direct

evaporative cooling, this may not be a significant effect compared to that of the higher ventilation rate. The impact of direct evaporative cooling vs. refrigerated air conditioning was studied by Stock et al. (1993) in homes in El Paso and Houston, Texas. They found that the average indoor/outdoor ratio of ozone concentrations was much higher in homes with evaporative cooling compared to those with refrigerated air-conditioning systems (0.7 vs. 0.1).

The most promising remediation method available is the use of activated carbon filters, which have been shown to be successful in significantly reducing ozone levels (Mueller et al. 1973; Weschler et al. 1991). It may be possible that these could be incorporated into evaporative coolers. However, such factors as engineering practicality, cost, efficiency, and service life would need to be tested under actual conditions to determine if this option is viable.

Nitrogen and Sulfur Oxides

As with ozone, nitrogen and sulfur oxides are produced primarily from outdoor sources. However, nitrogen dioxide and nitrous and nitric acids are also combustion by-products of gas cooking stoves and heaters and can accumulate indoors as a result of improper ventilation. Nitrogen and sulfur oxides react with water on indoor surfaces to form acid aerosols, which are generally found in higher concentrations indoors (Leaderer et al. 1993). Although nitrogen and sulfur oxides are common pollutants, surprisingly little work has been done thus far to determine the respiratory toxicity of their acid aerosols. Their acidic nature, reactivity, and aqueous solubility, however, suggest that respiratory damage is possible (Brauer et al. 1993) and increased indoor humidity does seem to increase the heterogeneous reaction on surfaces. In one chamber study it was found that at the highest relative humidity tested (80%), nitrous acid (HONO) concentrations were approximately 8% of the observed NO_2 level, while 30% and 45% relative humidities resulted in $HONO/NO_2$ ratios of 0.9% and 2.7%, respectively (Brauer et al. 1993). In terms of direct evaporative cooling, again the issue of high ventilation rates may be a significant factor to consider if outdoor air levels of nitrogen and sulfur oxides are high.

DISCUSSION

To the maximum extent possible, building standards should reflect the knowledge available when they are written. The subject of humidity limits is a complex one and many questions remain at this time. Nonetheless, there is a wide range of research under way through numerous disciplines, much of which can inform the preparation of standards. This paper summarizes this research and suggests a format for putting new information into a building standards context.

A paper by Sterling et al. (1985) also addresses the topic of humidity and health in buildings and is the only cited reference pertinent to humidity in ASHRAE Standard 62-1989. This paper includes a figure that has received wide circulation within the HVAC engineering profession. It graphically depicts humidity impact zones using bars that decrease in width, suggesting a decrease in the effect for each of the eight environmental health factors addressed. These bars converge for all of the eight categories into a narrow recommended "optimum" zone between 40% and 60% RH; both low- and high-humidity effects are addressed. There is clearly a need for such a summary because it has appeared in numerous journals and conferences (Arundel et al. 1986, 1992). It is also arrestingly drawn and easy to grasp, which adds to its appeal. This figure is the basis of the recommendation in ASHRAE Standard 62-1989 that humidity in the occupied space should be between 30% and 60% RH.

There are issues that may be raised with a figure that attempts to combine the influences of so many factors. One concern is that it does not assign relative weights (severities) for the different health factors. Another is that the practicality of the recommended humidity limits in various climates and building types is not assessed. In practice, these issues need to be addressed. As an example, ASHRAE Standard 62 overrides (without explanation) the recommended lower limit of 40%, lowering it to 30% although, based on the figure, the impact of this action is not much different than that of raising the upper limit from 60% to 70%. This may have represented a value judgment by ASHRAE about the relative severity of the different health effects—differences that are not expressed in the figure. Conversely, the change may have been forced by practical reality in conditioning the indoor environments of buildings.

In defense of the figure, it is ultimately necessary for designers to make decisions combining disparate elements even without complete justification. Someone needs to draw a line in the sand. The figure does this for them.

A more severe criticism is based on the factual substantiation of the health impact zones presented. As the figure is drawn and captioned, these zones imply linear relationships between humidity and health effects that are not supported by the literature. In addition, the focus on ambient RH to the exclusion of other environmental conditions is misleading in that it suggests that RH is the controlling environmental factor for all of the pollutants listed without regard to climate and the conditions of the building system.

