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PREFACE

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The Hazard Evaluations and Technical Assistance Branch also provides, upon request, technical and consultative assistance to Federal, State, and local agencies; labor; industry; and other groups or individuals to control occupational health hazards and to prevent related trauma and disease. Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

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This report was prepared by Nancy Clark Burton and Kenneth F. Martinez, of the Hazard Evaluations and Technical Assistance Branch, Division of Surveillance, Hazard Evaluations and Field Studies (DSHEFS). Field assistance was provided by Gregory Kinnes. Analytical support was provided by James Arnold (ergosterol analysis), Analytical Research and Development Branch, Division of Physical Sciences and Engineering, the Health Effects Laboratory Division, P & K Microbiology Services, Inc., and Data Chem Laboratories. Desktop publishing was performed by Ellen E. Blythe and Nichole Herbert. Review and preparation for printing was performed by Penny Arthur.

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SUMMARY

In April 1997, the National Institute for Occupational Safety and Health (NIOSH) received a management request for a health hazard evaluation (HHE) at the Social Security Administration (SSA) facility in Batavia, Ohio. The requestor asked NIOSH to evaluate indoor environmental conditions including ventilation and possible exposure to microbial contaminants due to frequent flooding of the building. A walk–through inspection of the building was conducted on June 17, 1997, and some general indicators of indoor environmental quality (IEQ) were measured (carbon dioxide [CO_2] concentration, temperature, and relative humidity [RH]). An exposure assessment for potential fungal contamination was conducted on July 15–16, 1997. Area air samples were collected for culturable bacteria and fungi, endotoxins (a component in cell membranes of gram–negative bacteria), ergosterol (a component in cell membranes of fungi), and total fungal spores. Three dust samples were also collected and analyzed for culturable fungi, and CO_2 concentration, temperature, and RH measurements were repeated. Informal voluntary interviews were conducted with 9 of the 17 employees to gather information on the workplace environment and symptoms that they associated with the workplace.

The SSA facility is ventilated by two household furnace/air–conditioning units. There was no provision for bringing in outside air through the mechanical ventilation systems. There were visible gaps around the edges of the low–efficiency glass fiber filters allowing unfiltered air to be recirculated. An active culvert, that overflows during very heavy rains, runs at an angle underneath the rear and side of the building.

Indoor CO_2 concentrations ranged from 1300 to 2275 parts per million (ppm) exceeding IEQ CO_2 guidelines established by NIOSH and the American Society of Heating, Refrigerating, and Air–Conditioning Engineers (ASHRAE) of 800 and 1000 ppm, respectively. Air temperatures ranged from 72 to 81°F. During the second site visit, the indoor temperatures exceeded the ASHRAE guideline for summer months. RH levels ranged from 32% to 47% and were within acceptable limits. The most prevalent symptoms and health concerns reported by employees were sinus problems, coughing, aggravation of existing allergies, nasal stuffiness, and headaches.

The geometric mean fungal air concentrations within the building ranged from 68 to 161 colony forming units per cubic meter of air (CFU/m^3). Outside the building, the geometric mean fungal air concentration was 1368 CFU/m^3. *Cladosporium* was the predominant genera identified for both outdoor and indoor culturable air samples. Acceptable levels of airborne microorganisms have not been established, primarily due to the varying immunogenic susceptibilities of individuals. Higher percentages of *Aspergillus* and *Penicillium* species were detected indoors when compared to outdoors, probably from insufficient remediation activities following flooding. Endotoxin levels

ranged from non–detectable to 2.7 endotoxin units per cubic meter (EU/m^3) in indoor air samples compare to outdoor concentrations of 0.8 EU/m^3. Ergosterol was not detected in the area air samples.

The monitoring data collected during this evaluation indicate that there is a need to improve general ventilation within the SSA building and that there appears to be residual microbial contamination from past flooding incidents. Recommendations for improving the general ventilation systems, carpet cleaning, and clean–up after water incursion episodes are included in the report.

Keywords: SIC Code 9441 (Administration of Social, Human Resource, and Income Maintenance), indoor environmental quality, IEQ, flooding, microbial sampling, fungi, spores, endotoxins, *Aspergillus*, *Cladosporium*, *Penicillium*, carbon dioxide, temperature, relative humidity.

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INTRODUCTION

In April 1997, the National Institute for Occupational Safety and Health (NIOSH) received a management request for a health hazard evaluation (HHE) at the Social Security Administration (SSA) facility in Batavia, Ohio. The request asked NIOSH to evaluate indoor environmental conditions including ventilation and possible exposure to microbial contaminants due to frequent flooding of the building. Employees were reporting a high incidence of allergies, colds, and respiratory infections. An initial walk–through inspection of the building was conducted on June 17, 1997, and an exposure assessment for potential fungal contamination was conducted on July 15–16, 1997.

BACKGROUND

The SSA facility, which was built in the early 1970s, is a single story concrete block building with a concrete floor and metal roof. Most floor surfaces were carpeted except in the multi-purpose (lunch) room. There are 17 employees in the building. The building is served by two household heating, ventilation, and air–conditioning (HVAC) package units. There is no provision for bringing in outside air through the mechanical ventilation systems. Recirculated air is distributed through ceiling supply diffusers. Ceiling fans, to improve air circulation throughout the work space, were installed in 1996.

There have been several flooding incidents at this facility. An active culvert, that overflows during very heavy rains, runs at an angle underneath the rear and side of the building. In 1996, an underground drainage system was installed around the edges of the building to help control flooding. The building sits at the bottom of a hill where run–off from rain storms is occasionally greater than the culvert can handle and, as a result, portions of the building are flooded. This occurred in between the two site visits on June 18, 1997. Employees reported that, in 1994, mushrooms appeared along the inside side wall where the culvert had overflowed and water had entered the building.

METHODS

Indoor Environmental Quality

The ventilation system for the building was examined and indicators of indoor environmental quality (IEQ) were measured. These indicators were carbon dioxide (CO_2) concentration, temperature, and relative humidity (RH). Measurements were made at seven and five inside locations (first and second surveys, respectively), and outside the building as shown in Figure 1. For the first survey, measurements were made at eight locations in the morning (about 9:50 a.m. to 10:20 a.m.), mid–day (about 1:00 p.m. to 1:30 p.m.), and late afternoon (about 3:30 p.m. to 4:00 p.m.). For the second survey, measurements were made at six locations in the morning (about 8:30 a.m. to 9:00 a.m.), mid–day (about 10:30 a.m. to 11:00 a.m.), early afternoon (about 1:00 p.m. to 1:30 p.m.), and late afternoon (about 3:15 p.m. to 3:45 p.m.). Smoke tubes were used to visualize airflow patterns in the evaluated area and to determine potential pollutant pathways into and throughout this building. To determine moisture content, conductivity measurements were made of the solid building materials using a Delmhorst Model BD-8 conductivity meter.

