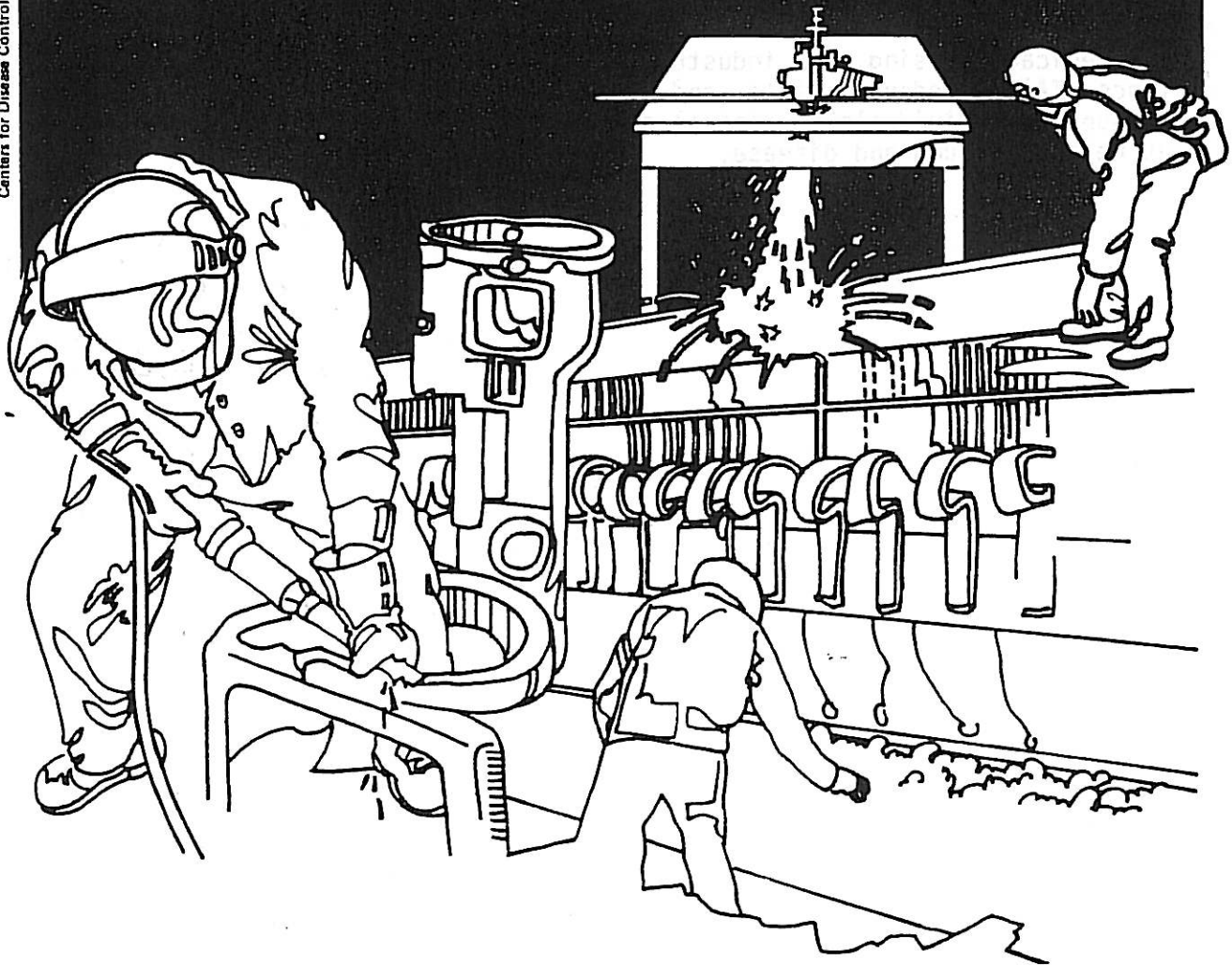


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NIOSH



Health Hazard Evaluation Report

GHEA 82-168-1302
HUBERT H HUMPHREY BUILDING
WASHINGTON, D.C.

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) 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.

GHETA 82-168-1302
APRIL 1983
HUBERT H HUMPHREY BUILDING
WASHINGTON, D.C.

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I. SUMMARY

On March 15, 1982, the National Institute for Occupational Safety and Health (NIOSH) received a request for a health hazard evaluation (HHE) from George E. Hardy, Jr., M.D., Assistant Director, Centers for Disease Control (CDC), Washington, D.C., concerning persistent flu-like illness in Public Health Service employees at the Hubert Humphrey Building.

A walk-through inspection of the building was performed on March 16, 1982. After a case definition had been established, a questionnaire was distributed to define the distribution of disease. Air sampling for bacteria and fungi as well as for other possible contaminants was performed. Bulk samples of dust from the heating/ventilation/air-conditioning (HVAC) system, and carpet, ceiling and water samples were analyzed for predominant microorganisms. Nasal swabs for isolation of amoebae were obtained. Sera were drawn from cases and controls for precipitin testing. Spirometry was performed before and after employees were exposed to the air in the building during a workshift. Single-breath carbon monoxide-diffusing capacity (DLco) was measured once.

On the basis of the questionnaire, it was postulated that affected persons experienced ongoing respiratory exposure to an unidentified antigen associated with water leaks and that this antigen caused the symptoms, which included - headaches, myalgias, chills, chest tightness, fever and nausea. Cases had lower single-breath carbon-monoxide diffusing capacity than controls, indicating the presence of lung disease.

On the basis of our investigation, there is a health hazard in corridor 7B of the Hubert H. Humphrey Building. A hypersensitivity pneumonitis-like syndrome is associated with water leaks and, unlike previously reported situations, is not associated with the HVAC system. The offending agent has not been identified. Recommendations are contained in the body of this report.

Keywords: SIC: 9431 admin. of public health programs, hypersensitivity pneumonitis, office buildings

II. BACKGROUND

Hypersensitivity pneumonitis and humidifier fever associated with buildings have been described in several reports in the last 13 years and have been the subject of several NIOSH health hazard evaluations (# 81-007, Hyattsville, Md.; # 80-19, Boston, Mass.; and # 82-031, Knoxville, Tenn.). These conditions have been associated primarily with air-handling systems containing water sumps (1). More recently, a wide variety of environmental sources including an automobile air conditioner and a disposable filter system in air handlers have been implicated (2, and PJ Baxter, unpublished observations). Outbreaks have been attributed to various organisms, including bacteria (3), fungi (4), and amoebae (5).

