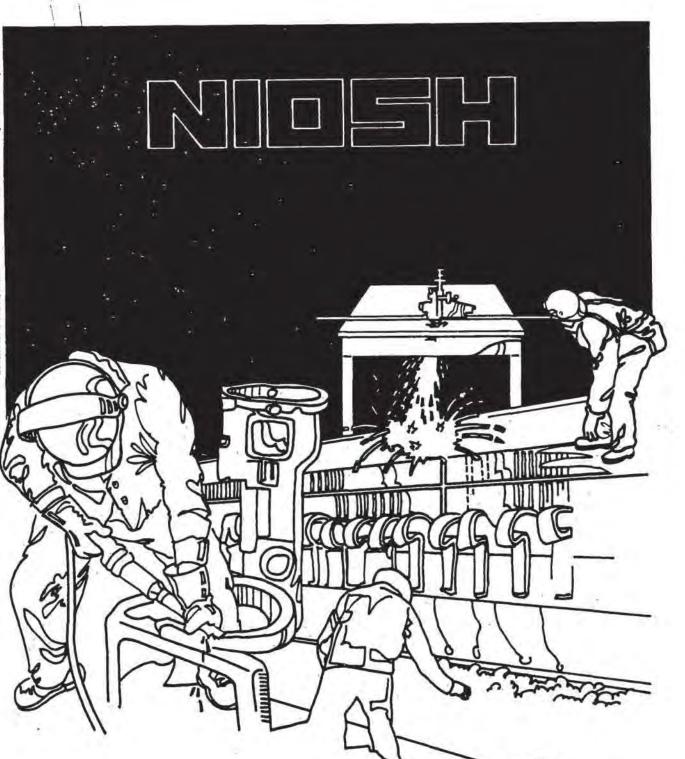
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service
Centers for Disease Control

National Institute for Occupational Safety and Health



Health Hazard Evaluation Report

HETA 82-228-1447 COCA COLA COMPANY HIGHTSTOWN, NEW JERSEY HETA 82-228-1447 APRIL 1984 COCA COLA COMPANY HIGHTSTOWN, NEW JERSEY INVESTIGATORS:
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SUMMARY

In May, 1982 the National Institute for Occupational Safety and Health (NIOSH) was requested by Local 11 of the International Brotherhood of Teamsters to evaluate possible effects on the respiratory system from a spray used in a can-lining process at the Coca Cola Company plant in Hightstown, New Jersey. The evaluation was assigned to the New Jersey State Department of Health, Occupational Health Program, under a Cooperative Agreement. The request for an evaluation was prompted by the development of asthmatic symptoms in a man who was compelled to transfer from his job as a mechanic on the can assembly line. Several other workers were reporting respiratory complaints.

In order to determine if exposures to the can-liner spray (a waterborne epoxy resin) were related to respiratory illness, Occupational Health Program Staff conducted a walkthrough inspection in May 1982, and a follow-up for industrial hygiene and medical testing from March 7 to March 14, 1983. During the medical testing, all fourteen individuals who work in the spray liner area participated. Four control persons from an adjacent can-filling plant without spray liner exposure were also included. Participating workers received a physician interview and examination, pulmonary function tests before and after the work shift on four days during the test week, and blood studies for complete blood count, sedimentation rate and antibody detection. In addition, six exposed workers were issued portable peak flow meters and kept diaries of their peak flow and symptoms every three hours for a period including two weekends and the intervening work week.

As a parallel part of the evaluation, the original worker who was transferred away from the area was given clinical testing outside the workplace. Inhalation challenge studies using dimethylethanolamine (DMEA), an amine compound found in the spray liner and reported to cause asthma, were conducted at the Deborah Heart and Lung Center.

Industrial hygiene sampling included air sampling for DMEA in the breathing zone of nine employees. Area air samples for DMEA were also collected adjacent to the ovens and spray machines. Previous data on butanol and butylcellosolve concentrations, obtained just prior to the HHE by the New Jersey State Department of Health, were also reviewed.

