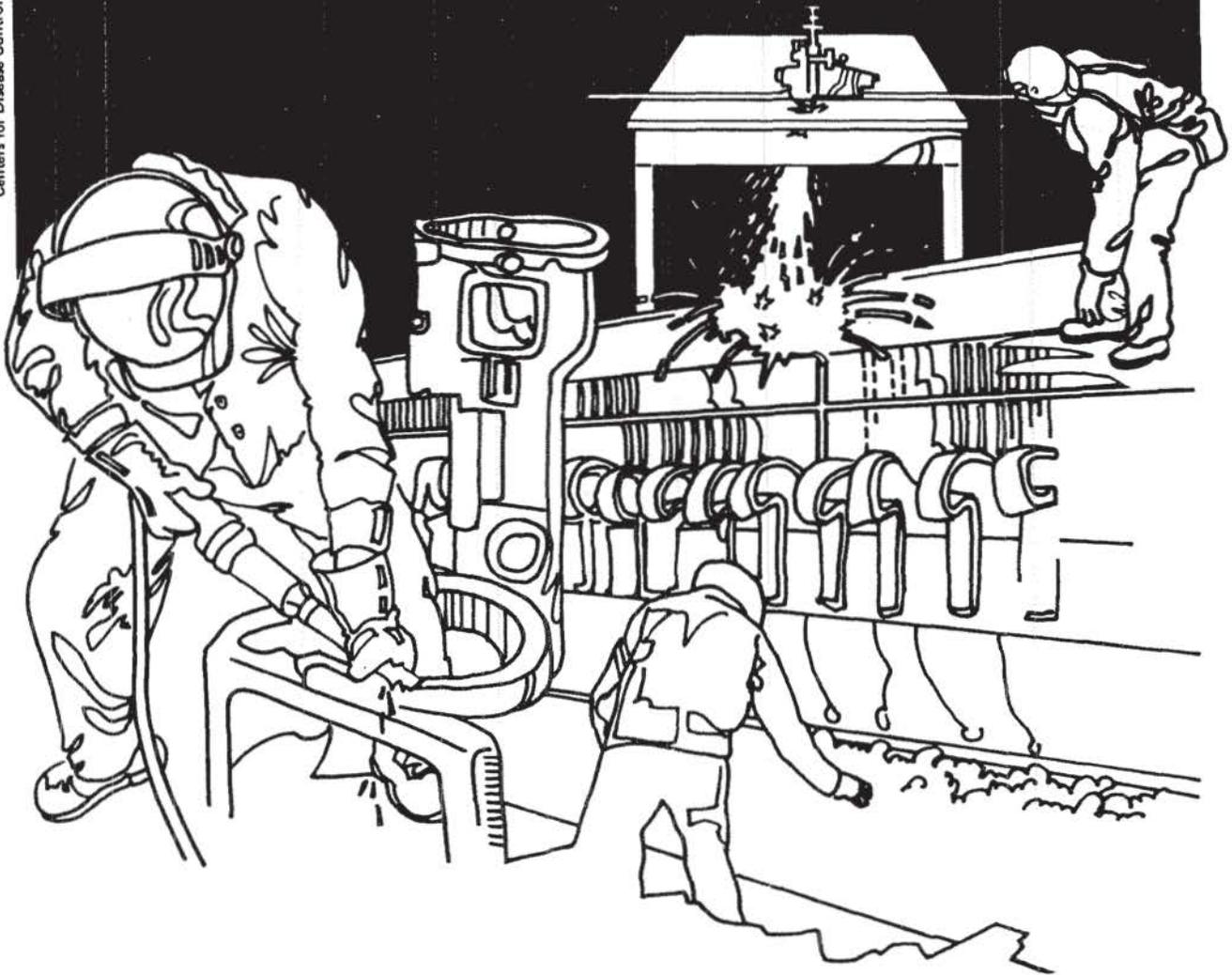


U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES ■ Public Health Service
Centers for Disease Control ■ National Institute for Occupational Safety and Health

NIOSH



Health Hazard Evaluation Report

HEA 82-147-1783
CONVERTERS INK COMPANY
LINDEN, NEW JERSEY

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.

HETA 82-147 - 1783
MARCH, 1987
CONVERTERS INK COMPANY
LINDEN, NEW JERSEY

NIOSH INVESTIGATORS
Peter Gann, M.D.
Carol Wilkinson, M.D.
Kathleen O'Leary, M.S.
Isabel Guerrero, M.D.

SUMMARY

In February, 1982, NIOSH received a request for a Health Hazard Evaluation at Converters Ink Company in Linden, New Jersey. The request was initiated by the employer and was assigned to the New Jersey State Department of Health, Occupational Health Program, under a Cooperative Agreement. The problem concerned an outbreak of fever, chills and respiratory symptoms experienced by employees in a laboratory where inks and printing products are tested.