Infiltrative lung disease (6) (i.e. extrinsic allergic alveolitis, also called hypersensitivity pneumonitis) associated with ventilation systems has been observed in 3 distinct forms: An acute form with a high attack rate, variable symptoms after a defined one-time exposure; a subacute form with a temporal pattern related to repeated exposures to antigens; a chronic form with respiratory and constitutional symptoms which develop insidiously. All three forms have been associated with both restrictive and obstructive ventilatory impairment. They also commonly manifest non-respiratory symptoms.

The Hubert Humphrey Building was partially completed in 1975 when it was occupied by the Department of Health and Human Services (then the Department of Health, Education and Welfare). Although it was constructed as an open-space office building, it was subsequently subdivided into individual offices. Offices of the Department of Health and Human Services occupy six of its eight floors. It is currently occupied by 1,372 employees. Each floor has eight corridors, lettered A through H (Figure 1).

The building has a central HVAC system controlled by a computer. Because the HVAC system was not modified when the office space was subdivided, there have been problems with ventilation imbalance. Water is piped from a cooling tower on the roof to a centrifugal chiller. The chilled water is piped to central and peripheral coils on each floor. Steam heat is piped from the General Services Administration (GSA) plant to over 900 peripheral (window) units and to reheating coils in the air supply ducts on each floor. The steam vaporizer for humidity control has apparently never been used. Treated air passes through air handlers that distribute it to each floor of the building (Figure 1). The same pattern of air distribution holds true for all floors except the sixth, where the office suite of the Secretary of Health and Human Services has a separate ventilation system. Supply air is distributed to offices through ducts and slotted diffusers in false ceilings. Return air is collected in a plenum formed by the false ceiling. Air enters this plenum through slots around ceiling light fixtures. The air interchange between the air handlers-- because of positive and negative pressures in different parts of the building and through mixing of return air--would be sufficient to distribute an airborne agent throughout the building once it has entered the HVAC system.

The Hubert Humphrey Building has been the subject of two recent HHE's. Hazard Evaluation and Technical Assistance (HETA) # 81-267 was concerned with poor indoor air quality; Health Hazard Evaluation (HHE) # 80-108-762 with air quality, possible drinking water contamination, and water leaks, thought to be due to improper building construction and the lack of a grease trap under the dishwasher in the cafeteria kitchen above corridor 7B. No illness was documented during either prior investigation.

There have been recurring problems with water leaks in corridor 7B. The cafeteria kitchen on the eighth floor is directly above this corridor, and its water-drainage system runs, together with sewage from toilets, through the plenum of that corridor. The cafeteria dishwasher has no grease traps, and grease has periodically clogged the drain pipes, causing water to back up and overflow. Because of faulty floor construction, this backed-up water has penetrated the ceiling and leaked through to the seventh floor. These leaks were temporarily stopped when the plumbing shop reamed out the pipes. The cause of one flooding incident was the cafeteria ice machine, also over corridor 7B. There is no exact record of when leaks occurred over the years. In late 1981, the building manager, the GSA engineering and plumbing departments, and an outside contractor analyzed the structural problems. A memo dated March 4, 1982, from the GSA engineering department to the GSA Humphrey Building manager, defined the problems and recommended procedural and structural corrections.

On January 27, 1982, when the pipe system was reamed with a sharp pipe reamer, flooding occurred in one of the CDC offices and other areas of 7B. Over the following months, several persons in this corridor experienced an illness with myalgias, fever, chills, chest tightness, headaches, and nausea. All these symptoms improved on weekends.

NIOSH was asked to perform an investigation. Staff from the Division of Respiratory Disease Studies met with the building manager and the safety officer and with plumbing, maintenance and ventilation engineers.

III. MATERIALS AND METHODS

Epidemiology

The illness, as described by the first two employees to report symptoms (the index cases), resembled subacute hypersensitivity pneumonitis. Since previous reports have suggested that air handling systems may be associated with this disease (1) and since this building also had water leaks, either the HVAC system or the leaks could have been associated with this illness. If the illness were associated with the HVAC system, there should have been no clustering of disease in any part of the building. If it were associated with water leaks, the disease would have been clustered in the wet areas. In order to identify the responsible factors, the NIOSH team distributed questionnaires to three groups of employees on March 17, 1982: Group I consisted of the employees in corridor 7B, the area with the water leaks from the cafeteria; group II consisted of the employees in 7G and 7H, the other PHS corridors on the same floor; and group III consisted of employees in 4G, who were not employed by PHS and who worked on a different floor.

The self-administered questionnaire was designed to determine the prevalence of 6 symptoms (headaches, myalgias, chest tightness, chills, fever, and nausea) and their temporal relation to the workplace. Risk factors such as smoking, allergies, and distance from water leaks were also examined. The questionnaire elicited information about diarrhea, colds, productive cough, and arthritis, but not about seasonal variation. The questionnaire was similar to one used by Arnow and Fink (7) in their investigation of building-associated disease and by Castellan (Health Hazard Evaluation #80-22) in an investigation of vegetable dust associated disease.

Medical

Tests of lung function were performed on 57 volunteers between March 29 and March 31: 26 from group II and 31 from group I, including 8 of the 12 individuals who met our case definition (See IV below). Four cases were not tested: 1 was ill and not at work that week, 1 was out of town the week of the testing, 1 was not identified until after testing had been completed, and 1 did not volunteer for testing.

Spirometry meeting American Thoracic Society criteria (8) was performed on a workday before and after the participating employees were exposed to the air in the Humphrey Building for at least 6 hours. An Ohio Medical Products model 840 waterless electronic spirometer was used.* Flow and volume signals were recorded on FM analog tape and later processed on a LSI-11 microcomputer (Digital Equipment Corp.). Five forced expiratory (FEV₁) maneuvers were obtained on each individual. All values were corrected to body temperature, atmospheric pressure, saturated with water vapor (BTPS). Predicted values were those described by Knudson and co-workers (9)

Single-breath carbon-monoxide diffusing capacity (DLco) was measured on a Hewlett-Packard System 47305 DCO S.B. Controller, 47313A helium analyzer, and 47312A carbon monoxide analyzer. Predicted values were obtained from Cotes (10).

Chest x-rays were arranged through the Clinical Center of the National Institutes of Health (NIH). Nine of the 12 persons who met the case definition volunteered to have x-rays. They were also offered bronchial lavage studies, but none volunteered for this examination.

