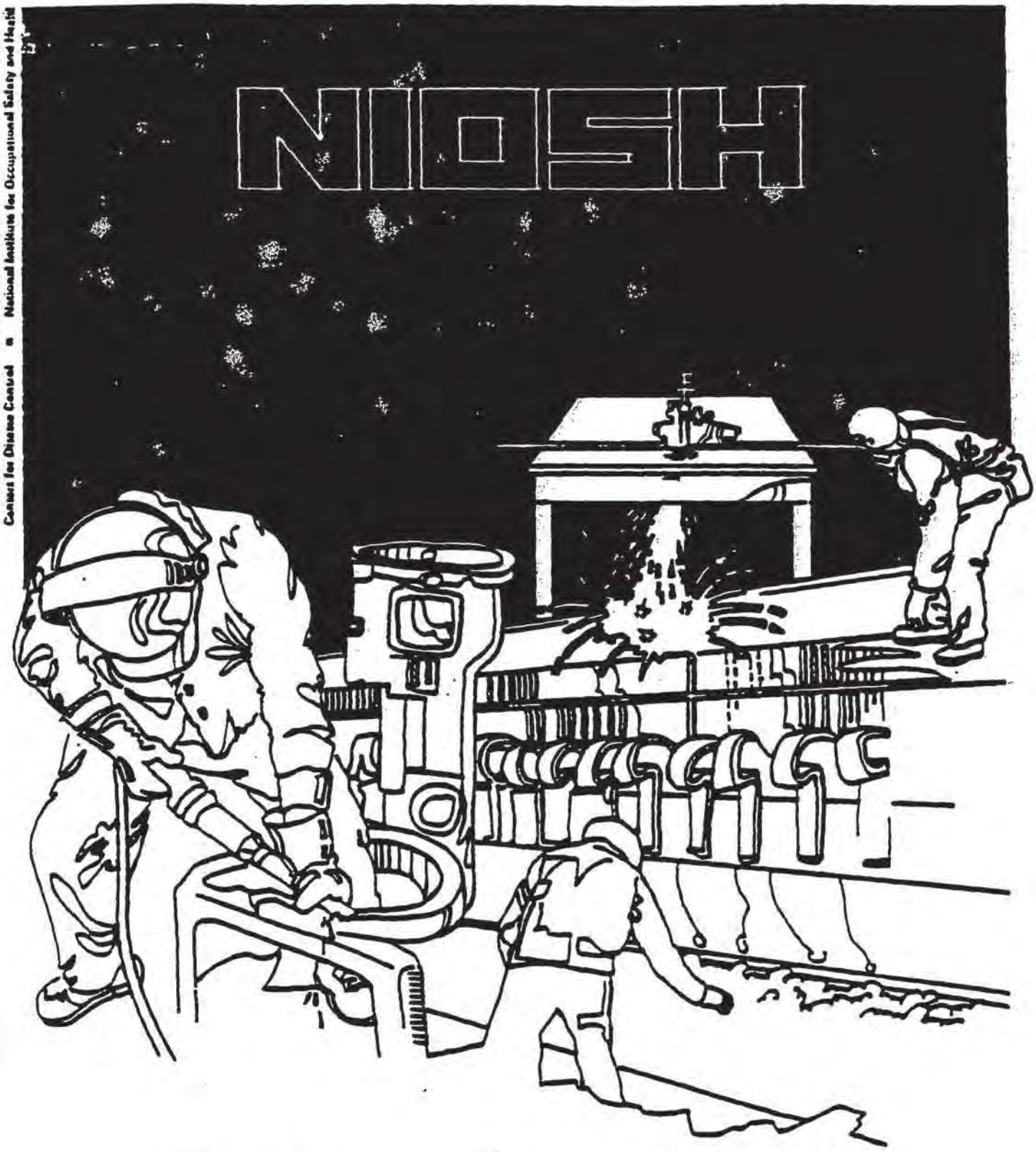
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES • Public Health Service
Centers for Disease Control • National Institute for Occupational Safety and Health

NIOSH



Health Hazard Evaluation Report

HETA 84-163-1657
SIOUXPREME EGG PRODUCTS
SIOUX CENTER, IOWA

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.

HETA 84-163-1657
January 1986
SIOUXPREME EGG PRODUCTS
SIOUX CENTER, IOWA

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I. SUMMARY

On January 30, 1984, the National Institute for Occupational Safety and Health (NIOSH) received a request to conduct a health hazard evaluation from workers at Siouxpreme Egg Products, Sioux Center, Iowa. This plant processes raw eggs into egg yolk powder and liquid egg white. Several workers were experiencing asthmatic-like symptoms, without watering of the eyes or any noticeable skin involvement. The request listed several cleaners, sanitizers, and germicides as possible chemicals that may be causing the symptoms. An initial site visit was conducted during the week of March 29, 1984. On August 14 and 15, 1984, air sampling was conducted throughout the plant for chloride and iodide ions, acid gases (hydrogen chloride and sulfuric acid), and total and respirable egg dust. Noise measurements were also taken throughout the plant. Questionnaires were administered to 91 plant employees.

Employees' exposure to any of the four chemical contaminants studied were well below OSHA, NIOSH, and ACGIH exposure guidelines. Area samples for chloride ranged from detected but not quantified to 24.3 micrograms per cubic meter (ugm/m^3), and for iodide ranged from none detected to 45.4 ugm/m^3. One personal sample showed a chloride level of 12.9 ugm/m^3 and an iodide level of 20.7 ugm/m^3. Two samples for HCl showed 5.6 and 21.7 ugm/m^3, and for H_2SO_4 showed 36.5 and 90.1 ugm/m^3. Personal total dust results in the sifting and packaging room were 12.8 milligrams per cubic meter (mg/m^3) and 7.3 mg/m^3 on the one worker during the two days sampled. Area total and respirable dust results were 1.2 mg/m^3 and 0.94 mg/m^3 total and 0.07 mg/m^3 and 0.03 mg/m^3 respirable. Two personal dust samples were 7.3 and 12.8 mg/m^3, which straddle the ACGIH guideline of 10 mg/m^3 total dust. The area respirable dust levels were 0.03 and 0.07 mg/m^3, which is below the ACGIH guideline of 5 mg/m^3. Analysis of one set of dust samples demonstrated it to be 50 percent protein, the amino acid composition of which resembled egg yolk protein.

Noise levels ranged from a high of 98 dBA in the sifter powder room to 85 dBA in the pick and prewash area. Six of the nine measurements exceeded 90 dBA. Octave band measurements suggested that the major source of the noise was compressed air discharging and pumps.

During the week of March 11, 1985, thirty-one employees were evaluated for occupational asthma. Five of these 31 were determined to have occupational asthma, the underlying mechanism for which was IgE mediated hypersensitivity to egg protein. Four of five asthmatics were non-atopic, that is, they did not have evidence of a familial predisposition to develop allergic responses. There were an additional seven employees examined by a physician, in whom the diagnosis of occupational asthma was considered as a possibliity, but for whom the clinical information was insufficient at the time of the examination to substantiate the diagnosis. It is possible that some of these employees may progress to express fully the clinical picture of occupational asthma.

On the basis of these data, NIOSH investigators have determined that a health hazard existed for employees of Siouxpreme Egg Products, Inc., from occupational exposure to egg protein. At least five workers were demonstrated to have occupational asthma due to IgE mediated hypersensitivity to egg proteins. In addition, a potential health hazard existed from noise and dust (determined to be egg protein) levels that exceeded the prevailing exposure criteria. Recommendations to reduce exposures and improve screening of at-risk workers are made in Section IX.

KEYWORDS: SIC 2017, egg protein, egg dust, occupational asthma, chlorine, iodine.

II. INTRODUCTION

On January 11 and 17, 1984, the Iowa Bureau of Labor conducted an inspection at Siouxpreme Egg Products in response to employee complaints of asthma. On February 8, 1984, they notified the plant manager that their air sampling for iodine showed less than detectable quantities; and, therefore, no citation would be issued at that time. On January 30, 1984, the National Institute for Occupational Safety and Health (NIOSH) received a request to conduct a health hazard evaluation from workers at Siouxpreme Egg Products, Sioux Center, Iowa. Several workers were experiencing asthma-like symptoms, without watering of the eyes or any noticeable skin involvement. The request listed several cleaners, sanitizers, and germicides as possible chemicals that may be causing the symptoms.

On March 29, 1984, NIOSH investigators conducted an initial site visit to validate the request, to conduct a walk-through survey of the plant, to collect additional information about the plant processes, and to talk with employees concerning their symptoms. During the week of August 13, 1984, NIOSH investigators administered questionnaires to employees and conducted environmental an survey. From the administered questionnaires, a study cohort was selected, and NIOSH investigators returned to the plant during the week of March 11, 1985 to conduct a comprehensive medical evaluation of the workers. Individual medical results of the March 11, 1985 survey were reported to each participant by mail in November, 1985. The plant manager was also notified by telephone of the summary medical results in November, 1985.

III. BACKGROUND

Siouxpreme Egg Products is a separate corporation formed by Sonstegard Foods, Inc. of Howard Lake, Minnesota. It processes raw eggs into egg yolk powder and liquid egg white. All of the products produced by the plant carry the USDA inspected label, which means that USDA inspectors are in the plant at all times checking for compliance with USDA guidelines. Siouxpreme Eggs started operations in 1981 in an old structure that was previously used to process and spray dry dairy cream. The facility contains a total of 35,000 $ft.^2$ under roof, with 10,000 $ft.^2$ of the space utilized as warehouse. A schematic layout of the plant is shown in Figure 1. No ventilation, except to maintain comfort, is provided except in the egg breaking room where filtered air is supplied to the room to maintain positive pressure in accordance with USDA guidelines.

Operations at the plant are structured to be in compliance with USDA guidelines. The first shift reports to work at 7:00 a.m. and works continually until 12:00 noon. At 12:00 noon, personnel operating the six egg washing and breaking machines take a 30-minute break while the machines are partially disassembled and cleaned. Fresh wash water and detergent are added to the machines, and floors and surfaces are washed down and sprayed with a sanitizer. Equipment that transfers the liquid egg product is also disassembled and cleaned. These operators then report back to the machines at 12:30 for another five-hour period. The first shift ends at 5:30 p.m., and the machines and areas are again washed down for a new crew that reports for work as the second shift at 6:00 p.m. This shift, mostly college students, operates for only one five-hour cycle ending at 11:00 p.m. The third shift reports at 11:00 p.m. and works until 6:00 a.m., completely disassembling and cleaning the machines and all other areas. Approximately 60-70 people work the first shift in the following job categories:

1. Office and supervisory staff (7) - Provide supervision to staff and overall plant management.

2. Forklift operators (2) - Unload trucks and move eggs into washing area.

3. Loaders (12-14) - Load eggs from pallets and boxes onto machines.

4. Operators and candling (6-8) - Sort and inspect eggs from washer. Work 45 minutes at station and 15 minutes at other duties away from station.