The evaluation consisted of a walkthrough, two follow-up visits (one for environmental sampling and one for medical evaluation of employees) and the re-examination of six of the originally ill workers. Microbiological and immunologic studies were performed at the Medical College of Wisconsin and the New Jersey Department of Health Laboratories. This report presents the results from both the medical and environmental evaluations.

A health hazard was found to exist due to exposure of laboratory and office workers to water aerosol from a machine used to test the fade resistance of inks. Questionnaires, pulmonary function tests, serum precipitin tests, and fungal cultures were used to identify the illness among 14 workers as humidifier lung, a type of hypersensitivity pneumonitis. The illness appears to have been largely acute and self-limited hypersensitivity pneumonitis. The antigen responsible for the illness is unknown, but it is certainly contained in stagnant water from the machine's humidifier. A gel diffusion test for serum precipitins to the stagnant water was found to be highly specific for the illness.

On the basis of a walkthrough evaluation and serial medical evaluations of employees, it has been determined that a form of acute hypersensitivity pneumonitis developed at Converters Ink Company due to exposure of laboratory and office workers to water aerosol from a machine used to test the fade resistance of inks. Recommendations to relieve workers from recurrence of this disorder are provided at the end of this report.

Key Words: SIC 2893 production of inks, respiratory complaints,
hypersensitivity pneumonitis

BACKGROUND

Converters Ink is a company specializing in the production of flexigraphic and rotogravure printing inks. The offices, with 13 employees, and the laboratory, with 16 employees, are located in the same single-story building. The production plant is located in a separate but interconnected building.

On February 10, 1982 the laboratory workers noted a strong unpleasant "dead animal" odor emanating from the back corner room which is used for chemical storage and also contains a Fade-Ometer machine for testing the fade-resistance of inks. Upon returning to work on February 11, the lab and office workers discovered that approximately 14 of their number had experienced an illness following work the previous day characterized by fever, weakness, chills, headache and chest discomfort. Most of the workers had recovered by the next morning, but a few continued to have symptoms for several days.

It was reported that a similar episode had occurred on July 23, 1981. An odor was noticed in the Fade-Ometer room, and several workers experienced a flu-like illness that evening. Symptoms were more severe in the February episode.

A floor diagram of the laboratory and office is shown in Figure 1. The Fade-Ometer, which is schematicized in Figure 2, is a metal enclosure housing a carbon arc lamp and circular rack for holding strips of test material. This type of instrument is the standard for determining the effects of light on all types of materials under controlled conditions of temperature and relative humidity. The typical temperature in the test chamber during operation is 57° C and the relative humidity normally fluctuates between 37 and 40%. The base of the instrument houses a fan, a conditioning chamber, and a humidifier with float valve. The humidifier draws water from a pan in the conditioning chamber where evaporation is occurring. The fan then blows the vapor, with some aerosol, into the test chamber. The humidified air is vented from the top of the instruments; the fan is on continuously during operation. The manufacturer's recommended maintenance for the conditioning and humidification unit consists of periodically draining the chamber and wiping with a soft cloth.

The Fade-Ometer was used on an irregular basis whenever certain inks must be tested. It was operated approximately twice per month and then usually for a period of 24 to 72 hours. The instrument at Converters Ink was put into operation in February, 1980. Prior to February 10, 1982, the machine had not been turned on for several weeks.

Subsequent to an interim report as filed in January, 1983, six workers who became ill during the hypersensitivity outbreak were invited to have followup lung function studies at a hospital in Elizabeth, New Jersey. Four were done in April, 1983, and two more in August.

EVALUATION METHODS

A. Environmental

An industrial hygienist visited Converters Ink on February 19, 1982. A walkthrough of the laboratory and office was performed in order to exclude the possibility that any laboratory chemicals could have been associated with the outbreaks of illness. Sampling for air contaminants was not believed to be justified and was not conducted.

A medical communicable disease investigator inspected the area on February 25, 1982, and obtained numerous culture specimens from water samples and room and instrument surfaces. These specimens were then delivered to the Department of Health Laboratories. A small (approximately 6 ounce) sample of water had been taken from the Fade-Ometer by Converter's personnel on February 12. A portion of this sample was sent to a private laboratory for fungal and bacterial analysis.

B. Medical

NIOSH and New Jersey State Department of Health investigators were notified of the second outbreak on February 17, and two physicians visited the site the following day. Rapid evaluation was indicated and therefore, all affected employees were sent to the company's contracted clinic on February 19 for chest x-rays, complete blood counts and erythrocyte sedimentation rates (ESRs). Pulmonary function tests were conducted on the affected employees on March 23 at Alexian Brothers Hospital in Elizabeth, New Jersey.

Medical investigators returned to the site on March 3 and conducted an interview using a standard questionnaire. Approximately 15 cc of venous blood was collected on each person interviewed. Subjects included 23 laboratory and office personnel and 16 workers from the adjacent plant who had experienced no complaints and were not exposed to the noxious odor in the laboratory. Blood samples were sent to Dr. Jordan Fink at the Medical College of Wisconsin for gel diffusion analysis of serum precipitins. Sera from the workers who were ill were also analyzed for antibodies to Legionella pneumophila by the New Jersey State Department of Health Laboratories.

