

**HETA 92-0244-2373
JANUARY 1994
KAISER NORTHLAKE ATRIUM
ATLANTA, GEORGIA**

**NIOSH INVESTIGATORS:
Scott Deitchman, M.D., M.P.H.
Kenneth Martinez, M.S.E.E.
Susan Upham, M.D., M.P.H.**

I. SUMMARY

On May 4, 1992, the National Institute for Occupational Safety and Health (NIOSH) received a request from employees of Kaiser Permanente in Atlanta, Georgia, to conduct a Health Hazard Evaluation at the Northlake Atrium building. The requesting employees were concerned about health effects from exposure to *Stachybotrys* fungus in the building; the obstetrics/gynecology area was described as being of particular concern. On August 7, 1992, a NIOSH industrial hygienist and two medical officers conducted a walk-through survey of the facility, inspected building fixtures and the ventilation system, reviewed the medical records of selected employees, and interviewed employees and medical staff.

Employees described symptoms such as cough, sneezing, urticaria, and shortness of breath, and described illnesses including otitis, sinusitis, asthma, viral meningitis, and pseudotumor cerebri. Some of the employees expressed their belief that these symptoms and illnesses had been caused by exposures to mycotoxins from *Stachybotrys* mold. This organism had been detected during sampling by an outside consultant. Medical records reviewed by NIOSH medical officers did not report evidence of the mucosal and blood abnormalities which are associated with stachybotrytoxicosis.

Kaiser management reported extensive remediation efforts were conducted in response to an earlier report by a private consultant. Employees reported additional clean-up activities were carried out immediately before the scheduled NIOSH visit. NIOSH investigators collected a sample of fungal contamination for culture and identification; which was subsequently identified as predominantly *Acremonium*, with lesser colonies of *Penicillium*, *Aspergillus*, *Alternaria*, and unidentified yeasts.

Although *Stachybotrys* fungal contamination was identified in samples collected by a private consultant, different species were present in the sample collected (in a different location) by NIOSH investigators. Much of the previously-reported fungal contamination had been cleaned prior to the time of the NIOSH visit, but some fungal contamination remained. The results of the NIOSH investigation and a review of the medical literature do not support the suggestion that the symptoms experienced by Kaiser employees were caused by exposure to *Stachybotrys* mycotoxins. Some of the symptoms, however, may be related to allergic reactions to other molds still present in the working environment. Remediation efforts should be continued to eliminate mold exposures.

KEYWORDS: SIC 8011 (Offices and Clinics of Doctors of Medicine), indoor environmental quality, bioaerosols, fungi, *Stachybotrys*.

II. INTRODUCTION

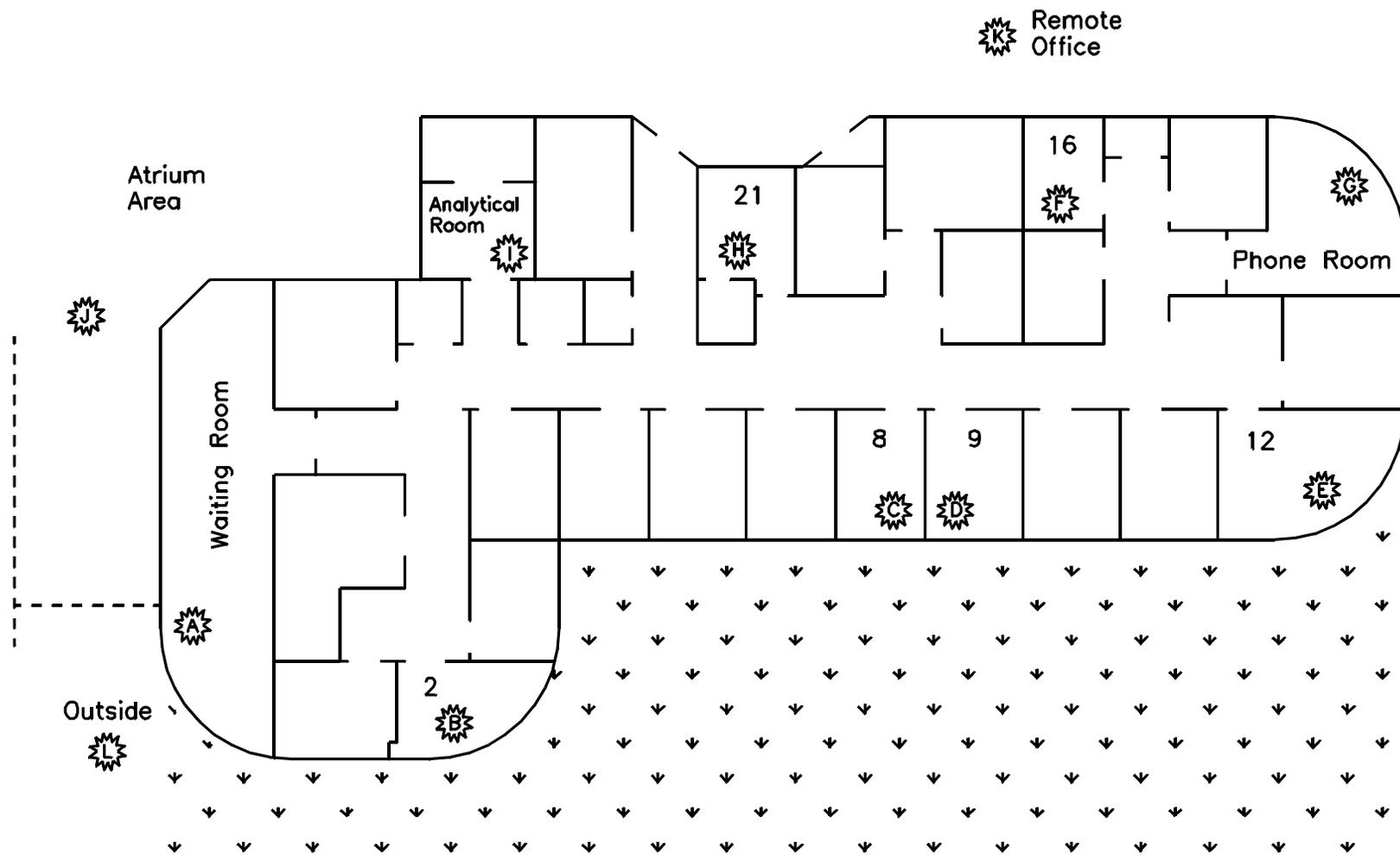
On May 4, 1992, the National Institute for Occupational Safety and Health (NIOSH) received a request from employees of Kaiser Permanente to conduct a Health Hazard Evaluation of the Northlake Atrium building. *Stachybotrys* fungus was cited as the hazardous exposure of concern, and the employees who submitted the request indicated their concern that this exposure was responsible for their health symptoms (which were not described in the request). These concerns were particularly focussed upon the obstetrics/gynecology area. In accordance with the request, the identities of the requesting employees were kept confidential. On August 7, 1992, a NIOSH survey team (consisting of an industrial hygienist and two medical officers) conducted a site visit at the building.

Background

The Kaiser Northlake Atrium building is a two-story, glass and metal curtain wall structure in a mixed suburban/commercial area in Atlanta, Georgia. The building houses medical clinics, offices, a pharmacy, and a medical records storage facility. Windows are available on most exterior building surfaces. A sketch (not to scale) of the evaluated area of the building is shown in Figure 1. The evaluation area was composed of a reception/waiting area, examination rooms, private and multi-person offices, and a diagnostic laboratory facility. The private and multi-person offices are primarily located in the exterior rooms, offering visual access to windows. All areas are carpeted.