In addition, the specific points at which these zones begin and end are not consistently supported by the references provided. The recommendations for bacteria (RH below 60%), viruses (RH below 70%), and fungi (RH below 60%) are not supported by the discussion or the references given in the original paper or its more recent versions. The literature cited for the mite recommendation (below 50% RH) is incomplete, and seasonal effects, which may be a significant factor, are not addressed. The limits for allergic rhinitis are presumably based on the potential for mites and fungal growth (for upper humidity limits), although most of the discussion concerns problems of mist humidifiers and low humidities. The chemical interactions category (RH below 30%) appears to be based entirely on two studies of formaldehyde offgassing rates (the

only other chemical interaction mentioned in the text is the conversion of sulfur and nitrogen dioxides to acids, for which no specific limits were cited). In light of the recent reductions in the formaldehyde levels within materials, this limit may be outdated. The use of the term "ozone production" as a pollutant category is confusing and possibly based on the incorrect assumption that ozone production from office lighting and equipment is a significant pollutant source.

For the purpose of setting humidity standards, the figure is clearly inadequate. To promote good building design it is important to identify the specific physical causes and solutions to health hazards and to regulate design practice to avoid them.

Table 1 summarizes the results of this review. It addresses the humidity requirements, the actual site of contamination, and the means of control for each of the biological pollutants.

CONCLUSIONS

- Most of the identified biological health agents grow on the surfaces of the building, its systems, and its furnishings, or in standing water within or outside the building. None of the agents grows in the air of the occupied space or the mechanical system. Their growth is therefore only indirectly related to the atmospheric humidity measured in the occupied space or the ducts of its mechanical system. To control these, one needs to ensure that the surfaces remain dry. There are a number of ways to achieve this in the design, furnishing, and operation of buildings. It is also necessary to avoid producing aerosols of water from the mechanical system or humidifiers. How this is done is independent of the level of indoor air humidity.
- The single exception to this is dust mite contamination (particularly *D. farinae*), which appears to be directly related to ambient RH. Controlled laboratory studies suggest that optimal conditions for growth are 70% to 80% RH at 25°C. A number of field studies have found mite contamination in residences with ambient RHs as low as 50%. Rather than ambient RH, however, the more relevant factor may be the RH within the microhabitat of the mite (within a few millimeters of the horizontal surfaces on which they lie). In the laboratory, surface and air temperatures can be controlled to provide equilibrium conditions; however, in actual environments equilibrium conditions seldom exist. In one field study, the RH within carpets was found to be more than 9% higher than the space RH, suggesting lower temperatures or local moisture production that may benefit mites. This difference between surface and ambient RH may possibly explain the discrepancy between the laboratory and field findings. Remedial methods are designed to address contamination at the source. For example, one of the most widely recommended remedial methods is to encase mattresses with a semipermeable polyurethane cover. This discourages moisture from getting into the bedding where the mites live. Researchers have also suggested that electric blankets, which can lower the RH within bedding, are effective in reducing mite levels. In terms of seasonal effects, it is not yet known whether use of evaporative cooling a few months out of the year, sometimes only during the day, leads to increased mites. In general, since mite contamination occurs primarily in residences and affects only a subset of the population, it may be that, when necessary, they should be controlled by other means such as cleaning and covering bedding, treatment or removal of carpets, and insulation of cooled floor slabs under carpets.
- Fungal contamination occurs primarily as a result of condensation on surfaces and/or water damage. Field and laboratory studies suggest that fungal growth does not become an issue below 70% or even 80% RH unless there are other factors influencing their growth on building surfaces. Studies that reported problems at lower RH values appeared to have problems that could be corrected otherwise. In setting a maximum limit to air humidity in the space, there is little, if any, evidence from field studies that provides a reason for distinguishing 60% relative humidity from 70%.
- The health impacts of nonbiological health agents are hard to assess at this time. Formaldehyde generation is exacerbated in some materials by higher humidity. Because of the greater awareness of the adverse health effects, new building products and furnishings generate far less formaldehyde than before. The effect of this change will need to be evaluated. For a given ventilation rate, indoor ozone concentrations may decrease as humidity increases due to an increased rate of surface reactions. However, at the high ventilation rates associated with direct evaporative cooling, the level of ozone will not be significantly offset by the higher humidity levels. Oxides of nitrogen and sulfur are primarily of outdoor origin, but the severity of their effects on health may increase with higher humidity levels. At this point there is little evidence in the literature to suggest that this is a significant health effect.
- The other significant source of biological health agents is humans harboring infectious diseases. This source (primarily the respiratory tract but also the skin) is largely independent of the humidity level in the space. However, the spread of infectious disease agents depends somewhat on atmospheric humidity in the space, in that aerosol evaporation rates and deposition rates may affect viability of antigens, bacteria, and viruses enclosed in water droplets. Space humidity may also affect the settling rate of dust particles to which bacteria are attached. The viability of these dustborne organisms also varies with humidity, with viability optima occurring throughout the range of RH. For each of the above considerations, there is little evidence to suggest that any humidity between 50% and 90% is significantly better than any other in reducing the viability or number of suspended infectious disease organisms, as well as the susceptibility of the human receptor.