Real–time CO_2 concentrations were measured using a Gastech Model RI–411A, portable CO_2 indicator. This battery–operated instrument uses a non–dispersive infrared absorption detector to measure CO_2 in the range of 0–4975 parts per million (ppm), with an accuracy of ±25 ppm. Instrument zeroing and calibration were performed prior to use with zero air and a known concentration of CO_2 span gas (800 ppm).

Real–time temperature and humidity measurements were made using a Vaisala, Model HM 34, battery–operated meter. This meter is capable of providing direct readings for dry–bulb temperature and RH, ranging from –4 to 140°F and 0 to 100%,

respectively. Instrument calibration is performed monthly using primary standards.

Microbiological Sampling

To determine concentrations of culturable airborne fungi, an Andersen single–stage viable cascade impactor was used at a calibrated flowrate of 28.3 liters per minute (Lpm). The Andersen single–stage impactor is designed to collect particles 0.65 micrometers (μm) or larger. Samples were collected in sets of four replicates using malt extract agar (MEA) for general fungal isolation and dichloran glycerol agar (DG18) for the isolation of xerophilic fungi (those fungi that are tolerant of low moisture conditions). Sampling time periods for each of the five inside and one outside sites were randomly allocated over the two days of monitoring. All inside samples were collected over a ten–minute time period and all outside samples were collected over a five–minute time frame. Sample plates were incubated at 25°C. The taxa and rank of the collected microorganisms were determined by morphological characteristics.

Area air samples for endotoxins (a component in cell membranes of gram–negative bacteria), ergosterol (a component in cell membranes of fungi), and total fungal spores were collected at the six sampling locations for the two days of the second survey. Twelve endotoxin samples were collected on tared 5.0 μm pore size, 37 millimeter (mm) polyvinyl chloride filters using a calibrated flowrate of 2 Lpm. The samples were analyzed for endotoxin content using the Kinetic–QCL assay kit (BioWhittaker, Walkerville, Maryland) according to the manufacturer's recommended procedures. For these analyses, 10 endotoxin units (EU) are equivalent to one nanogram of endotoxin. The limit of detection (LOD) for the analyses was 0.5 EU per sample.

Twelve ergosterol samples were collected on 25 mm polytetrafluoroethylene (PTFE) filters with 0.2 μm pore size, using a calibrated flowrate of 1.5 Lpm. Control samples with known concentrations of ergosterol were run with the field samples. The recovery of ergosterol from each filter sample was facilitated with methylene chloride. Filter extracts were analyzed by high performance liquid chromatography with an ultraviolet detector set at 282 nanometers. The field samples were run in two separate sets and the method LODs were 0.04 and 0.05 micrograms (μg) per sample, respectively. The MDCs were 0.07 and 0.09 micrograms per cubic meter ($\mu g/m^3$) for the first and second sample sets, respectively, using a sample volume of 567 liters.

Twelve total fungal spore count samples were collected on open–faced 0.8 μm pore size, 37 mm mixed cellulose ester filters using a calibrated flowrate of 2 Lpm. Filters were cleared with acetone vapor, mounted in cotton blue/lactic acid, and scanned at 400x magnification using bright field or phase contrast illumination. One hundred fields were counted for each sample. Only particles greater than 2 μm in diameter were considered as possible fungal spores.

Three samples of dust were collected on mixed cellulose ester filters from the mailroom rug, the side employee entrance rug, and the rug next to the floorboard under the desk beside the side employee entrance. One square foot area of rug was vacuumed for each sample with the air sampling pump used for culturable air monitoring. The dust samples were submitted for culturable fungal analysis and were processed, extracted, and inoculated on MEA and DG18 media. The plates were incubated at 25°C, and the taxa and rank of the organisms were identified.

Employee Interviews

Informal voluntary interviews were conducted with 9 of the 17 employees to gather information on the workplace environment and symptoms that they associated with the workplace. All employees at the work site were offered the opportunity to talk to NIOSH investigators.

EVALUATION CRITERIA

Indoor Environmental Quality

A number of published studies have reported a high prevalence of symptoms among occupants of office buildings.[1,2,3,4,5] NIOSH investigators have completed over 1200 investigations of the indoor environment in a wide variety of settings since 1971. However, the great majority of these investigations have been conducted since 1979.

The symptoms reported 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.

Scientists investigating indoor environmental problems believe that there are multiple factors contributing to building–related occupant complaints.[6,7] Among these factors are imprecisely defined characteristics of (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.[4,5,6,7,8] Reports are not conclusive as to whether increases of outdoor air above currently recommended amounts are beneficial.[9] However, rates lower than these amounts appear to increase the rates of complaints and symptoms in some studies.[10] Design, maintenance, and operation of HVAC systems are critical to their proper functioning and provision of healthy and thermally comfortable indoor environments. Indoor environmental pollutants can arise from either indoor or outdoor sources.[11]

There are also reports describing results which show that occupant perceptions of the indoor environment are more closely related to the occurrence of symptoms than the measurement of any indoor contaminant or condition.[12] Some studies have shown relationships between psychological, social, and organizational factors in the workplace and the occurrence of symptoms and comfort complaints.[13,14]

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 irritant 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 office furnishings, office machines, structural components of the building and contents, tobacco smoke, microbiological contamination, and outside air pollutants; comfort problems due to improper temperature and RH conditions, poor lighting, and unacceptable noise levels; adverse ergonomic conditions; and job–related psychosocial stressors. In most cases, however, no environmental cause of the reported health effects could be determined.

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.[15,16,17] With few exceptions, pollutant

concentrations observed in the office work environment 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 and thermal comfort guidelines.[18,19] The ACGIH has also developed a manual of guidelines for approaching investigations of building–related symptoms that might be caused by airborne living organisms or their effluents.[20]

Measurement of indoor environmental contaminants has rarely proved to be helpful, in the general case, in determining the cause of symptoms and complaints except where there are strong or unusual sources, or a proved relationship between a contaminant and a building–related illness. However, measuring ventilation and comfort indicators such as (CO_2), temperature, and RH is useful in the early stages of an investigation in providing information relative to the proper functioning and control of HVAC systems.

Carbon Dioxide

Carbon dioxide is a normal constituent of exhaled breath and, if monitored, can be used as a screening technique to evaluate whether adequate quantities of outside air are being introduced into an occupied space. ASHRAE's most recently published ventilation standard, ASHRAE 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 15 cfm/person for reception areas, classrooms, libraries, auditoriums, and corridors.[19] Maintaining the recommended ASHRAE outdoor air supply rates when the outdoor air is of good quality, and there are no significant indoor emission sources, should provide for acceptable indoor air quality.