Conditioning of the indoor air is accomplished through eight water-source heat pumps located in the space above the drop ceiling (serving as a return plenum). Each heat pump services a "zone" controlled by a single thermostat; each zone includes a small group of adjacent rooms (two to five). Air is returned through rectangular ceiling grills to the ceiling plenum. One hundred percent return air (0% outside air) enters each heat pump through low efficiency, metal mesh filters where it is then recirculated to the occupied spaces. Based on a physical inspection by the investigating team and reports from building maintenance personnel, the ventilation systems were designed to provide 100% recirculated air. Dedicated exhausts to the outside are located in the rest rooms and two specialty rooms (diagnostic laboratories).

According to employees, in November 1989 the present obstetrics/gynecology area began service. At the time of occupancy the building already had a history of water leakage, and employees reported that during heavy rainstorms they were obliged to catch leaking water with basins. Within a year, the employees began to notice fungal growth on the wallpaper and the presence of odors of mold or mildew. The employees contacted the area office of the Occupational Safety and Health Administration (OSHA), and an OSHA compliance officer conducted a site visit on January 7, 1992. The OSHA investigation included a review of sampling for microorganisms in the ventilation system conducted during site visits of December 23 and February 13, 1992, by a consultant hired by Kaiser. Among the microbiological species identified in this sampling were fungi of the genus *Stachybotrys*. This finding led to the issuance of an OSHA citation for failure to maintain clean and



NOTE: Star indicates real-time measurement sample locations.

Figure 1. Plan Drawing of Evaluation Area

sanitary conditions. The employees, concerned about possible health effects from exposure to *Stachybotrys* fungi, were encouraged by the OSHA compliance officer to submit a request for a NIOSH health hazard evaluation.

III. EVALUATION CRITERIA

A. Microbiological Contaminants

Because the employees were chiefly concerned about occupational exposure to *Stachybotrys* and/or its mycotoxins, we present a detailed review of this fungus and its reported health effects.

1. Biology and ecology of *Stachybotrys*

Fungi of the genus *Stachybotrys* are found worldwide. *Stachybotrys* is a saprophyte (i.e., grows on dead or decomposing matter) known to destroy cellulose. It is usually described as dark brown/black or sooty in appearance and is composed of conidiophores capped by dark conidium (spores).¹ It has been isolated from soil and a wide variety of substances rich in cellulose such as hay, wood pulp, cotton, grains, various dead plant components, paper, glue in book binderies, and plant fiber processing manufacturers.²

Stachybotrys species tend to be uncommon in "healthy" work or home environments. Several studies of viable mold spore counts using various sampling techniques (Rotorods, Anderson sampling) obtained from homes in southern California revealed a frequency of isolation of *Stachybotrys* ranging from 2.9% to 7.1%. This may vary, however, depending on the local mold flora, weather, and outdoor activity present (e.g., mowing the lawn, landscaping, etc).³

The frequency with which *Stachybotrys* is found in buildings with mold problems has varied among different studies. This may depend partly on the sampling technique utilized. Since this fungus competes poorly on typical agar media, it may not be detected unless cellulose based agar or moist filter paper medium are used. Studies of buildings with mold problems using Rotorods, Anderson sampling, and moist sterile filter paper have found *Stachybotrys* in up to 19% of the buildings sampled; the higher yields were in buildings with known problems with mold contamination.^{3,4}

Various strains of *Stachybotrys* have somewhat different growth requirements; the temperature range for optimum growth is 72 to 82 degrees Fahrenheit, and the minimum humidity required for spore germination is 96.3% to 98.5%.² Buildings where *Stachybotrys* growth problems have been reported typically had materials subject to chronic water damage (e.g., due to leaking roofs or plumbing, floods, air conditioner condensation, etc.) and appropriate conditions of temperature. Examples of building materials, which have been found as growth substrates for *Stachybotrys*, have included: jute carpet backing, which repeatedly has been wet; cold air return ducts containing moisture, lint and carpet fibers; wood fiber ceiling board; and moist urea formaldehyde foam insulation in contact with gyproc paper.^{3,5,6} Other potential sources for fungal growth, all of which maintain a consistent source of moisture, include humidifiers (vaporizers, water spray conditioners), evaporative coolers, self-defrosting refrigerators, flush toilets, air conditioners and HVAC systems.⁷

Stachybotrys is one of many fungi which produces chemicals called trichothecene mycotoxins. These substances have been responsible for illness in animals and man and have allegedly been used in chemical warfare (described as "yellow rain") in Cambodia,

Laos, and Afghanistan.⁸ Studies of *Stachybotrys* species have revealed that approximately two-thirds of isolates were found to produce these toxins, with each productive strain elaborating several different toxins.^{1,9,10,11,12} Sorenson and associates demonstrated that these mycotoxins could be found in the aerosolized spores of this fungus, indicating the potential for inhalational exposure to these compounds.¹³

2. Veterinary experience with *Stachybotrys*

Animal disease produced by *Stachybotrys* fungi is called stachybotryotoxicosis, and is well known to veterinarians. It has severely affected large and small animals, especially in the early 1900's in Russia and Europe. It was established that the ingestion of mold-contaminated feeds (hay, grains, etc.) was responsible for the resultant disease. Laboratory studies revealed that the severity of the illness was dose-dependent and that the trichothecene mycotoxins elaborated by the fungi were the responsible agents. Eventually, the disease was controlled by improved containment or treatment of animal foodstuffs. It is important to note that these reports all involve substantial exposures, either from eating mold-contaminated feed or from sleeping on visibly moldy bedding.¹

Several different types of stachybotryotoxicosis can be distinguished, depending on the dose of toxin ingested, length of ingestion, and type of animal afflicted. The dermal manifestations are characterized by oral ulcerations, hyperemia, edema, and tissue necrosis of varying severity. Systemic toxicity which can occur includes fever, compromise of the immune and blood-forming systems, anorexia, hemorrhage of the internal organs, cardiac arrhythmia, sepsis, neurological abnormalities, and frequently death.^{1,2,9}

3. Human disease due to *Stachybotrys*

Fungal infection is a condition where a fungus causes disease by actually growing on a person; for example, athlete's foot is an infection caused by the growth of a fungus on the skin of the foot. In our review of the medical literature we found no information describing human infection by *Stachybotrys*. Instead, the potential for human disease caused by *Stachybotrys* includes allergy to the fungus and toxicity (poisoning) from exposure to its products. Data on the allergic and toxic forms of the disease are limited, but several studies and case reports implicate *Stachybotrys* and its mycotoxins as causes of certain human illnesses.

a. Allergies and allergic asthma

Data on the allergic manifestations of *Stachybotrys* are very limited. Only one case study suggested an allergic basis for disease due to *Stachybotrys*. In this case, a 4½ year-old child with asthma experienced some relief of his symptoms upon removal from his home. Investigation of the home revealed a history of repeated water damage resulting in extensive *Stachybotrys* mold growth on the jute-backed carpet. The authors reported a "dramatic reduction" of this child's asthma symptoms after removal and cleaning of the affected materials and surfaces.³

b. Stachybotryotoxicosis

The toxic manifestations of *Stachybotrys* are caused by absorption of the toxins produced by the fungus. There are several potential routes of exposure to the trichothecene toxins produced by this fungus, including absorption from skin contact, inhalation, and oral ingestion. There are reports of local skin irritation due to handling of material contaminated by this fungus, but whether or not systemic effects occur due to skin absorption is unknown. Most sources propose the inhalational route as the most likely entryway of the spores and their toxins into the body in occupational exposures. Occupations usually identified at risk for this disease are those involving work at farms, cottonseed oil plants, grain elevators, plants used for reprocessing moldy grains, malt grain processors, textile mills using plant fibers, and binder twine factories. Rarely, individuals who used straw for fuel or slept on mattresses filled with contaminated straw have developed this illness.^{2,4,9} Because these situations involved close contact with mold-contaminated materials, the people involved probably received greater exposures to mold spores than would be expected in the average indoor environment.