Results of the medical evaluations indicated that one worker, in addition to the original case, may have a lung problem as a result of exposure to the spray liner, based upon symptoms and baseline pulmonary function. In addition, thirteen of the fourteen exposed workers reported transient symptoms of respiratory irritation consistent with the effects of an inhaled amine. Peak flow results were negative for occupational asthma in all six workers tested. Immunologic studies showed potentially significant specific antibody levels in three individuals: two exposed symptomatic workers, including the original case, and one non-exposed control. It was also noted that five of the exposed workers and none of the controls had abnormal baseline pulmonary functions. This raised a question regarding the effects of long-term low level exposure to lung toxins in the can manufacturing plant. Review of past company-administered lung function studies for these five employees indicated that gradual deterioration in lung function was unlikely, but could not be totally ruled out due to unreliable data.

Industrial hygiene results indicated that DMEA could be detected in only one of the air samples, an area sample from the top of a drying oven. The concentration detected was 1.08 ppm. There is no OSHA exposure limit specifically for DMEA. Levels of butanol and butyl cellosolve, solvent vehicles in the spray liner, were well below the OSHA standards for these compounds.

Improvements in general ventilation were made in the spray liner areas in October, 1982. These improvements led to a decrease in the incidence of respiratory irritation due to the spray liner.

Based on these results NIOSK concluded that there was a health hazard from exposure to waterborne epoxy spray at Coca Cola Foods, Hightstown. The hazard involved lung sensitization in at least one worker and transient respiratory irritation in nearly all others directly exposed. Dimethylethanolamine (DMEA) is believed to be the epoxy ingredient responsible for the case of occupational allergy and is suspected as the cause of irritation. Current levels of epoxy spray appear to be very low, and have been reduced following improvements made in ventilation and enclosure of the process. Further steps are needed to decrease the risk of respiratory allergy developing in other workers and to diminish the risk of unusual short-term exposure during spills, leaks or ventilation failures. Recommendations to achieve these objectives are made in Section VIII of this report.

KEYWORDS: SIC 2086, asthma, dimethylethanolamine, butanol, butyl cellosolve, epoxy, RAST testing.

II. INTRODUCTION

In May 1982, NIOSH received a request for a health hazard evaluation at the Coca Cola Food Division can manufacturing plant in Hightstown, New Jersey. The request was initiated by Local 11 of the Teamsters and the evaluation was assigned to the New Jersey State Department of Health under a Cooperative Agreement with NIOSH. The requestors asked NIOSH to determine if a respiratory hazard existed due to water-based epoxy spray used to line the inside of beverage cans.

III. BACKGROUND

The can manufacturing plant of the Coca Cola Foods Division in Hightstown is a single, large building constructed in 1967 for the production on aluminum cans to contain fruit drinks. The cans assembled in the plant are filled in an adjacent Coca Cola plant. The total non-clerical workforce in the can assembly plant numbers forty people. The schematic diagram in Figure 1 illustrates the layout of the plant. Two can assembly lines are operated during the day shift; only one is operated during the evening shift. The can assembly process begins with flat sheets of aluminum which are rolled into shape and then strip-soldered. A water-based epoxy seam sealer is applied. The cans are then coated internally with a water-based epoxy compound, and heat-treated for drying. Tops and bottoms are added to the cans in a separate building, prior to filling and labelling.

The "spray liner area" was defined as the area from the solder machines to the far wall where the spray machines are located. During full operation, nine workers are required in this area. Each line has a body-maker working at the solder machines and a spray operator who stacks the discs to be attached as can bottoms and keeps watch on the spraying process. Each line also has a line mechanic who moves about the line making repairs or adjustments. Two quality control technicians are usually working in an enclosed booth between the two liners, and a utility person may be present in the area.

The two spray machines, which are indentical, contain a nozzle, at approximately neck level, which draws the epoxy from a storage tank under the floor. Cans are fed by overhead conveyer into the machine at a rapid speed, and the nozzle moves in and out of the can while the can is momentarily spun to spread the spray evenly. Directly above the nozzle is a slotted ventilation duct with a flange to deflect spray. The nozzle apparatus is not enclosed and can be observed totally during operation. Beneath each nozzle is a bucket to collect overspray. Spray operators periodically empty these buckets back into the underfloor tanks for recycling. In addition to the epoxy spray, workers in the can assembly area may be exposed to dimethylformamide (DMF) and methyl isobutyl ketone (MIBK) which are used intermittently for cleaning.