Indoor CO_2 concentrations are normally higher than the generally constant ambient CO_2 concentration (range 300-350 ppm). CO_2 concentration is used as an indicator of the adequacy of outside air supplied to occupied areas. When indoor CO_2 concentrations exceed 800 ppm in areas where the only known source is exhaled breath, inadequate ventilation is suspected.[21] Elevated CO_2 concentrations suggest that other indoor contaminants may also be increased. It is important to note that CO_2 is not an effective indicator of ventilation adequacy if the ventilated area is not occupied at its usual level.

Temperature and Relative Humidity

Temperature and RH measurements are often collected as part of an indoor environmental quality (IEQ) investigation because these parameters affect the perception of comfort in an indoor environment. The perception of thermal comfort is related to one's metabolic heat production, the transfer of heat to the environment, physiological adjustments, and body temperature.[22] Heat transfer from the body to the environment is influenced by factors such as temperature, humidity, air movement, personal activities, and clothing. The American National Standards Institute (ANSI)/ASHRAE Standard 55-1981 specifies conditions in which 80% or more of the occupants would be expected to find the environment thermally acceptable.[18] Assuming slow air movement and 50% RH, the operative temperatures recommended by ASHRAE range from 68–74°F in the winter, and from 73–79°F in the summer. The difference between the two is largely due to seasonal clothing selection. ASHRAE also recommends that RH be maintained between 30 and 60% RH.[18] Excessive humidities can support the growth of microorganisms, some of which may be pathogenic or allergenic.

Microorganisms

Microorganisms (including fungi and bacteria) are normal inhabitants of the environment. The saprophytic varieties (those utilizing non–living organic matter as a food source) inhabit soil, vegetation, water, or any reservoir that can provide an adequate supply of a nutrient substrate. Under the appropriate conditions (optimum temperature, pH, and with sufficient moisture and available nutrients)

saprophytic microorganism populations can be amplified. Through various mechanisms, these organisms can then be disseminated as individual cells or in association with soil or dust particles or water droplets. In the outdoor environment, the levels of microbial aerosols will vary according to the geographic location, climatic conditions, and surrounding activity. In a "normal" indoor environment, where there is no unusual source of microorganisms, the level of microorganisms may vary somewhat as a function of the cleanliness of the HVAC system and the numbers and activity level of the occupants. Generally, the indoor levels are expected to be below the outdoor levels (depending on HVAC system filter efficiency) with consistently similar ranking among the microbial species.[23,24]

Some individuals manifest increased immunologic responses to antigenic agents encountered in the environment. These responses and the subsequent expression of allergic disease is based, partly, on a genetic predisposition.[25] Allergic diseases which have been reported to be associated with exposures in indoor environments include allergic rhinitis (nasal allergy), allergic asthma, allergic bronchopulmonary aspergillosis (ABPA), and extrinsic allergic alveolitis (hypersensitivity pneumonitis).[23] Allergic respiratory diseases resulting from exposures to microbial agents have been documented in agricultural, biotechnology, office, and home environments.[26,27,28,29,30,31,32,33]

Symptoms vary with the type of allergic disease: (1) allergic rhinitis is characterized by episodes of sneezing, itching of the nose, eyes, palate, or pharynx, nasal stuffiness with partial or total airflow obstruction, and rhinorrhea with postnasal drainage; (2) allergic asthma is characterized by episodic or prolonged wheezing and shortness of breath due to bronchial narrowing; and (3) ABPA is characterized by the production of IgE and IgG antibodies with symptoms of cough (which is sometimes productive of mucous), fatigue, low grade fever, and wheezing.[23,34] Heavy exposures to airborne microorganisms can result in an acute form of extrinsic allergic alveolitis which is characterized by chills, fever, malaise, cough, and dyspnea (shortness of breath) appearing 4 to 8 hours after exposure. Onset of the chronic form of extrinsic allergic alveolitis is thought to be induced by a continuous low–level exposure, and onset occurs without chills, fever, or malaise but is characterized by progressive shortness of breath with weight loss.[35] However, despite these relatively well–defined diseases which have been reported to occur in office environments, as described previously, symptoms most commonly encountered by office workers are generally not associated with any particular medical diagnosis or etiologic agent.

Acceptable levels of airborne microorganisms have not been established, primarily because allergic reactions can occur even with relatively low air concentrations of allergens, and individuals differ with respect to immunogenic susceptibilities. The current strategy for on–site evaluation of environmental microbial contamination involves an inspection to identify sources (reservoirs) of microbial growth and potential routes of dissemination. In those locations where contamination is visibly evident or suspected, bulk samples may be collected to identify the predominant species (fungi, bacteria, and thermoactinomycetes). In limited situations, air samples for microorganisms may be collected to document the airborne presence of a suspected microbial contaminant. Ergosterol is a component of fungal cell walls and air samples may be collected as an indicator of biomass.[36,37,38]

Bacterial Endotoxin

A bacterial endotoxin is a lipopolysaccharide compound from the outer cell membrane of gram–negative bacteria, which occur abundantly in organic dusts.[39] It has been shown that the biological properties of endotoxin vary depending upon the bacterial species from which they are derived, as well as upon the state of the growth cycle of the bacteria.[40] Endotoxins have a wide range of biological activities involving inflammatory, hemodynamic, and immunological responses. Of the most importance to occupational exposures are the activities of endotoxin in the lung.[41] The primary target cell for endotoxin–induced damage by

inhalation is the pulmonary macrophage. Human macrophages in particular have been shown to be extremely sensitive to the effects of endotoxin in vitro.[42] Endotoxin, either soluble or associated with particulate matter, will activate the macrophage, causing the cell to produce a host of mediators.[41]

Clinically, little is known about the response to inhaled endotoxins. Exposure of previously unexposed persons to airborne endotoxin can result in acute fever, dyspnea, coughing, and small reductions in forced expiratory volume in one second (FEV_1), although some investigators have not been able to demonstrate acute changes in FEV_1.[41] The effects of repeated exposure to aerosols of endotoxins in humans are not known. Some animal studies have demonstrated a chronic inflammatory response characterized by goblet cell hyperplasia and increased mucous production. This suggests that repeated exposure may cause a syndrome similar, if not identical, to chronic bronchitis.[41]

Occupational exposure criteria have not been established for bacterial endotoxin by either OSHA, NIOSH, or ACGIH. However, Jacobs has reported that a sufficient toxicological data base is believed to exist for establishing an occupational limit for endotoxin based on acute changes in pulmonary function.[41] Eight–hour time–weighted average (TWA) concentrations have been suggested for airway inflammation with increased airway reactivity (200 endotoxin units per cubic meter [EU/m^3]), for over–shift decline in FEV_1 (2,000 EU/m^3), for chest tightness (3,000 EU/m^3), and for toxic pneumonitis (10,000–20,000 EU/m^3).[40]

RESULTS

Indoor Environmental Quality

Environmental measurements are presented in Figures 2–10. During the first survey, indoor CO_2 concentrations (Figure 2) at the seven inside monitoring locations ranged from 1500 to 2125 ppm. The outdoor CO_2 concentration was 350 to 400 ppm throughout the day. During the second survey, indoor CO_2 concentrations (Figures 3 and 4) at the five inside monitoring locations ranged from 1300 to 2275 ppm. The outdoor CO_2 concentration was 350 to 475 ppm throughout the two days. All of the indoor CO_2 concentrations were higher than the NIOSH and ACGIH guidelines of 800 ppm and 1000 ppm, respectively. All three days of sampling showed a progressive build–up of CO_2 throughout the day.