Review of the pulmonary function results led to two individuals returning in to Alexian Brothers Hospital for repeat spirometry plus determination of single-breath diffusing capacity and static lung volumes.

Six workers who became ill during the outbreak in February were invited to have follow-up lung function studies. Four were studied in April, 1983 and two more in August. All six had previously shown borderline or frank restrictive findings on their initial pulmonary function tests.

EVALUATION CRITERIA

A case definition based upon the questionnaire was established. A case was defined as any Converters Ink Employee experiencing an acute illness beginning on February 10 or 11 characterized by extreme fatigue, weakness, fever, chills, or lower respiratory

symptoms. Fourteen of 29 laboratory/ office workers met these criteria for an overall attack rate of 48%. None of the 19 workers interviewed from the adjacent manufacturing plant became ill on the days in questions. There was no difficulty in distinguishing cases due to the abrupt onset of the illness.

RESULTS

A. Environmental

Although dozens of chemicals were found to be in use in the laboratory, including propanol, ethanol, ammonia, pigments and resins, none were introduced during the weeks the outbreaks occurred and none were being used in a manner that suggested any possible association with the pattern of illness observed.

The microbiological findings are summarized in Table 1. Legionella was not found in any of the cultures. Enterobacter cloacae was the sole bacterium isolated from the stagnant water sample. The fungi isolated from water and surface samples represent a typical spectrum for the geographic area and do not include any unusual pathogens. The significance of the fungal isolations, however, is discussed further under medical findings.

B. Medical

Tables II and III show the frequency of various reported symptoms among cases and a description of attack rates. Figure 3 shows the distribution of time of onset for the cases.

It was noted that nine workers who became ill February had also been ill during the similar episode the previous July. All nine reported that the symptoms were more severe the second time and that in particular, respiratory effects such as shortness of breath, cough and chest heaviness were more prominent. Table IV shows the relationships between the occurrence of July and February illnesses among individuals. It can be seen that out of five workers who were normal in July and were present again in February, four were normal again. Out of ten workers who were sick in July and were present in February, nine became sick again. Thus, a high degree of correlation between illness status on both occasions was observed.

The work locations of the cases are shown in Figure 1. A lack of cases among workers at the "front" lab benches (#1 and #2) is noted. The duties of the chemists at these benches do not differ from the rest of the group.

Complete blood counts and erythrocyte sedimentation rates were available on 17 persons. Total white cells and differential were all normal. There was no evidence for eosinophilia in the ill group. Sedimentation rates, however, appeared to be elevated. Of 13 ill persons who were tested, 6 had mild or moderate sedimentation rate elevations, ranging from 14 to 40 mm/hr. Only 4 non-ill persons had sedimentation rate determinations, and all were normal.

Chest x-rays did not reveal instances of intrinsic lung disease in any of the cases except when chronic disease such as COPD was known to exist.

Pulmonary function tests were obtained on thirteen of the cases. Evidence for restrictive lung disease (FVC, forced vital capacity, less than 80% of predicted) was found in two cases. In five other cases, the FVC was between 80 and 85% of predicted. There was no correlation between reporting of respiratory symptoms and pulmonary function results. The two persons with decreased FVC were tested again, including static lung volumes and single-breath diffusing capacity. In one case the abnormal FVC appeared to be explained largely by chronic heart disease. However, in the second case, FVC remained decreased and the carbon monoxide time constant was reduced to 73% of predicted.

Of the six who were given follow up pulmonary function testing in 1983, one had minimal restriction believed to be due to obesity. One had a mild restrictive abnormality due to congestive heart failure. Three no longer showed restrictive indices. The only case that raised concern about possible chronic hypersensitivity pneumonitis was clinically well but continued to have an FVC that was 67% of predicted.

Serological studies for antibodies to *Legionella pneumophila* serogroups 1 through 6 were performed on 17 samples. Only one person, who was ill on both occasions, displayed any reaction; in this case to groups 2 and 6.

Precipitin studies were performed blindly on sera from laboratory/office workers, and manufacturing plant workers. Using an agar gel double diffusion method, sera were tested 1) against all types of fungi grown from the Fade-Ometer stagnant water sample ("crude antigens") 2) against laboratory-prepared fungal extracts from organisms known to be associated with hypersensitivity diseases ("lab antigens"), and 3) against an extract from the stagnant water sample itself. The results, divided into ill + exposed, total exposed, and non-exposed groups, are shown in Table V. Significant differences in prevalence of precipitins were not observed for any of the specific fungal antigens. However, a striking difference in precipitins to the stagnant water sample was observed. Seven people from the ill + exposed group demonstrated precipitins, whereas none from the other two groups did so. The sensitivity of this precipitin test for the illness, therefore, was 50%, its specificity was 100%.