Stachybotryotoxicosis in man is generally uncommon and not fatal. The severity of the disease is dose-dependent and symptoms usually resolve with removal from exposure. Initially patients experience severe mucous membrane irritation associated with headache, dizziness, weakness, vomiting, diarrhea, abdominal pain, fever, sweating, tachycardia, cyanosis, dry cough, shortness of breath, and chest pain. Later manifestations include suppression of the hematologic and immune systems leading to petechiae, skin necrosis, hemorrhage of the mucous membranes or gastrointestinal tract and sepsis. In chemical warfare, where "yellow rain" was spread among its victims by airplane, the clinical experience was prolonged and severe, frequently leading to death. If death does not ensue, a gradual recovery occurs over the next several months.^{1,2,4,9,8}

Another disorder has been described by several different authors and may be a form of mycotoxin toxicity. The initial report described 10 patients who were all exposed to heavy concentrations of fungi while cleaning mold from the top of farm silos; the exposure was so heavy that the patients were covered with white dust mostly consisting of fungi. The affected workers experienced fever, chills, severe cough, and an increased white blood count. When doctors listened to their lungs, abnormal sounds were heard, but chest x-rays were normal in some patients and showed lung inflammation in others. All the patients recovered. The authors suggested that the cause of the disease might have been inhaled mycotoxins and described the disease by the term "pulmonary mycotoxicosis."¹⁴ A later report described 29 nine cases of this disease in which patients exposed to fungal dust in farm silos experienced symptoms and signs including fever, myalgia, chest tightness, cough, headache, malaise, and dyspnea. These symptoms were usually associated with normal chest exams and chest x-rays, negative tests for precipitating antibodies, normal pulmonary function tests, and only mildly abnormal oxygenation. The symptoms usually resolved without serious complications upon removal from the source of fungal exposure. It should

be noted that the authors' attempts to detect mycotoxins from the organic dust in these exposures were unsuccessful, leading them to question whether mycotoxins were truly the cause.¹⁵ Neither report included any identification of fungal species involved, but they are cited here because they discuss a proposed route of exposure to mycotoxins.

Only one published investigation has described an outbreak of stachybotryotoxicosis secondary to mold contamination in a home. A family of five experienced cold and flu symptoms, sore throats, diarrhea, headaches, dermatitis, patches of hair loss, and fatigue. Medical investigations of their conditions did not reveal any causes. In their home, a cold air return duct and an area of wood fiber board were contaminated with heavy growth of *Stachybotrys*. When the mold was cleaned up, the family members' symptoms resolved. The authors inferred that mycotoxins from the mold were responsible for the symptoms, although the report does not describe any biological testing for mycotoxins in the people affected.⁵

4. Summary

In summary, *Stachybotrys* is a common fungus in the environment which may on occasion be found in higher concentration in chronically wet environments. Because of its fastidious nature, special measures need to be taken in order to successfully identify it. It produces trichothecenes, known toxins to man and animal.

The effects of this mold in mammals are known in the veterinary field, where it has posed a significant threat to livestock in European and Asian farming regions in the past. The potential effects on humans vary, with the most significant being that of toxicity due to its trichothecene toxins. The usual setting of human disease is that of inhalation due to close contact with contaminated materials, although one report suggested that it could occur under conditions of mold contamination due to chronic water exposure in a home environment. Whether or not *Stachybotrys* can be the causative agent for allergic disorders is less clear, as only one case report of asthma secondary to this fungus is present in the literature.

B. Indoor environmental criteria

NIOSH investigators have completed over 1100 investigations of the occupational indoor environment in a wide variety of non-industrial settings. The majority of these investigations have been conducted since 1979.

The symptoms and health complaints reported to NIOSH by building occupants have been diverse and usually not suggestive of any particular medical diagnosis or readily associated with a causative agent. A typical spectrum of symptoms has included headaches, unusual fatigue, varying degrees of itching or burning eyes, irritations of the skin, nasal congestion, dry or irritated throats, and other respiratory irritations. Typically, the workplace environment has been implicated because workers report that their symptoms lessen or resolve when they leave the building.

A number of published studies have reported a high prevalence of symptoms among occupants of office buildings.^{16,17,18,19,20} Scientists investigating indoor environmental problems believe that there are multiple factors contributing to building-related occupant complaints.^{21,22} Among these factors are imprecisely-defined characteristics of heating, ventilating, and air-conditioning (HVAC) systems, cumulative effects of exposure to low concentrations of multiple chemical pollutants, odors, elevated concentrations of particulate matter, microbiological contamination, and physical factors such as thermal comfort, lighting, and noise.^{23,24,25,26,27,28} Indoor environmental pollutants can arise from either outdoor sources or indoor sources.

There are also reports describing results which show that occupant perceptions of the indoor environment are more closely related than any measured indoor contaminant or condition to the occurrence of symptoms.^{29,30,31} Some studies have shown relationships between psychological, social, and organizational factors in the workplace and the occurrence of symptoms and comfort complaints.^{31,32,33,34} Less often, an illness may be found to be specifically related to something in the building environment. Some examples of potentially building-related illnesses are allergic rhinitis, allergic asthma, hypersensitivity pneumonitis, Legionnaires' disease, Pontiac fever, carbon monoxide poisoning, and reaction to boiler corrosion inhibitors. The first three conditions can be caused by various microorganisms or other organic material. Legionnaires' disease and Pontiac fever are caused by *Legionella* bacteria. Sources of carbon monoxide include vehicle exhaust and inadequately-ventilated kerosene heaters or other fuel-burning appliances. Exposure to boiler additives can occur if boiler steam is used for humidification or is released by accident.

Problems that NIOSH investigators have found in the non-industrial indoor environment have included: poor air quality due to ventilation system deficiencies, overcrowding, volatile organic chemicals from furnishings, emissions from office machines, structural components of the building and contents, tobacco smoke, microbiological contamination, and outside air pollutants; comfort problems due to improper temperature and relative humidity (RH) conditions, poor lighting, and unacceptable noise levels; adverse ergonomic conditions; and job-related psychosocial stressors. In most cases, however, these problems could not be directly linked to the reported health effects.

Standards specifically for the non-industrial indoor environment do not exist. NIOSH, the Occupational Safety and Health Administration (OSHA), and the American Conference of Governmental Industrial Hygienists (ACGIH) have published regulatory standards or recommended limits for occupational exposures.^{35,36,37} With few exceptions, pollutant concentrations observed in non-industrial indoor environments fall well below these published occupational standards or recommended exposure limits. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) has published recommended building ventilation design criteria and thermal comfort guidelines.^{38,39} The ACGIH has also developed a manual of guidelines for approaching investigations of building-related complaints that might be caused by airborne living organisms or their effluents.⁴⁰

Measurement of indoor environmental contaminants has rarely proved to be helpful in determining the cause of symptoms and complaints except where there are strong or unusual sources, or a proven relationship between contaminants and specific building-related illnesses. The low-level concentrations of particles and variable mixtures of organic materials usually found are difficult to interpret and usually impossible to causally link to observed and reported health symptoms. However, measuring ventilation and comfort indicators such as CO₂, temperature and RH, has proven useful in the early stages of an investigation in providing information relative to the proper functioning and control of HVAC systems.