During the first survey, indoor temperatures (Figure 5) ranged from 72 to 76°F. Outside temperatures were 74 to 80°F. During the second survey, indoor temperatures (Figures 6 and 7) ranged from 74 to 81°F. Outside temperatures were 71 to 95°F. Inside RHs during the first survey (Figure 8) ranged from 33 to 48% and outside RHs ranged from 65 to 84%. During the second survey, inside RHs (Figures 9 and 10) ranged from 32 to 47% and outside RHs ranged from 31 to 82%.

Microbial Analyses

Bulk Dust Samples

Microbial analysis of the three dust/debris samples indicated fungal levels ranging from 3.5×10^4 to 1.3×10^5 CFU/gm as shown in Table 1. The predominant fungal genera identified include *Alternaria*, *Aureobasidium pullulans*, *Cladosporium*, *Phoma*, and yeasts. *Aspergillus versicolor* was detected in two samples at low concentrations. *A. versicolor* has been implicated as a toxicologically significant fungus which is normally found where there is an abundance of moisture available.

Total Spores

The results of air sampling for total spores are listed in Table 2. Greater relative numbers of total spores were observed for the samples collected outdoors when compared to those collected indoors. *Cladosporium* was the main genera identified for both outdoor and indoor samples.

Culturable Air Samples

The results of the culturable air sampling for fungi are shown in Table 3. No significant differences existed in concentration or in the predominant taxa between the two types of nutrient media (MEA and DG18) used at each of the sampling locations; therefore, the data collected using the two media at each location was pooled. The geometric mean fungal concentrations within the building ranged from 68 colony forming units per cubic meter (CFU/m^3), with a geometric standard deviation of 2.8, on the side wall cabinet to 161 CFU/m^3 (geometric standard deviation of 3.2) in the mailroom. Outside the building, the geometric mean fungal concentration was 1368 CFU/m^3 (geometric standard deviation of 1.2). All of the indoor fungal concentrations were below the outdoor geometric mean fungal concentration. *Cladosporium* was the predominant genera identified for both outdoor and indoor samples.

The taxonomic ranking (i.e., the ranking of predominant genera according to frequency of occurrence) among the lesser genera of the indoor locations was dissimilar to the taxonomic ranking observed outdoors. A higher percentage of *Penicillium* species was identified indoors (range: 12 to 18%) than outdoors (4%). Also, *Aspergillus* species were identified indoors (range: 3 to 11%) while none were detected outdoors.

Endotoxins

Endotoxin levels are presented in Table 4. The 10 interior endotoxin levels ranged from non–detectable to 2.69 EU/m^3. Outdoor endotoxin concentrations were 0.8 EU/m^3 for both days.

Ergosterol

Ergosterol was not detected in any of the 12 area air samples at minimum detectable concentrations (MDCs) of 0.07 and 0.09 $\mu g/m^3$ for the first and second sample sets, respectively.

Observations

The household furnace units were lined with fibrous glass on the outside. Water from the condensate pans was visibly draining from the two air–conditioning units during both site visits. Low–efficiency glass fiber filters were used and there were visible gaps around the edges of the filters which would allow unfiltered air to be recirculated. Smoke tube patterns showed that there was little or no air movement in or out of the building when the doors were opened or closed. This is consistent with the lack of provision of outdoor air. There were no water stains on the ceiling tiles or walls. The conductivity meter showed evidence of moisture in the concrete surrounding the doorways indicating past water incursion.

Informal Interviews

Four of the employees reported sinus problems including congestion, headaches, and infections; three reported allergy problems; three reported coughing at work which did not occur at home; one reported general nasal stuffiness that occurred in the office, and one reported headaches which the employee thought were related to working conditions.

DISCUSSION AND CONCLUSIONS

Indoor CO_2 concentrations ranged from 1300 to 2275 ppm during the two surveys. CO_2 measurements over 800 –1000 ppm indicate a potential problem with air circulation and/or distribution within those offices.[19] The elevated CO_2 concentrations are likely due to the HVAC units not providing outside air to the building. During the first visit, the temperature and RH measurements were within the acceptable seasonal ranges of operative temperature and humidity suggested by ASHRAE.[18] The temperature increased and RH decreased or remained stable during the workday in the office

areas. During the second site visit, the temperatures in the late afternoon exceeded the maximum ASHRAE guideline for summer months. RH levels were within acceptable levels. The furnace filters did not fit correctly. This could result in filtration short–circuiting, i.e., particles passing around the filter and not being removed. Also, the filters were less than 20% efficient, making the ability to efficiently filter small particles, i.e. fungal spores, improbable. More than half of the employees reported symptoms which they associated with the workplace environment.

The survival and growth of microorganisms in environmental reservoirs requires a suitable nutrient source, water, and appropriate temperatures. The results from this investigation are similar to another HHE investigation in which water incursion was also documented.[43] The bulk dust samples did not reveal any significant reservoirs of fungal contamination (i.e. large active colonies) inside the building, however, *Aspergillus versicolor*, a mycotoxin producer, was detected in two samples at low concentrations. The presence of spores in the indoor environment is also a result of material that is brought in by occupants that accumulates over time. The genera of the spores identified in the indoor total spore results were similar to those identified in outdoor samples. However, the higher percentages of *Aspergillus* and *Penicillium* species detected indoors, when compared to outdoors, may be associated with unidentified fungal "sinks", e.g., residual organisms from ineffective clean–up after flooding. Also, the identification of yeast colonies in the bulk samples are characteristic of microbial proliferation due to high moisture availability. Due to the past history of flooding in this building, steps should be taken to prevent flooding and to thoroughly remediate those areas that have experienced water damage to remove any residual microbial contamination. The endotoxin concentrations were below suggested criteria that is based on changes in pulmonary function. Although fungi were detected in the other air sample using different collection media, ergosterol was not detected in the air samples collected during the second site visit probably due to the analytical method's lack of sensitivity.

RECOMMENDATIONS

(1) A minimum amount of outside air should be provided at all times while the building is occupied. ASHRAE recommends that 20 CFM of outside air be provided per person (employees and clients).[19] The HVAC filters should be upgraded to a higher efficiency to provide better particulate filtration. There are several commercially available higher efficiency filters. A ventilation engineer should be consulted to ensure that the furnace units can handle the increased pressure drop.