5. Breaking operators (6-8) - Operate breaking machine, making decisions on each egg, separating it into yolk, white or whole egg if separation not complete. Work 45 minutes at station and 15 minutes washing down breaking room area.

6. Inedible egg cleanup (1) - Gather up broken and waste eggs for separation of shell and liquid product for disposal.

7. Drying Operators (2) - Operate dryer and package product.

8. Cooling and pasteurizing operators (2) - Operate and monitor cooling and pasteurizing systems. Also blend additives into liquid streams before drying.

9. Flat washing (1) - Wash egg separators or flats before they are returned to egg supplier.

10. Maintenance (4) - Maintain and repair all equipment and facilities.

11. Laborers (6) - Miscellaneous duties in all areas of plant, including backup slots on process lines.

12. Cleanup crew - Selected workers who may have other duties during the regular shift.

The second shift utilizes a full complement of operational people, with a reduction in supervisory, labor, and maintenance staff. The employees at the plant are not represented by a union. The workforce has very little turnover. The plant manager has the responsibility for the occupational safety and health program for the plant. No uniforms are required in the plant, although employees are encouraged to wear rubber boots because of the constant presence of water on the floors. Workers in the breaking room must wear hair nets, and females are not allowed to wear any makeup because of USDA regulations. No pre-employment physicals are required of new employees, and traumatic injuries are referred to a local clinic. Insurance protection is provided for the plant by St. Paul Insurance of Des Moines, Iowa. They have made some site visits to the plant as a service to the plant owners.

IV. PROCESS DESCRIPTION

On the average, approximately 1.5 million eggs are processed each working day. The six semi-trailer loads are off-loaded from the loading dock area to the whole egg-in-shell warehouse area using propane-powered forklifts. The eggs come from a variety of sources, but most come from cage laying operations in Texas, Arkansas and Missouri. The eggs also are a variety of grades since most would not grade out for supermarket use. Some are also dirty with a variety of material, and others are cracked or in bad condition because they have not been refrigerated. From the warehouse, the eggs are moved on the pallets to the loading and washing room for transfer by hand to egg washing machines. Thirty-six eggs (6X6 format) on separators or flats are examined and placed on the loading chute. Eggs that are cracked or broken are removed by hand and placed in the inedible tray, and ones that are overly dirty but usable are sorted onto another flat and sent to the pre-wash area. The 36 eggs from each flat are picked up by a series of suction cups on an arm and transferred over to the conveyor going into the washing tunnel. The eggs travel through the washer where they are spray washed with a mixture of detergent compound water. The detergent in use during the study was "Best Eggs-Plus" (mono(trichloro)-tetra-(mono potassium) dichloro-penta-s-triazone and anhydrous sodium metasilicate). The

wash water is recirculated continually during the five-hour work cycle, and the solution is changed during the 30-minute cleanup period. After passing through a clean water rinse, the eggs are sprayed with a sanitizer containing iodine as the active ingredient. The brand name of the rinse sanitizer was "Bac-Stop" (Butoxy monoether of polyoxypropylene-polyoxyethylene glycol-Iodine complex (providing 1.75% titratable iodine)). Chlorine was used previously, but this was thought to be the source of the employees' complaint; so it was changed. The eggs then pass over the candling table where they are again examined. Dirty eggs are returned to the pre-wash area or sent through the washer again. Cracked or broken eggs, as well as those with visible interior spots, are thrown in the "non-edible" container.

The flats or egg separator are returned to the flat washing area for cleaning and drying prior to being returned to the egg suppliers. The flats are washed with a product called "Simbol". "Simbol" contains sodium hydroxide and chlorinated isocyanates.

The washed and candled eggs pass into the adjacent breaking room on the same continuous conveyor. The eggs fall into a continuous chain that grips the egg, holds and separates the shell and drops the contents into a separating cup. As the cup passes by, the operator makes a decision on the thoroughness of the break. If the separation of yolk and white is clean, the operator lets the cup pass by, and the resultant products are egg yolks and egg whites. If the separation is not good or the yolk is broken, the operator must trip each cup which sends the whole egg product into another system. Most of the egg whites are pumped to refrigerated storage where they are eventually loaded into bulk trucks for transport to other users. The egg yolk and whole eggs are pasteurized and refrigerated before they are sent to the drying room.

Liquid whole eggs or yolks are pumped to the drying area. Additives such as sugar, powdered milk, corn syrup, salt, soybean oil, and Zeolex 7 are added directly to the stream flow before it reaches the high pressure spray pump. The liquid is pumped through four nozzles into the large air drying oven. The water is evaporated, and the dryed product falls to the floor of the dryer where it is moved by chain and bar conveyor to a screw conveyor on one side of the oven. The dried material is picked up by vacuum and transported overhead to a cyclone separator. The product is removed and passed down to a sifter in the packaging room. The 12 or so products that can be produced are then weighed out into packages and sealed for storage in the warehouse prior to shipment.

V. METHODS

A. ENVIRONMENTAL

On August 14 and 15, 1985, air sampling was conducted throughout the plant. The sampling focused on the three most apparent agents in the workplace, including: (1) Halogen ions (chloride and iodide); (2) acid gases (HCl and H_2SO_4); and (3) egg dust. Twenty-two day shift area samples using midget impingers containing $NaHCO_3$ were collected from the transfer room, flatwashing room and the office area during the two work days. The volume of air sampled was approximately one-half cubic meter of air. Separate aliquots of these samples were analyzed, using a Dionex Model 2010i ion chromatograph. An AS-4 anion separator was utilized for chloride analysis, and the AS5 anion separator for iodide analysis. One personal sample using an impinger was collected from a machine operator in the transfer room and analyzed for chloride and iodide.

Two air samples for acid gases were collected from the transfer room adjacent to machines #2 and #5 only on day 2. These samples were collected using a silica gel solid sorbent tube and analyzed according to NIOSH Method #7903, using a Dionex 2010i ion chromatograph and a conductivity detector. Chloride and sulfate ions served as the quantitative indicators of the presence of hydrochloric acid (HCL) and sulfuric acid (H_2SO4).

Two total dust air samples and four respirable dust air samples were collected in the sifter and packaging room during the day shift on both days. The samples were collected on tared filters, and the quantity of dust present was determined gravimetrically. One bulk dust sample was analyzed for total protein[21] and amino acid profile.[22] Five Hi-Vol air samples were analyzed for amino acid profile.

Because of what seemed to be excessive noise levels that were experienced during the initial survey, noise measurements were taken throughout the plant, utilizing a hand-held General Radio GR1982 Precision Sound-Level Meter and Analyzer. Levels were obtained during various stages of the work cycle to gain some insight into possible sources of the noise, as well as to assist in the development of meaningful noise abatement recommendations.

B. MEDICAL

Ninety-one employees of Siouxpreme Eggs completed a questionnaire, which elicited the following information: demographics, occupational history, personal habits, past medical history, and symptoms suggestive of asthma. Respondents were characterized into two groups, based upon responses to three questions:

1. Within the past month, has your breathing sounded wheezy or whistling?

2. Within the past month, have you had episodes of shortness of breath?

3. Withing the past month, has your chest ever felt "tight"?

The two groups were:

Group 1: Responded "yes" to at least one of questions 1, 2, and 3.

Group 2: Responded "no" to all three questions.

All group 1 respondants, plus a like number of group 2 respondents, were asked to participate in a follow-up medical evaluation, the intent of which was to determine what the symptoms represented (such as asthma, for instance), and if the symptoms might reasonably be attributed to workplace exposures.

The follow-up evaluation consisted of:

1. an assessment of pulmonary function by measurement of forced vital capacity and one-second forced expiratory volume (FVC and FEV1),

2. an assessment of respiratory airways reactivity by serial measurements over seven days of peak expiratory flow rates,

3. an assessment of allergy to egg proteins by the administration of 9 prick skin tests and the measurement of IgE and IgG antibody titers to 8 egg proteins,

4. an assessment of atopy (familial predisposition to develop allergic responses) by clinical history and by administration of 5 prick skin tests to common allergens, and

5. the taking of a clinical history and performing of a focused physical examination by a physician, trained in internal medicine and board-certified in occupational medicine. The physician was kept unaware, at the time of his examination, of the results from the questionnaire, pulmonary function tests, peak flow determinations, and allergy tests.

VI. EVALUATION CRITERIA

A. ENVIRONMENTAL

As a guide to the evaluation of the hazards posed by workplace exposures, NIOSH field staff employ environmental evaluation criteria for assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to 10 hours per day, 40 hours per week, for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy).

In addition, some hazardous substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled at the level set by the evaluation criterion. These combined effects are often not considered in the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes and, thus, potentially increase the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent become available.

The primary sources of environmental evaluation criteria for the workplace are: 1) NIOSH Criteria Documents and recommendations, 2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLV's), and 3) the U.S. Department of Labor (OSHA) occupational health standards. Often, the NIOSH recommendations and ACGIH TLV's are lower than the corresponding OSHA standards. The OSHA standards also may be required to take into account the feasibility of controlling exposures in various industries where the agents are used; the NIOSH-recommended standards, by contrast, are based solely on concerns relating to the prevention of occupational disease. In evaluating the exposure levels and the recommendations for reducing these levels

found in this report, it should be noted that industry is legally required to meet those levels specified by an OSHA standard.

A time-weighted average (TWA) exposure refers to the average airborne concentration of a substance during a normal 8- to 10-hour workday. Some substances have recommended short-term exposure limits or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from high short-term exposures.