In December, 1981, the spray liner was changed from a vinyl-based lacquer to a water-based epoxy. Subsequently, in March, 1982, one line mechanic was forced to leave work due to chest constriction, shortness of breath, cough, and skin rash. Pulmonary function tests performed during his recovery period and shortly after a trial return to work appeared to indicate a work related respiratory problem. Compared to pre-return levels, FEV 1 and FVC following return to work fell by 19% and 24% respectively. The history indicated that symptoms were worsened by immediate exposure to the new spray. Eventually, an inhalation challenge test was conducted in July, 1982 at Deborah Heart and Lung Center in Browns Mills, New Jersey under the supervision of Dr. David Murphy. The individual inhaled a mist containing a 2% solution of dimethylethanolamine (DMEA). DMEA is a highly reactive additive which comprises 2% of the spray liner by volume, and has been associated with occupational asthma in a case report (1). A delayed reaction, characterized by a fall in both FVC and FEV, as well as chest tightness, rash and slight temperature elevation, was observed. It was concluded that the patient had an occupational immunologic lung disease which needed further clarification. However, the patient was unavailable for further inhalation studies. Pulmonary function testing seven months after job transfer indicated continuing asthma and bronchconstriction related to exercise. Despite transfer to a new building, this employee may still have been exposed to very small amounts of DMEA due to plant emissions and occasional visits to personnel offices in the can assembly building. After a one month absence from work, airway reactivity returned to normal.

The waterborne epoxy spray liner contained 8% butyl alcohol and 8% butyl cellosolve in addition to 2% DMEA. The epoxy is a standard high molecular weight polymer made from epichlorhydrin and bis-phenol A. The monomer content and presence of any other additives is unknown. According to the manufacturer, phthalic anhydride is not present. In March, 1982, prior to the initiation of the HHE request, the New Jersey State Department of Health was involved in evaluating exposure to butyl alcohol and butylcellosolve at the plant.

IV METHODS

A. Environmental

To quantify the exposure of employees working at or near the spray machine to dimethylethanolamine, air monitoring was performed on three occasions - March 7, 9 and 11, 1983. Nine employees - 2 spray machine operators, 2 body maker operators, 2 can line mechanics, 2 inspectors, and one utility man-were monitored for a full shift. All employees who were sampled worked on the first shift. These employees wore precalibrated MSA C-200, Dupont P-2500 and Dupont P-4000 sampling pumps with 150 mg silica gel tubes. The New Jersey Department of Health Laboratory performed the analysis for DMEA using the approved NIOSH analytical methodology.

On separate days the flow rate used to collect DMEA was varied from 200 cc min. to 1 liter/min. Various flow rates were used because NIOSH sampling methodology designed specifically for DMEA does not exist. The sampling methods for closely related compounds called for flow rates of 200 cc/min in silica gel tubes. However, because DMEA is a low percentage component of the spray liner and has a relatively low volatility, little of this substance might become airborne. We therefore decided to sample at 1.0 liter/min for one shift to maximize our ability to collect the DMEA. The collection efficiency for DMEA at various flow rates is unknown.

In addition to the personal monitoring which was conducted, areas near the spray machines and adjacent to the ovens were also sampled. Dupont P-4000 sampling pumps calibrated to 200 cc/min were used. The sampling medium was silica gel.

On March 9, 1982, the New Jersey State Department of Health conducted an evaluation of employees exposures to 1-butanol and butoxyethanol (butylcellosolve). Personal air samples were collected for 8 hours on spray machine operators, body maker operators and can line mechanics. Area samples were also collected using personal sampling pumps and 150 mg. charcoal tubes according to the sampline methodology specified in the NIOSH Manual of Sampling Data Sheets.