(2) If water incursion occurs, it should be dealt with immediately. Water should be removed immediately from furnishings, carpets, and construction materials. Fans and heat sources should be used to dry carpets and other applicable surfaces within 24 hours or such porous materials should be discarded. Any soft materials that become wet with sewage contaminated water should be promptly discarded.

(3) Carpets that have been water–damaged and were not promptly remediated should be removed to prevent them from serving as a reservoir for microbial contamination. The underlying concrete floor should be treated with an Environmental Protection Agency (EPA)–approved chemical disinfectant to reduce the risk of microorganism regrowth before the installation of new carpeting.[44] Remediation workers should use personal protective equipment (PPE) appropriate for the hazards to which they are exposed. Remediation work on small, localized patches of microbial growth should be conducted using appropriate respirators (i.e. a minimum of a NIOSH–approved N-95 respirator), eye protection, and gloves. For respirator use, OSHA requires a respiratory protection program that includes the following components: written standard operating procedures, user instruction and training, cleaning and disinfection, storage, inspection, surveillance of work area conditions, respirator fit testing, user checks, evaluation of respirator

protection program, medical review, and use of certified respirators.[45] The remediation zone should be under negative pressure with respect to adjacent areas to ensure containment. Isolation barriers should be used to contain airborne spores and other biological matter.

REFERENCES

1. 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.

2. Gammage RR, Kaye SV, eds [1985]. Indoor air and human health: Proceedings of the Seventh Life Sciences Symposium. Chelsea, MI: Lewis Publishers, Inc.

3. 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.

4. Kreiss K [1989]. The epidemiology of building–related complaints and illness. Occupational Medicine: State of the Art Reviews 4(4):575–592.

5. 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.

6. 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.

7. Molhave L, Bach B, Pedersen OF [1986]. Human reactions to low concentrations of volatile organic compounds. Environ Int 12:167–176.

8. Burge HA [1989]. Indoor air and infectious disease. Occupational Medicine: State of the Art Reviews 4(4):713–722.

9. Nagda NI, Koontz MD, Albrecht RJ [1991]. Effect of ventilation rate in a health building. 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.

10. Jaakkola JJK, Heinonen OP, Seppänen O [1991]. Mechanical ventilation in office buildings and the sick building syndrome. An experimental and epidemiological study. Indoor Air 1(2):111–121.

11. Levin H [1989]. Building materials and indoor air quality. Occupational Medicine: State of the Art Reviews 4(4):667–694.

12. NIOSH [1991]. Hazard evaluation and technical assistance report: Library of Congress, 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. HHE 88–364–2104.

13. Boxer PA [1990]. Indoor air quality: a psychosocial perspective. JOM 32(5):425-428.

14. Baker DB [1989]. Social and organizational factors in office building–associated illness. Occupational Medicine: State of the Art Reviews 4(4):607–624.

15. NIOSH [1992]. 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.

16. Code of Federal Regulations [1997]. 29 CFR 1910.1000. Washington, DC: U.S. Government Printing Office, Federal Register.

17. ACGIH [1998]. 1998 TLVs® and BEIs®: Threshold limit values for chemical substances and physical agents; Biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

18. ASHRAE [1981]. Thermal environmental conditions for human occupancy. American National Standards Institute/ASHRAE standard 55–1981. Atlanta, GA: American Society for Heating, Refrigerating, and Air–Conditioning Engineers, Inc.

19. ASHRAE [1989]. Ventilation for acceptable indoor air quality, standard 62–1989. Atlanta, GA: American Society of Heating, Refrigerating, and Air–Conditioning Engineers, Inc.

20. ACGIH [1989]. Guidelines for the assessment of bioaerosols in the indoor environment. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

21. 59 Federal Register 15969 [1994]. Occupational Safety and Health Administration: indoor air quality; proposed rule. To be codified at 29 Code of Federal Regulations, Parts 1910, 1915, 1926, and 1928. Washington, D.C.: U.S. Government Printing Office.

22. NIOSH [1986]. Criteria for a recommended standard: occupational exposure to hot environments, revised criteria. 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. 86-13.

23. Burge HA [1988]. Environmental allergy: definition, causes, control. Engineering Solutions to Indoor Air Problems. Atlanta, GA: American Society of Heating, Refrigeration and Air–Conditioning Engineers, pp. 3–9.

24. Morey MR, Feeley JC [1990]. The landlord, tenant, and investigator: their needs, concerns and viewpoints. Biological Contaminants in Indoor Environments. Baltimore, MD: American Society for Testing and Materials, pp. 1–20.

25. Pickering CA [1992]. Immune respiratory disease associated with the inadequate control of indoor air quality. Indoor Environ 1:157–161.

26. Vinken W, Roels P [1984]. Hypersensitivity pneumonitis to *Aspergillus fumigatus* in compost. Thorax 39:74–74.

27. Malmberg P, Rask–Andersen A, Palmgren U, Höglund S, Kolmodin–Hedman B, Stålenheim G [1985]. Exposure to microorganisms, febrile and airway–obstructive symptoms, immune status and lung function of Swedish farmers. Scand J Work Environ Health 11:287–293.

28. Topping MD, Scarsbrick DA, Luczynska CM, Clarke EC, Seaton A [1985]. Clinical and immunological reactions to *Aspergillus niger* among workers at a biotechnology plant. British J Ind Med 42:312–318.

29. Edwards JH [1980]. Microbial and immunological investigations and remedial action after an outbreak of humidifier fever. British J Ind Med 37:55–62.

30. Weiss NS, Soleymani Y [1971]. Hypersensitivity lung disease caused by contamination of an air–conditioning system. Annals of Allergy 29:154-156.

31. Hodgson MJ, Morey PR, Attfield M, Sorenson W, Fink JN, Rhodes WW, Visvesvara GS [1985]. Pulmonary disease associated with cafeteria flooding. Archives Environ Health 40(2):96–101.

32. Fink JN, Banaszak EF, Thiede WH, Barboriak JJ [1971]. Interstitial pneumonitis due to hypersensitivity to an organism contaminating a heating system. Annals Internal Med 74:80–83.

33. Banazak EF, Barboriak J, Fink J, Scanlon G, Schlueter EP, Sosman A, Thiede W, Unger G

[1974]. Epidemiologic studies relating thermophilic fungi and hypersensitivity lung syndrome. Am Review Resp Disease 110:585–591.

34. Kaliner M, Eggleston PA, Mathews KP [1987]. Rhinitis and asthma. JAMA 258(20):2851–2873.

35. Jordan FN, deShazo R [1987]. Immunologic aspects of granulomatous and interstitial lung diseases. JAMA 258(20):2938–2944.