The following occupational exposure limits have some application to understanding the significance of the air sampling results:

Occupational Exposure Limit

Substance	OSHA[1]	ACGIH[2]	NIOSH[3]
Chlorine	3 mg/m^3(c)	3 mg/m^3	3 mg/m^3
Iodine	1 mg/m^3(c)	1 mg/m^3(c)	1 mg/m^3
HCl	7 mg/m^3(c)	7 mg/m^3(c)	7 mg/m^3
H_2SO_4	1 mg/m^3	1 mg/m^3	1 mg/m^3
Total Dust	15 mg/m^3	10 mg/m^3	
Respirable Dust	5 mg/m^3	5 mg/m^3	

(c) = ceiling concentration

The dust limits are directly applicable to evaluating the significance of the air sampling results in the sifter and packaging room areas.

The other four chemical standards can at best be called indicators. The sampling and analytical methods utilized in this study were the ones recommended for use when sampling the air for the four contaminants in a gaseous state. After the contaminants are collected in a liquid or sorbent media, a specific ion in the desorption solution is quantified. The gaseous concentration is then calculated, assuming that the detected ion is representative of the total molecule. However, during this study the detection of specific ions cannot be used to calculate back to gas

concentration because the specific ions are part of a different molecule. Since there are no standards for halogen organic complexes like those used in this plant, a straight-forward interpretation of the results is not feasible.

A worst case approach would be to consider that all of the contaminants were gaseous parts of the gaseous molecule and that their effects are additive. This concept has some merit in that the four chemicals all have been shown to demonstrate irritant-type symptoms. This approach is also suggested in the ACGIH Threshold Limit Values booklet for 1985-86. This method utilizes the following formula:

$$\frac{C1}{T_1} + \frac{C2}{T_2} + \frac{C3}{T_3} + \frac{C4}{T_4} = 1$$

where C_1 = concentration of chemical one
where T_2 = TLV for chemical

As the formula implies, if the sum exceeds one, a problem may exist.

Exposure to intense noise causes hearing losses which may be temporary, permanent, or a combination of the two. These impairments are reflected by elevated thresholds of audibility for discreet frequency sounds, with the increase in decibels (dB) required to hear such sounds being used as a measure of the loss. Temporary hearing losses, also called auditory fatigue, represent threshold losses which are recoverable after a period of time away from the noise. Such losses may occur after only a few minutes of exposure to intense noise. With prolonged and repeated exposures (months or years) to the same noise level, there may be only partial recovery of the threshold losses, the residual loss being indicative of a developing permanent hearing impairment. There is abundant epidemiological and laboratory evidence that protracted noise exposure above 90 decibels (dBA) causes hearing loss in a portion of the exposed population.

OSHA's existing standard for occupational exposure to noise (29 CFR 1910.95) specifies a maximum permissible noise exposure level of 90 dBA for a duration of eight hours, with higher levels allowed for shorter durations. NIOSH, in its Criteria for a Recommended Standard, proposed a limit of 5 dBA less than the OSHA standard. Time-weight average noise limits as a function of exposure duration are shown as follows:

Duration of Exposure (hours/day)	Sound Level, dBA NIOSH	OSHA
16	80	--
8	85	90
4	90	95
2	95	100
1	100	105
1/2	105	110
1/4	110	115*
1/8	115	--
		140 (dB)**

*No exposure to continuous noise above 115 dBA
**No exposure to impact or impulse noise above 140 dB peak sound pressure level (SPL)

B. MEDICAL

The American Thoracic Society has defined asthma as "a disease characterized by the increased responsiveness of the trachea and bronchi to various stimuli and manifested by widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy."[4] Scadding defines asthma as "a disease characterized by wide variations over short periods of time in resistance to flow in intrapulmonary airways."[5] From either definition, it is clear that the key concepts are "widespread narrowing of the airways" and changes in "resistance to (air) flow", "changes in severity", and "various stimuli".

In these definitions, symptoms are not mentioned. However, the seeking of a medical diagnosis clearly is prompted by an individual's symptoms. Asthma is usually diagnosed in a person for whom episodes of wheezing and shortness of breath, with demonstrable increases in resistance to airflow in the pulmonary airways, are interspersed between symptom-free (or relatively symptom-free) intervals. Thus, we have taken as a case-definition of asthma, any participant with symptoms of episodic wheezing and/or shortness of breath, who has demonstrable physiologically significant fluctuations in airflow resistance. We have determined the presence of the latter by serial measurements of peak expiratory flow rate (PEFR) over a seven day period. PEFRs vary throughout the day in normal persons. However, asthmatic persons are said "usually to show a difference of at least 15%

between mean morning and evening values."[6] Along these lines, we have taken as evidence of a physiologically significant variation in airways resistance, a 20% difference on any one day's PEFR measurements, comparing maximum and minimum values. For example, in an individual whose maximum and minimum PEFRs on any particular day were 510 and 330 liters per second, respectively, then the variation in PEFR on that day would be (510-330)/510=35.2%, and the individual would be taken to have evidence of physiologically significant (and reversible) airways obstruction on that day. If at the time of the decrement in PEFR the individual also had symptoms of wheezing and/or shortness of breath, then he/she would be classified as a case of asthma.

This definition of a "case" of asthma does not in itself impute cause. Neither do the definitions given above by the American Thoracic Society and by Scadding. Indeed, the "various stimuli" of the American Thoracic Society's definition are wide-ranging, and consist only in part of airborne allergens. To impute cause to the asthma, we must have additional evidence of some type. To impute an occupational cause to the asthma, that evidence must include a compatible temporal pattern to the asthmatic attacks, or evidence of allergy to a substance found in the workplace. We have taken as evidence of a compatible pattern, symptoms of wheezing and/or shortness of breath that occur in association with workplace exposures, that abate upon leaving the worksite, that are less frequent (preferably non-existent) while at home, and that are less frequent (preferably non-existent) on days off and on vacation. We take as evidence of allergy, positive skin prick tests to egg components, and/or elevated IgE antibody titers to egg proteins by RAST.

<u>In summary</u>, for the reasons outlined above, we define a case of asthma as an individual who has a 20% or greater decrement in PEFR on any one day, with symptoms of wheezing and/or shortness of breath. We impute an occupational etiology if the person has at least one positive skin test to egg protein, and/or elevated IgE titers to egg proteins. The occupational etiology is reinforced by the observation that the asthma-compatible symptoms occur temporally in association with workplace exposures, abate upon leaving the worksite, are less frequent (or non-existent) while at home, and are less frequent (preferably non-existent) on days off-work and on vacation.

VII. RESULTS

A. ENVIRONMENTAL

The air sampling results for chloride and iodide utilizing impingers are shown in TABLE I. The 12 area sample results for chloride ranged from detected but not quantified to 24.3 ugm/m^3 during the two-day sampling period at the six sampling stations. Three results were detected but not quantified, while eight samples had quantifiable levels of chloride. One sample was destroyed during the collection period. The same 12 samples for iodide ions ranged from none detected to 45.4 ugm/m^3. Seven results showed no detectable quantities of iodide and all showed a detected but not quantified level. Therefore, only three quantifiable levels of iodide were found. One personal sample was collected on day 2 that showed a chloride result of 12.9 ugm/m^3 and an iodide result of 20.7 ugm/m^3.

The air sampling results for acid gases are shown in TABLE II. The results for HCl and H_2SO_4 were both determined using the same tube. Loading machine #2 showed 21.7 ugm/m^3 of HCl and 90.1 ugm/m^3 of H_2SO_4 on day 1. The concentrations by loading machine #5 were 5.6 ugm/m^3 and 36.5 ugm/m^3, respectively. The HCl and H_2SO_4 levels were quantified by looking at chloride and sulfate.

The air sampling dust results are shown in TABLE III. Personal total dust results in the sifting and packaging room were 12.8 mg/m^3 and 7.3 mg/m^3 on the one worker during the two days sampled. Area total and respirable dust results were 1.2 mg/m^3 and .94 mg/m^3 total and .07 mg/m^3 and .03 mg/m^3 respirable. The egg dust is very light and subject to high static charges. Therefore, the reliability of these results may be questionable.

The bulk dust sample was determined to be 50.5 percent protein. The amino acid analysis of 3 of 5 Hi-Vol filter samples resembled the profile of the bulk dust sample, and resembled reference standards for egg yolk protein.[23] Two of 5 Hi-Vol smaples contained insufficient material for analysis.

The noise measurements are shown in TABLE IV. They range from a high of 98 dBA in the sifter powder room to 85 dBA in the pick and prewash area. Six of the nine measurements exceeded 90 dBA. Octave band measurements were made in three areas, and the results are shown in TABLE IV also.

B. MEDICAL

Ninety-one persons completed the self-administered questionnaire. Sixty-eight answered "no" to all three questions, and were classified as belonging to group 2. Twenty-three responded "yes" to at least one question, and were classified as belonging to group 1. Thirteen of group 1 respondents, and 18 of group 2 respondents, participated in the follow-up evaluation. Overall, a total of 31 Siouxpreme Egg employees were so evaluated. Not every participant underwent the full set of examinations.

Results of the follow-up evaluation are summarized in TABLE V. Based upon clinical history and physical examination alone, and without knowledge of the pulmonary function tests, peak expiratory flow rates, or allergy tests, the examining physician diagnosed possible occupational or non-occupational asthma in 12 of 31 participants (ID nos. 04, 08, 09, 10, 12, 13, 19, 23, 24, 25, 28, 30). As summarized in TABLE VII, when the additional information was available, the diagnostic impression was modified in only three participants (ID nos. 10, 14, 19). The only significant reclassification of diagnostic impression occurred for ID no. 10, whose diagnosis was changed <u>from</u> irritant respiratory symptoms vs. non-occupational asthma, <u>to</u> occupational asthma. The reasons for each modification of diagnostic impression are given in TABLE VII.

Six of thirty-one participants (ID nos. 09, 10, 12, 13, 14, 25) had at least one day of peak expiratory flow tests (PEFR), on which the difference between minimum and maximum recorded peak expiratory flow exceeded 20 percent of the maximum. Five of these six had concomitant symptoms of cough, wheezing, chest tightness, and/or shortness of breath. One of the six (ID no. 14) was asymptomatic at all times. The five persons with a peak expiratory flow rate variability of 20 percent or more, with concomitant symptoms, were diagnosed to have asthma (ID nos. 09, 10, 12, 13, 25). Four of these five had at least <u>two</u> (not just one, as suggested above under our diagnostic criteria) positive skin tests to egg proteins <u>and</u> at least <u>two</u> (not just one) elevated RAST antibody titers to egg proteins (ID nos. 09, 10, 12, 13). The fifth (ID no. 25) had two positive skin tests to egg proteins. One of these five (ID no. 12) had elevated IgE levels overall. IgE levels in the other four were normal. Thus, all five participants who were symptomatic at the time of their documented decrements in peak expiratory flow, had evidence of reaginic (IgE) allergy to egg proteins. In addition, each of these five reported that their symptoms followed activities at work, while performing these activities or within one hour of commencing those activities, that they less frequently occurred

away from work, and that they less frequently (2 of 5) or never (3 of 5) occurred while on vacation. We concluded that these five individuals (ID nos. 09, 10, 12, 13, 25) had occupational asthma, from workplace exposure to egg proteins.