DISCUSSION

The differential diagnoses at the start of this outbreak investigation included the following:

1. Non-pneumonic, short-incubation period Legionellosis ("Pontiac Fever")
2. Polymer fume fever
3. Hypersensitivity Pneumonitis ("Humidifier Fever")

Legionellosis was unlikely due to the absence of microbiological and immunological evidence. The prevalence and degree of pulmonary symptoms in this outbreak were also slightly greater than that previously reported for Pontiac Fever. Furthermore, the illness recurred in several individuals whereas infection with *Legionella* would be expected to provide at least relative immunity. (1) Polymer fume fever is a flu-like illness caused by exposure to the thermal decomposition products of fluorinated polymers such as polytetrafluoroethylene (Teflon). (2) A piece of Teflon tape was found attached to the glass bell of the carbon arc lamp. However, despite the intense heat in that area, the tape was intact and did not appear to have been burnt.

The evidence points towards hypersensitivity pneumonitis (also referred to as extrinsic allergic alveolitis) as the likely cause of this outbreak. The term "humidifier lung" has sometimes been used to describe similar outbreaks associated with air conditioners, humidifiers, or vaporizers. (3) Various microorganisms have been implicated, as shown in Table VI, but evidence for a specific causative organism is often difficult to obtain, and there exists no agreement as to the necessary characteristics for including humidifier lung. The disease process does involve an immune reaction to the offending agent, rather than an actual infection. A Type III hypersensitivity reaction, with formation of specific IgG antibodies, is believed to occur.

The diagnosis of humidifier lung in this outbreak is supported by several factors. First, the character and short duration of the chief symptoms was highly typical. The close clustering of times of onset was also typical. The tendency for the illness to recur among the same individuals in a relatively homogeneous work population suggest an all-or-nothing sensitization phenomenon. The overall attack rate, however, was higher than might be expected in an immune type of outbreak; this requires further discussion. The apparent "skipping" of one of the lab benches is also unexplained, although conceivably this could be due to varying deposition of aerosol around the room. The finding of cases among office workers could either be due to their practice of occasionally entering the laboratory or to the penetration of aerosol into the offices through the drop ceiling or open doors. The general flow of air in the building is from the Fade-Ometer side to the office side where a return air vent is located.

The intermittent use of the Fade-Ometer means that water may remain for several weeks in the tray below the humidifier. When the machine is turned on, the water is warmed to an ideal temperature for the growth of microorganisms, and the fan is capable of providing a steady stream of contaminated aerosol droplets for a period of one to three days. These droplets exit through a vent in the top of the machine and enter the general circulation of the room. Prior to the February outbreak, the water had not been drained and replaced for one year.

The laboratory data was also consistent with a diagnosis of hypersensitivity pneumonitis. Erythrocyte sedimentation rates appeared to be elevated, even 9 days after onset of the illness. Total white counts and chest x-rays were unremarkable. The pulmonary function data indicated 2 individuals with definite restrictive lung disease, and 5 with borderline values. The strongest evidence, however, is obtained from the precipitins to stagnant water extract which were identified only in cases. Analysis for precipitins has often proved vague or misleading when investigating

possible outbreaks of hypersensitivity pneumonitis. (4) A single causative organism can rarely be identified, and although exposed persons tend to have a higher prevalence of antibodies to certain cultured organisms than non-exposed persons, the correlation between presence of antibodies and clinical illness is often poor. In this case, fungi cultured from the machine, including thermophiles, did not appear to be involved. However, a precipitin test using an extract from the suspect water sample was highly specific for illness. These data suggest that an antigen, as yet unidentified, is present in the stagnant water sample and could be responsible for the outbreak. Protozoans have been implicated in previous outbreaks, and cannot be ruled out here as they were not extensively searched for. Extremely fastidious fungi or bacteria, or the involvement of endotoxin from *Enterobacter*, are further possibilities.

Humidifier lung, like other types of hypersensitivity pneumonitis, has two distinct clinical forms: acute and chronic. The symptoms in this outbreak are consistent with the acute form. However, two features suggest that a chronic, insidious reaction cannot be excluded. First, the high attack rate may be due to prolonged exposure to the unknown antigen, allowing sensitized individuals to be exposed repeatedly. Second, the finding of decreased or borderline forced vital capacities several days after the acute illness may be explained by a mild degree of interstitial lung change normally associated with chronic hypersensitivity pneumonitis. Evidence against the presence of the chronic form is the largely negative results of follow-up pulmonary function testing over a year following the last outbreak.

As of January, 1983, the illness has not recurred. Also prior to this date, a wall exhaust fan was installed near the ceiling in the Fade-Ometer room, and greater care was taken during the operation of the machine to ensure that water did not stagnate. In addition, an algicide was added to the humidifier water.