- Direct evaporative cooling through porous media appears to be benign in that field and laboratory studies suggest that biological organisms in the cooling water appear not to be aerosolized or transmitted downstream. The wet pads may have benefits over dry filters in removing incoming pollutants. However, this needs to be experimentally investigated. In addition, the once-through ventilation required by such systems should act to dissipate the concentration of infectious organisms in the air, since such organisms are almost always internally generated. This process also needs to be systematically evaluated.
- Finally, very little of the literature on health effects is expressed in terms of risk to the occupant: first, the likelihood of humidity-influenced pollutants occurring in the building and then the likelihood of the pollutant affecting the occupant.

ACKNOWLEDGMENTS

The authors would like to thank Alison Kwok, a Ph.D. candidate at the University of California at Berkeley, for help with the literature search. We also wish to thank William Fisk of the Indoor Environment Program at Lawrence Berkeley Laboratory and Professor Wayne Wilcox of the University of California Forest Products Laboratory for their detailed reviews of the manuscript. The research reported here was funded by the California Institute for Energy Efficiency (CIEE), a research unit of the University of California. Publication of the research does not imply CIEE endorsement of or agreement with these findings, nor that of any CIEE sponsor.

REFERENCES

Abe, K. 1993. A method for numerical characterization of indoor climates by a biosensor using xerophilic fungus. *Indoor Air* 3: 344-348.

Airoldi, T., and W. Litsky. 1972. Factors contributing to the microbial contamination of cold-water humidifiers. *American Journal of Medical Technology* 38(12): 491-495.

Anderson, I., and J. Korsgaard. 1986. Asthma and the indoor environment: Assessment of the health implications of high indoor air humidity. *Environment International* 12: pp. 121-127.

Anderson, J.D., F.A. Dark, and S. Peto. 1968. The effect of aerosolation upon survival and potassium retention by various bacteria. *Journal of General Microbiology* 52: pp. 99-105.

Arlian, L.G. 1992. Water balance and humidity requirements of house dust mites. *Experimental and Applied Acarology* 16: 15-35.

Arlian, L.G., I.L. Bernstein, J.S. and Gallagher. 1982. The prevalence of house dust mites, dermatophagoids, and associated environmental conditions in homes in Ohio. *Journal of Allergy and Clinical Immunology* 69(6): 527-532.

Arlian, L.G., D. Bernstein, I.L. Bernstein, S. Friedman, A. Grant, P. Lieberman, et al. 1992. Prevalence of dust mites in the homes of people with asthma living in eight different geographic areas in the United States. *Journal of Allergy and Clinical Immunology* 90(3): 292-300.

Arundel, A.V., E.M. Sterling, J.H. Biggin, and T.D. Sterling. 1986. Indirect health effects of relative humidity in indoor environments. *Environmental Health Perspectives* 65: 351-361.

Arundel, A.V., E.M. Sterling, J.H. Biggin, and T.D. Sterling. 1992. Indirect health effects of relative humidity in indoor environments. *Desiccant Cooling and Dehumidification*, pp. 3-12. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

ASHRAE. 1989. *ANSI/ASHRAE Standard 62-1989, Ventilation for acceptable indoor air.* Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

ASHRAE. 1992. *ANSI/ASHRAE Standard 55-1992, Thermal environmental conditions for human occupancy.* Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

August, P.J. 1984. House dust mite causes atopic eczema. A preliminary study. *British Journal of Dermatology* 3(suppl 26): 10-11.

Avari, G.P., and D. Allsopp. 1983. The combined effect of pH, solutes, and water activity on the growth of some xerophilic *Aspergillus* species. *Biodeterioration 5,* T.A. Oxley and S. Barry, eds., pp. 548-556. New York: John Wiley & Sons Ltd.

Ayerst, G. 1969. The effects of moisture and temperature on growth and spore germination in some fungi. *Journal of Stored Produce Research* 5: 127-141.

Bates, J.M., D.A. Rorek, and M.H. Ballantye. 1993. Dust mite counts and mite allergens in family homes before and after dry extraction carpet cleaning. *Indoor Air '93, Proceedings of the 6th International Conference on Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 2, pp. 33-38.

Bayer, C.W., and C.C. Downing. 1992. Indoor humidity in schools with insufficient humidity control. *Environments for People: Proceeding of IAQ '92,* pp. 197-200, San Francisco. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Bayer, C.W., and S. Crow. 1993. Detection and characterization of microbially produced volatile organic compounds. *Indoor Air '93: Proceedings of the 6th International Conference on Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 6, pp. 297-302.