To reinforce this diagnosis, based upon PEFR measurements (plus symptoms), we have plotted the daily peak flow rates for each case of asthma. (See figures at end of report. Note that the vertical scale on each graph is not the same.) On each figure is a plot of the maximum and minimum peak flow measurements by day (labeled along the x-axis from 1 to 7). At the bottom we have written-in whether the case reported working with eggs on that day, whether the person was taking any medication, and what the PEFR variation was on that day:

Refer to the figure for subject ID no. 5009 (ID no. 09 in tables 1-3). On each day while he/she reported working with eggs, the peak flow variation was a minimum of 23.5%. On the two days during which he/she reported not working with eggs, the peak flow variation is reduced to 5.7 and 9.1 percent. On three days on which he/she had a PEFR variability of greater than 20%, he/she also took Primatene by inhalation. This person has a pattern of PEFR variation diagnostic of occupational asthma.

Refer to the figure for subject ID no. 5025 (ID no. 25 in tables 1-3). On only one day did he/she have PEFR variability greater than 20%. This was on day 2, when he/she was awakened at 4:30 A.M. with cough, chest tightness, shortness of breath, and wheezing, and a significant PEFR decrement. This is called a delayed-onset asthmatic reaction, and is not infrequently seen among occupational asthmatics.

Refer to the figures for ID nos. 5010, 5012, and 5013 (ID nos. 10, 12, and 13 in tables 1-3). They each had at least two days of PEFR measurements that showed variability of at least 20%. ID nos. 5010 and 5012 were taking on 6 of 7 days a theophylline preparation. ID no. 5012 was also taking a beta-2 agonist (Proventyl). Despite these medications, PEFR variability diagnostic of asthma was observed.

Three of the five occupational asthmatics were non-smokers (ID nos. 10, 13, 25). The other two both had less than 5 pack-year smoking histories. One of the five (ID no. 10) had an atopic history, and was noted as well to have a positive skin test to ragweed.

The five occupational asthmatics reported working between 13 and 48 months at the plant. Two had held jobs in the powder room, which entailed significant dust exposure. The other three reported work in the transfer and the break room, where dust exposure was less than in the powder room. The two occupational asthmatics exposed to the higher dust levels of the powder room noticed onset of symptoms associated with work within 2 months of hire. The three who worked in the less dusty jobs reported onset of symptoms 6 months, 36 months, and between 4 and 16 months (this person gave year but not month of onset of symptoms, hence the 12 month range cited) after hire.

All diagnosed asthmatics were group 1 participants (i.e., symptomatic at the time of the original questionnaire, of chest tightness, wheezing, or shortness of breath within the preceding month). None of the group 2 participants were diagnosed to have asthma. Of the seven participants (in addition to those five diagnosed as having occupational asthma) in whom the examining physician considered (in the absence of knowledge about the additional examination results) occupational or non-occupational asthma as possible diagnoses, one (ID no. 24) was felt to have occupational asthma, one (ID no. 04) was felt to have either occupational or non-occupational asthma, two (ID nos. 19, 30) were felt to have either occupational asthma or irritant respiratory symptoms, and three (ID nos. 08, 23, 28) were felt to have either bronchitis or non-occupational asthma. Of the four in whom occupational asthma was considered to be a possibility (ID nos. 04, 19, 24, 30), only one had pulmonary function tests suggestive of airways obstruction (ID no. 24). Three had negative skin tests and RAST titers to egg proteins, while the fourth had negative skin tests, without RAST determinations. Of the three with bronchitis vs. non-occupational asthma as their probable diagnoses, one had pulmonary function test evidence of obstruction. This person was an 18 pack-year smoker.

VIII. DISCUSSION

A. ENVIRONMENTAL

Employees' exposure to any of the four chemical contaminants study were well below any recognized time-weighted average exposure guideline. The worst possible exposure scenario, considering simultaneous exposure to the four contaminants, can be calculated as follows from data in TABLES I and II:

	Exposure Conc.	Recommended Exposure Limit
Chloride	12.9 ugm/m^3	3 mg/m^3
Iodide	20.9	1 mg/m^3
HCl	21.7	7 mg/m^3
H_2SO_4	90.1	1 mg/m^3

$$\frac{.0129}{3} + \frac{.0209}{1} + \frac{.0217}{7} + \frac{.0901}{1} = \text{guidance value}$$

$$.004 + .021 + .003 + .090 = 0.118$$

If the guidance value exceeded one, the exposure would be considered potentially hazardous. Since it is well below this value, there is very little chance that the exposures will cause any health effect.

One of the personal dust exposure levels exceeded the ACGIH guideline of 10 mg/m^3 total dust. On the second day, the value was close to 10, and we would estimate that it exceeds 10 on most workdays when the packaging operation runs all day. The area respirable dust levels were very low, indicating that most of the dust is non-respirable. It is our judgment that a hazard exists in this area. At times, the workers wore disposable respirators in the area, but in the judgement of the NIOSH industrial hygienist, these afforded very little protection.

High noise levels were noted during the two-day survey. Several areas had levels exceeding the OSHA standard of 90 dBA. Based on the octave band analysis and some very simple observations, the major source of the noise appeared to be compressed air discharging and pumps. As indicated in TABLE IV, the sound pressure level in the breaking room was reduced to 86 dBA when the machines were shut down for cleaning.

When workers are exposed to sound levels exceeding the OSHA standard, feasible engineering or administrative controls should be implemented to reduce levels to permissible limits. OSHA has recently issued a hearing conservation amendment to its noise standard. For workers exposed at or above a TWA of 85 dBA, the amendment will require noise exposure monitoring, employee education, and audiometric testing. Reviews of audiograms have to be made by an audiologist or otolaryngologist or a qualified physician in their absence. Employees also must be notified of monitoring results within 21 days. Employee records must be kept by the employer for up to five years after termination of

employment. Finally, for those employees exposed to noise levels exceeding 90 dBA for eight hours and/or where audiometric testing results indicate a hearing loss, hearing protection must be worn.

B. MEDICAL

Chicken egg white is a common allergen, ingestion of which may provoke in atopic individuals pruritis (itching) and exacerbation of atopic dermatitis, rhinitis, urticaria (hives), angioedema, and bronchial asthma. Various proteins constitute approximately eleven percent of the total weight of an egg. There are approximately 40 different proteins, many of which have not been characterized [7] Thirteen proteins have been found to cause production of IgE antibodies in egg-allergic individuals.[8] Various investigators have classified ovalbumin,[8,9] ovomucoid,[8,9,10,11,12,13] and ovotransferrin,[8,9] as major allergens in egg white. Lysozyme appears to be of lesser importance.[8,10,12,13] Egg yolk also contains proteins that are antigenically related to proteins in egg white.[15]

Sensitivity to specific allergens may be associated with specific clinical manifestations and route of exposure. Egg-sensitive atopics who react with bronchial asthma from egg inhalation exhibit stronger crossed radioimmunoelectrophoresis reactions to ovomucoid and higher RAST values to egg white, than asymptomatic egg-sensitive (i.e., RAST positive) controls.[15]

Egg-sensitive patients tend to be more severely affected by atopic diseases and to suffer from more allergies than atopics sensitive to other allergens.[16,17,18] It has been suggested that egg allergy is associated with a tendency to high total IgE synthesis and liability to develop multiple allergies. In the series of studies cited immediately above, differences in severity of atopic dermatitis and frequency of allergic rhinitis between egg-allergic and non-egg-allergic atopics disappeared when groups were matched with respect to total IgE levels. However, the difference in severity of bronchial asthma was still present.

IgG antibodies to ovalbumin have been shown not to inhibit IgE binding (to ovalbumin), suggesting that "blocking" antibody is not of significance.[15]

We have demonstrated IgE mediated asthma related to occupational exposure to egg proteins among 5 workers at Siouxpreme Egg Products, Inc. These 5 represent a minimum estimate of the actual number of workers with occupational asthma or who will develop occupational asthma as a consequence of their exposures, since the participant sample consisted of 13 out of 23 symptomatic

(determined by questionnaire response) workers; and individuals who were symptomatic, or who were diagnosed as having possible or early occupational asthma by the examining physician, but who did not have immunologic or pulmonary function tests diagnostic of occupational asthma by our criteria, could well develop those changes at some time after our set of examinations. Four of five asthmatics were non-atopic. Therefore, we may reasonably conclude that these four were not necessarily familially pre-disposed to develop allergic reactions. That is, there appears to be no basis to state that they brought their allergy to their worksite, and work exposures merely brought out an underlying problem. (Workers who are atopic, but have no history of asthma, are said to be at increased risk of developing occupational asthma in some situations. This, at least, is the prevailing notion for workers exposed to enzyme detergents and laboratory animals.)

We have been able to find only one other report of allergy to inhaled egg protein.[19] In this report, 8 of 13 bakery workers who sprayed a 25% mixture of egg white and yolk in water on meat rolls, developed respiratory symptoms. Only 5 of 8 were reported to complain of shortness of breath. Four of 5 also complained of wheeze, chest tightness, and shortness of breath. Only one had pulmonary function test changes of reversible airways obstruction. Four of 8 symptomatics were also atopic, all of them with increased IgE levels. Five symptomatics had increased IgE titers to one or more egg proteins. In contrast to this report, we have found five symptomatic individuals with pulmonary function changes diagnostic of reversible airways obstruction; four out of five being non-atopic; all with evidence of IgE mediated allergic reactions to egg proteins; and four out of five having normal serum IgE levels.

IX. RECOMMENDATIONS

1. Every worker with asthma related to workplace exposure to egg protein should be offered a work assignment that will minimize inhalational egg exposure. Each such worker should be assessed by a physician conversant in the management of the asthmatic patient, and receive optimal therapy. (ID nos. 5009, 5013, and 5025 were on no medication except Primatene or Actifed, which is sub-optimal to ineffective therapy.) Since the asthma is IgE mediated, the managing physician might consider a trial of cromolyn, preceded by a beta-2 agonist by inhalation. Cromolyn inhibits the IgE mediated response, and may provide specific therapy for this problem. The managing physician should consult standard reference material, such as the Physician's Desk Reference, for indications, contraindications, and dosage.