CONCLUSION

A health hazard was found to exist at Converters Ink Company in Linden, New Jersey due to exposure of laboratory and office workers to water aerosol from a machine used to test the fade resistance of inks. Questionnaires, pulmonary function tests, serum precipitin tests, and fungal cultures were used to identify the illness among 14 workers as humidifier lung, a type of hypersensitivity pneumonitis. The illness appears to be largely acute and self-limited, and chronic hypersensitivity pneumonitis, though documented in other situations, appears not to have developed in this group. The antigen responsible for the illness is unknown, but is certainly contained in stagnant water from the machine's humidifier. A gel diffusion test for serum precipitins to the stagnant water was found to be highly specific for the illness.

RECOMMENDATIONS

Recommendations made in January, 1983, at Converter's Ink, which are still applicable, included:

1. Follow-up examination including spirometry was recommended to the one worker who had an unexplained restrictive pulmonary function test.

2. Water should not be permitted to stagnate in the Fade-Ometer. A continual flow of water through the humidifier system is recommended.
3. At least once a month, water should be drained from the Fade-Ometer and surfaces should be cleared with a bacteriostatic and fungostatic solution.
4. An algicide should be added to the Fade-Ometer water at all times to reduce the presence of organic matter.
5. Illness among the laboratory/office workers should be monitored by self-reporting and at the first sign of a new outbreak, NIOSH and State Department of Health physicians should be notified immediately.

REFERENCES

- (1) Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: Isolation of the etiologic agent and demonstration of its mode of transmission, *American Journal of Epidemiology*, 114:337-347, 1981.
- (2) Dwiggins GA, Wiggins MC, and Dwyer MJ, Exposure to polytetrafluoro-ethylene decomposition products during the initial use of some pad heaters, *Amer. Ind. Hyg. Assoc. Journal*, 42:319-320, April, 1981.
- (3) Gainer M, Lieberman P, Fink JN and Lockwood DG, Humidifier lung: on outbreak in office workers, *Chest* 77:183-187, February, 1980.
- (4) Scribner GH, Barboniak JJ, and Fink JN, Prevalence of precipitins in groups at risk of developing hypersensitivity pneumonitis, *Clinical Allergy*, 10:91-95, 1980.
- (5) Banaszak EF, Thiede WH, and Fink JN, Hypersensitivity pneumonitis due to contamination of an air conditioner, *N. Engl. J. Med.*, 283:271, 1970.
- (6) Edwards JH, Microbial and immunological investigations and remedial action after an outbreak and humidifier fever, *Br. J. Indust. Med.*; 37:55, 1980.
- (7) Rylander R, Haglind P, Lundholm M, et al. Humidifier fever and endotoxin exposure, *Clinical Allergy* 8:511, 1978.
- (8) Hales CA, Rubin RH, Case records of the Massachusetts General Hospital, Case 47-1979, *N. Engl. J. Med.* 301:1168, 1979.
- (9) Bernstein RS, Sorenson WG, Garabrant D, et al. Exposure to respirable, airborne Penicillin from a contaminated ventilation system, unpublished report.
- (10) Parrott WF, Blyth W, Another causal factor in the production of humidifier fever, *J. Soc. Occup. Med.*, 30:63, 1980.
- (11) Johnson CL, Bernstein IL, Gallagher JS, et al. Familial hypersensitivity pneumonitis induced by *Bacillus subtilis*, *Am. Rev. Respir. Dis.*, 122:339, 1980.

AUTHORSHIP AND ACKNOWLEDGEMENTS

Report prepared by: Peter Gann, M.D.
presently with the University of
Massachusetts Department of Family and
Community Medicine

Timothy Liveright, M.D.
New Jersey State Department of Health

Evaluation conducted by: Peter Gann, M.D.

Carl Wilkinson, M.D.
presently in private practice

Environmental evaluation: Isabel Guerrero, M.D.
presently practicing with the New Jersey
State Department of Corrections

Kathleen O'Leary, M.S.
New Jersey State Department of Health

Immunology testing: Jordan N. Fink, M.D.
Medical College of Wisconsin
Milwaukee, Wisconsin

DISTRIBUTION AND AVAILABILITY OF THIS REPORT

Copies of this report are currently available, upon request, from NIOSH, Division of Technical Services, Information Resources and Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through National Technical Information Service (NTIS), Springfield, Virginia 22161.