B. Medical

The walkthrough investigation established that the workers with the greatest risk for exposure to the spray liner were all those working on the two assembly lines: 3 line operators, 4 line mechanics, 3 body operators, 3 inspectors, and 1 utility person. Four workers from the can-filling plant adjacent to the can assembly plant were selected as controls. All 18 subjects were given an interview by a physician who collected information on past and present work history, symptoms (skin, upper and lower respiratory), past medical history, smoking, and family history. Each person was given a physical examination of the chest, head, and exposed skin.

Blood samples for specific IgE and IgG (types of allergic antibody) to DMEA were collected by venipuncture. Separated serum was sent to the Division of Immunology, University of Cincinnati Medical Center, where radioallergosorbent tests (RAST) for specific IgE were carried out using DMEA alone, DMEA coupled with human serum albumin, and DMEA coupled with Sepharose gel. For specific IgG, an enzyme-linked immunosorbent assay (ELISA) was done using DMEA and DMEA-albumin conjugate. Total serum IgE was also measured. A blood sample was sent to Metpath Labs for a complete blood count and sedimentation rate.

Pulmonary function studies were performed using a Collins survey spirometer with an Apex 420 microprocessor incorporating the predicted values of Morris (2). Timed vital capacities were measured before and after the shift on Monday, Wednesday, Friday and the following Monday. Second shift workers had preshift testing only. Six workers with possible work-related symptoms were given Mini-Wright peak flow meters to record their own peak flow every three hours for a period of nine days (two complete weekends and the intervening work week). Each peak flow participant kept a diary of results and symptoms.

V. EVALUATION CRITERIA

When TLV's (Threshold Limit Values) or environmental criterial are mentioned, they refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, 8 hours a day, without adverse effects. TLV refer to time-weighted average concentrations for an 8-hour workday and 40-hour workweek. The American Conference of Governmental Industrial Hygiensts (ACGIH) emphasizes that TLV's should be used only as a guide in the control of health hazards and not to establish the line between safe and dangerous concentrations.

There are three major sources for environmental criteria: 1) NIOSH recommended evaluation criteria, which are usually published as Criteria Documents; 2) ACGIH Threshold Limit Values; and 3) OSHA standards.

The Water-Borne Spray Liner used at Coca Cola contained a number of constituents only a few of which are known. Butylalcohol and butyl cellosolve each comprise 8% of the product. Two percent of the spray liner is dimethyle-thanolamine. The system itself is an epoxy resin based on the reaction of epichlorohydrin and bisphenol A.

<u>Dimethylethanolamine (DMEA)</u> - There is currently no environmental or occupational standard proposed by the ACGIH, OSHA or NIOSH regarding exposure to DMEA. The health effects of DMEA also have not been well studied. However, a related compound, diethylethanolamine (DEAE) has a ACGIH recommended TLV of 10 ppm and an OSHA permissable exposure limit of 10 ppm. The health effects

of ethanolamine compounds in general include irritation of the eyes, mucous membranes and skin. The liquid is a severe skin irritant and in animal sudies it has been shown to be a skin sensitizer. There is also evidence that dimethyl ethanolamine can cause asthma and/or rhinitis, as previously mentioned.

<u>Butyl cellosolve</u> - is a solvent which can enter the body through inhalation or by skin contact. It is an irritant of the eyes and mucous membranes and severe exposure may cause hemolysis. The ACGIH TWA is 25 ppm. The current OSHA standard is 50 ppm.

The NIOSH Current Intelligence Bulletin No. 39 (May 2, 1983) discusses new data from animal studies which indicate that exposure to glycol ethers related to butyl cellosolve, specifically methyl and ethyl cellosolve, have a variety of adverse effects on reproduction. These effects included testicular atrophy, congenital defects, and fetal toxicity or death. Butyl cellosolve itself has not been thoroughly studied in this regard.

<u>Butyl Alcohol</u> - is a solvent which can enter the body through inhalation or by skin contact. It is an irritant of the eyes and mucous membranes; it may cause central nervous system depression at very high concentrations. Contact dermatitis involving the fingers and hands may occur owing to a defatting action of the liquid. The ACGIH TWA is 50 ppm for n-butyl alcohol and 100 ppm for sec- and tertbutyl alcohol. The current OSHA standards are 100 ppm, 150 ppm and 100 ppm's respectively.