36. Gardner RM, Tindall GW, Cline SM, Brown KL [1993]. Ergosterol determination in activated sludge and its application as a biochemical maker for monitoring fungal biomass. J Microb Methods 17:49-60.

37. Gessner MQ, Bauchrowitz MA, Escautier M [1991]. Extraction and quanitification of ergosterol as a measure of fungal biomass in leaf litter. Microb Ecol 22:285-291.

38. Grant WD, West AW [1986]. Measurement of ergosterol, diamionopimelic acid, and glucosamine in soil: evaluation as indicators of microbial biomass. J Microb Methods 6:47-53.

39. Hagmar L, Schütz A, Hallberg T, Sjöholm A [1990]. Health effects of exposure to endotoxins and organic dust in poultry slaughter–house workers. Int Arch Occup Environ Health 62:159–164.

40. Rylander R [1987]. The role of endotoxin for reactions after exposure to cotton dust. Am J Ind Med 12:687–697.

41. Jacobs RR [1989]. Airborne endotoxins: an association with occupational lung disease. Appl Occup Environ Hyg 4:50–56.

42. Olenchock SA [1985]. Endotoxins in occupationally related airborne dusts. Govern Lab 1:28–30.

43. NIOSH [1998]. Hazard evaluation and technical assistance report: Cle Elum–Roslyn High School, Cle Elum, Washington. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, NIOSH Report No. HHE 97–0045–2676.

44. IICRC [1995]. Standard and reference guide for professional water damage restoration S500–94. Vancouver, Washington: Institute of Inspection, Cleaning and Restoration Certification.

45. Code of Federal Regulations [1998]. 29 CFR 1910.134. Washington, DC: U.S. Government Printing Office, Federal Register.

Table 1
Microbiological Results of Bulk Samples (Dust/Debris)
Social Security Administration
Batavia, Ohio
HETA 97–0177–2727

Sample Location	Fungi (DG18)*		Fungi (MEA)**	
	CFU/gm	Taxa Rank	CFU/gm	Taxa Rank
Carpet near sidewall next to employee entrance	1.3×10^5	Clad>>A. ver=Epi=Pen	7.4×10^4	Alt=Clad=Paec=Pith
Carpet next to furnace/employee entrance	4.5×10^4	Y>>Pho>Alt>Clad>Aur=Epi	4.1×10^4	Y=Aur>Clad=Rho >Pho
Mailroom	3.8×10^4	Clad>Yeasts>Pho>Epi	3.5×10^4	Y>Clad>Rho>A. ver

DG18 = Dichloran Glycerol Agar
MEA = Malt Extract Agar
Alt = *Alternaria*
A.ver = *Aspergillus versicolor*
Aur = *Aureobasidium pullulans*
Clad = *Cladosporium*
Epi = *Epicoccum nigrum*
Paec = *Paecilomyces variotii*
Pen = *Penicillium*
Pho = *Phoma*
Pith = *Pithomyces*
Rho = *Rhodotorula*
Y = Unidentified Yeasts

Table 2
Total Spore Air Sampling Results
Social Security Administration
Batavia, Ohio
HETA 97–0177–2727

Sample Location	Date	Concentrations (spores/m^3)	Taxonomic Rank
Mailroom	7/15/97	2600	asc=Clad
	7/16/97	ND	
Employee Side Door	7/15/97	2600	Asp./Pen.–like=bas
	7/16/97	2600	Clad=unk
Sidewall Cabinet	7/15/97	2600	bas=Clad
	7/16/97	ND	
Cabinet/Security	7/15/97	9000	Clad>Alt=asc=Pith
	7/16/97	1300	bas
Lunchroom	7/15/97	1200	Clad
	7/16/97	ND	
Outdoor	7/15/97	19000	Clad>bas>asc>Alt
	7/16/97	26000	Clad>bas>Gan>asc=myx=unk
Minimum Detectable Concentration (MDC)		1300	

ND = not detected
Alt = *Alternaria*
asc = ascospores
Asp./Pen.-like = *Aspergillus /Penicillium*
bas = basidiospores
Clad = *Cladosporium*
Gan = *Ganoderma*
myx = myxomycetes
Pith = *Pithomyces*
unk = unknown

Table 3
Culturable Air Sampling Results
Social Security Administration
Batavia, Ohio
HETA 97–0177–2727

Sample Location	Fungal Concentrations Geometric Mean (CFU/m^3)*	Geometric Standard Deviation	Percentage *Aspergillus*	Percentage *Cladosporium*	Percentage *Penicillium*	Number of Samples
Lunchroom	78	1.2	5%	58%	12%	16
Side Door	70	1.3	3%	60%	13%	16
Side Cabinet	68	2.8	11%	57%	16%	16
Outdoor	1368	1.2	0%	86%	4%	16
Cabinet/Security	94	1.3	5%	54%	18%	16
Mailroom	161	3.2	4%	51%	14%	16

*** CFU/m^3 = Colony forming units per cubic meter**

Table 4
Endotoxin Air Sampling Results
Social Security Administration
Batavia, Ohio
HETA 97–0177–2727

Sample Location	Sampling Time	Sample Volume (m^3)*	Concentration (EU/m^3)**
Lunchroom	7:58 a.m. - 3:26 p.m.	0.90	ND^
Furnace/Door	8:00 a.m. - 3:28 p.m.	0.90	2.33
Sidewall	8:03 a.m. - 3:30 p.m.	0.89	2.69
Security	8:06 a.m. - 3:31 p.m.	0.89	0.79
Mailroom	8:10 a.m. - 3:34 p.m.	0.89	1.01
Outdoors	8:14 a.m. - 3:35 p.m.	0.88	0.80
Furnace/Door	8:29 a.m. - 3:59 p.m.	0.90	2.33
Mailroom	8:37 a.m. - 4:05 p.m.	0.90	2.00
Sidewall	8:31 a.m. - 4:01 p.m.	0.90	ND
Lunchroom	8:26 a.m. - 3:54 p.m.	0.90	ND
Outdoors	8:40 a.m. - 4:08 p.m.	0.90	0.78
Security	8:34 a.m.- 4:03 p.m.	0.90	2.22
Minimum Detectable Concentration (MDC)		0.88	0.57