Beaumont, F., H.F. Kauffman, H.J. Sluiter, and K. de Vries. 1985. Volumetric aerobiological survey of conidial fungi in the North-East Netherlands. II. Comparison of aerobiological data and skin tests with mould extracts in an asthmatic population. *Allergy* 40: 181-186.

TABLE 1 BIOLOGICAL POLLUTANTS IN INDOOR AIR
Summary of Environmental Requirements and Recommended Remediation Procedures

	Health Implications	Environmental Requirements	Common Sites of Contamination	Frequently Recommended Remediation Procedures
Dust Mites	**Allergic reactions:** asthma, rhinitis, dermatitis **Dust mite allergens:** mite body parts and fecal matter are the most common source of allergic reactions within house dust **Species most commonly associated with disease:** *Dermatophagoides* *D. pteronyssinus* - more common in humid regions *D. farinae* - more common in relatively dry regions *Euroglyphus* - generally rare, found only in humid regions	**Source of nutrients:** shed human and animal skin scales **Source of water:** • Uptake water vapor directly from air • RH of 70-80% at 25°C is optimal for growth **Critical relative humidity:** 55% at 15°C to 75% at 35°C (For more detailed information on the humidity requirements for mites see Arlian, 1992)	• Mattresses/bedding • Thick carpeting • Heavily used upholstered furniture (Dust mites are more commonly associated with residences rather than commercial buildings)	• Removal of contaminated carpeting, bedding, and/or furniture • Frequent hot-water cleaning of bedding • Encasement of mattresses with semi-permeable vinyl casing • Special vacuuming procedures (e.g., HEPA filters, central vacuum system with outside equipment) • Surface treatments (e.g., benzyl benzoate) • Reduction of ambient humidity or specifically within the microhabitat of mites (e.g., through the use of electric blankets, radiant heating of carpeted floor surfaces, etc.)
Fungi	**Allergic reactions:** asthma, rhinitis, dermatitis (most common: *Alternaria, Aspergillus, Cladosporium,* and *Penicillium)* **Hypersensitivity Pneumonitis** (e.g., *Cladosporium, Sepula)* **Mycotoxicosis** (e.g., *Aspergillus, Penicillium, Stachybotrys atra, Trichoderma virde)* **Infectious disease** (e.g., *Aspergillus fumigatus, Cryptococcus)*	**Source of nutrients** - organic debris, dirt, organic building materials **Source of water:** moisture on and within surfaces • Specific water requirement varies from species to species. • Growth of xerophilic species such as *Aspergillus* is likely to begin at an ERH of 75% to 80%. (For more detailed information on humidity requirements see Flannigan 1992 and IEA Annex 14 guidelines.)	• Moisture damaged building materials (walls, carpeting, books, etc.) • Within fiberglass duct lining in which condensation has occurred • On wall surfaces with high ERH or on which condensation has occurred • Within poorly maintained conditioning systems containing water (e.g., humidifiers, cooling coil drip pans)	• Removal of damaged material where possible (i.e. carpeting, duct liners, wallpaper, etc.) • Cleaning of water resistant materials with chlorine bleach • Proper maintenance and operation of conditioning systems (e.g., cleaning and disinfecting cooling coils and drain pans, continual operation of forced air systems to avoid condensation) • Proper construction techniques to avoid water damage (e.g., proper placement of vapor barriers to avoid condensation within walls, design of drainage systems to avoid flooding and water incursion) • Adequate ventilation to reduce internal humidity loads
Bacteria and Viruses	**Infectious disease: person-to-person** (e.g., common cold, flu, measles, TB, etc.) **Infectious disease: building related (e.g., *Legionella pneumophila)*** **Hypersensitivity pneumonitis** (most commonly associated with ***thermophilic actinomycetes)***	• Virulent bacteria and viruses are transferred to hosts through droplet nuclei expelled when coughing/sneezing or formed within contaminated building systems that form aerosols • Bacteria and viruses that grow outside of the human host require liquid water for growth (For detailed information, see Cox 1989.)	• Infected humans • Improperly maintained building systems that have the potential to form aerosols: (e.g., spray-type humidifies, cooling towers etc.)	**Person-to-person spread of airborne infection:** • Isolation of contaminated persons • Increase fresh air exchange rate **Growth within building systems:** • Remove source of contamination (e.g., replace system, biocide treatment, etc.) • Routine cleaning of water systems and/or filters • Relocate intake vents (in the case of cooling tower contamination)

Becker, R. 1984. Condensation and mould growth in dwellings—Parametric and field study. *Buildings and Environment* 19(4): 243-250.

Bencko, V., J. Melichercik, V. Melichercikova, and Z. Wirth. 1993. Microbial growth control in spray humidifiers of health facilities. *Indoor Air* 3: 20-25.