NIOSH and the Environmental Protection Agency (EPA) jointly published a manual on building air quality, written to help prevent environmental problems in buildings and solve problems when they occur.⁴¹ This manual suggests that indoor environmental quality (IEQ) is a constantly changing interaction of a complex set of factors. Four of the most important elements involved in the development of IEQ problems are: (1) a source of odors or contaminant; (2) a problem with the design or operation of the HVAC system; (3) a pathway between the contaminant source and the location of the complaint; and (4) the building occupants. A basic understanding of these factors is critical to preventing,

investigating, and resolving IEQ problems.

The basis for measurements made during this evaluation are listed below.

1. Carbon Dioxide

Carbon dioxide (CO₂) is a normal constituent of exhaled breath, and if monitored, may be useful as a screening technique to evaluate whether adequate quantities of fresh air are being introduced into an occupied space. The ANSI/ASHRAE Standard 62-1989, Ventilation for Acceptable Indoor Air Quality, recommends outdoor air supply rates of 20 cubic feet per minute per person (cfm/person) for office spaces and conference rooms, 15 cfm/person for reception areas, and 60 cfm/person for smoking lounges, and provides estimated maximum occupancy figures for each area.³⁸

Indoor CO₂ concentrations are normally higher than the generally-constant ambient CO₂ concentration (range 300-350 ppm). When indoor CO₂ concentrations exceed 1000 ppm in areas where the only known source is exhaled breath, inadequate ventilation is suspected. Elevated CO₂ concentrations suggest that other indoor contaminants may also be increased.

2. Temperature and Relative Humidity

The perception of comfort is related to one's metabolic heat production, the transfer of heat to the environment, physiological adjustments, and body temperatures. Heat transfer from the body to the environment is influenced by factors such as temperature, humidity, air movement, personal activities, and clothing. ANSI/ASHRAE Standard 55-1981 specifies conditions in which 80% or more of the occupants would be expected to find the environment thermally comfortable.³⁹

IV. METHODS

An opening conference was held with representatives of labor and management, during which time the history of the problem was discussed. After the opening conference, a walk-through tour of the facility was conducted with particular emphasis on the obstetrics/gynecology clinic. Following that, the industrial hygienist and the medical officers conducted separate components of the investigation. The hygienist inspected various heat pump units in the evaluated area and collected real-time measurements for carbon dioxide (CO₂), temperature, and relative humidity (RH). The medical officers interviewed employees, spoke with a Kaiser physician, and reviewed medical records. A closing conference was held at the end of the day.

A. Industrial Hygiene Investigation

1. Methods and Materials

Direct measurements for temperature, humidity, and CO₂ were collected at each sample location for three rounds of sampling beginning at approximately 11:30 a.m., followed by subsequent sampling rounds at 1:30 p.m. and 3:00 p.m. Carbon dioxide was measured using a Gastech RI 411 CO₂ monitor (Gastech, Inc., Newark, California) that was calibrated before and after the day's samples were collected using 800 parts per million (ppm) CO₂ in nitrogen (Alphagaz, Division of Liquid Air Corporation, Cambridge, Maryland) as a calibrant. Temperature and RH were measured using a Vaisala HM 34 temperature and humidity meter (Vaisala Oy, Helsinki, Finland). Two bulk samples of wallpaper (analyzed for

microbial content) were collected from an exposed wallpaper surface on an exterior facing indoor location on the second floor.

2. Results

Observation of visible surfaces (i.e., carpets, wallpaper, furniture, etc.) in the evaluated area did not reveal microbial amplification (growth) which would indicate a continued microbiological presence. Reports from maintenance personnel indicated that building remediation was initiated subsequent to the initial consultant's report, which indicated the presence of *Stachybotrys sp.* in bulk and air samples. Remediation efforts included exterior strategies designed to arrest the incursion of "ground waters" into the building; the clean-up of contaminated surfaces (including removal with a vacuum installed with a high efficiency particulate air [HEPA] filter), and the decontamination (5 to 10% bleach solution) of surfaces after clean-up attempts. Air sampling data from the consultant's follow-up visit did not reveal continued *Stachybotrys sp.* in bulk or air samples.

Analysis of bulk samples collected (outside the evaluated area) during the NIOSH site visit did not reveal the presence of *Stachybotrys sp.* The microbiological analysis results are reported in Table I. The fungi identified included *Acremonium* (predominant genus), *Aspergillus*, *Alternaria*, *Cladosporium*, and *Penicillium*. Although these fungal taxa are normal constituents of most indoor and outdoor environments, visual evidence of amplification was observed on both bulk samples. The quantity of fungi observed at this location indicates the possible incursion of water into other areas of the building and the subsequent association with microbiological contamination.

A physical inspection of select heat pumps servicing the evaluated area did not reveal any visible evidence that would indicate a microbial contamination source. The filters appeared free of debris accumulation, the interior unit insulation was in good condition, and the heating/cooling coils, and the area directly beneath, were absent of standing water and/or

Table I. Results of Bulk Sample Analysis

SAMPLE LOCATION	TOTAL FUNGI*	TAXA RANK	TOTAL BACTERIA*	TAXA RANK
Wallpaper Sample #1a	106,000	Acre>>Asp=Alt>Yea	18,000	Staph>Pseudo
Wallpaper Sample #1b	460,000	Acre=Asp=Pen=Clad	ND	
Wallpaper Sample #2a	50,000	Acre>>Pen>Asp>Yea	2,500	Staph
Wallpaper Sample #2b	360,000,000	Acre>>Asp>Pen	10,000	Pseudo
a = clean area on sample b = "dirty" area on sample	Acre = <i>Acremonium</i> Asp = <i>Aspergillus</i> Alt = <i>Alternaria</i> Clad = <i>Cladosporium</i> Pen = <i>Penicillium</i> Yea = unidentified yeasts		Staph = <i>Staphylococcus</i> Pseudo = <i>Pseudomonas</i>	

*Results presented as Colony Forming Unit per gram of material (CFU/gm)

"slime." The filters appeared to be well-seated in their tracks. The heat pumps and atrium heating, ventilating, and air-conditioning units were reported to be on a three month preventive maintenance schedule.