*** m^3 = cubic meter**
**** EU/m^3 = endotoxin units per cubic meter**
^ ND = not de

Figure 3 - Carbon Dioxide Measurements - 7/15/97

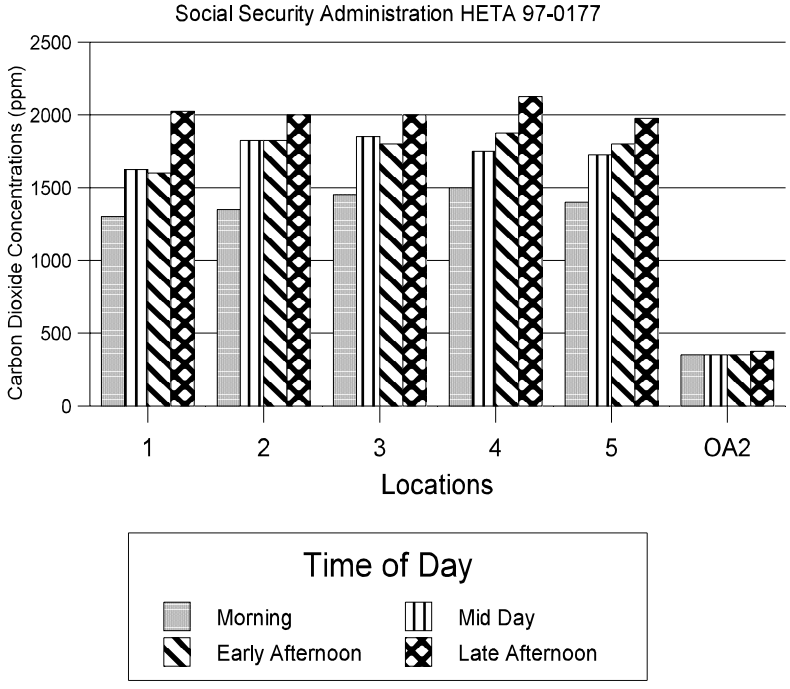


Figure 4 - Carbon Dioxide Measurements - 7/16/97

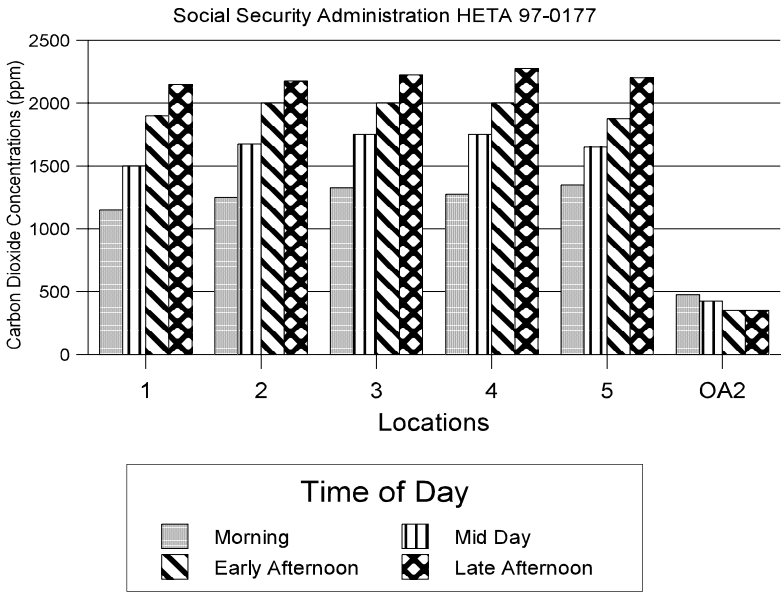


Figure 5 - Temperature Measurements - 6/17/97

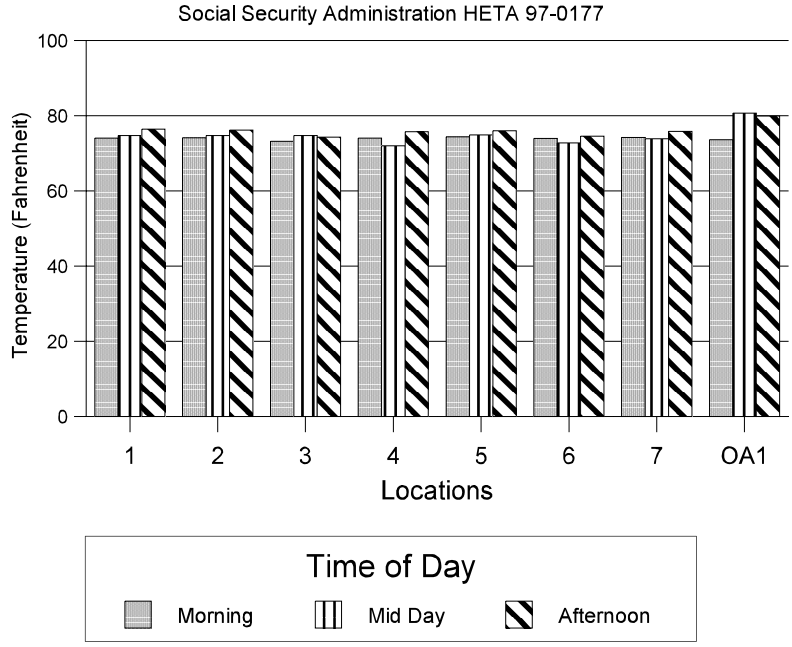


Figure 6 - Temperature Measurements - 7/15/97

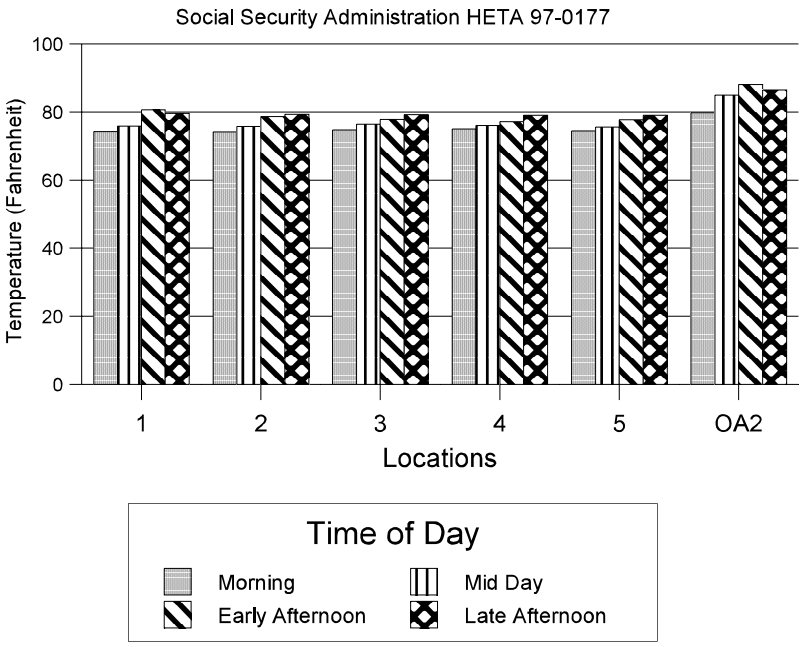


Figure 7 - Temperature Measurements - 7/16/97

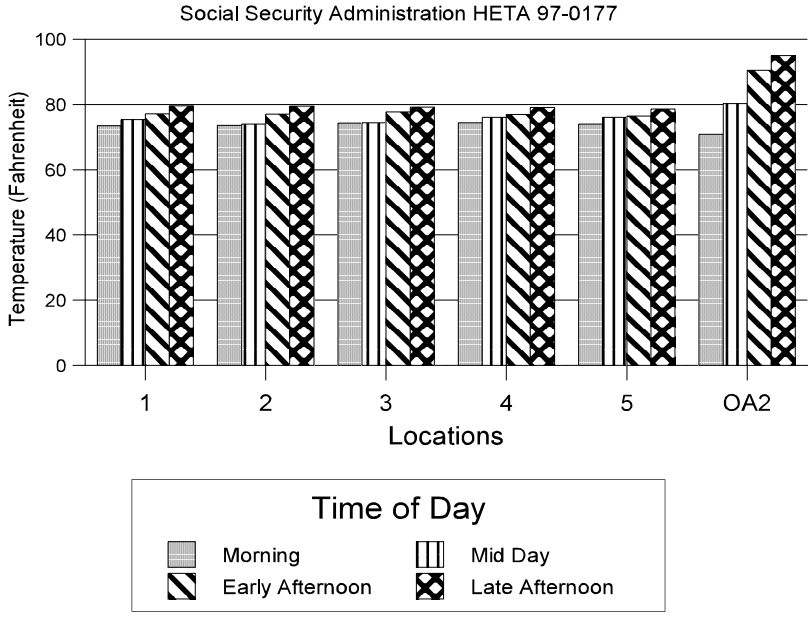