Berglund, B., B. Brunekreep, H. Knopple, T. Lindvall, L. Maroni, et al. 1992. Effects of indoor air pollution on human health. *Indoor Air* 2: 2-25.

Bjurman, J. 1993. Thermal insulation materials, microorganisms and the sick building syndrome. *Indoor Air '93: Proceedings of the 6th International Conference on Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 4, pp. 339-344.

Block, S.S. 1953. Humidity requirements for mold growth. *Applied Microbiology* 1: 287-293.

Brauer, M., T.R. Rasmussen, and S.K. Kjaergaard. 1993. Indoor nitrous acid, formation and human exposure effects. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 3, pp. 141-146.

Brundrett, G.W. 1990. *Criteria for moisture control.* London: Butterworth & Co.

Burge, H.A. 1988. Environmental allergy, definition, causes, control. *Proceedings of IAQ '88.* Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Burge, H.A. 1989. Indoor air and infectious disease. *Occupational Medicine: State of the Art Reviews* 4(4): 713-721.

Burge, H.A. 1995. Aerobiology of the indoor environment. *Occupational Medicine: State of the Art Reviews* 10(1): 27-40.

Burge, H.A., W.R. Solomon, and J.R. Boise. 1980. Microbial prevalence in domestic humidifiers. *Applied and Environmental Microbiology* 39(4): 840-844.

Burge, H.A., J.C. Feeley et al. 1991. Indoor air pollution and infectious diseases. *Indoor Air Pollution*, J.M. Samet and J.D. Spengler, eds., pp. 273-284. Baltimore, Md.: Johns Hopkins University Press.

CL. 1994. Personal communications. Berkeley, Calif.: Center Laboratories.

Cabrera, P., G. Julia-Serda, F.C. Rodriguez, J.B.D. Camminero, and T. Carrillo. 1995. Reduction of house dust mite allergens after dehumidifier use. *Journal of Allergy and Clinical Immunology* 95(2): 635-636.

Colloff, M.J. 1992. Exposure to house dust mites in homes of people with atopic dermatitis. *British Journal of Dermatology* 127(4): 322-327.

Covelli, H.D., J. Kleeman, J.E. Martin, W.L. Landau, R.L. Hughes et al. 1973. Bacterial emission from both vapor and aerosol humidifiers. *American Review of Respiratory Disease* 108: 698-701.

Cox, C.S. 1966. The survival of *Escherichia coli* sprayed into air and into nitrogen from distilled water and from solutions of protecting agents, as a function of relative humidity. *Journal of General Microbiology* 43: 383-399.

Cox, C.S. 1989. Airborne bacteria and viruses. *Science Progress* 73: 469-500.

deBoer, R., and L.P. van der Geest. 1990. House dust mite (Progylphidae) populations in mattresses, and their control by electric blankets. *Experimental and Applied Acarology* 9(1-2): 113-122.

Ellingson, A.R., R.A. LeDoux, P.K. Vedanthan, and R.W. Weber. 1995. The prevalence of Dermatophagoldes mite allergen in Colorado homes utilizing central evaporative coolers. *Journal of Allergy and Clinical Immunology* 96(4): 473-479.

Ezeonu, I.M., J.A. Noble, R.B. Simmons, D.L. Price, S.A. Crow, D.G. Ahearn, et al. 1994. Effect of relative humidity on fungal colonization of fiberglass insulation. *Applied and Environmental Microbiology* 60(6): 2149-2151.

Fernandez-Caldas, E., W.L. Trudeau, and D.K. Ledford. 1994. Environmental control of indoor biologic agents. *Journal of Allergy and Clinical Immunology* 94(2): 404-412.

Fink, J.N., G.T. Hensley, D.P. Schleuter, and G.F. Unger. 1976. Interstitial lung disease due to contamination of forced air systems. *Annals of Internal Medicine* 84: 406-413.

Flannigan, B. 1992. Approaches to assessment of the microbial flora of buildings. *Environments for People: Proceedings of IAQ '92,* September, San Francisco, pp. 139-145. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Flannigan, B., and D. Miller. n.d. Health implications of fungi in indoor environments—An overview. Unpublished.

Flannigan, B., E.M. McCabe, and F. McGarry. 1991. Allergenic and toxigenic micro-organisms in houses. *Journal of Applied Bacteriology,* Symposium Supplement, vol. 70, pp. 61S-73S.

Flynn, D.D., and L.J. Goldberg. 1971. Effect of relative humidity on aerosol persistence of *Streptococcus salivarius. Archives of Environmental Health* 23: 40-42.

Foarde, K., P. Dulaney, E. Cole, D. VanOsdell, D. Ensor, and J. Chang. 1993. Assessment of fungal growth on ceiling tiles under environmentally characterized conditions. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 4, pp. 357-362.