2. Each worker who develops episodic wheezing and shortness of breath should be evaluated for workplace-related asthma. The diagnosis requires a compatible history, with documentation of reversible episodic airways obstruction. The mini-Wright's peak expiratory flow meter appears to provide useful information in this regard. If occupational asthma is diagnosed, recommendation 1 would then apply.

3. The diagnosis of occupational asthma should be considered in persons who are otherwise asymptomatic, but who are awakened in the evening by cough, shortness of breath, wheezing, or chest tightness. This may be a late or delayed asthmatic reaction, as evidenced by ID no. 5025, and should be evaluated. Again, the mini-Wright's peak expiratory flow meter appears to provide useful information in this regard.

4. There does not appear to be any basis from this study to recommend pre-employment testing for evidence of atopy, since 4 of 5 occupational asthmatics here were non-atopic. The supposition that the problem each worker has suffered is related to a predisposition to develop asthma, cannot be substantiated by this study.

5. This occupational problem requires further study. A re-evaluation of the symptomatic workers who were not diagnosed as definite cases of occupational asthma (these are ID nos. 04, 08, 19, 23, 24, 28, and 30), is desirable. An evaluation of the ten group 1 respondents who did not participate in the evaluation is also desirable, for those non-participants who are still employees of Siouxpreme Egg Products, Inc.

6. Persons in whom IgE mediated hypersensitivity reactions have been documented should not receive immunizations with vaccines grown in eggs. The vaccine most likely to be offered to an adult is the influenza vaccine. Yellow-fever vaccine is also manufactured in eggs. According to the Centers for Disease Control: "Although current influenza vaccines contain only a small quantity of egg protein, on rare occasions, vaccine can induce hypersensitivity reactions. Individuals with anaphylactic hypersensitivity to eggs should <u>not</u> be given influenza vaccine. Such persons include those who, on eating eggs, develop swelling of the lips or tongue or experience acute respiratory distress or collapse."[20] In addition, in our opinions, such persons also include those who, from inhalational exposure to egg protein, have developed evidence of occupational asthma, or other allergic response. In the event an egg-sensitive individual is judged to require

influenza vaccination, then that person's allergic sensitization should be brought to the attention of the physician, and the immunization should be carried out under the supervision of an allergist.

7. A hearing conservation program that conforms to all the requirements of the Occupational Safety and Health Administration should be implemented immediately.

8. Local exhaust ventilation should be provided in the sifter and packaging room, to control dust during filling operations. The discharge from a roof mounted exhaust fan should be away from all possible air inlets to prevent reentrainment of egg dust. This system should adequately protect workers from large quantities of egg dust, but will not protect the sensitized worker from exposure to low levels of egg dust.

9. Although the chloride and iodide ions in the egg washing area do not appear to be the cause of the asthmatic symptoms, the ventilation system from these machines could be connected directly to a roof mounted fan and not just to a vertical 4'x4' ventilation shaft. This would provide more positive removal of the decontamination mist.

References:

1. General Industry Occupational Safety and Health Standards (29CFR1910), OSHA 2206, March 1, 1983.

2. Threshold Limit Values for 1985-1986. American Conference of Governmental Industrial Hygienists, ISBN: 0-936712-61-9.

3. NIOSH/OSHA Pocket Guide to Chemical Hazards, NIOSH Publication 78-210, September, 1978.

4. American Thoracic Society. Chronic bronchitis, asthma, and pulmonary emphysema. A statement by the Committee on Diagnostic Standards for Nontuberculous Respiratory Disease. Am. Rev. Resp. Dis. 1962;85:762-768.

5. Scadding JG. Definition and clinical categories of asthma. In Asthma. THJ Clark and S Godfrey, eds. Chapman and Hall, London, 1983, pp. 1-11.

6. Rees H, ABC of asthma. Definition and diagnosis. Br. Med. J. 1984;288:1370-1372.

7. Vadehra DV, Nath KR. Eggs as a source of protein. C.R.C. Crit. Rev. Food Tech. 1973;193-309.

8. Langeland T. A clinical and immunological study of allergy to hen's egg white. III. Allergens in hen's egg white sutdied by cross-radioimmunoelectrophoresis. Allergy 1982;37:521-530.

9. Langeland T, Harbitz O. A clinical and immunological study of allergy to hen's egg white. V. Purification and identification of a major allergen (antigen 22) in hen's egg white. Allergy 1983;38:131-139.

10. Virtue CM, Whittig HJ. Allergenicity of egg white fractions as determined by histamine release from human lung tissue. Fed. Proc. Am. Soc. Exp. Biol. 1970;29:576.

11. Schloss MO. A case of allergy to common foods. Am. J. Dis. Child. 1912;3:341-362.

12. Bleumink E, Young E. Studies on the atopic allergens in hen's egg. I. Identification of the skin reactive fraction in egg white. Int. Arch. Allergy 1969;35:1-19.

13. Bleumink E, Young E. Studies on the atopic allergens in hen's egg. II. Further characterization of the skin reactive fraction in egg white: Immunoelectrophoretic studies. Int. Arch. Allergy 1971;40:72-88.

14. Langeland T. A clinical and immunological study of allergy to hen's egg white. VI. Occurrence of proteins cross-reacting with allergens in hen's egg white as studied in egg white from turkey, duck, goose, seagull, and in hen egg yolk, and hen and chicken sera and flesh. Allergy 1983;38:399-412.

15. Langeland T. A clinical and immunological study of allergy to hen's egg white. IV. Specific IgE-antibodies to individual allergens in hen's egg white related to clinical and immunological parameters in egg-allergic patients. Allergy 1983;38:493-500.

16. Langeland T. A clinical and immunological study of allergy to hen's egg white. I. A clinical study of egg allergy. Clinical Allergy 1983;13:371-382.

17. Buffum WP, Settipane GA. Prognosis of asthma in childhood. Am. J. Dis. Child. 1966;122:214.

23. Schur S, Hyde JS, Wypych JI. Egg white sensitivity and atopic eczema. J. Allergy Clin. Immunol. 1974;54:174.

19. Edwards JH, McConnochie K, Trotman DM, Collins G, Saunders MJ, Latham SM. Allergy to inhaled egg material. Clinical Allergy. 1983;13:427-432.

20. Centers for Disease Control. Recommendation of the Immunization Practices Advisory Committee (ACIP): Prevention and control of influenza. Morbidity and Mortality Weekly Report. 1985;34:266.

21. Official Methods of Analysis. Thirteenth edition. Method 2.057, AOAC, Washington, D.C., 1980.

22. Analytical Chemistry. 1958;30:1190-1206.

23. U.S.D.A. Handbook 8-1.

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Copies of this report are currently available upon request from NIOSH, Division of Standards Development and Technology Transfer, Publications Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), 5285 Port Royal, Springfield, Virginia 22161. Information regarding its availability through NTIS can be obtained from NIOSH Publications Office at the Cincinnati address. Copies of this report have been sent to:

1. Siouxpreme Egg Products, Inc., Sioux Center, Iowa
2. Requestor
3. NIOSH, Region VII
4. OSHA, Region VII
5. Grading Branch, Poultry Division, AMS, U.S. Department of Agriculture, Washington, D.C.

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

TABLE I

IMPINGER RESULTS

SIOUXPREME EGG PRODUCTS
HETA 84-163

Location	Day 1		Day 2	
	Chloride	Iodide	Chloride	Iodide
	ugm/m^3	ugm/^{3}	ugm/m^3	ugm/m^3
A. Loading Room				
Machine #2	4.98*	45.4	7.97	41.6
Machine #5	6.14	28.8	--	--
B. Breaking Room				
Machine #2	18.8	ND	10.4	ND
Machine #5	10.8	ND	9.9	ND
C. Flat Washing	7.27	(3.07*)	24.3	ND
D. Office Area	(3.41*)	ND	(3.14*)	ND
E. Personal Sample Loading Room	--	--	12.9	20.7

ND = None detected
* = Detected but not quantified
-- = No sample

TABLE II

ACID GASES

SIOUXPREME EGG PRODUCTS
HETA 84-163

	HCl*	H2SO4**
	ugm/m^3	ugm/m^3
Loading Machine #2	21.7	90.1
Loading Machine #5	5.6	36.5

*As chloride
**As sulfate

TABLE III

DUST RESULTS

SIOUXPREME EGG PRODUCTS
HETA 84-163

	Day 1	Day 2
	mg/m^3	mg/m^3
Personal Total Dust (10 mg/m^3)	12.8	7.3
Area Total Dust	1.2	.94
Area Respirable Dust (5 mg/m^3)	.07	.03

TABLE IV

NOISE MEASUREMENTS

SIOUXPREME EGG PRODUCTS
HETA 84-163

General Room Noise

Location	Sound Pressure Level (dBA)
Flat Washing	88
Loading Room	91
Pick and Prewash	85
Breaking Room	94
Hopper Loading Area	95
Drying Room by Air Handlers	94
Drying Room by High Pressure Pumps	90
Sifter & Powder Loading Room	98
Breaking Room with Machines Shut Down	86

Octave Band Analysis
(dB)

	Frequency									
Location	31.5	63	125	250	500	1000	2000	4K	8K	16K
Loading Room	80	85	82	86	85	87	85	82	78	72
Breaking Room	79	76	89	91	87	87	88	88	88	84
Sifter & Powder Loading	88	90	90	95	93	92	90	92	89	80

TABLE V
Summary of Clinical Data

ID	Atopy	Physician's Diagnosis	Pack-yrs of Smoking
01	N	Normal	0
02	Y	Normal	2.5
03	N	Normal	0
04	Y	Occupation asthma vs. Non-occupational asthma	5
05	Y	Normal	2.25
06	N	Normal	2.5
07	Y	Irritant respiratory sx.	0.5
08	Y	Bronchitis vs. Non-occupational asthma	
09	N	Occupation asthma	3.5
10	Y	Irritant respiratory sx. vs. Non-occupational asthma	0
11	N	Normal	0
12	N	Occupational asthma	4.8
13	N	Occupational asthma	0
14	N	Normal	15
15	Y	Coronary artery disease	60
16	N	Upper respiratory tract infection	9
17	N	Normal	0
18	Y	Normal	0

TABLE V (continued)

ID	Atopy	Physician's Diagnosis	Pack-yrs of Smoking
19	N	(Early) Occupational asthma vs. Irritant respiratory symptoms	1
20	N	Normal	0
21	Y	Normal	0
22	N	Normal	0
23	Y	Non-occupational asthma vs. Bronchitis	18
24	Y	Occupation asthma	0
25	N	Occupational asthma	0
26	N	Normal	0
27	N	Normal	0.25
28	Y	Non-occupational asthma vs. Bronchitis	5
29	Y	Normal	4.5
30	Y	Occupational asthma vs. Irritant respiratory symptoms	0
31	N	Normal	0

The "Physician Diagnosis" was the most probable diagnosis, determined from clinical history and examination alone at the time of the survey, without the knowledge of the results of pulmonary function tests, peak expiratory flow rates, skin tests, IgE or IgG determinations. Three diagnoses were modified, subsequent to knowledge of the additional data, as noted in Table 3.