Industrial Hygiene

On March 16 and 17, viable organisms from corridor 7B were collected with Andersen 2000 samplers, operated at flow rates of 28.3 liters per minute for periods of 10 or 20 minutes. Rose bengal streptomycin and Sabouraud agar (modified with 50 µg/ml of streptomycin and 50 µg/ml of penicillin), both selective for fungi, were used. Samples were collected both with the HVAC

*Use of trade names throughout this document is for identification only and does not imply endorsement by the National Institute for Occupational Safety and Health, the Centers for Disease Control, the Public Health Service, or the Department of Health and Human Services.

system shut down and running. Relative humidity measurements were made by sling psychrometer during periods when the Andersen samplers were running. Bulk samples for isolation of bacteria, fungi, and protozoa were collected from the carpets, false ceilings, and outer surfaces of pipe-traps in corridor 7B. A sample of water was collected from the dishwasher drainage lines. Predominant protozoa were isolated from bulk samples by the Center for Infectious Diseases, CDC. Predominant bacteria and fungi were isolated from bulk samples by the Medical College of Wisconsin. Blood samples were obtained from 10 cases and 14 controls who worked in corridor 7B. Serologic studies using crude extracts of organisms cultured from bulk samples as well as other panels of antigens were carried out at the Center for Infectious Diseases, CDC and the Medical College of Wisconsin.

Respirable dust samples were collected at various locations in 7B with a one-inch cyclone (Bendix Model 240, polyvinyl chloride 47 mm filter, with a nominal pore diameter of 0.8 μ m) operating at a flow rate of 66 liters per minute. The estimated diameter of particles collected at 50% cyclone efficiency was approximately 4 μ m (11). In addition, airborne particulate matter was collected with samplers with open-faced filter cassettes (type AA filters, with a nominal pore size of 0.8 μ m and a flow rate of 2 liters per minute). Sampling was carried out both with the HVAC system shut down and running.

Air quality was examined in several areas of corridor 7B with a Miran Infrared Analyzer. Colorimetric indicator tubes were used to test for the presence of contaminant gases. Specific indicator tubes were used for carbon tetrachloride, hydrocarbons, ozone, ammonia, carbon monoxide, perchloroethylene, chlorine, carbon dioxide, and methanol-ethanol. Other possible air contaminants were collected in a large silica gel tube and in a large charcoal tube, desorbed with CS₂ and ethanol respectively, and screened by gas chromatography.

A second industrial hygiene survey was conducted on May 6 and 7. Viable airborne organisms in corridor 7B were collected on an Andersen 2000 sampler (28.3 liters per minute) during times when the HVAC system was operating. Rose bengal streptomycin and trypticase soy agar media were used to collect fungi and bacteria, respectively. Viable organisms were also collected on settling plates (3 minutes) in corridor 7B and elsewhere in the building. High-volume samplers (8 x 10 inch glass fiber filter, both respirable and non-respirable dust collected) with a flow rate of 1.98 m³ per minute were used to collect airborne antigens in corridor 7B. Because a previous study in the same building (#81-267) had recognized pesticide application as a possible health hazard, air samples were obtained in corridors 7B and 7H to determine if pesticides were present in this office environment. Information provided by GSA indicated that at least 7 insecticides including the organophosphates diazinon and dursban, the carbamates baygon and bendiocarb, and the pyrethrins resmethrin and pyrethrum have been used in the Humphrey Building. A sampling train consisting of a pre-extracted glass fiber filter (13 mm) followed by a chromosorb 102 tube (air flow=2 liters per minute) was used to sample for these insecticides. For diazinon and dursban glass fiber filters and chromosorb 102 tubes were desorbed with toluene and analyzed on a gas chromatograph with an

electron capture detector. The limit of detection for diazinon and dursban was 0.005 µg per sample for both the chromosorb tubes and the glass fiber filters. For baygon, bendiocarb, resmethrin, and pyrethrum, glass fiber filters and chromosorb 102 tubes were desorbed with acetonitrile and analyzed by high resolution liquid chromatography. The limit of detection was 1.0 µg per sample for baygon, pyrethrum, and resmethrin and 1.5 µg per sample for bendiocarb. GSA records also indicated that two rodenticides including zinc phosphide and one bat poison have been used in this building. As a possible indication of exposure to an airborne rodenticide (e.g., zinc phosphide) air samples were collected in corridor 7B and 7H on cellulose ester filters in an open face configuration at a flow rate of 2 liters per minute. The filters were analyzed for zinc by atomic absorption spectroscopy according to NIOSH Method P and CAM 173 (12). The limit of detection for zinc was 1 µg per filter. Other possible air contaminants in corridor 7B were collected in large charcoal tubes and on chromosorb 106 resin at flow rates varying from 0.96 to 2.0 liters per minute. Charcoal and chromosorb 106 tubes were desorbed with carbon disulfide and methanol respectively and screened by gas chromatography. Charcoal tubes were further analyzed by mass spectroscopy.

The HVAC system was inspected on May 7 and May 24. Several dust samples were collected from main air handlers and from perimeter units for microbiological analyses.

IV. EVALUATION CRITERIA

The index cases described relief of symptoms on weekends; both had more than three symptoms, the number used in previous outbreaks for "probable" disease (7 and D Reed, personal communication). We therefore defined possible disease as the presence of any 2 symptoms (headaches, chills, chest tightness, muscle aches, fever, and/or nausea) which either exhibited periodicity during a workweek and relief on weekends or were more likely to be present at the end of a week than at the beginning. We did not consider symptoms as indicating possible or probable disease if they were reported to occur on workdays, interrupted by workdays without symptoms. We defined probable disease as the presence of at least three symptoms with similar periodicity. Cases were defined as instances of probable or possible disease. Sporadic symptoms were defined as symptoms with onset after the employee began work in the Humphrey Building, which recurred with a frequency of more than once every 2 weeks, but which did not meet criteria for periodicity and could not be accounted for by exposures outside the Humphrey Building. Unrelated symptoms were defined as symptoms with onset before the employee began working in the Humphrey Building or that could be explained by other causes.

In an effort to eliminate reporting bias that could result from our restrictive disease definition, the distribution of all symptoms possibly related to work in the Building (i.e., all symptoms occurring more often than every 2 weeks) was calculated by two or more and one or no symptoms.

V. RESULTS

A. Epidemiology

The response rate 24 hours after the questionnaires were distributed did not differ significantly between corridors: 7B, 29 of 41 (70%); 7 G and H, 35 of 54 (62%); and 4 G and H, 28 of 40 (70%). Seven possible and three probable cases had responded. During our spirometry and DLco testing, we solicited volunteers. The total number of questionnaires returned at the end of our data collection period was 41 on 7B, 38 on 7G and H, and 29 on 4G. By using the number of persons assigned to desks in these corridors as denominators, response rates were calculated to be 80% (41/51), 55% (38/69), and 72% (29/41), respectively. These were significantly different ($\chi^2 = 8.85$, 2 df, $p < 0.02$). Table 1 shows the numbers of persons with possible and probable disease who worked on the different corridors (7B vs. 7G, 7H vs. 4G and H).