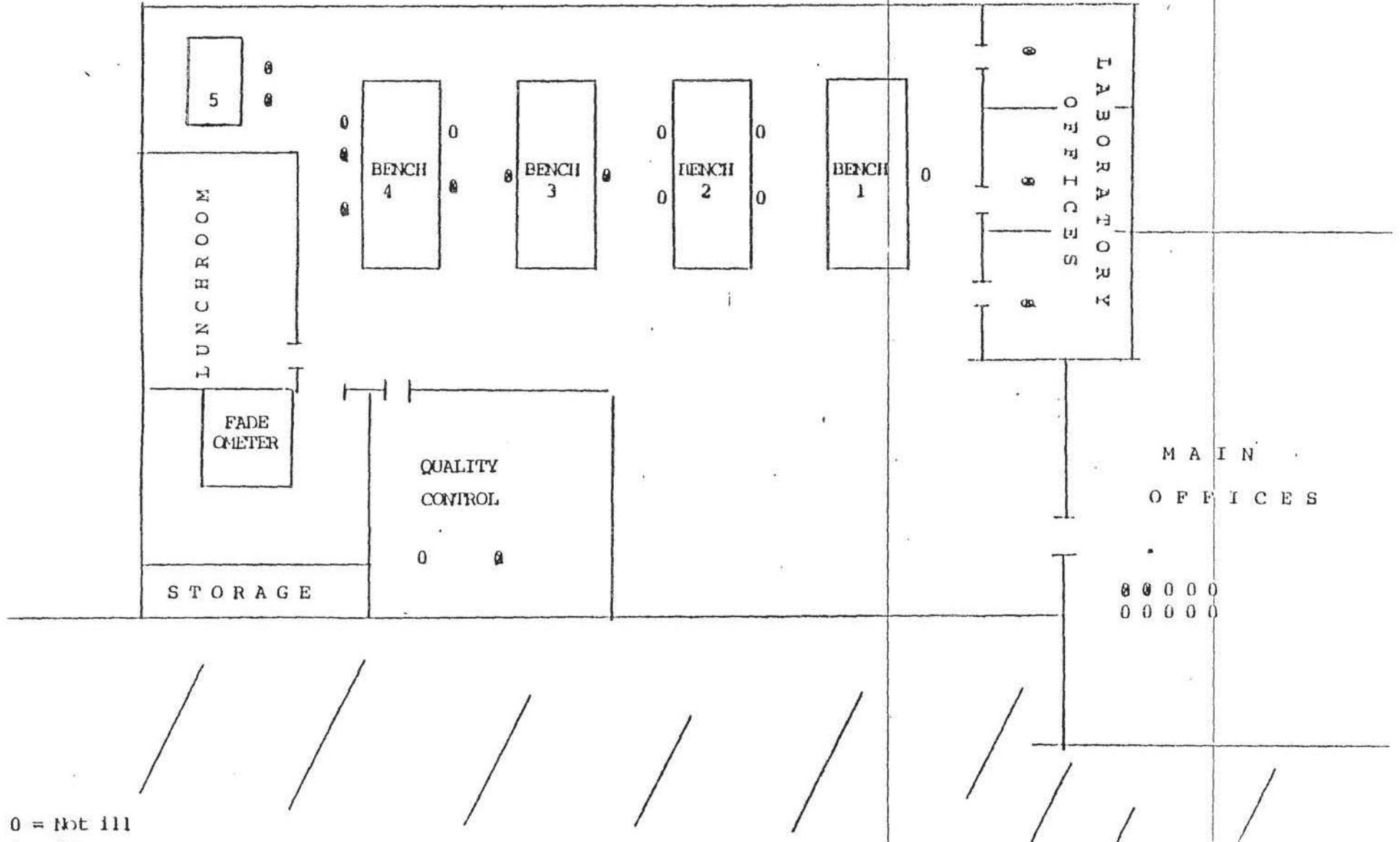
Copies of this report have been sent to:

Atlas Electric Company, Chicago (manufacturer of instrument)
Anthony Cappuccio, Converters Ink
U.S. Department of Labor, OSHA, Region II, New York, NY

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

FIGURE 1

FLOOR DIAGRAM: LAB/OFFICES AT CONVERTER INK
WITH LOCATION OF CASES



0 = Not III
= III

APPROX 6'