Figure 8 - Relative Humidity Measurements - 6/17/97

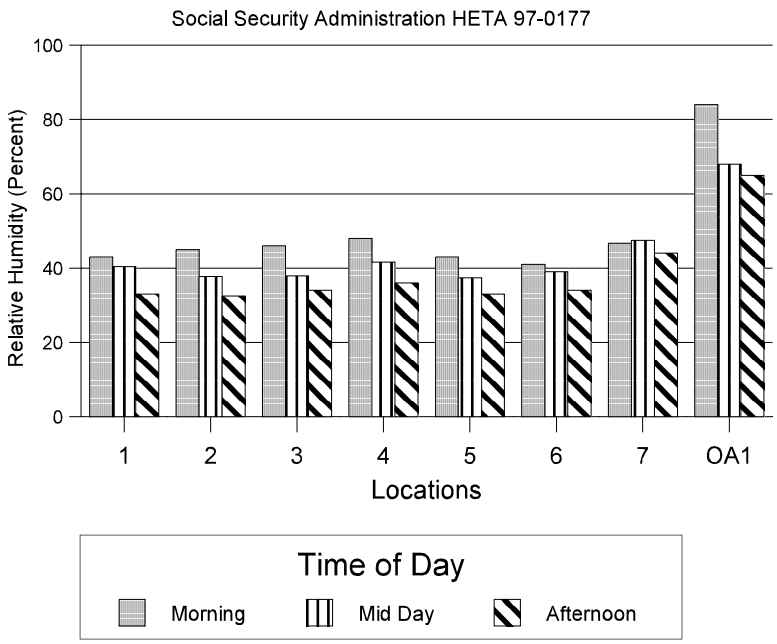


Figure 9 - Relative Humidity Measurements - 7/15/97

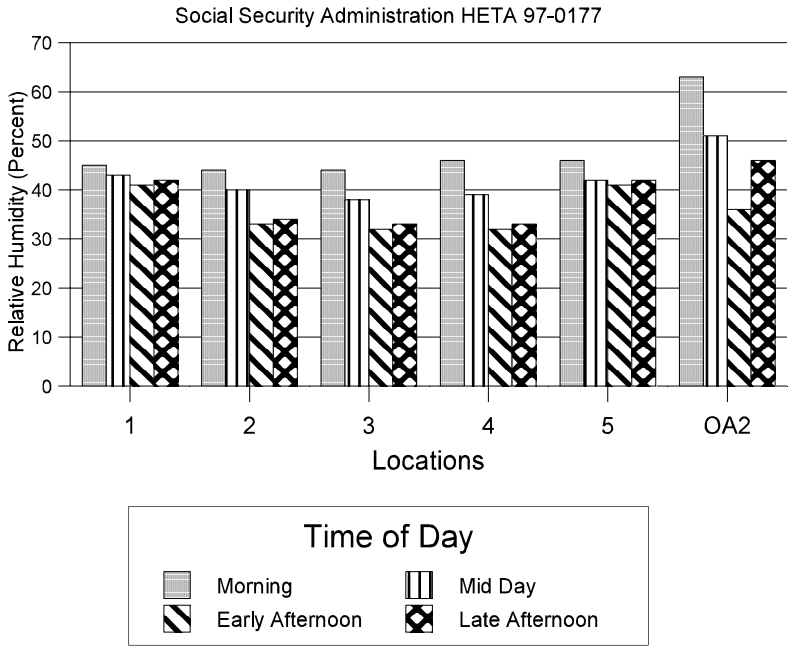
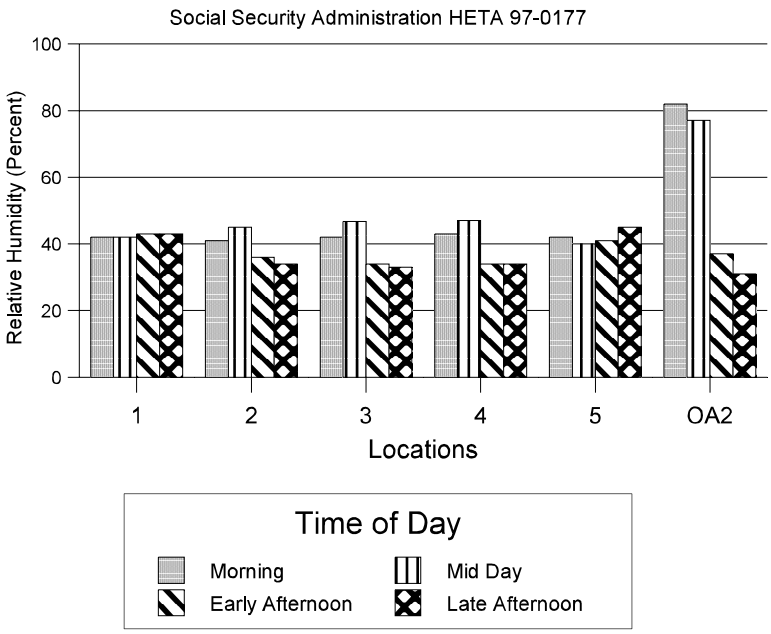


Figure 10 - Relative Humidity Measurements - 7/16/97



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Occupational Safety and Health Concerns

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or visit the NIOSH Homepage at:
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