TABLE VI
Summary of Clinical Data

ID	FEV1	FVC	FEV1/FVC	PEF Variability	Skin Test	RAST (IgE)	ELISA (IgG)
01	D	N	N	5.7 - 7.4	N	.	.
02	N	N	N	.	N	N	N
03	N	N	N	.	N	N	Whole egg Egg yolk Conalbumin Lysozyme Ovomucoid
04	N	N	N	2.1 - 9.6	N	.	.
05	N	N	N	1.8 - 11.1	Whole egg Conalbumin	N	Conalbumin Lysozyme
06	N	N	N	2.8 - 12.8	N	.	.
07	.	.	.	2.0 - 6.1	Whole egg	N	Whole egg Conalbumin Lysozyme
08	N	N	N	4.9 - 12.1	N	N	Whole egg Egg yolk Conalbumin Lysozyme Ovomucoid
09	D	N	D	5.6 - 45.0	Conalbumin Lysozyme	Conalbumin Egg white	Conalbumin Lysozyme

(continued)

KEY: N = Normal or Negative
D = Decreased
Y = Yes
. = missing, insufficient, or invalid data

TABLE VI (continued)
Summary of Clinical Data

ID	FEV1	FVC	FEV1/FVC	PEF Variability	Skin Test	RAST (IgE)	ELISA (IgG)
10	N	N	D	10.5 - 30.9	Whole egg	Whole egg	Whole egg
						Egg yolk	Egg yolk
						Conalbumin	Conalbumin
					Ovalbumin	Ovalbumin	Ovalbumin
					Lysozyme		Lysozyme
					Ovomucoid	Ovomucoid	Ovomucoid
					Whole egg		
						Egg white	
						Egg yolk	
							Human serum albumin
					Ragweed		
11	N	N	N	2.2 - 4.4	N	N	Whole egg
							Lysozyme
12*	D	D	D	32.5 - 51.7		Whole egg	Whole egg
						Egg yolk	
						Conalbumin	Conalbumin
						Ovalbumin	
					Lysozyme		Lysozyme
					Ovomucoid	Ovomucoid	
						Egg white	
						Egg yolk	
13	N	N	N	11.3 - 25.4		Whole egg	Whole egg
						Egg yolk	Egg yolk
					Conalbumin	Conalbumin	Conalbumin
						Ovalbumin	Ovalbumin
					Lysozyme	Lysozyme	Lysozyme
					Ovomucoid	Ovomucoid	Ovomucoid
					Whole egg		
						Egg white	
					Egg yolk	Egg white	

(continued)

* = Total IgE = 1000

KEY: N = Normal or Negative
D = Decreased
Y = Yes
. = missing, insufficient, or invalid data

TABLE VI (continued)
Summary of Clinical Data

ID	FEV1	FVC	FEV1/FVC	PEF Variability	Skin Test	RAST (IgE)	ELISA (IgG)
14	N	N	N	6.2 - 54.5	N	N	Egg yolk Lysozyme
15	D	N	D	0 - 6.3	N	N	N
16	N	N	N	5.7 - 16.4	N	.	.
17	N	N	N	0 - 6.3	N	.	.
18	N	N	N	0 - 4.8	N	N	Whole egg Egg yolk Conalbumin Lysozyme Ovomucoid
19	N	N	N	.	N	N	Lysozyme Human serum albumin
20	N	N	N	0 - 5.2	Ovomucoid	.	.
21	N	N	N	1.6 - 7.9	N	N	Conalbumin Lysozyme
22	N	N	N	.	N	N	Lysozyme
23	D	D	N	.	N	N	Lysozyme
24	D	D	N	.	N	N	Lysozyme Ovomucoid Egg white

(continued)

KEY: N = Normal or Negative
D = Decreased
Y = Yes
. = missing, insufficient, or invalid data

TABLE VI (continued)
Summary of Clinical Data

ID	FEV1	FVC	FEV1/FVC	PEF Variability	Skin Test	RAST (IgE)	ELISA (IgG)
25	N	N	N	4 - 21.2	Conalbumin Ovalbumin	N	Whole egg Egg yolk Conalbumin Lysozyme
26	N	N	N	3.2 - 8	Ragweed Dust mite	N	Whole egg Egg yolk Conalbumin Lysozyme
27	N	N	N	4.9 - 9.3	N	N	Conalbumin Lysozyme
28	N	N	N	1.7 - 3.7	N	N	Conalbumin Lysozyme
29	N	N	N	3.3 - 9.8	N	N	Lysozyme
30	N	N	N	0 - 6.4	N	N	Lysozyme
31	N	N	N	3.2 - 8.3	N	N	Whole egg Lysozyme

KEY: N = Normal or Negative
D = Decreased
Y = Yes
. = missing, insufficient, or invalid data

TABLE VII
Changes in Physician's Diagnoses

ID	Original* Impression	Modified Impression	Reason for modification
10	Irritant resp. sx. vs. NOc-Asthma	Occ-Asthma	Decreased FEV1/FVC, PEF variability suggestive of episodic airways obstruction, positive skin tests and RAST (IgE)
14	Normal	Normal vs. don't know	PEF variability suggestive of episodic airways obstruction, in absence of respiratory symptoms
19	(Early) Occ-asthma vs. Irritant resp. sx.	Irritant resp. sx. vs. (Early) Occ-asthma	No evidence for airways obstruction, negative skin tests and RAST (IgE)

* See table V

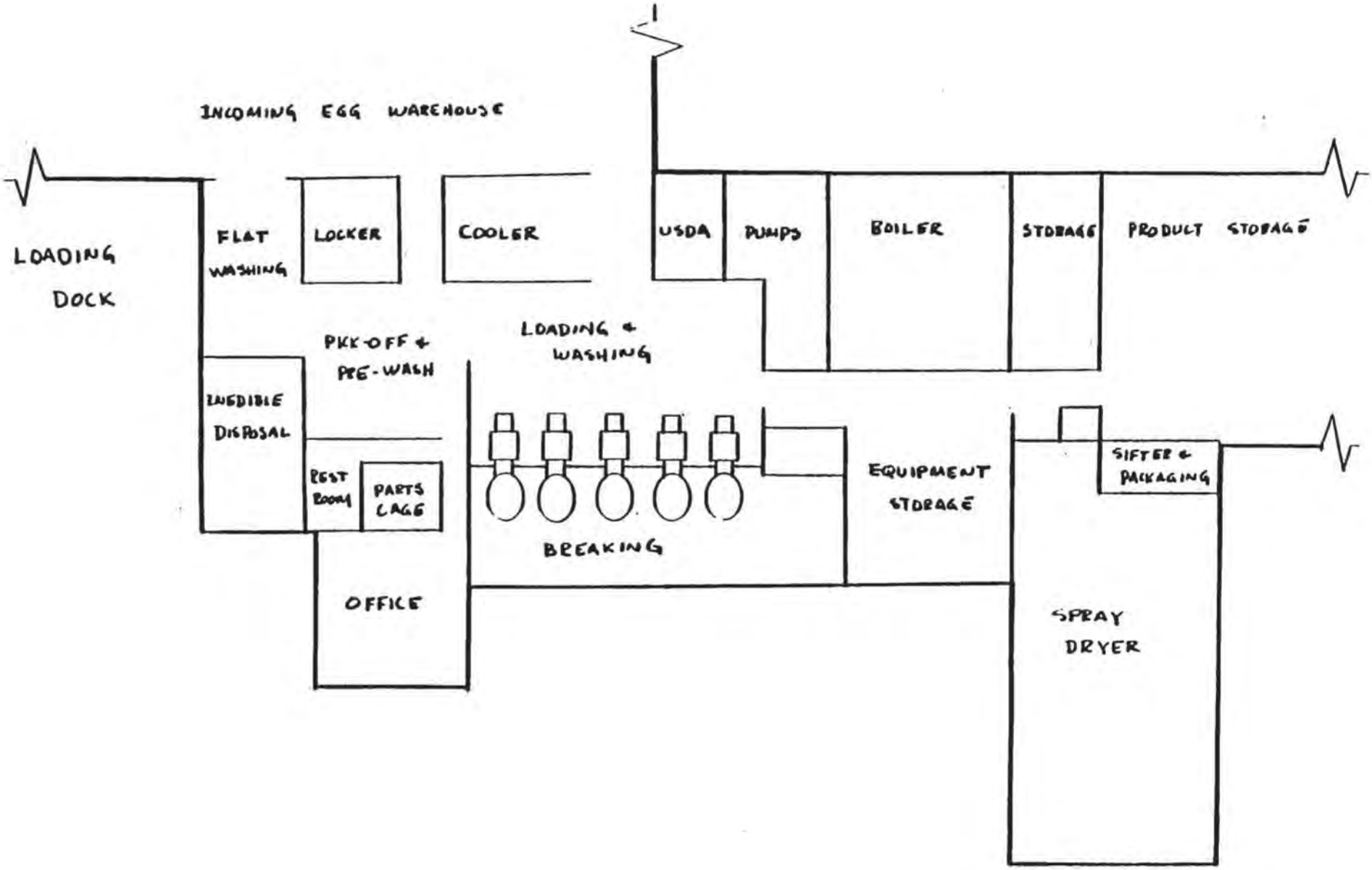
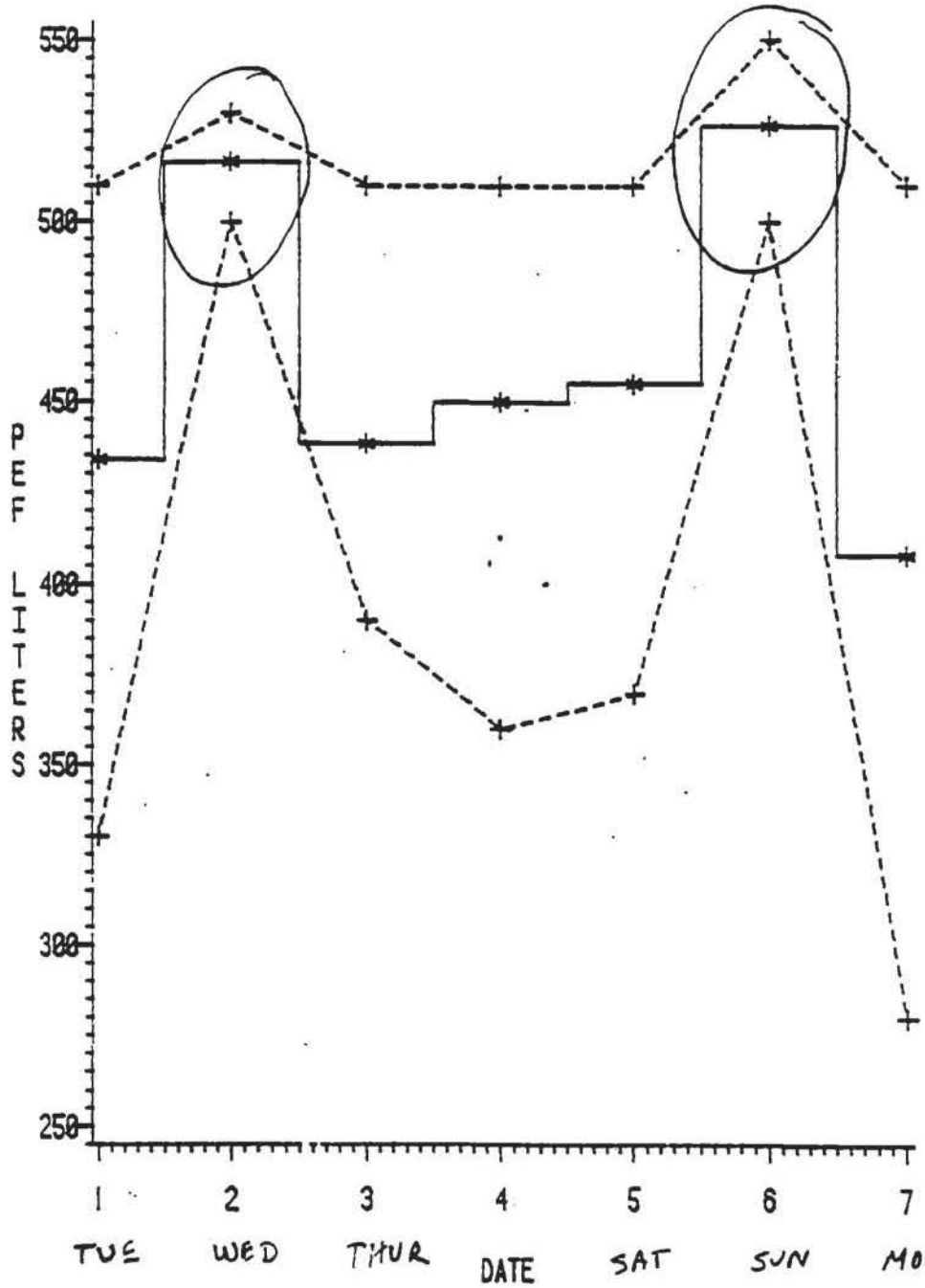