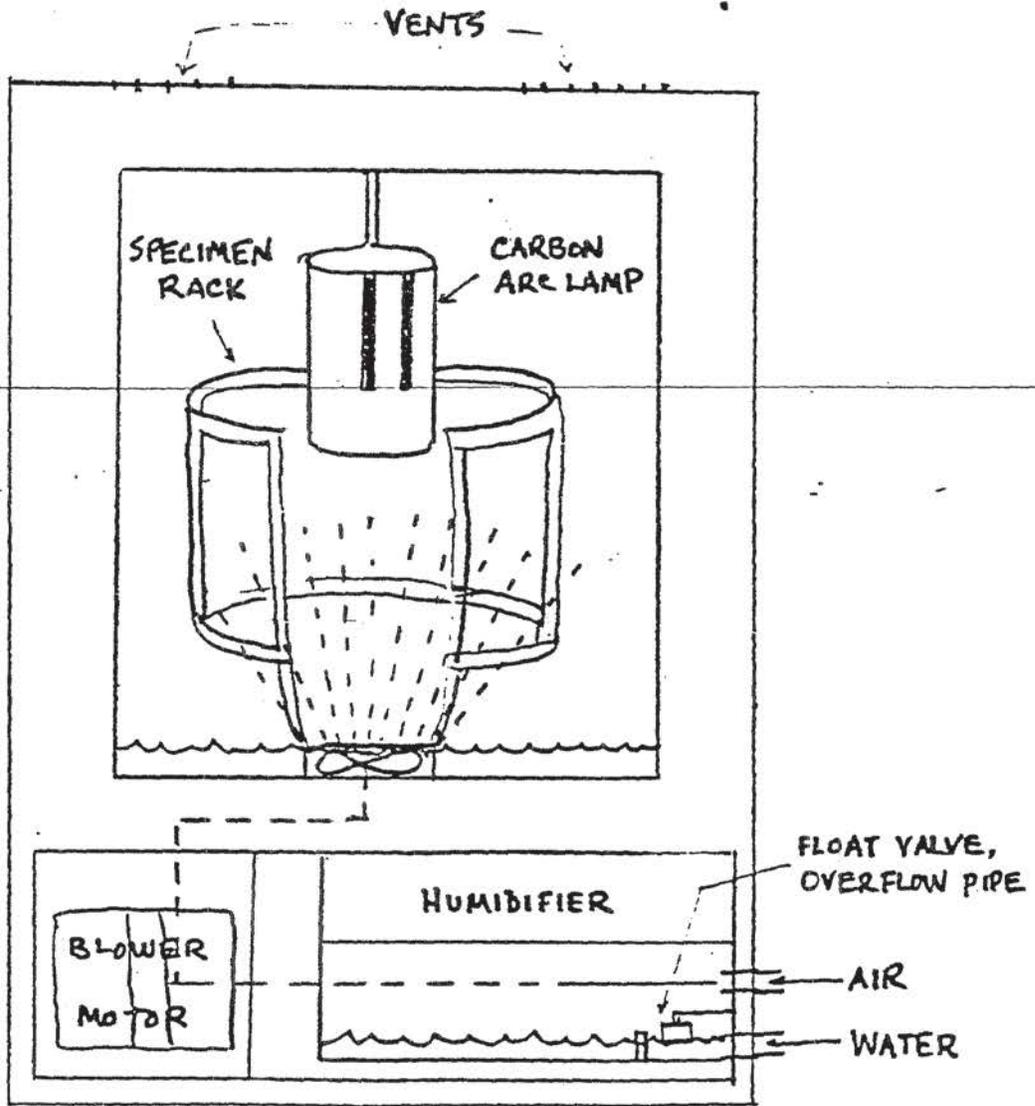


Figure 2

Schematic Diagram of Fade-Ometer
Converters Ink Company, Linden, New Jersey

Figure 3 - Illness Time of Onset

(n = 14)