Environmental CO₂ measurements are presented in Figure 2. Measurements were made at 12 locations throughout the evaluated area (Figure 1) in the Atrium area, in a remote office, and at one outdoor location. Carbon dioxide concentrations ranged from 875 to 1625 ppm during the morning measurement period (~11:30 a.m.), from 1075 to 1500 ppm during the mid-afternoon period (~1:30 p.m.), and from 1100 to 1650 ppm during the late afternoon period (~3:00 p.m.). The outdoor concentration was 350 ppm during all measurement periods. The highest measurement was 1650 ppm, taken in Room 2 during the

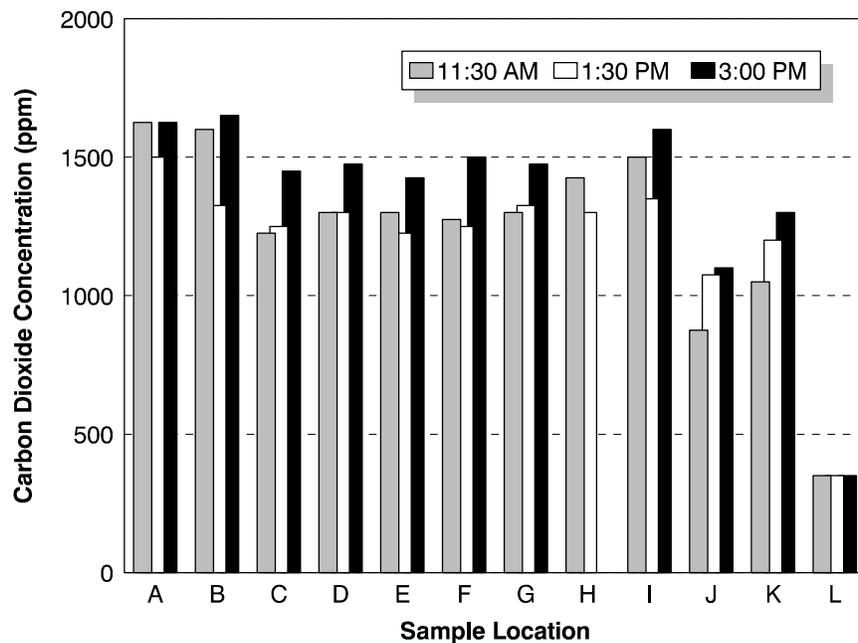


Figure 2. Carbon Dioxide Measurement Results

late afternoon period. All CO₂ measurements in the evaluation exceeded the ASHRAE comfort criterion level of 1000 ppm for indoor environments. The lack of outdoor air provision into the occupied spaces is responsible for the elevated CO₂ concentrations observed throughout the evaluated area. This condition was observed for other areas of the building (i.e., the Atrium area and the remote office location), and therefore not localized on the evaluation area alone.

Temperatures ranged from 72.1 to 74.2°F during the morning measurement period, 71 to 75°F during the mid-afternoon measurement period, and from 72.2 to 75.2°F during the late afternoon (Figure 3). The outdoor temperature ranged from 75.3 to 77.8°F during these time periods. The relative humidity (RH) levels in the building remained fairly consistent (mean value of 41% with a sample standard deviation of 3%) between locations and throughout the day (Figure 4). The outdoor RH was also consistent throughout the day (average value of 54% with a sample standard deviation of 1.4%). The indoor temperatures and relative humidities are within the limits recommended by ASHRAE, as shown in the thermal comfort chart (Figure 5). This chart specifies the acceptable (at least 90% would be expected to feel thermally comfortable) ranges of operative temperature and humidity for persons clothed in