FIGURE 1 PLANT LAYOUT - SIOUXPREME EGG PRODUCTS

DAILY PEAK FLOW

SUBJECT ID NUMBER=5009



DID NOT WORK WITH EGG PROTEIN ON WED. OR SUN.

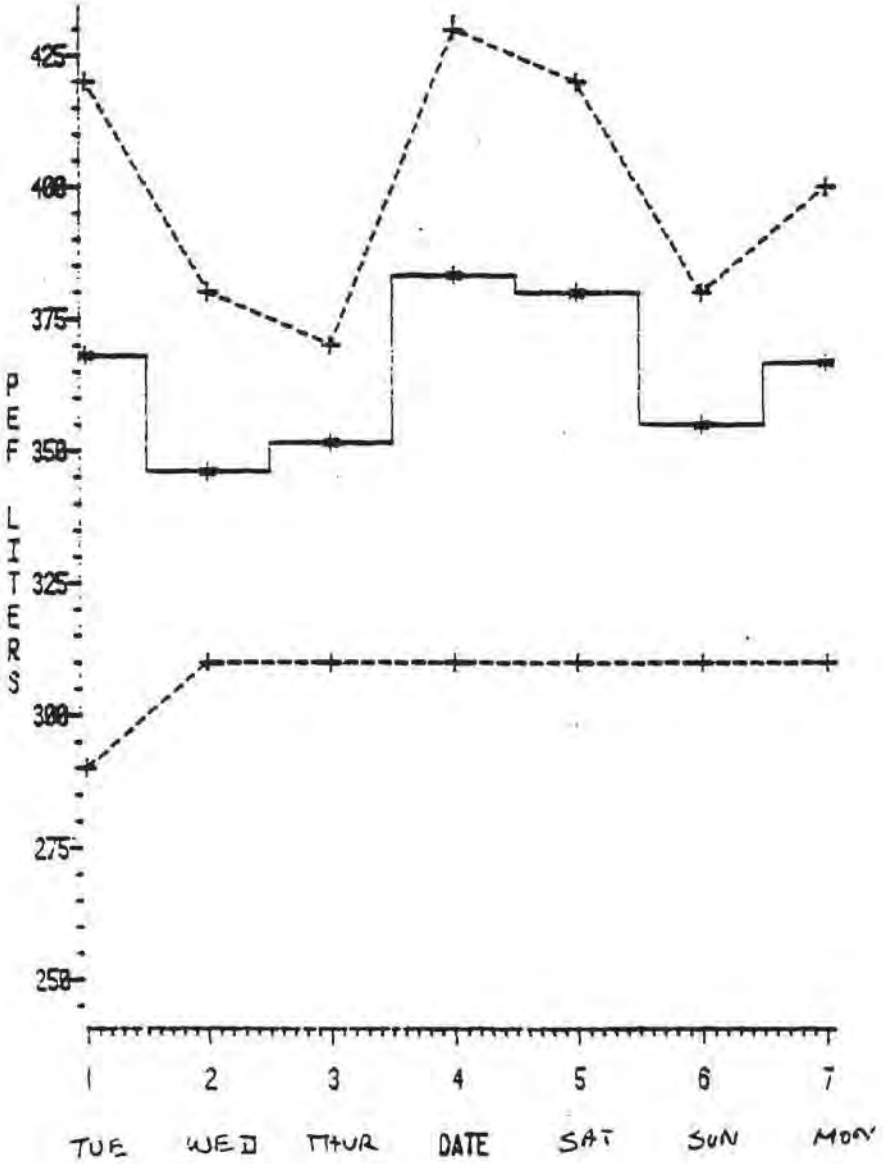
FOR MIN OR MAX FLOW

FOR MEAN FLOW

WORKED WITH EGGS?	Yes	No	Yes	Yes	Yes	No	Yes
MEDICATIONS	No	No	PRIMATENE	PRIMATENE	No	No	PRIMATENE
PEF VARIABILITY %	35.2	5.7	23.5	29.4	27.4	9.1	45.1

DAILY PEAK FLOW

SUBJECT ID NUMBER=5018

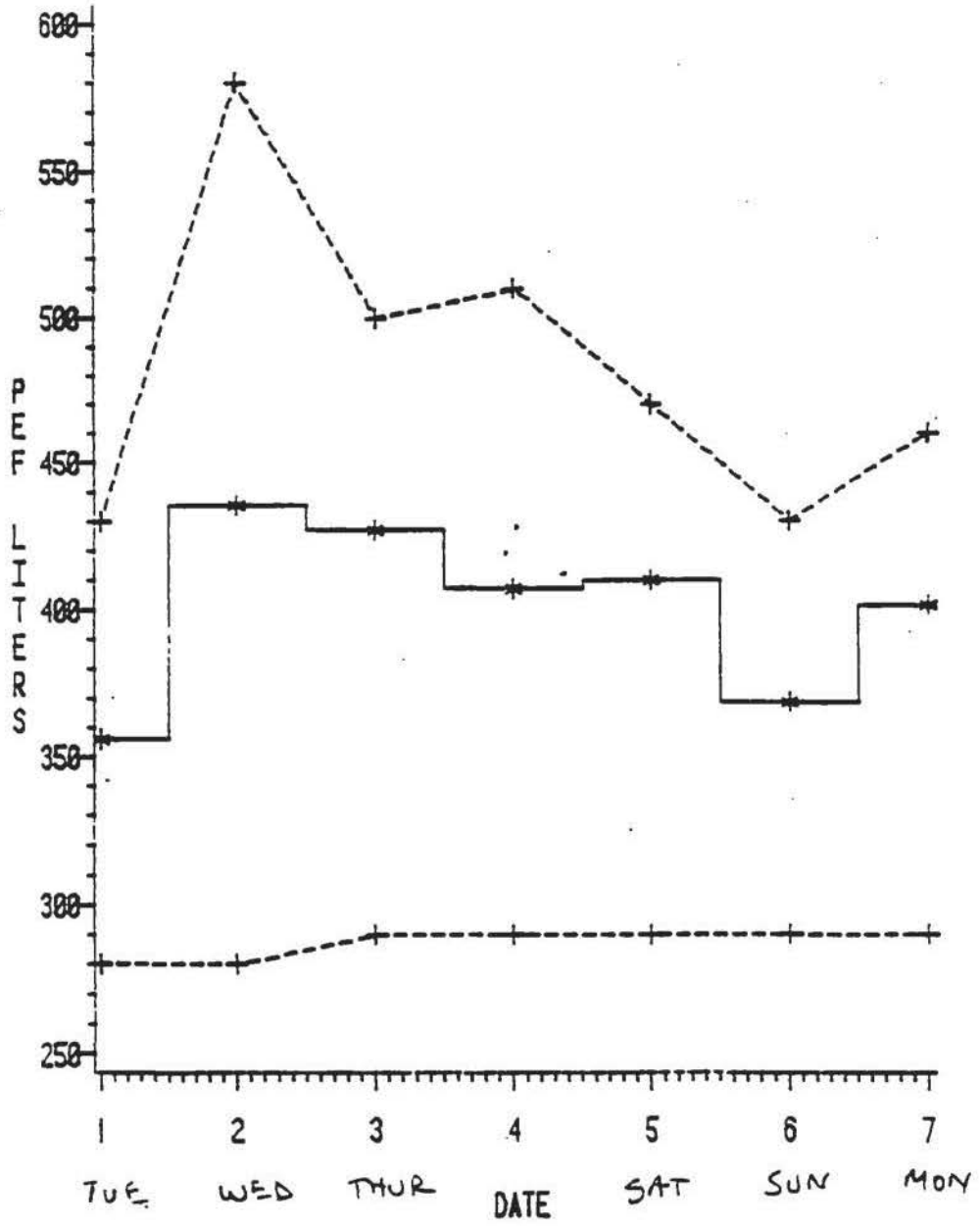


FOR MIN OR MAX FLOW
FOR MEAN FLOW

WORKED WITH EGGS?	YES	YES	YES	YES	No	No	No RESPONSE
MEDICATIONS	No	CONSTANT - T →					
PEF	31	18.4	15.8	28	26.2	18.4	22.5
VARIABILITY %							