Four persons who worked in corridor 7B, including both index cases, were identified as having probable disease (all had four or more symptoms) (Table 1). One had the first onset of symptoms after he began work in the building several years before, but experienced additional symptoms 2 months before the investigation. The desks of all four persons were located within 10 feet of a water leak. Eight persons who worked in corridor 7B were identified as having possible disease. Six of these worked within 15 feet of water leaks.

Two persons in corridor 4G and H had disease (Table 1); one with two and one with three symptoms. One had developed her symptoms years before when she worked on the seventh floor; the desk of the other was in the middle of a puddle caused by water leaks from another source.

Symptoms that began after an affected employee began working in the Humphrey Building and occurred more often than once every two weeks were viewed as possibly related to the building. The distribution of sporadic and periodic symptoms combined was similar to that of "disease" (Table 2). The difference in sex-specific attack rates (Table 3) was not significant.

Onset of disease was poorly defined. Table 4 shows duration of disease before the questionnaires. Responses to the questionnaire indicate that most employees affected experienced onset of symptoms about 2 months before the investigation. The answers were obviously approximations, but do indicate that there was a problem on that corridor before March.

We looked for risk factors for the development of disease, both in corridor B and among persons who had worked within 15 feet of a water leak in the last 2 months (Table 5). Sitting within 15 feet of a leak was the only statistically significant risk factor identified. Chronic bronchitis, smoking, history of allergies to a single substance, and air supply through air handler 6 were not risk factors for disease (Tables 5 and 6).

Of the 12 possible cases, 10 described periodic headaches, 5, myalgias; 5, chills; 7, chest tightness; 6, fever; and 5, nausea. Two had developed severe arthralgias. 11 persons were more likely to have had diarrhea on 5 or more days in the 3 months preceding the questionnaire (Table 5). Table 7

shows a tabulation of all symptoms. Persons in 7 G and H reporting an episode (duration greater than one week) of at least one of the symptoms were more likely to have worked in corridor 7B in the last two years (Table 8).

B. Medical

Similar percentages of employees in group I (51.0%) and group II (45.0%) consented to spirometry and DLco measurements. Percent-predicted DLco differed significantly between ill and asymptomatic persons within group I ($t=2.30$, 29 df, $p < 0.05$) and between ill and all non-ill persons studied ($t=2.63$; 56 df, $p < 0.01$). No one had a decrement of 10% or more in FEV₁ or FVC. The mean change in FEV₁ between groups I and II was not significantly different (Table 9). No chest x-rays showed changes compatible with diffuse interstitial fibrosis (chronic hypersensitivity pneumonitis) or alveolitis (acute pneumonia).

Amoebae were not recovered from any nasal swabs. Serologic studies were carried out against crude extracts of all the fungi and bacteria cultured from samples listed in Table 10 as well as against antigens often associated with hypersensitivity lung disease (e.g., Micropolyspora faeni, Saccharomonospora viridis, etc.). These tests failed to demonstrate any difference between case and control groups (Table 11). Similarly, individuals from the case group did not have higher antibody titers against Acanthamoeba polyphaga, A. castellanii, Naegleria gruberi or N. lovaniensis than controls.

C. Industrial Hygiene

The maximum number of airborne fungi collected with the Andersen sampler on March 16-17 was 63 colony forming units (CFU) per m³. In one case on March 17, no viable fungi were collected in this 6-stage sampler (Table 12). However, the number of airborne fungi collected was greater on March 16, when the HVAC system was turned off, than on March 17, when the ventilation system was fully operational. Changes in relative humidity between Andersen sampling periods were minimal (March 16 = 31%; March 17 = 37%-38%).

Relatively low numbers of airborne fungi and also bacteria were again collected with the Andersen viable sampler in corridor 7B on May 7. The highest number of CFU per m³ for fungi and bacteria was 49 and 28, respectively (Table 13). Relative humidity in Corridor 7B at the time of sampling varied from 59 to 67%. Settling plates were devoid of microorganisms except for one tripticase soy agar plate opened in the reheat area of air handler #12. This plate contained colonies of Bacillus sp., Streptomyces griseus, and aerobic spore forming organisms.

Predominant microorganisms isolated from 12 bulk samples collected from corridor 7B and from the HVAC system are listed in Table 10. With the exception of respirable dust collected on a polyvinylchloride filter, all of the samples from corridor 7B that were examined for protozoa contained Acanthamoeba polyphaga. Thermophiles were not found in samples from this location. In contrast, all samples from perimeter ventilation units and from main air handlers contained Thermoactinomyces sp. as predominant organisms.

Amoebae were absent from the only two HVAC samples examined for protozoa, the air supply plenums of air handlers 6 and 7.

The average concentration of respirable dust collected with the Bendix one-inch cyclone was $25 \mu\text{g per m}^3$ (std. dev. = $3 \mu\text{g per m}^3$). The location and time of collection appeared to have little influence on dust levels with concentrations of 21, 27, and $26 \mu\text{g per m}^3$ found in 708B (March 17), 709B2 (March 17) and 709B (March 16), respectively. Two high volume (total dust) samplers were operated continuously in 708B and 709B from 1900 hrs. on May 6 to 1300 hrs. on May 7. The time weighted average (TWA) airborne dust concentrations collected with these instruments in 708B and 709B were 23 and $35 \mu\text{g per m}^3$, respectively. Relative humidity measurements made at the two sampling locations over this period varied from 52 to 70%.

Examination by phase-contrast microscopy of type AA filters collected in Corridor 7B on March 16 and 17 showed that the number of fibers longer than 5 μm (3:1 aspect ratio) was equivalent to that of control filters. Asbestos fibers were not observed on filters from dust samplers in Corridor 7B.

All tests for contaminant gases with colorimetric indicator tubes were negative. Carbon dioxide was present (by indicator tube) at a concentration of 0.03% by volume (300 ppm), which is far below hazardous levels (OSHA PEL=5000 PPM). Analysis of charcoal and silica gel tubes showed no detectable contaminants. Comparison of Miran infrared spectra made at various times and locations in Corridor 7B showed that air quality was identical throughout the corridor, regardless of location and/or whether the HVAC was operational.