Epoxy Resins - are thermosetting resins based on the reactivity of the epoxide group. One type is made from epichlorohydrin and bisphenol A. The general health effects associated with exposure to epoxy resins include dermatitis, irritation of mucous membranes and asthmatic symptoms These compounds are well-known skin sensitizers. Additional specific hazards may be posed by unreacted monomeric constituents.

VI. RESULTS

A. Environmental

The air-monitoring results are presented in Table I. Only one sample showed detectable levels (detection limits for DMEA 90 liters - 0.378 ppm; 400 liters - 0.085 ppm). This level of 1.08 ppm was collected as an area sample near the entrance of the top oven of #2 line.

Exposures to 1-butanol ranged from 0.757 ppm to 3.44 ppm as an 8 hour time weighted average (TWA). This is far below both the OSHA permissible exposure limit (PEL) and the ACGIH limit for this substance. Exposures to butyl cellosolve ranged from 0.047 ppm to 0.185 ppm as an 8 hour TWA. The OSHA PEL for butyl cellosolve is 50 ppm.

B. Medical

The 14 exposed workers in the study had a mean age of 41; 13 were male. The 4 non-exposed workers had a mean age of 32, and 3 were male. The mean duration of exposure to can assembly processes was 10.4 years. Symptoms reported on the questionnaire are summarized in Table II. Eleven out of the 14 exposed workers reported lower respiratory symptoms (cough, wheezing or shortness of breath) during the previous year which were work-related. Work relatedness was determined by time of onset of symptoms and whether symptoms were worse at work and better during periods off. Six workers with lower respiratory symptoms developed coughing in the workplace shortly after introduction of the new spray liner in December, 1981; three also had wheezing. Three others reported onset of cough or other symptoms during the May-June, 1981 period.

Ten workers reported work-related upper respiratory symptoms (sore dry throat, nasal stuffiness, or irritated eyes) during the previous year. Sore, dry throat was the predominant symptom. Once again, the onset was nearly always immediately following introduction of the new spray liner, and symptoms were closely linked to acute exposure to the spray.

Two workers reported non-specific systemic symptoms (fever, chills or muscle aches) during the previous year which were unusual. These symptoms occurred in transient episodes and did not reoccur with every exposure to the spray. No exposed workers reported work-related skin conditions. Figure 2 shows the time periods for the occurrence of upper and lower respiratory symptoms. There were no symptoms reported by controls except for one worker with hand dermatitis related to peroxide exposure.

Most of the symptoms reported were mild and transitory in character. However, two individuals reported symptoms which were notably more severe and persistent. The first person, subject 1, had no previous history of asthma or other atopic illness, but developed cough, wheezing, shortness of breath, sore throat and itchy eyes one week after the new spray was introduced. Three episodes of fever, chills and muscle aches were also reported during the most recent three-month period; two episodes began on Monday mornings. Decreased exercise tolerance and relief of symptoms after taking oral bronchodilators was also reported. The second person, subject 4, had a history of summer hay fever for a few years, and reported transitory cough and wheezing each time after emptying buckets with the spray overflow. The major problem however, was a persistent nasal stuffiness, sore throat and eye irritation which began soon after return to work in the spray liner

area. These symptoms were described as being different in nature from the usual hay fever, and had occurred earlier in the year than any previous hay fever episodes. Three additional workers had episodes of cough, wheezing, chest tightness or sputum which appeared to be work-related and caused them to seek medical attention. All three were improved at the time of this study and were trying to avoid the spray liner area. Overall, the frequency and severity of symptoms had decreased from the time walkthrough interviews were conducted in June, 1982 until formal interviews done in April, 1983.