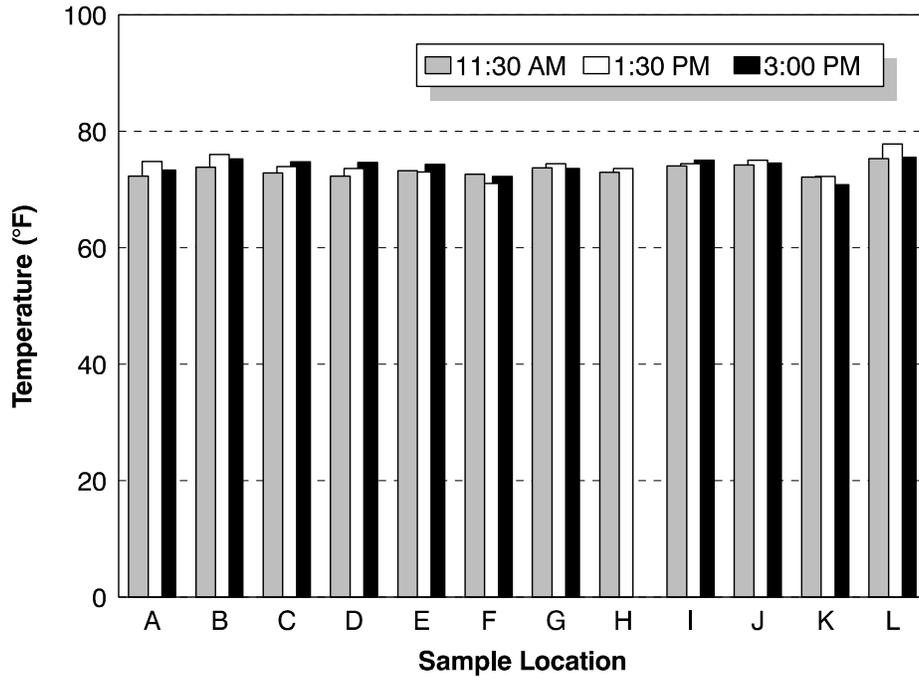


Figure 3. Temperature Measurement Results

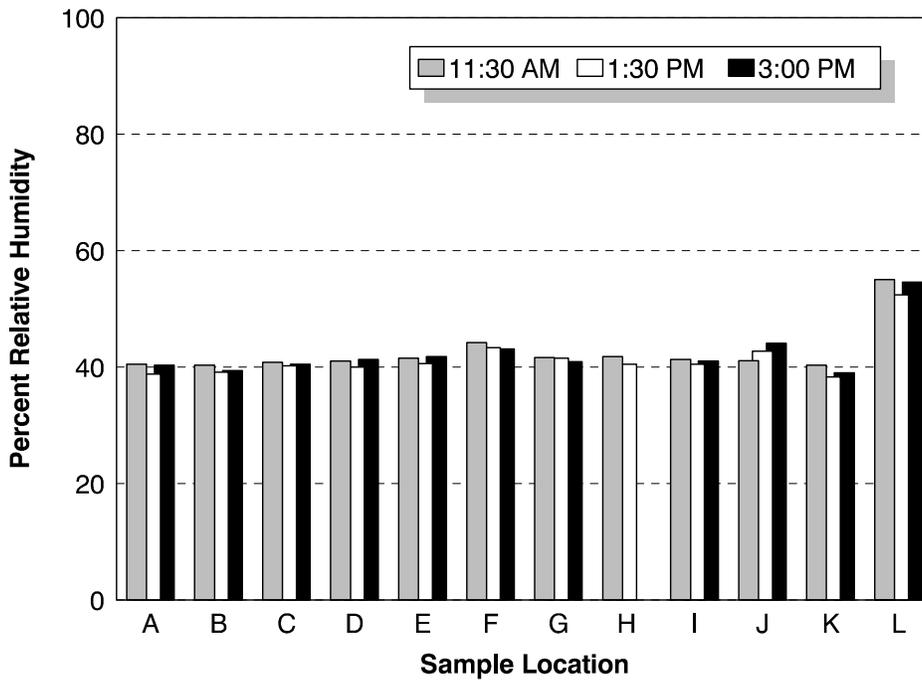


Figure 4. Relative Humidity Measurement Results

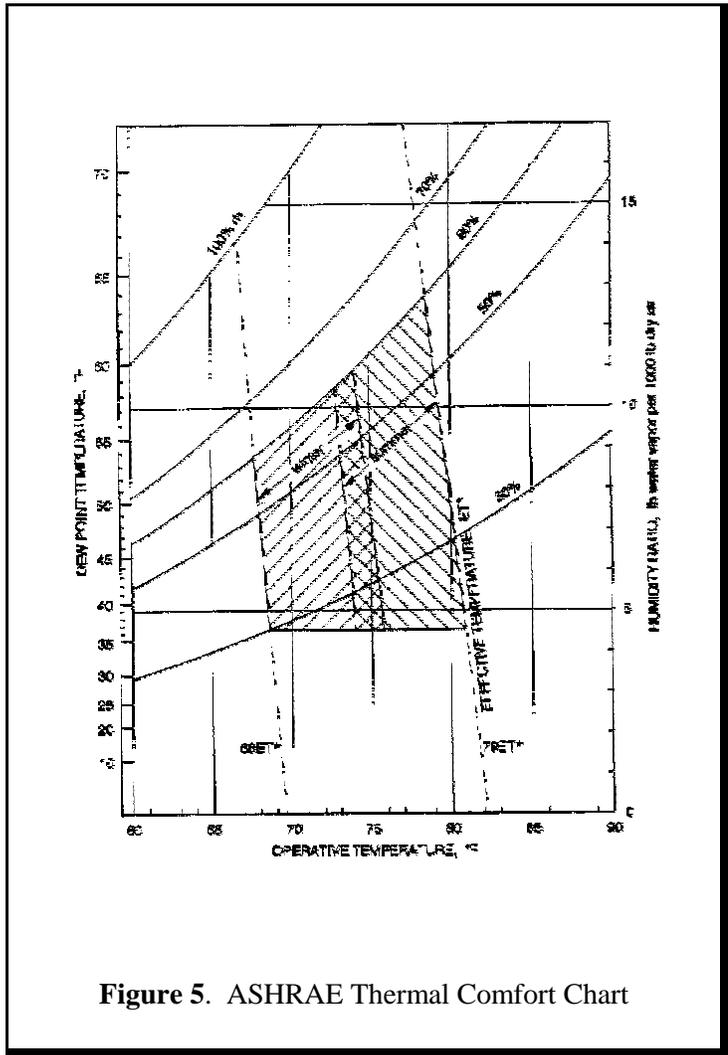


Figure 5. ASHRAE Thermal Comfort Chart

typical summer and winter clothing, performing mainly sedentary activity.

B. Medical Investigation

Eight workers were interviewed by the medical investigators during the visit; an additional four workers were interviewed by telephone. Their job categories included receptionists, clerks, medical assistants, physicians' assistants, and nurses. In order to evaluate the progress of the illnesses described by some employees, they were contacted by a NIOSH physician by telephone as late as mid-May 1993. Because cases were often described together by the employees, in this report the workers will be designated by alphabetical letters.

Three workers (A,B, and C) said they were not experiencing symptoms or illness at the time of the interview, although one had a single 3-day episode of a cough with a fever 8 months earlier; it resolved and had not reoccurred at the time of the interview. One worker (D) complained of frequent sneezing, and related a single episode of bronchitis which was resolved with antibiotic therapy.

Two workers described episodes of skin urticaria ("hives"). The episode affecting worker E occurred about 4 months before our visit, lasted for 2 weeks without respiratory symptoms and seemingly improved on weekends. It was accompanied by eye irritation, which was relieved by eye drops provided by a physician. The episode did not reoccur, although the worker also reported having "2 or 3" upper respiratory infections the previous winter, all of which were resolved with antibiotic and decongestant therapy. Worker F related a single episode of hives without respiratory or other symptoms, which lasted one day and was treated with antihistamines. This worker speculated that the hives were a reaction to some undetermined food.

One employee (G) related frequent episodes of sinus infection, one (H) described a case of otitis media (middle ear infection) and also complained of frequent fatigue.

One employee (I) related frequent episodes of both sinusitis and otitis, and was told by an allergist that these were caused by allergies to indoor substances. After an episode of several days of cough and shortness of breath, this worker saw a physician who after performing medical tests, diagnosed "granulomatous lung disease." However the worker was also told, after a biopsy, that this was neither tuberculosis nor sarcoidosis, which are the most frequent causes of granulomas in the lung (the term "granuloma" describes the appearance of the abnormal lung tissue when seen under a microscope). This employee also developed asthma and was being treated with appropriate medications. Although the NIOSH physicians were given copies of this worker's medical records, the records submitted described only an evaluation for allergies during which the worker reacted to a variety of allergens including weeds, molds, mites, and feathers.

Another employee (J) related a history of a lung condition that involved episodes of shortness of breath. In these episodes the employee was found to have abnormal blood gases and was hospitalized for several days, but the employee said that no cause was found. The employee also told of being diagnosed with chronic fatigue syndrome due to persistent weakness and related weakness and pain in both knees. This employee also stated that the illness had resulted in impaired ability to recall both long-term memories and short-term tasks (such as remembering to pay bills).

Employee K was hospitalized with a diagnosis of viral meningitis. This employee temporarily had persistent headaches after discharge from the hospital, but eventually the

case completely resolved. Employee K was more recently treated for sinusitis.

One employee (L) who initially presented with severe headache and visual changes was hospitalized with an initial diagnosis of viral meningitis. After additional investigation, this diagnosis was changed to pseudotumor cerebri, a disease in which a person's body produces too much of the fluid which normally surrounds the brain and spinal cord.

Several employees indicated that in 1 week two employees were discovered to have lost pregnancies to fetal demise. Both had left Kaiser employment before our visit. Several of the employees we spoke with said the fetal losses coincided in the same month as the onset of the illnesses of employees J and L.

V. DISCUSSION

The initial consultant's report (based on site visits of December 23 and February 13, 1992) states that various species of mold were found in the OB/GYN wing; when the mold was cultured. *Stachybotrys* was among the species identified. By the time of the NIOSH visit, however, the condition had been remediated. Although visible mold was found behind wallpaper on the second floor, *Stachybotrys* was not identified among the fungal taxa cultured. Because *Stachybotrys* or other mold species were not visibly apparent in the evaluated area, it was not possible to conduct measurements that would assess whether

Stachybotrys mycotoxins or materials carrying *Stachybotrys* mycotoxins, were present in the air of the building, or to quantify the workers' exposures to those mycotoxins if such were present.

Some of the employees contacted in the course of this evaluation indicated their concern, that workers were experiencing frequent infections and illnesses, because exposure to *Stachybotrys* toxin had caused immunosuppression (decreased function of the immune system) as described in animal and human exposures. However, immunologic function is typically carried out through a complex system of blood chemicals (antibodies) and white blood cells. A decrease in the number of white blood cells can result in a loss of immune function and increased susceptibility to infection. An example of this is seen in patients receiving certain anticancer drugs. As a side effect, these drugs cause a profound reduction in the number of white blood cells. Patients are very susceptible to infection until the treatment is stopped and the white blood cell count returns to normal. This phenomenon has been observed in animal stachybotrytoxicosis. For example, in the later stage of poisoning in the horse, the animal develops fever, the number of white cells in the blood drops further, and the animal frequently develops infections.¹

It should also be noted that this susceptibility to infection is a late stage, preceded by other obvious signs of poisoning. In horses exposed by eating mold-contaminated feed, initially inflammation or swelling is seen in the tissues of the nose, mouth, and eyes. Later sores are seen on the lips, tongue, and tonsils. The next stage includes first an increase and then a sharp decrease in the number of white blood cells, and disturbances of blood clotting.¹

In the medical records that we examined of Kaiser employees who have felt ill, however, the blood tests we reviewed did not show the profoundly low white blood counts which are associated with suppressed immunity in animal stachybotrytoxicosis. Neither did we hear or read reports of mucosal inflammation or disturbances of blood clotting. We therefore do not feel that *Stachybotrys* toxin exposure can account for the symptoms reported by Kaiser employees. It is thus unlikely that there is an association between the case of viral

meningitis and a mold-related workplace exposures. Pseudotumor cerebri, the disease experienced by employee L, is not known to be associated with infectious agents or workplace exposures. It is extremely unlikely that this case was related to mold exposures at work.