Sampling for pesticides and other possible organic air contaminants was carried out on May 7. The maximum amount of dursban and diazinon found in air samples collected in corridor 7B and 7H was less than the limit of detection ($<0.005 \mu\text{g per sample}$) of the analytical technique used. Considering the amount of air collected by each sampling train, this is equivalent to a concentration of less than $0.01 \mu\text{g per m}^3$ which is far below the ACGIH TWA - TLV (American Conference of Government Industrial Hygienists Time-Weighted Average-Threshold Limit Value) for diazinon of $100 \mu\text{g/m}^3$. The maximum amount of baygon, pyrethrum, and resmethrin found in air samples was less than $1.0 \mu\text{g per sample}$. For baygon and pyrethrum this is equivalent to a concentration of less than $2.2 \mu\text{g/m}^3$, several orders of magnitude less than their ACGIH TWA-TLVs. Analysis of cellulose ester filters collected in corridor B and H for zinc (by inference for zinc phosphide) showed that levels of this metal were equivalent to or less than the limit of detection ($1.0 \mu\text{g per filter}$) for the method employed.

Air samples from corridor B were collected on charcoal tubes (air volume = 0.7 m^3) and chromosorb 106 resin (air volume = 0.3 m^3). Analysis by gas chromatography revealed that the chromosorb 106 tubes collected no contaminants other than those found in blanks. Charcoal tube samples contained levels of contaminants log orders below their TLVs. Compounds identified by mass spectral analyses included toluene, xylene, and a series of mostly branched alkanes in the $\text{C}_{10}\text{-C}_{12}$ region.

The following observations were made on the HVAC system on May 7 and May 24. The amount of make up air entering each main air handler is

controlled automatically by a computer system incorporating interior and exterior temperature sensors. During hot summer and cold winter days the amount of make-up air is minimal (3-5%). However on cool spring and autumn days, the amount of make-up air may rise to 85%. Return plus make up air is passed through a self-moving glass fiber filter prior to entering the mixing plenum of each air handler. The glass fiber filter is moved at a speed such that it takes 4 days to traverse the filter holding slot. Thus, the particle arrestance of the filter media may be expected to vary according to the length of time (0 to 4 days) the glass fiber is exposed to the air stream. Mixed air then passes over a series of coils containing chilled water. Chilled water within the coils has no access to the air stream passing through each air handler. Chiller coils not only function in removing heat from the air stream but, during periods of warm humid weather, they also remove moisture from the air. The condensate pan servicing each chiller coil system transports water directly to a drain. Condensate pans, chiller coils and associated surfaces in each air handler were observed to be clean and well maintained.

Air exiting the chiller coil system passes through a centrifugal fan to an air supply plenum from where it is ducted to specific office areas as indicated in Figure I. It was observed that 5 of the main air handlers (#5, 6, 7, 8 and 17) were constructed so that fan motors are within the plenum housing the fan squirrel cage. The other air handlers were all characterized by having motors external to air handler ductwork. Fan blades, fan motors within plenums and metal and concrete surfaces within plenums appeared, for the most part, clean and adequately maintained. However, the air supply plenums downstream from fans in air handlers #6 and 7 (both on south side of building) had at one or more times in the past been thoroughly flooded. This was evidenced by the brown-tan stains covering 10 to 12 meters of the otherwise white outside surface of each supply plenum. Rust and some dirt were found on the inside surface of these plenums. At that time (May 24) the inside surfaces of air supply plenums 6 and 7 were dry. Air supply plenums of air handlers 3 and 4 showed no external evidence of previous water damage. It should be noted that air supplied to corridor 7B originates jointly from air handlers 3, 4, 6 and 7.

VI. DISCUSSION

The disease definitions used in this study are restrictive. Other investigators have not insisted on "periodicity" as part of their disease definition (7 and D Reed, personal communication). Thinking that reporting bias might have influenced the distribution of disease, we re-analyzed symptom distribution under the assumptions that: (1) our "more than once every 2 weeks" was the equivalent of Arnow and Fink's "sometimes or often" categories which they used as their frequency definition and (2) that two or more symptoms would indicate disease as opposed to one or none. This would eliminate periodicity throughout a work week and imply chronic rather than subacute disease. Corridor 7B was still associated with significantly more complaints. Since both index cases described periodicity and since use of a less specific definition did not change the distribution of disease, ours appears to be a legitimate working case definition.

Although there were similar response rates among the three groups during the initial survey, the response rate for the final analysis was significantly

higher in corridor 7B. Persons in 7B may have had greater interest in the study. There are more persons in 7G and 7H who had additional offices in other buildings than there were in 7B. In addition, many persons were out of town or at meetings. Both analysis in the field and final analysis at NIOSH indicated that the source of the problem was on 7B. Therefore, since the distribution of symptoms did not significantly change when more persons volunteered for spirometry and their questionnaires were incorporated into the pool, response bias does not appear to be a problem.

Outbreaks such as this have been primarily associated with air conditioning systems or home humidifiers. If the disease had been associated with the HVAC system or one of the air handlers, it would have been distributed differently. That it could occur by chance in the distribution found in the Humphrey Building is exceedingly unlikely. However, such clustering has been observed in other buildings (W. W. Rhodes, unpublished results). The kind of HVAC system used in the Humphrey Building has to date not been associated with hypersensitivity pneumonitis. This system is of a kind that has been recommended to prevent disease(13).

In this instance disease appears associated with water leaks. All persons on corridor 7B with recent onset of disease sat within 15 feet of water leaks from the cafeteria. The office of one person in 4G with recent onset of disease had water-soaked rugs from external building leaks. The person with pre-existing disease had developed it on the seventh floor. A recirculating agent may account for the symptoms of these two employees. Two persons with a previous, similar disease on corridor 7B experienced symptoms that resolved when their offices were moved. Persons in corridors 4 and 7G were more likely to have had a disease of more than 1 week's duration in the last 2 years if they had worked on corridor 7B. On direct questioning, several ill persons stated that their symptoms improved when they were working away from their usual stations on 7B and that their symptoms worsened upon re-exposure. All of the above implicate 7B, and specifically water leaks, as the culprit in this outbreak.

On the basis of the distribution of disease in the building and the association with water leaks in the affected corridor, the cafeteria water leaks appear responsible for the symptoms experienced by employees in corridor 7B.

Although 9 of the 12 affected persons had onset of disease or new symptoms in the 2 months preceding our initial survey, 3 had developed disease 1, 2, and 3 years before. This raises the possibility of an intermittent source outbreak. We did not correlate numbers of symptoms, periodic symptoms, or disease with duration of building occupancy. The numbers of subjects are too small for meaningful regression analyses.