DAILY PEAK FLOW

SUBJECT ID NUMBER=5012



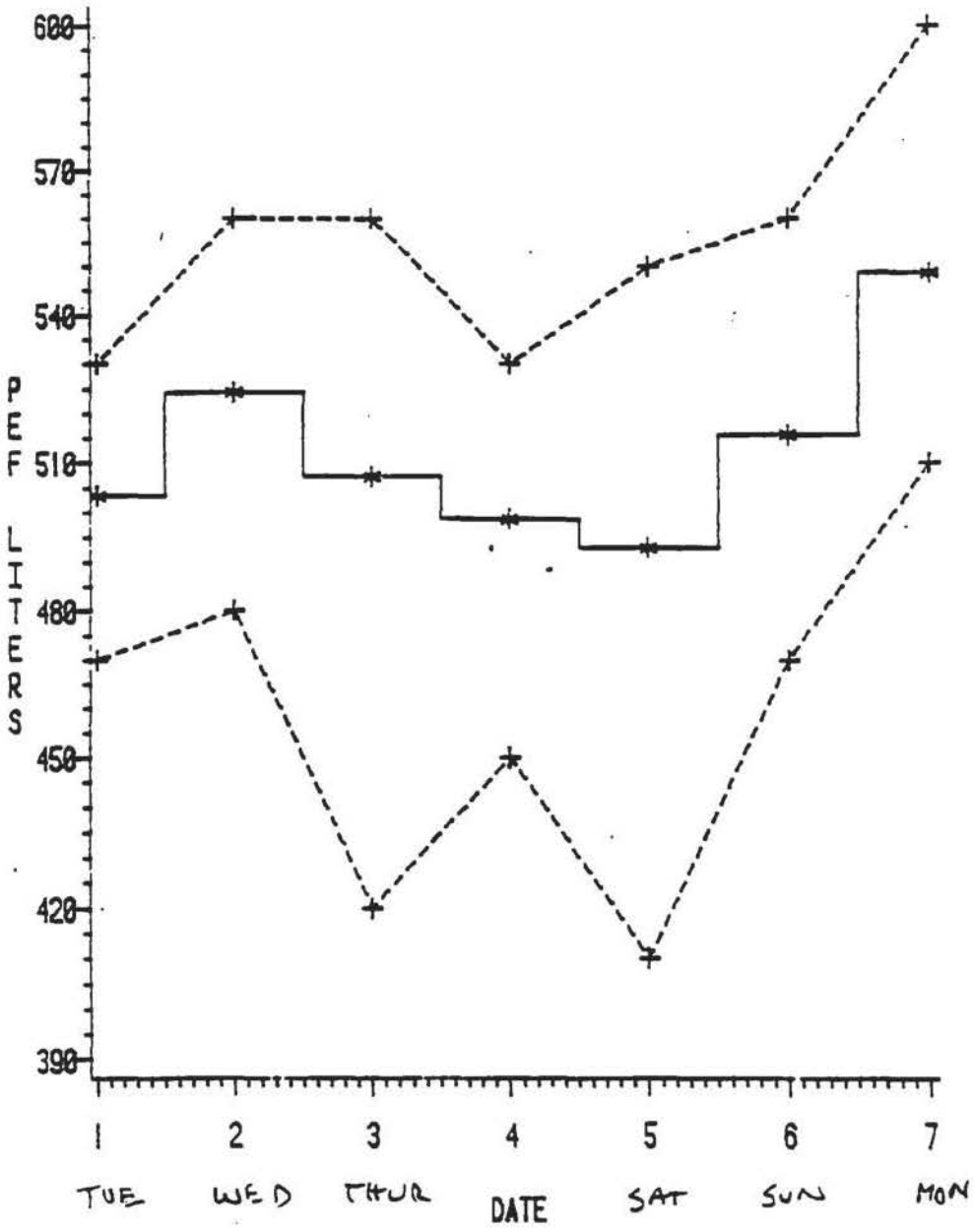
+——+ FOR MIN OR MAX FLOW

—— FOR MEAN FLOW

WORKED WITH EGGS?	YES	YES	YES	YES	YES	NO	YES
MEDICATIONS	CECLOR PROVENTYL	PROVENTYL CONSTANT-T	→	→	→	NONE	PROVENTYL CONSTANT-T
PEF	34.9	51.7	42	43.1	38.3	31.6	37
VARIABILITY %							

DAILY PEAK FLOW

SUBJECT ID NUMBER=5013



+——+ FOR MIN OR MAX FLOW

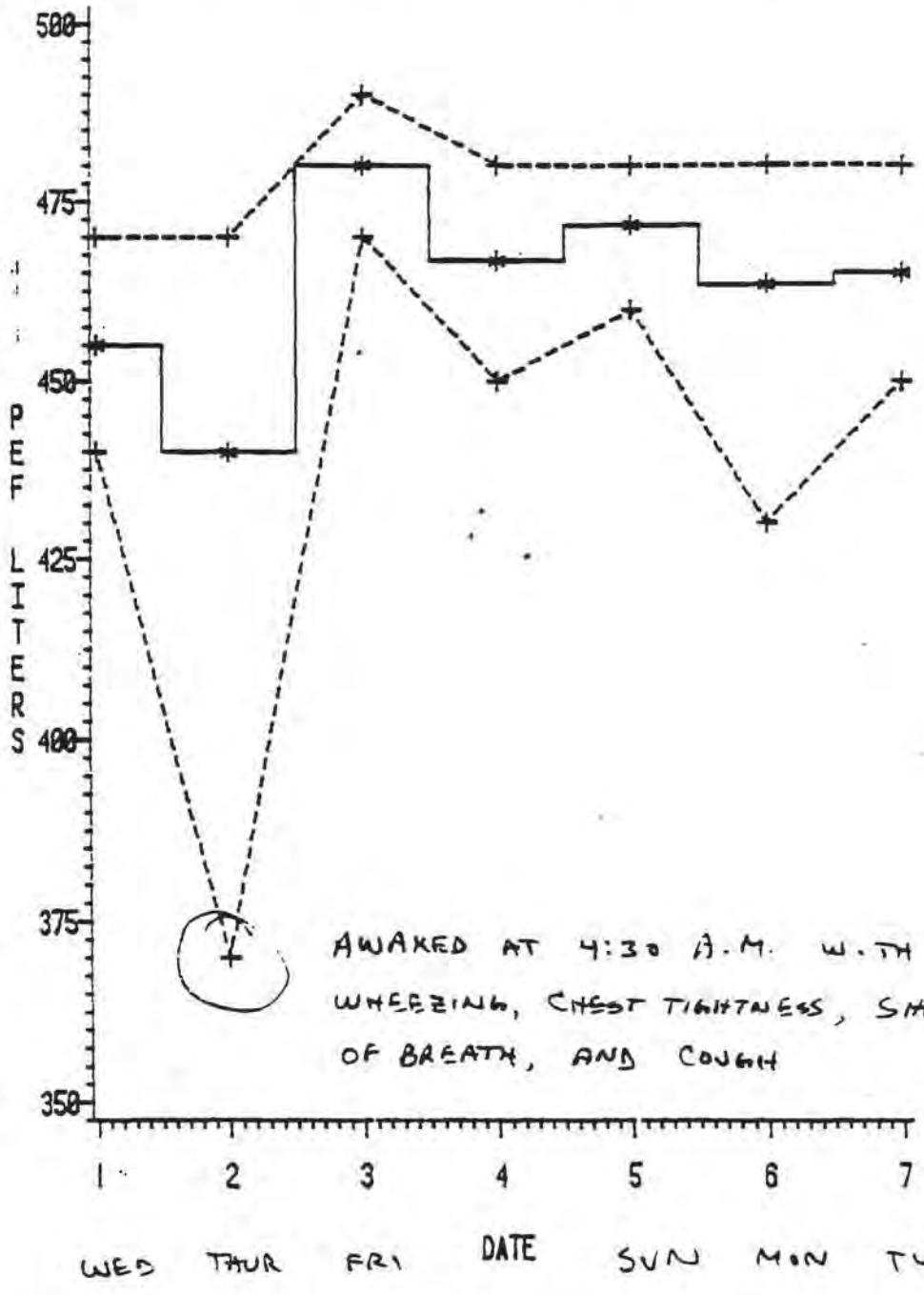
—— FOR MEAN FLOW

WORKED WITH EGGS?	YES	YES	YES	YES	YES	YES	YES
MEDICATIONS?	PRIMATENE ⟶			No	No	No	No
PEF VARIABILITY	11.3	14.3	25	15.1	25.4	16.1	15.0

M.D.

DAILY PEAK FLOW

SUBJECT ID NUMBER=5025



FOR MIN OR MAX FLOW

FOR MEAN FLOW

WORKED WITH ENNS	YES	YES	YES	YES	NO	YES	YES
MEDICATIONS	ACTIFED →						NONE
PEF VARIABILITY %	6.4	21.3	4.1	6.3	4.2	10.4	6.3