Friedman, F.M., H.M. Friedman, and G.T. O'Connor. 1992. Prevalence of dust mite allergens in homes and workplaces of the Upper Connecticut River Valley of New England. *Allergy Proceedings* 13(5): 259-262.

Gammage, R.B. 1990. Exposure to formaldehyde in indoor air. *Risk Analysis* 10(1): 77-83.

Godish, T., and J. Rouch. 1986. Mitigation of residential formaldedyde contamination by indoor climate control.

American Industrial Hygiene Association Journal 47: 792-797.

Grant, C., C.A. Hunter, B. Flannigan, and A.F. Bravery. 1989. *International biodeterioration* 25: 259-284.

Gravesen, S. 1979. Fungi as a cause of allergic disease. *Allergy* 34: 135-154.

Green, G.H. 1985. Indoor relative humidities in winter and the related absenteeism. *ASHRAE Transactions* 91(1).

Griffin, D.H. 1981. *Fungal physiology.* New York: Wiley and Sons.

Hambleton, P., M.G. Broster, P.J. Dennis, R.F. Henstridge, J.W. Conlan et al. 1983. Survival of virulent *Legionella pneumophila* in aerosols. *J. Hyg. Camb.* 90: 451-460.

Hart, B.J., and L. Whitehead. 1990. Ecology of house dust mites in Oxfordshire. *Clinical and Experimental Allergy* 20: 203-209.

Harving, H., J. Korsgaard, R. Dahl, H.I. Beck, P. Bjerring et al. 1990. House dust mites and atopic dermatitis. A case-control study on the significance of house dust mites as etiolofic allergens in atopic dermatitis. *Annals of Allergy* 65: 25-31.

Harving, H., J. Korsgaard, and R. Dahl. 1994. House-dust mite exposure reduction in specially designed, mechanically ventilated "healthy" homes. *Allergy* 49(9): 713-718.

Hens, H., and E. Senave. 1990. Condensation and energy, Zolder case study. *Journal Ventilation System Performance*, pp. 81-99, 11 AIVC Conference, September 18-21, Belgirate, Italy.

Hodges, G.R., J.N. Fink, and D.P. Schlueter. 1974. Hypersensitivity pneumonitis caused by a contaminated cool-mist vaporizer. *Annals of Internal Medicine* 80: 501-504.

Hodgson, M.J. 1989. Clinical diagnosis and management of building-related illness and the sick building syndrome. *Occupational Medicine: State of the Art Reviews* 4(4): 593-606.

Htut, T. 1994. Airborne house dust mite allergen: Are short-duration avoidance measures helpful? *Indoor Environment* 3: 48-53.

Hung, L.L., C.S. Yang, F.J. Dougherty, F.A. Lewis, F.A. Zampiello, and L. Magniaracina. 1992. Dust mite and cat dander allergens in office buildings in the mid-Atlantic region. *Environments for People: Proceedings of IAQ '92,* October 18-21, San Francisco, pp. 87-93.

Hyvarinen, A., T. Reponen, T. Husman, J. Ruuskanen, and A. Nevalainen. 1993. Characterizing mold problem buildings—Concentrations and flora of viable fungi. *Indoor Air* 3: 337-343.

IEA. 1991. IEA Annex 14 (Condensation and Energy), *Guidelines and practice: Energy conservation in building and community systems.* Paris: International Energy Agency.

Kalliokoski, P., T. Juutinen, P. Pasanen, and A. Pasanen. 1993. Fungal growth and activity on moist building materials. *Indoor Air '93: Proceedings of the 6th International Conference on Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 4, pp. 299-303.

Kalra, S., S.J. Owen, J. Hepworth, and A. Woodcock. 1990. Airborne house dust mite antigen after vacuum cleaning. *Lancet* 336: 449.

Korsgaard, J. 1983a. Mite asthma and residency, a case-control study on the impact of exposure to house-dust mites in dwellings. *American Review of Respiratory Disease* 128: 231-235.

Korsgaard, J. 1983b. House-dust mites and absolute indoor humidity. *Allergy* 38(2): 85-92.

LaForce, F.M. 1986. Airborne infections and modern building technology. *Environment International* 12: 137-146.

Lang, J.D., and M.S. Mulla. 1977. Distribution and abundance of house dust mites, Dermataphagoides, in different climatic zones of southern California. *Environmental Entomology* 6(2): 213-216.

Lang, J.D., and M.S. Mulla. 1978. Seasonal dynamics of house dust mites, Dermatophagoides, in homes in southern California. *Environmental Entomology* 7(2): 281-286.

Leaderer, B.P., J. Sullivan, P. Koutrakis, and J.M. Wolfson. 1993. A passive monitor for the measurement of nitrous acid and sulfur dioxide. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 2, pp. 233-238.