Hypersensitivity pneumonitis can have obstructive and restrictive components(14). Simple spirometry (FEV_1 , FVC) and measurements of diffusing capacity (DL_{CO}) (15) have been employed to assess these abnormalities. In this study no differences were observed for DL_{CO} and shift changes in FEV_1 and FVC between employees in corridor 7B and those in corridor 7G/H (Table 9). Comparison of DL_{CO} values limited to corridor 7B workers, however, showed

a statistically significant difference between employees meeting our case definition and those without disease. While similar FEV₁ and FVC comparisons among 7B workers were not significant, the DLco decline is noteworthy and provides a physiologic correlate to disease defined on the basis of symptoms.

The maximum level of airborne fungi and bacteria found in corridor 7B on March 16-17 and on May 7 was low, being less than 100 CFU per m³ (Tables 12 and 13). In some office buildings with hypersensitivity pneumonitis or other respiratory tract diseases, levels of microorganisms exceeding 500 or even 4000 CFU per m³ have been reported (HHE #80-19, Boston, Mass.; HHE #82-031, Knoxville, Tenn.). However, in these buildings it is difficult to relate disease prevalence with airborne levels of microorganisms because the latter is significantly influenced by such variables as temperature, humidity and season (16), filtration by the HVAC system and the time of sampling relative to disease onset.

Several of the agents associated with hypersensitivity pneumonitis in the past, including A. polyphaga, Aureobasidium sp., and Thermoactinomyces sp. (13,17) were isolated in the Humphrey Building (Table 10). However, less than a third of cases and controls had precipitin reactions to these agents (Table 11). The presence of precipitin reactions is commonly accepted as evidence of exposure, necessary to but not sufficient for the development of disease (18). This implies that the agents listed in Table 11 are not etiologically involved in the disease in the Humphrey Building.

After our investigation and during the clean up of corridor 7B in the third week of May, large amounts of dust were liberated when office partitions were handled. Similar illness occurred in previously ill individuals and 2 persons were hospitalized. Even though our analyses were unsuccessful in identifying the agent, it may still have been present in office partitions in May. There are many potential reasons for the difficulty in identifying the specific disease agent, and include: (A) The agent may be an organism other than the predominant ones that were isolated from the environmental samples. (B) The agent may not be viable and therefore was not cultured from bulk samples and was absent from the panel of antigens used in serologic studies. (C) The exact etiology of this disease may be demonstrable only by provocative challenge which was not attempted in this study.

The maximum concentration of airborne dust in corridor 7B was 35 µg per m³ which is well below the ASHRAE limit of 75 µg (19) and comparable with that (37 µg per m³) found in other office buildings (# 80-122-1117, Atlanta, Georgia). Environmental sampling indicated that other air contaminants were present at below hazardous levels. Toluene, xylene, fibrous dust, pesticides, etc., were present at very low levels or were not detectable. We cannot, however, say whether some or all of these materials

present in minimal concentrations can act together synergistically to produce disease.

VII. RECOMMENDATIONS

Clean-up procedures in the reported outbreaks of building-associated hypersensitivity pneumonitis have been controversial and sometimes ineffective (1). To remove the source of the disease--i.e., the antigen-- still in the surroundings, the following measures are recommended for corridor 7B:

- (1) discard the carpeting;
- (2) discard the ceiling panels where water had leaked; clean the outside surface of the pipes from which the leaks originated; clean or discard the accessory water trays for the ceiling drains;
- (3) scrub the floor with bleach and let it dry completely;
- (4) clean all wall partitions and upholstered furniture with a vacuum incorporating a high-efficiency particulate air (HEPA) filter;
- (5) after the above steps are carried out, replace the carpeting;
- (6) vacuum all office materials that need to be reused;
- (7) carry out the structural and procedural recommendations of the GSA engineering department (their memo dated March 4, 1982). Water leaks in 7B and elsewhere must be prevented. The above clean-up procedures will be ineffective if water leaks are not prevented.
- (8) serious consideration should be given to the recommendations concerning operation of the HVAC system made to GSA (letter to P. Gilson, dated October 12, 1982) by W.W. Rhodes, Ph.D., P.E.

Employees should reoccupy corridor 7B only after all the remedial measures recommended above have been accomplished. Consideration should be given to the creation of a simple system of medical surveillance to monitor health consequences of the above remedial measures. This may be accomplished by a simple system of illness reports where individuals, work station, date, and nature of complaints are recorded. These illness reports should be reviewed by someone capable of identifying incipient disease clusters by place and time.

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TABLE 1

DISTRIBUTION OF DISEASE

	Corridor 7B(a) Group I	Corridor 7G/H(b) Group II	Corridor 4G/H(c) Group III
<u>Probable Disease</u>			
yes	4	0	0
no	37	38	29

3x2 contingency table: $X^2=6.74$; 2 df⁺ $p < 0.05(*)$
a x b: $p=0.002$ (Fisher's exact test)

Possible Disease (excluding probable disease cases)

yes	8	0	2
no	29	38	27

3x2 contingency table: $X^2=6.5$, 2 df, $p < 0.05(*)$
a x b: $p=0.001$ (Fisher's exact test)

Disease Combined

yes	12	0	2
no	29	38	27

3x2 contingency table: $X^2=16.2$; 2df, $p < 0.001(*)$
a x b: $p=0.001$ (Fisher's exact test)

(+) Degrees of freedom

(*) Multiple contingency tables for X^2 testing are controversial where individual groups number less than five

TABLE 2

DISTRIBUTION OF SYMPTOMS POSSIBLY RELATED TO THE BUILDING

a. Total number of persons with symptoms possibly related to the building
(implying no need for periodicity for disease definition)

number of symptoms per person	2 or more	1 or less	
Group I (7B)	17	24	n=41
Group II (7 G+H)	4	34	n=38
Group III (4 G)	6	23	n=29

3x2 contingency table: two or more symptoms versus one or no symptoms: $\chi^2 = 6.36$; 2 df, $p < 0.05^*$

TABLE 3

SEX-SPECIFIC ATTACK RATES

	Male		Female	
	ill	not ill	ill	not ill
7B	6	8	6	21
7 G and H	0	14	0	21
4 G and H	0	14	2	15

$\chi^2(7B)=1.03$, n.s.