Pre-shift and post shift spirometry failed to detect any individuals with a significant drop in performance either over the shift or over the work week. Baseline spirometry values, however, were depressed in five individuals. Their results are displayed in Table III. It was noted that only one of this group had a history of cigarette smoking. Pulmonary function tests were performed periodically on these individuals; the time trends in FVC and FEV₁ are plotted in Figures 3 and 4. We see no gradual drop in either variable over time; as we might see if chronic deterioriation in lung function due to prolonged exposure to a toxin was occurring. Furthermore, we note that all five individuals had some low measurement observed prior to the introduction of the new spray liner in late 1981. This interpretation is weakened by a great fluctuation in values in the same individuals from one year to the next. The likeliest explanation for this is poor spirometric technique and/or poor subject cooperation.

Subject 1, who may have developed symptoms of asthma coincident with the introduction of the new spray liner, is of particular interest. The values are relatively stable, implying good cooperation, and show a 650 cc decrease in FEV₁ in May, 1979 (the last test before introduction of the new spray liner in December,

1981) compared to the previous test. The FEV₁/FVC ratio in May, 1979 was a normal 80%, however.

Peak flow diaries indicated that none of the six individuals tested had dips in peak flow of any significance, including the one worker with possible work-related asthma, subject 1.

Complete blood counts and sedimentation rates were all normal. Total IgE was elevated for three individuals: one with possible work-related asthma (subject 1) one control (subject 18) and the index case who was challenge tested. Significant levels of DMEA-specific IgE were found using the DMEA-Sepharose RAST for the worker with possible work-related rhinitis (subject 4), one control (subject 18) and the challenge test confirmed case. ELISA tests for specific IgG were all negative.

VII. DISCUSSION AND CONCLUSIONS

The nature and high prevalence of symptoms and the clustered times of onset indicate that the spray liner was causing non-specific irritation of the respiratory tract. In most cases, this respiratory irritation was reversible and has diminished in recent months following worker avoidance of the area and introduction of changes designed to reduce exposure. These changes include spray deflectors on the spray machines, buckets for catching spray overflow, and boosted general ventilation. High volume personal and area air samples for dimethylethanolamine (DMEA) taken during April, 1983 showed levels beneath the limit of detection with only one exception. The evaluation focused upon DMEA because it was a major ingredient of the epoxy spray and, compared to other known constituents, was believed to have the highest potential for respiratory toxicity. It is likely that the air levels of DMEA during 1982, before engineering changes, were higher. The low levels at present coincide with the drop in acute symptoms of respiratory irritation being reported.

DMEA was also of interest due to its ability to cause respiratory sensitization. A case of occupational asthma, confirmed by inhalation challenge testing, has been reported in a painter exposed to spray paint containing 2% DMEA (1). Asthma due to similar amine compounds such as ethylenediamine and aminoethyl ethanolamine have also been reported (3,4). A recent study from Sweden found increased bronchial reactivity and respiratory symptoms in workers exposed to amines in the polyurethane foam industry (5). Several cases of asthma associated with curing or hardening agents in epoxy resin systems were discussed by Fawett and co-workers (6). One case was due to an amine compound (amines are generally used in "cold-cure" epoxies); the others were due to either phthalic or trimellitic anhydride.

It is our opinion that the index subject studied in this evaluation displayed an immunologic lung response to DMEA. A bronchoconstrictive reaction as well as a possible systemic delayed reaction were found following inhalation challenge with dilute DMEA. A delayed recovery following removal from exposure, characterized by fatigue, shortness of breath, and exercise - induced bronchoconstriction was noted. Serum tests for DMEA specific antibodies failed to detect any IgG, but did detect low levels of specific IgE and a moderate elevation in total IgE. The evaluation on this individual is limited by the lack of data on response to inhalation of control substances, the absence of data on chest X-ray and pulmonary function 10 to 24 hours after DMEA challenge, and the possibility that an increased total IgE might explain an elevated specific IgE due to non-specific binding in a newly-developed RAST assay. The phenomenon of a single small molecular weight compound causing both an asthmatic response and a "flu like" or hypersensitivity pneumonitis-type response has been seen with trimellitic anhydride (TMA) and certain isocyanates. (7,8)