Lintner, T.J., and K.A. Brame. 1993. The effects of season, climate, and air-conditioning on the prevalence of Dermatophagoides mite allergens in household dust. *Journal of Allergy and Clinical Immunology* 91(4): 862-867.

Macher, J.M. 1994. Personal communication.

Macher, J.M., and J.R. Girman. 1990. Multiplication of microorganisms in an evaporative cooler and possible indoor air contamination. *Environment International* 16: 203-211.

Marbury, M.C., and R.A. Krieger. 1991. Formaldehyde. *Indoor Air Pollution: A Health Perspective*, J.M. Samet and J.D. Spengler, eds., pp. 223-251. Baltimore, Md.: Johns Hopkins University Press.

Mbithi, J.N., V.S. Springthorpe, and S.A. Sattar. 1991. The effect of relative humidity and air temperature on survival of Hepatitis A virus on environmental surfaces. *Applied and Environmental Microbiology* 57(5): 1394-1399.

McDonald, L.G., and E. Tovey. 1992. The role of water temperature and laundry procedures in reducing house dust mite populations and allergen content of bedding. *Journal of Allergy and Clinical Immunology* 90(4, pt 1): 599-608.

Menzies, R., R. Tamblyn, P. Comtois, C. Reed, J. Pasztor, et al. 1992. Case-control study of microenvironmental

exposures to aero-allergens as a cause of respiratory symptoms—Part of the sick building syndrome (SBS) symptom complex. *Environments for People: Proceedings of IAQ '92,* October, San Francisco. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Meyer, B. 1986. Formaldehyde exposure from building products. *Environment International* 12: 283-288.

Miller, J.D. 1992. Fungi and the building engineer. *Environments for People: Proceedings of IAQ '92,* October, San Francisco, pp. 147-159. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Moore-Landecker, E. 1982. *Fundamentals of the fungi,* 2d ed. Englewood Cliffs, N.J.: Prentice-Hall.

Morey, P.R. 1988. Microorganisms in buildings and HVAC systems, a summary of 21 environmental studies. *Proceedings of IAQ '88,* pp. 10-24. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Morey, P.R., C.M. Williams et al. 1991. Is porous insulation inside an HVAC system compatible with a healthy building? *Healthy Buildings: Proceeding of IAQ '91,* September, Washington, D.C. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

Mueller, F.X., L. Loeb, and W.H. Mapes. 1973. Decomposition rates of ozone in living areas. *Environmental Science and Technology* 7(4): 342-346.

Murray, A.B., and P. Zuk. 1979. The seasonal variation in a population of house dust mites in a North American city. *Journal of Allergy and Clinical Immunology* 64(4): 266-269.

Murray, A.B., A.C. Ferguson, and B.J. Morrison. 1985. Sensitization to house dust mites in different climatic areas. *Journal of Allergy and Clinical Immunology* 76(1): 108-112.

Nevalainen, A. 1993. Microbial contamination of buildings. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 4, pp. 3-12.

Nevalainen, A., A.L. Pasanen, M. Nininen, T. Reponen, P. Kalliokoski, and M.J. Jantunen. 1991. The indoor air quality in Finnish homes with mold problems. *Environment International* 17: 299-302.

O'Rourke, M.K., L. Fiorentino, D. Clark, M. Ladd, and S. Rogan. 1993. Building characteristics and importance of house dust mite exposures in the Sonoran desert, USA. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 4, pp. 155-160.

Pasanen, A.L., P. Kallioloski, P. Pasanen, M.J. Jantunen, and A. Nevalainen. 1991. Laboratory studies on the relationship between fungal growth and atmospheric temperature and humidity. *Environment International* 17: 225-228.

Pasanen, A.L., H. Heinonen-Tanski, P. Kalliokoski, and M.J. Jantunen. 1992. Fungal microcolonies on indoor surfaces—An explanation for the base-level fungal spore counts in indoor air. *Atmospheric Environment* 26B: 117-120.

Pasanen, P., A. Pasanen, and M. Jantunen. 1993. Water condensation promotes fungal growth in ventilation ducts. *Indoor Air* 3: 106-112.

Platts-Mills, T.A.E., M.L. Hayden, M.D. Chapman, and S.R. Wilkins. 1987. Seasonal variation in dust mites and grass-pollen allergens in dust from the houses of patients with asthma. *Journal of Allergy and Clinical Immunology* 79(5): 781-791.

Platts-Mills, T.A.E., A.L. de Weck et al. 1989. Dust mite allergens and asthma—A worldwide problem. *Journal of Allergy and Clinical Immunology* 83(1-3): 416-427.