DURATION OF SYMPTOMS
(As of March 17, 1982)

21

TABLE 5
RISK FACTORS FOR THE DEVELOPMENT OF AND ASSOCIATION WITH DISEASE

I. For all persons in 7B

a. Proximity to a water leak in the last two months vs. recent or new symptoms (2 persons had previous disease with new symptoms and had moved)

Disease		
	yes	no
yes	10	2
no	14	15

p=0.038 (Fisher's exact test, one-tailed)

b. Current Smokers

Disease		
	yes	no
yes	5	7
no	6	23

$\chi^2=0.92$; p=0.3

c. History of allergies

Disease		
	yes	no
yes	6	4
no	9	20

$\chi^2=1.58$; P=0.2

Two persons did not respond to this question.

d. Chronic Bronchitis (3 months of cough or phlegm production in 2 years) vs. disease

Disease		
	yes	no
yes	4	8
no	5	24

$\chi^2=0.51$; P=0.5

TABLE 5 (continued)

II. For persons sitting within 15 feet of a water leak

e.		smoking	
		yes	no
	yes	4	6
	Disease		
	no	3	12
	P=0.25 (Fisher's exact test)		
f.		chronic bronchitis	
		yes	no
	yes	3	7
	Disease		
	no	1	14
	P=0.13 (Fisher's exact test)		

III. Miscellaneous for all persons on 7B

g.		Diarrhea on 5 or more days in 3 months	
		yes	no
	yes	4	8
	Disease		
	no	1	28
	P=0.002 (Fisher's exact test)		

TABLE 6

NUMBER OF CASES BY FLOOR AND AIR HANDLER

Air handlers	1/2	3/4	6	7
<hr/>				
Floor involved				
7	--	1	6	5
4	2	--	--	--
<hr/>				

TABLE 7
DISTRIBUTION OF SYMPTOMS BY GROUPS

	<u>Any Symptom</u>	<u>Headaches</u>	<u>Myalgias</u>	<u>Chills</u>	<u>Chest Tightness</u>	<u>Fever</u>	<u>Nausea</u>
<u>7B</u>							
Number with possibly related symptoms	29	16	13	6	7	8	8
Periodic	16	10	7	5	6	7	5
Sporadic	5	5	5	1	1	1	3
Daily	2	1	---	---	---	---	---

Unrelated	7	3	4	---	---	---	---
<u>7G and 7H</u>							
Number with possibly related symptoms	24	10	4	2	2	3	3
Periodic	5	4	---	---	---	---	1
Sporadic	12	6	2	2	1	3	2
Daily	3	---	2	---	1	---	---

Unrelated	8	4	2	1	2	1	---
<u>4G and 4H</u>							
Number with possibly related symptoms	9	4	1	2	1	2	2
Periodic	2	---	---	1	1	1	1
Sporadic	8	4	1	1	---	1	1
Daily	---	---	---	---	---	---	---

Unrelated	5	0	3	1	1	---	---

These are frequencies of symptoms. Individuals could have more than one symptom and could have related and unrelated symptoms at the same time.

TABLE 8

PREVIOUS ILLNESS OF SIMILAR SYMPTOMS

Previous periods of illness of greater than one week's
duration among persons in corridor 7 G + H

		yes	no
Sitting in Corridor 7B	yes	4	2
	no	2	30

$p = 0.002$ (Fisher's exact test)

TABLE 9
TEST OF LUNG FUNCTION

SINGLE-BREATH CARBON-MONOXIDE DIFFUSING CAPACITY
IN PER CENT PREDICTED (After Cotes)

	Group I	Group II
Mean	99.3	101.0
Standard Deviation	20.4	14.2
Number of subjects	31	26

$t = 0.48, n.s.$

SINGLE-BREATH CARBON-MONOXIDE DIFFUSING CAPACITY
IN PER CENT PREDICTEDV (After Cotes)

	Cases	Non-cases in Group I
Mean	86	104.0
Standard Deviation	26.3	16.1
Number of subjects	8	23

$t = 2.30; 29 \text{ df}, p < 0.05$

CHANGE IN FEV₁ in PER CENT

	Group I	Group II
Mean	-0.47	+0.3
Standard Deviation	3.5	3.6
Number of subjects	32	29
$t =$	0.85	n.s.

CHANGE IN FVC IN PER CENT

	Group I	Group II
Mean	0.2	0.4
Standard Deviation	2.9	3.6
Number of subjects	32	29
$t =$	-0.2	n.s.

TABLE 10

PREDOMINANT ORGANISMS ISOLATED FROM BULK SAMPLES
COLLECTED IN CORRIDOR 7B AND IN THE HVAC SYSTEM

SAMPLES FROM CORRIDOR 7B		SAMPLES FROM THE HVAC SYSTEM	
<u>Sample source</u>	<u>Organisms</u>	<u>Sample source</u>	<u>Organisms</u>
False Ceiling tile 709B	<u>Monosporium apiospermum</u> no thermophiles	Dirt on chilled water coils, Air handler #7	<u>Thermoactinomyces candidus</u> <u>Penicillium</u> sp.
Carpet 708B*	<u>Rhodotorula</u> sp. <u>Aureobasidium</u> sp. <u>Acanthamoeba polyphaga</u> No thermophiles	Self moving filter, Air handler #7	<u>Thermoactinomyces candidus</u> <u>Thermoactinomyces vulgaris</u> <u>Aspergillus fumigatus</u> <u>Aspergillus niger</u> <u>Paecilomyces</u> sp.
Carpet 709B*	<u>Acanthamoeba polyphaga</u> No thermophiles	Filter dirt, peri- meter unit in 717H	<u>Thermoactinomyces candidus</u> <u>Aspergillus niger</u> <u>Rhodotorula</u> sp. <u>Mucor</u> sp.
Pipe dirt 709B*	<u>Monosporium apiospermum</u> <u>Acanthamoeba polyphaga</u> No thermophiles	Panel back dirt, perimeter unit in 407H	<u>Thermoactinomyces candidus</u> <u>Aspergillus niger</u> <u>Penicillium</u> sp.
Water sample from flood in 709B	<u>Acanthamoeba polyphaga</u> <u>Colpoda</u> sp. <u>Mastigamoeba</u> sp.	Air supply plenum* downstream from air handler #6	<u>Thermoactinomyces candidus</u> No amoebas
Respirable dust on PVC filter in 709B2	No amoebae	Air supply plenum* downstream from air handler #7	<u>Thermoactinomyces candidus</u> No amoebas

*Samples analyzed for bacteria, fungi and amoebas.