In addition to the index case, there are two other workers in the spray liner area who are suspected of having an occupational allergy. Subject 1 may have developed asthma at the time of the spray liner change due to occupational triggers. However, non-occupational causes for this asthma cannot yet be ruled out, and peak flow data did not indicate a work-related change in peak flow. In some cases of occupational asthma, peak flow becomes fixed at a depressed level and does not vary greatly with exposure. Only inhalation challenge or testing of lung performance offer a prolonged removal from exposure could determine conclusively if occupational asthma due to the spray liner is present. In the second case, subject 4 developed a worsened allergic rhinitis after first being exposed to

the new spray liner. Subject 4 also had a significant elevation of DMEA specific IgE antibody without an increase in total IgE. The RAST method has not previously been applied to DMEA and results should be interpreted with caution. Once again challenge testing would be required to make a final conclusion regarding DMEA-induced allergic rhinitis in this individual. Simply removing subject 4 from exposure and observing for improvement would not be helpful since factors outside the workplace are already known to play a role in the allergic rhinitis.

The observation of five out of thirteen can assembly workers with borderline or abnormal pulmonary function is considered unusual, particularly in view of their light smoking history overall. Nevertheless a chronic effect of the can assembly work on pulmonary function can neither be confirmed nor denied on the basis of our results. Previous pulmonary function testing appears to be unreliable.

We conclude that the waterborne epoxy spray liner, and DMEA in particular, are capable of causing an allergic response in the respiratory system of can assembly workers. This conclusion is based on the finding of one established case and two others which are suspected. The occurrence of this allergic response is probably rare, but would be expected to increase when exposure is intense or prolonged. It is also concluded that the spray liner causes non-specific respiratory irritation at exposure levels above that required to trigger allergic response in a sensitive individual. This non-specific effect could also be due to DMEA and is readily controlled by elementary ventilation and shielding techniques. Currently, levels of epoxy spray in the work area are very low during routine operations. Significant exposure is more likely to occur during spills or maintenance activities.

VIII. RECOMMENDATIONS

Based on the information presented above, NIOSH recommends the following preventive measures.

- Methods for more completely enclosing the spray nozzle area should be explored. Although current air levels are low, even small amounts of exposure may trigger symptoms in sensitive individuals. Furthermore, periodic failure of the local ventilation may occur and enclosure in that event would prevent accumulation of toxic air contaminants. The local ventilation should be checked routinely on a regular maintenance schedule, preferably with a velometer to measure air flow. The observation of visible epoxy spray mist outside the immediate area of the nozzle and it's enclosure should signal the need for maintenance and adjustment.
- 2. Respirators (NIOSH-approved half-face chemical cartridge air purifying respirators equipped with organic vapor cartridges and high-efficiency particulate filters) should be worn while workers are in direct contact with the spray liner, such as during maintenance operations or during a spill or malfunction.
- 3. The air intakes for the air conditioners in the inspector's room should be relocated so as not to face the spray machines. Air turnover in this room, which is an enclosed space presumably to reduce noise, can be boosted through the use of small fans located in the wall opposite the spray area. The practice of inspectors spraying cans individually for spray weight measurements should be replaced by the checking of automatically-sprayed cans, where exposures can be better controlled.

- 4. Medical monitoring of can assembly workers is essential and should be directed in part towards the early recognition of occupational allergy or respiratory irritation. A periodic medical interview covering allergic and respiratory symptoms is essential. Referral to physicians who are experts in the evaluation of occupational diseases including allergy should be made available when such problems are suspected.
- 5. The periodic lung function testing currently being conducted should be standardized to conform with the recommendations of the American Thoracic Society (9). This will allow lung function to be compared from year to year. If poor subject effort is suspected, the tests should be repeated. All workers should be informed of their results, whether normal, abnormal or uninterpretable, by the responsible testing physician.