Puhakka, E., and J. Karkkainen. 1993. Formaldehyde concentrations in indoor air in new buildings; factors that affect the prevalence of formaldehyde. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air Quality and Climate,* July 4-8, Helsinki, Finland, vol. 2, pp. 147-152.

Reed, C.E., and M.C. Swanson. 1986. Indoor allergens, identification and quantification. *Environment International* 12: 115-120.

Reiser, J., D. Ingram, E.B. Mitchell, and J.O. Warner. 1990. House dust mite allergen levels and an anti-mite mattress spray (natamycin) in the treatment of childhood asthma. *Clinical and Experimental Allergy* 20(5): 561-567.

Seltzer, J.M. 1995. Biologic contaminants. *Occupational Medicine: State-of-the-Art Reviews* 10(1): 1-25.

Smith, P.W., and R.M. Massanari. 1977. Room humidifiers as the source of Actinetobacter infections. *JAMA* 237(8): 795-797.

Smith, T.F., L.B. Kelly, P.W. Heymann, S.R. Wilkins, and T.A.E. Platts-Mills. 1985. Natural exposure and serum antibodies to house dust mite-allergic children with asthma in Atlanta. *Journal of Allergy and Clinical Immunology* 76(6): 782-788.

Solomon, W.R. 1974. Fungus aerosols arising from cold-mist vaporizers. *Journal of Allergy and Clinical Immunology* 54(4): 222-228.

Sorenson, W.G. 1989. Health impact of mycotoxins in the home and workplace: An overview. In *Biodeterioration Research 2,* C.E. O'Rear and G.C. Llewellyn, eds., pp. 201-215. New York: Plenum Press.

Sterling, E.M., A. Arundel, and T.D. Sterling. 1985. Criteria for human exposure to humidity in occupied buildings. *ASHRAE Transactions* 91(1).

Stock, T.H., and E.A. Venso. 1993. The impact of residential evaporative air cooling on indoor exposure to ozone. *Indoor Air '93: Proceedings of the 6th International*

Conference on Indoor Air and Climate, July 4-8, Helsinki, Finland, vol. 4, pp. 251-256.

TenWolde, A., and W.B. Rose. 1993. Criteria for humidity in the building and the building envelope. *Bugs, Mold and Rot II: Proceedings of Workshop on Control of Humidity for Health, Artifacts and Buildings*, November 16-17, pp. 63-65. Oak Ridge, Tenn.: Oak Ridge National Laboratory.

Thogersen, K., L. Gunnarsen, and P.A. Nielsen. 1993. The effects on indoor air quality by water-damaged chipboards. *Indoor Air '93: Proceedings of the 6th International Conference on Indoor Air and Climate,* July 4-8, Helsinki, Finland, vol. 2, pp. 537-542.

Tsongas, G. 1991. A field study of indoor moisture problems and damage in new Pacific Northwest homes. *Healthy Buildings: Proceeding of IAQ '91*, September, Washington, D.C., pp. 202-209. Atlanta: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

van Netten, C., C. Shirtleffe, and J. Svec. 1989. Temperature and humidity dependence of formaldehyde release from selected building materials. *Bulletin of Environmental Contamination and Toxicology* 42: 558-565.

Verhoeff, A.P., R.T. van Strien, J.H. van Wijnen, and B. Brunekreef. 1995. Damp housing and childhood respiratory symptoms: The role of sensitization to dust mites and molds. *American Journal of Epidemiology* 141(2): 103-110.

Voorhorst, R., M.I.A. Spieksma-Boezeman, and F.TH.M. Spieksma. 1964. Is a mite (Dermatophagoides) the producer of the house-dust allergen? *Allergy und Asthma Band* 6: 329-334.

Weschler, C.J., V.N. Datta, and H.C. Shields. 1991. Indoor ozone exposures from the infiltration of outdoor ozone. *Indoor Air Pollution: Radon, Bioaerosols and VOC's*, J.G. Kay, G.E. Keller, and J.F. Miller, eds. pp. 83-99.

Wickman, M., S.L. Nordvall, G. Pershagen, J. Sundell, and B. Schwartz. 1991. House dust mites sensitization in cihildren and residential characteristics in a temperate region. *Journal of Allergy and Clinical Immunology* 88(1): 89-95.

Wickman, M., S.L. Nordvall, G. Pershagen, J. Korsggard, N. Johansen, and J. Sundell. 1994a. Mite allergens during 18 months of intervention. *Allergy* 49(2): 114-119.

Wickman, M., G. Emenius, A.C. Egmar, G. Axelsson, and G. Pershahen. 1994b. Reduced mite allergen levels in dwellings with mechanical exhaust and supply ventilation. *Clinical and Experimental Allergy* 24(2): 109-114.

Wilcox, W. 1995. Personal communication.