One of the principles of toxicology, the science that describes the effect of poisons, is that most poisons have a dose-response effect. This means that the more poison someone absorbs, the greater, or more serious, will be the resulting health effect. For example, if a person breathes a small amount of carbon monoxide there is no visible effect but larger amounts cause nausea and headache, and so on up to coma and finally death. Reports indicate that tricothecene poisoning in animals is dose-dependant.⁴ In addition, studies of the effect of tricothecenes in extracts from *Stachybotrys* include tests in which the toxins were applied to cultures of rat cells. A dose-response was seen in the effect of the extract. As more extract was applied, a greater effect on cell function was seen.¹³ This suggests that a dose-response effect is likely in at least some of the toxic effects of *Stachybotrys* mycotoxins in humans.

In this regard, then, the reports of *Stachybotrys* exposure in humans must be examined in order to compare the exposures reported in those cases with the likely exposures in the Kaiser Northlake Atrium building. The case reports of "pulmonary mycotoxicosis" all involved heavy exposures to airborne dust from stored animal feed contaminated by bacteria and fungi. Some of the exposed farmers described dust concentrations so thick that they could see no more than 1 or 2 feet.^{14,15} Russian reports of human stachybotrytoxicosis describe exposures to dust aerosols laden with mold spores in typically dusty work environments such as cottonseed oil plants, grain elevators, textile mills, and grain mills.⁹ It is doubtful that workers at Kaiser Northlake were ever exposed to airborne fungal levels as high as those which presumably occurred in these incidents.

The inferences from these reports do not support the conclusion that worker health effects at Kaiser Northlake were caused by exposure to airborne mycotoxins elaborated by *Stachybotrys*. However, this must be balanced with a report from another research group, who detected *Stachybotrys* mycotoxins in mold scraped from a ceiling board and in fungi recovered in air samples from a home in which a family was experiencing a variety of symptoms. Unfortunately, the authors did not test the family members in order to see if they had absorbed detectable levels of the toxins. However, the family's symptoms resolved after the visible fungal contamination was removed. The authors take this as implicit evidence that the illnesses were caused by exposure to *Stachybotrys* mycotoxins.⁵ This is consistent with Russian studies of people heavily exposed to mold spores, where patients rapidly recovered when they were removed from exposure to the source of the mycotoxins.⁹

If Kaiser employees had incurred environmental exposures to *Stachybotrys* mycotoxins exposure, the previously cited investigations suggest that their symptoms would have resolved when exposures ceased. Such has not been the case. Workers at Kaiser Northlake continued to describe health symptoms when *Stachybotrys* was no longer detected in the workplace.

We believe that the available evidence does not support the conclusion that *Stachybotrys* mycotoxin exposures were responsible for the health symptoms of workers at Kaiser Northlake. Despite this conclusion, there were still problems in the workplace that could contribute to worker health symptoms. Many workers described lengthy histories of sinus infections, which they related to working in the Kaiser facility. Our investigation demonstrated that mold was still present underneath wallpaper in the facility. In addition, most of the workers we interviewed described the unusual cleaning process that was conducted in the facility the day before we arrived. We therefore assume that mold

contamination may be more prevalent at other times. One worker's medical records described tests which demonstrated allergies to several types of molds. Others might have similar allergies. Exposures of sensitive employees to mold allergens will cause allergic reactions including inflammation of the mucosa with itching and runny nose. Continuing or frequent exposures may cause a chronic state of inflammation of the nose and paranasal sinuses. This inflammation can cause an increased susceptibility to bacterial infection. Different investigations have suggested that allergies can be associated with up to 32% of cases of acute sinusitis and up to 67% of cases of chronic sinusitis.⁴² Thus the mold contamination seen at Kaiser Northlake can result in adverse health effects even if mycotoxins are not present.

VI. RECOMMENDATIONS

Results of the medical evaluation, environmental measurements, and physical inspection of building components and systems revealed persistent mold contamination as well as ventilation system deficiencies. Based on the results and observations of this evaluation, the following recommendations are offered to correct those deficiencies and optimize employee comfort.

1. Kaiser Northlake administration should continue its efforts to prevent mold growth in reservoirs, amplification, and mold dissemination in the facility. The extensive remediation efforts which were conducted in response to the consultant's report and in advance of the NIOSH visit are commendable. Continued vigilance of this nature should pores of all kinds.
2. Based on a physical inspection by the investigating team and reports from building maintenance personnel, the ventilation systems were designed to provide 100% recirculated air (0% outside air). The lack of outside air supply into the occupied spaces is responsible for the elevated CO₂ concentrations observed throughout the evaluated area. The ventilation systems should re-designed to provide a minimum amount of outside air that will conform to the ASHRAE guideline of 20 cubic feet per minute of outside air per person for office environments. Additionally, ASHRAE has established criteria for acceptable air quality for other environments (i.e., medical examination rooms, diagnostic laboratories, waiting area, etc.) that require special consideration. These environments should be assessed as to their ability to meet the ASHRAE criteria.³⁸
3. Steps should be taken to increase the efficiency of the filters in the heat pumps to promote the removal of airborne contaminant particles. The metal mesh filters currently being used have very low efficiency (approximately 10%) and require a diligent preventative maintenance program to ensure particulate collection efficiency. Contact the heat pump and various filter manufacturers to ensure the selection of an alternative filter that will not burden the system fan beyond its capabilities. Higher efficiency (not necessarily "high efficiency") air filters will be advantageous because they will contribute to the success of the administration's plan to minimize mold reservoirs and amplification and dissemination of mold in the facility.

VII. REFERENCES

1. Hintikka, E [1977]. Stachybotryotoxicosis as a veterinary problem. In: Rodricks J, Hesseltine C, Mehlman M, eds. Mycotoxins in human and animal health. Park Forest South, IL: Pathotox Publishers, pp. 277-284.
2. Forgacs J [1972]. Stachybotryotoxicosis. In: Kadis S, Ciegler A, and Ajl S, eds. Microbial toxins. Vol. 8. New York, NY: Academic Press, pp. 95-128.
3. Kozak P. Jr., Gallup J, Cummins L, Gillman S [1985]. Endogenous mold exposure: environmental risk to atopic and nonatopic patients. Chapter 10. In: Gammage R, Kaye S, Jacobs V, eds. Indoor air and human health. Chelsea, MI: Lewis Publishers, pp. 149-170.
4. Jarvis B [1990]. Mycotoxins and indoor air quality. In: Morey P, Feeley J Jr., Ohen J eds. Biological contaminants in indoor environments, ASTM STP. Philadelphia: American Society for Testing and Materials, pp. 201-211.
5. Croft W, Jarvis B, Yatawara C [1986]. Airborne outbreak of trichothecene toxicosis. Atmosph Environ 20: 549-552.
6. Bisset J [1987]. Fungi associated with urea-formaldehyde foam insulation in Canada. Mycopathologia 99: 47-56.
7. Burge H [1985]. Indoor sources for airborne microbes. In: Gammage R, Kaye S, Jacobs V eds. Indoor air and human health. Chelsea, MI: Lewis Publishers, Inc., pp. 139-148.
8. Ellenhorn M, Barceloux D [1988]. Trichothecenes. In: Medical toxicology, part 5. Natural toxins. New York, NY: Elsevier, pp. 1312-1314.
9. Newberne P [1974]. Mycotoxins: toxicity, carcinogenicity, and the influence of various nutritional conditions. Environmental Health Perspect 9:1-32.
10. Bata A, Harrach B, Ujszaszi K, Kis-Tamas A, Lasztity R [1985]. Macrocyclic trichothecene toxins produced by *Stachybotrys atra* strains isolated in middle Europe. Appl Environ Microbiol 49: 678 - 681.
11. Harrach B, Nummi M, Niku-Paavola M, Mirocha C, Palyusik M [1982]. Identification of "water soluble" toxins produced by a *Stachybotrys atra* strain from Finland. Appl Environ Microbiol 44:494-495.
12. El-Maghraby O, Bean G, Jarvis B, Aboul-Nasr M [1991]. Macrocyclic trichothecenes produced by *Stachybotrys* isolated from Egypt and Eastern Europe. Mycopathologia 113: 109-115.
13. Sorenson W, Frazer D, Jarvis B, Simpson J, Robinson V [1987]. Trichothecene mycotoxins in aerosolized conidia of *Stachybotrys atra*. Appl Environ Microbiol 53:1370-1375.
14. Emanuel D, Wenzel F, Lawton B [1975]. Pulmonary mycotoxicosis. Chest 67: 293-297.