TABLE 11

NUMBER OF PEOPLE WITH PRECIPITATING ANTIBODIES AGAINST SPECIFIC ANTIGENS

ANTIGEN	Humphrey Building Cases N=10	Humphrey Building Controls N=14
^a <u>Micropolyspora faeni</u>	0	0
^a <u>Thermoactinomyces vulgaris</u>	1	0
^a <u>Thermoactinomyces candidus</u>	0	3
^a <u>Saccharomonospora viridis</u>	3	2
^a Pigeon serum	0	1
^a <u>Aspergillus fumigatus</u> #507	0	1
^a <u>Aspergillus fumigatus</u> #515	0	1
^a <u>Aspergillus fumigatus</u> #534	0	0
^a <u>Penicillium notatum</u>	0	0
^a <u>Candida albicans</u>	1	4
^b <u>Aspergillus fumigatus</u>	1	0
^b <u>Aspergillus niger</u>	0	0
^b <u>Aureobasidium</u> sp.	3	2
^b <u>Bacillus</u> sp.	0	0
^b <u>Monospora</u> sp.	0	0
^b <u>Mucor</u> sp.	0	2
^b <u>Paecilomyces</u> sp.	1	2
^b <u>Penicillium</u> sp.	1	0
^b <u>Rhodotorula</u> sp.	0	0
^b <u>Streptomyces griseus</u>	0	0
^b <u>Thermoactinomyces candidus</u>	1	2
^b <u>Thermoactinomyces vulgaris</u>	0	2
^c <u>Naegleria gruberi</u>	0	1
^c <u>N. lovaniensis</u>	0	0
^c <u>Acanthamoeba polyphaga</u>	1	5
^c <u>Acanthamoeba castellanii</u>	1	2

^aStandard panel of antigens, Medical College of Wisconsin^bCrude extracts of antigens from Humphrey Building, Medical College of Wisconsin^cAntigen from Humphrey Building (A. polyphaga) and from stock cultures (A. castellanii, N. gruberi and N. Lovaniensis), Protozoal Diseases Branch, CDC.
N = 10 for cases; N = 14 for controls

TABLE 12
AIRBORNE FUNGI (CFU Per m³)

Media	Time and Location		
	709B March 16 early evening HVAC OFF	709B March 17 late morning HVAC ON	708B March 17 early afternoon HVAC ON
Sabouraud	35	11	2
	12	0	--
Rose bengal	63	21	3
	--	5	--

Controls = zero CFU per m³.

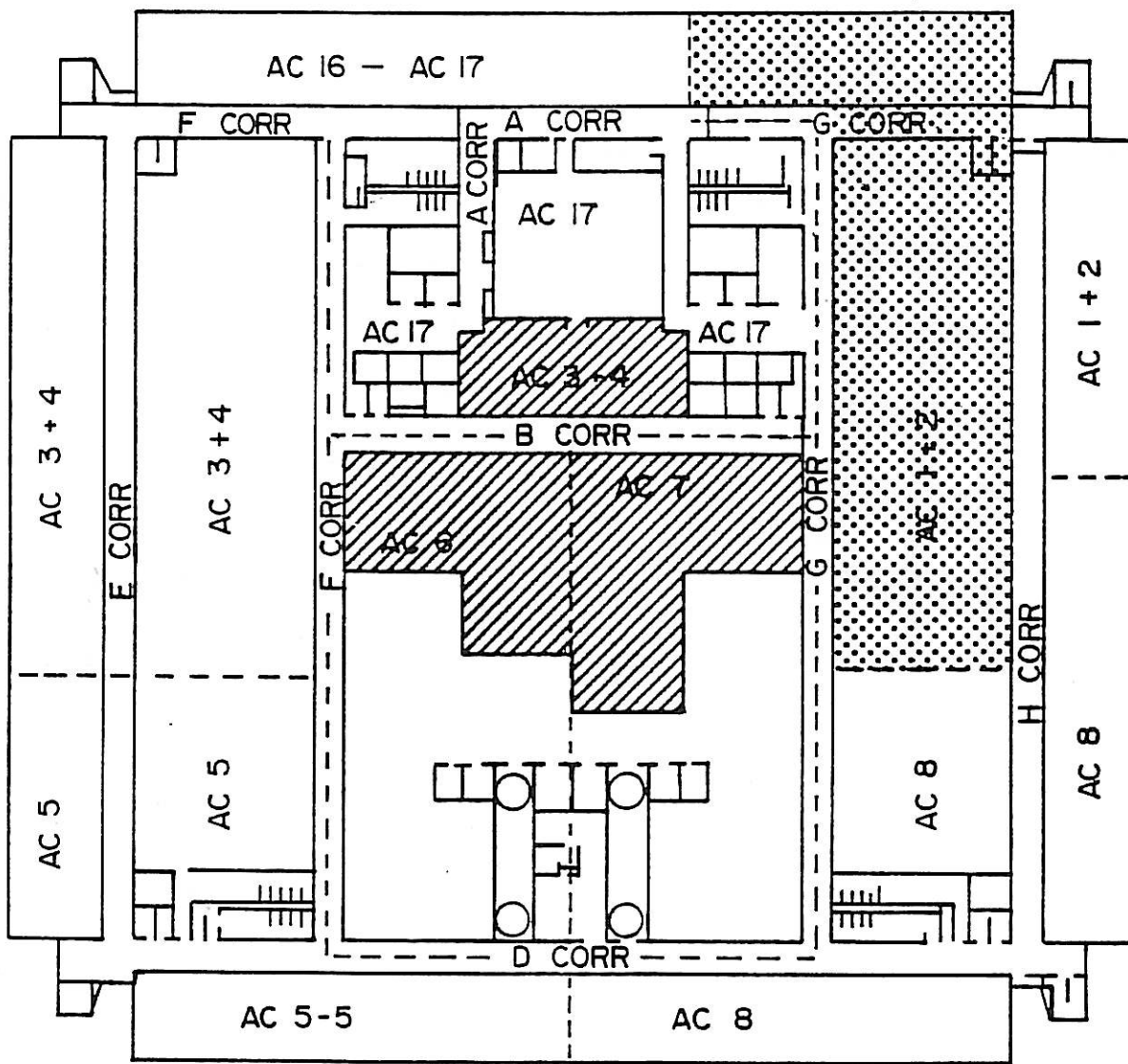
TABLE 13

AIRBORNE FUNGI OR BACTERIA (CFU per m³)
AT VARIOUS TIMES IN CORRIDOR 7B ON MAY 7

Sampling media for (a)	Time	CFU/m ³
Bacteria	7:55-8:15	28
Fungi	8:23-8:43	49
Fungi	13:34-13:54	37
Bacteria	13:59-14:19	11

(a) Trypticase soy agar for bacteria (37°C).
Rose bengal streptomycin agar for fungi (25°C).

FIGURE 1. SEVENTH FLOOR OF HUBERT H. HUMPHREY BUILDING:
LOCATION OF PHS EMPLOYEES BY AIR HANDLER



 Area occupied by PHS employees in 7B with water leaks from the cafeteria

 Area occupied by PHS employees in non-cafeteria leak associated zone

AC = Air Handlers

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