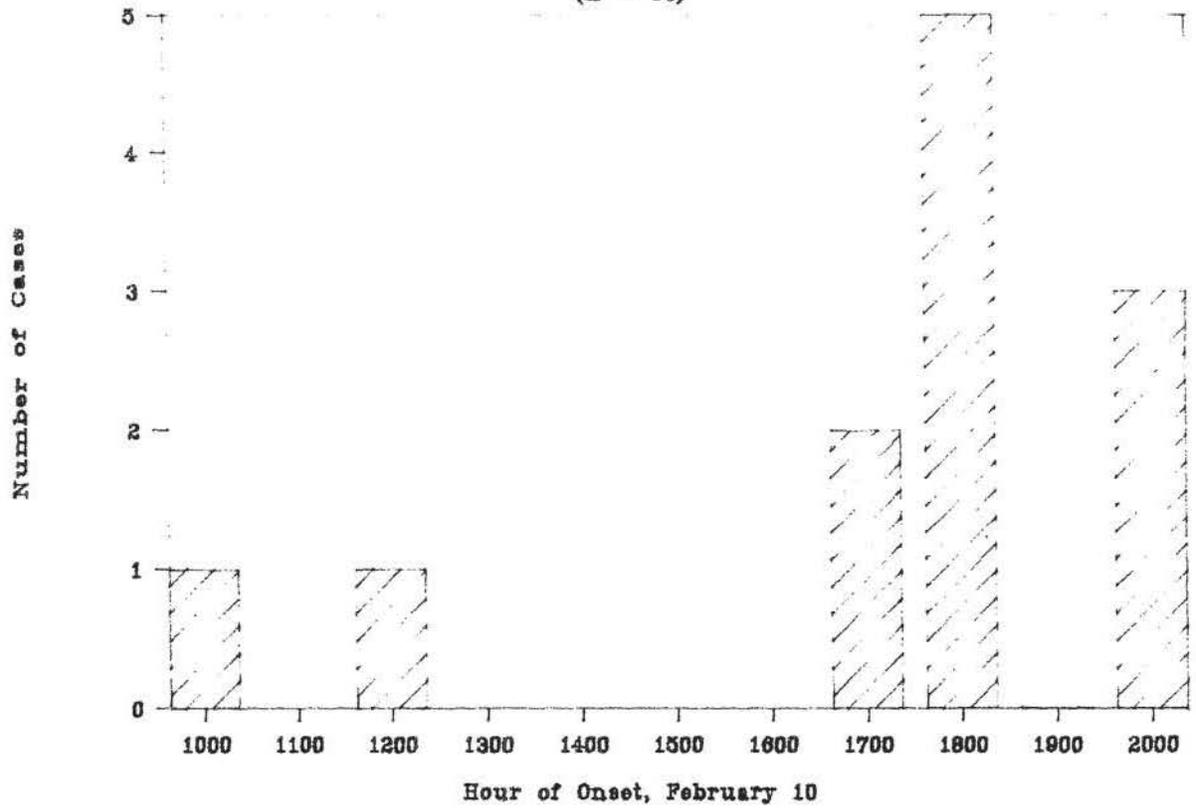


TABLE I

MICROBIOLOGICAL ISOLATES: CONVERTERS INK, LINDEN, NEW JERSEY

SOURCE	ISOLATES
Exterior and Interior of Fade-Ometer Vents	No growth obtained
Upper outlet, Fade-Ometer	Fusarium Rhodotorula, Penicillium, Cladosporium
Lower outlet, Fade-Ometer	Fusarium Rhodotorula, Penicillium, Cladosporium
Overflow pipe, Fade-Ometer	Fusarium Rhodotorula, Penicillium, Cladosporium
Window sill, Fade-Ometer Room	Rhodotorula, Alternaria, Aureobasiclium pullulans
Stagnant Water, Fade-Ometer Humidifier	Enterobacter cloacae, Aspergillus niger, Rhodotorula, Saccharomyces, Candida guillermondi

Note: Legionella growth was not obtained in any cultures. Growth of Thermophillic fungi was also not obtained

TABLE II
 REPORTED SYMPTOM FREQUENCIES, FEBRUARY 1982 OUTBREAK
 CONVERTERS INK, LINDEN, NEW JERSEY

Symptom	No. (%)	Symptom	No. (%)
chills	12 (86)	shortness of breath	8 (57)
easy fatigability	12 (86)	joint aches	8 (57)
weakness	11 (79)	muscle aches	7 (50)
fever	10 (71)	headache	5 (24)
chest heaviness	10 (71)	cough	3 (21)

TABLE III
 ATTACK RATES, FEBRUARY 1982 OUTBREAK
 CONVERTERS INK, LINDEN, NEW JERSEY

Work Area	No. At Risk	No. Ill	Attack Rate
Laboratory	16	9	56%
Lab. Office	3	3	100%
Main Offices	10	2	20%
Mfg. Plant	19	0	0
Overall	29	14	48%

TABLE IV: COMPARISON OF ILLNESS ON TWO OCCASIONS
 WORKERS PRESENT BOTH ON 7/81 AND 2/82
 CONVERTERS INK, LINDEN, NEW JERSEY

	February, 1982 Illness	
	absent	present
July, 1981 Illness		
absent	3	1
present	1	9

Risk Ratio = 4.5

TABLE V

SERUM PRECIPITIN RESULTS: CONVERTERS INK, LINDEN, NEW JERSEY

Antigen	Total Exposed (n=23)	Ill + Exposed (n=14)	Non-Exposed (n=16)
Lab antigens			
M. faeni	1	0	1
T. vulgaris	0	0	1
T. candidas	7	6	5
S. viridis	1	1	5
Pigeon serum	1	1	0
A. fumigatus	2	2	4
Pen. notatum	2	2	0
Can. abicans	1	1	1
<hr/>			
Stagnant Water	7	7	0
<hr/>			
Crude antigens			
Alternaria	17	10	10
A. Niger	0	0	1
Aurea pullulans	3	3	1
Cladosporium	1	1	1
Fusarium	3	0	2
Penicillium	4	2	5
Rhodoturula	1	0	2
Saccharomyces	1	1	3
Sabouraud medium	10	5	8

This includes the ill + exposed group

TABLE VI

ETIOLOGIC AGENTS IN BUILDING OUTBREAKS OF HYPERSENSITIVITY
PNEUMONITIS¹

Reference	Attack Rate	Agent	Source
(5)	4/27	<u>Microspolyspora faeni</u>	Water spray cooling system
(6)	9-20/50	Amoeba	Humidifier, ceiling dust
(7)	3/7	Flavobacteria, endotoxin	Humidifier
(8)	5/30-40	<u>Thermophilic actinomycetes</u>	Water in ventilation ductwork
(9)	2-3/14	<u>Penicillium</u>	Heater-coder unit
(10)	7/26	<u>Bacillus subtilis</u>	Humidifier
(11)	6/10	<u>Bacillus subtilis</u>	Wood dust, bathroom

adapted by permission from Dr. Michael Hodgson and Dr. Kathleen Kreiss, CDC

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
ROBERT A. TAFT LABORATORIES
4676 COLUMBIA PARKWAY, CINCINNATI, OHIO 45226

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE. \$300

Third Class Mail



POSTAGE AND FEES PAID
U.S. DEPARTMENT OF HHS
HHS 396