IX. REFERENCES

- Vallieres M, Cockcroft DW, Taylor DM, Dolovich J, and Hargreave FE.
 Dimethyletanolamine-induced asthma. Amer Rev Resp Dis 115:867-871,
 1977
- Morris, JF, Koski A, Johnson LC. Spirometric standards for healthy non-smoking adult. Amer Rev Resp Dis 103:57-67, 1971.
- Lam S, and Chan-Yeung M. Ethylenediamine-induced asthma. Amer Rev Resp Dis 121:151-155, 1980.
- 4. Peys J, Pickering CAC. Asthma due to inhaled chemical fumes-aminoethanolamine in aluminum soldering flux. Clin Allergy 2:180-196, 1972.
- 5. Belin L, Wass U, Audunsson G, and Mathiasson L. Amines: possible causative agents in the development of bronchial hyperreactivity in workers manufacturing polyurethanes from isocyanates. Brit J Ind Med 40:251-257, 1983.
- 6. Fawcett IW, Newman Taylor AJ, and Peys J. Asthma due to inhaled chemical agents-epoxy resin systems containing phthalic acid anhydride, trimellitic acid anhydride and triethylene tetramine. Clin Allergy 7:14, 1977.
- Patterson R, Zeiss CR and Pruzansky JJ. Immunology and immunopathology of trimellitic anhydride pulmonary reactions. J Allergy Clin Immunol 70:19-23, 1982.

- Charles J, Bernstein A, Jones B, et al. Hypersensitivity pneumonitis after exposure to isoyanates. Thorax 31:127-131, 1976.
- Gardner R, (Chairman). ATS statement: Snowbird workshops on standardization of spirometry. Amer Rev Resp Dis 119:831, 1979.

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XI. DISTRIBUTION AND AVAILABILITY OF REPORT

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

Copies of this report have been sent to:

- 1. Authorized representative of Teamsters, Local 11.
- 2. Coca Cola Company, Foods Division, Hightstown, New Jersey.
- Region II, NIOSH.
- OSHA, Region II.

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.

TABLE I

AIR SAMPLING RESULTS FOR DMEA

MARCH 7, 9, and 11, 1983

Total number of samples collected:			40
Total number of personal sa	imples collected:		32 all non detectable
Job titles sampled: Ca	an Line Mechanics	6	
	Body Maker Operators	6	
	Spray Machine Operators	11	
	Utility Man	3	
	Inspector	6	
Total number of area samples collected:			8
Location of areas sampled:	Spray Machine	4	non-detectable
	Doubling Box	1	non-detectable
	Upper Drying Oven	2	(1.08 ppm on one sample)
	Lower Drying Oven	1	non-detectable

- Collection Medium Silica Gel
- ** Range of Volumes Collected 90 liters 400 liters
- *** Levels of Detection
 - (a) At 90 liters 0.378 ppm
 - (b) At 400 liters 0.085 ppm

TABLE II WORK-RELATED SYMPTOMS REPORTED IN EPOXY SPRAY - EXPOSED WORKERS AND CONTROLS, COCA COLA FOODS, HIGHTSTOWN, NEW JERSEY

Subject	Lower	Upper	Fever Ache	Rash
1	+(W,C,SOB)	+(DT,E)	+	0
2	+(C,SOB)	+(DT)	-+-	0
3	+(c)	+(N)	0	0
4	+(W,C)	+(DT,E,N)	0	C
5	+(C)	+(DT)	0	0
6	С	+(DT,E,N)	c	O
7	0	+(DT)	0.	0
8	С	C	C	O
9	+(W,C)	+(DT)	0	C
10	+(C)	0	c	C
11	+(C)	0	O	C
12	+(C)	+(DT,N)	C	C
13	+(W,C,SOB)	+(DT,N)	0	C
14	+(C)	0	0	c
15	0	0	0	C
16	0	0	0	0
17	0	C	0	O
18	C	0	О	C
W - wheezing C - cough SOB - shortness	of breath	DT - dry throat E - eye irritati N - nasal conges		

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Table III

BASELINE PULMONARY FUNCTION ABNORMALITIES - CAN ASSEMBLY WORKERS

Subject	FVC* (% Predicted)	FEV ₁ * (% Predicted)	FEV1/FVC%*	Abnormality	Smoking
10	79	79	79	borderline restriction	18 pack-years
11	79	73	74	borderline obstruction	less than one pack-year
1	87	77	70	mild obstruction	never
13	. 38	76	64	mild restriction	never
12	86	76	64	mild obstruction	never

^{*}Average of at least 5 tests