15. May J, Stallones L, Darrow D, Pratt D [1986]. Organic dust toxicity (pulmonary mycotoxicosis) associated with silo unloading. *Thorax* 41:919- 923.
16. Kreiss KK, Hodgson MJ [1984]. Building associated epidemics. In: Walsh PJ, Dudney CS, Copenhaver ED, eds. *Indoor air quality*. Boca Raton, FL: CRC Press, pp 87-108.
17. Gammage RR, Kaye SV, eds. [1985]. *Indoor air and human health: Proceedings of the Seventh Life Sciences Symposium*. Chelsea, MI: Lewis Publishers, Inc.
18. Woods JE, Drewry GM, Morey PR [1987]. Office worker perceptions of indoor air quality effects on discomfort and performance. In: Seifert B, Esdorn H, Fischer M, et al., eds. *Indoor air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate*. Berlin Institute for Water, Soil and Air Hygiene.
19. Skov P, Valbjorn O [1987]. Danish indoor climate study group. The "sick" building syndrome in the office environment: The Danish town hall study. *Environ Int* 13:399-349.
20. Burge S, Hedge A, Wilson S, Bass JH, Robertson A [1987]. Sick building syndrome: a study of 4373 office workers. *Ann Occup Hyg* 31:493-504.
21. Kreiss K [1989]. The epidemiology of building-related complaints and illness. *Occupational Medicine: State of the Art Reviews*. 4(4):575-592.
22. Norbäck D, Michel I, Widstrom J [1990]. Indoor air quality and personal factors related to the sick building syndrome. *Scan J Work Environ Health* 16:121-128.
23. Morey PR, Shattuck DE [1989]. Role of ventilation in the causation of building-associated illnesses. *Occupational Medicine: State of the Art Reviews*. 4(4):625-642.
24. Mendell MJ, Smith AH [1990]. Consistent pattern of elevated symptoms in air-conditioned office buildings: A reanalysis of epidemiologic studies. *Am J Public Health*. 80(10):1193-1199.
25. Molhave L, Bach B, Pedersen OF [1986]. Human reactions during controlled exposures to low concentrations of organic gases and vapours known as normal indoor air pollutants. *Environ Int* 12:167-175.
26. Fanger PO [1989]. The new comfort equation for indoor air quality. *ASHRAE J* 31(10):33-38.
27. Burge HA [1989]. Indoor air and infectious disease. *Occupational Medicine: State of the Art Reviews* 4(4):713-722.
28. Robertson AS, McInnes M, Glass D, Dalton G, Burge PS [1989]. Building sickness, are symptoms related to the office lighting? *Ann Occup Hyg* 33(1):47-59.
29. Wallace LA, Nelson CJ, Dunteman G [1991]. Work place characteristics associated with health and comfort concerns in three office buildings in Washington, D.C. In: Geshwiler M, Montgomery L, and Moran M, eds. *Healthy buildings. Proceedings of the ASHRAE/ICBRSD conference IAQ'91*. Atlanta, GA. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.

30. Haghghat F, Donnini G, D'Addario R [1992]. Relationship between occupant discomfort as perceived and as measured objectively. *Indoor Environ* 1:112-118.
31. NIOSH [1991]. Hazard evaluation and technical assistance report: Library of Congress Madison Building, Washington, D.C. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, NIOSH Report No. HETA 88-364-2104 - Vol. III.
32. Skov P, Valbjørn O, Pedersen BV [1989]. Influence of personal characteristics, job related factors, and psychosocial factors on the sick building syndrome. *Scand J Work Environ Health* 15:286-295.
33. Boxer PA [1990]. Indoor air quality: A psychosocial perspective. *J Occup Med* 32(5):425-428.
34. Baker DB [1989]. Social and organizational factors in office building-associated illness. *Occupational Medicine: State of the Art Reviews*. 4(4):607-624.
35. CDC [1992]. NIOSH recommendations for occupational safety and health: Compendium of policy documents and statements. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. DHHS (NIOSH) Publication No. 92-100.
36. Code of Federal Regulations [1989]. OSHA Table Z-1-A. 29 CFR 1910.1000. Washington, DC: U.S. Government Printing Office, Federal Register.
37. ACGIH [1991]. 1991-1992 Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
38. ASHRAE [1990]. Ventilation for acceptable indoor air quality. Atlanta, GA: American Society of Heating, Refrigerating, and Air-conditioning Engineers. ANSI/ASHRAE Standard 62-1989.
39. ASHRAE [1992]. Thermal environmental conditions for human occupancy. Atlanta, GA: American Society for Heating, Refrigerating, and Air-conditioning Engineers. ANSI/ASHRAE Standard 55-1992.
40. ACGIH [1989]. Guidelines for the assessment of bioaerosols in the indoor environment. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.
41. NIOSH [1991]. Building air quality: a guide for building owners and facility managers. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 91-114.
42. Spector SL [1992]. The role of allergy in sinusitis in adults. *Journal of Allergy and Clinical Immunology* 90:518-20.

VIII. AUTHORSHIP and ACKNOWLEDGEMENT

Report Prepared by:

Scott Deitchman, M.D., M.P.H.
Supervisory Medical Officer
Medical Section
Cincinnati Office

Kenneth Martinez, M.S.E.E.
Industrial Hygiene Engineer
Industrial Hygiene Section
Cincinnati Office

Susan Upham, M.D., M.P.H.
Occupational Medicine Resident
University of Massachusetts

Originating Office:

Hazard Evaluations and Technical
Assistance Branch
Division of Surveillance, Hazard
Evaluations and Field Studies
National Institute for Occupational
Safety and Health
4676 Columbia Parkway
Cincinnati, Ohio 45226

IX. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report may be freely reproduced and are not copyrighted. Single copies of this report will be available for a period of 90 days after the date of this report from the NIOSH Publications Office, 4676 Columbia Parkway, Cincinnati, OH 45226. To expedite your request, include a self-addressed mailing label along with your written request. After this time, copies may be purchased from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161. Information regarding the NTIS stock number may be obtained from the NIOSH Publications Office at the Cincinnati address.

Copies of this report have been sent to:

1. Employee representative
2. Medical Facility Administrator, Kaiser Permanente Northlake Atrium Building
3. Counsel, Kaiser Foundation Health Plan, Inc.

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.