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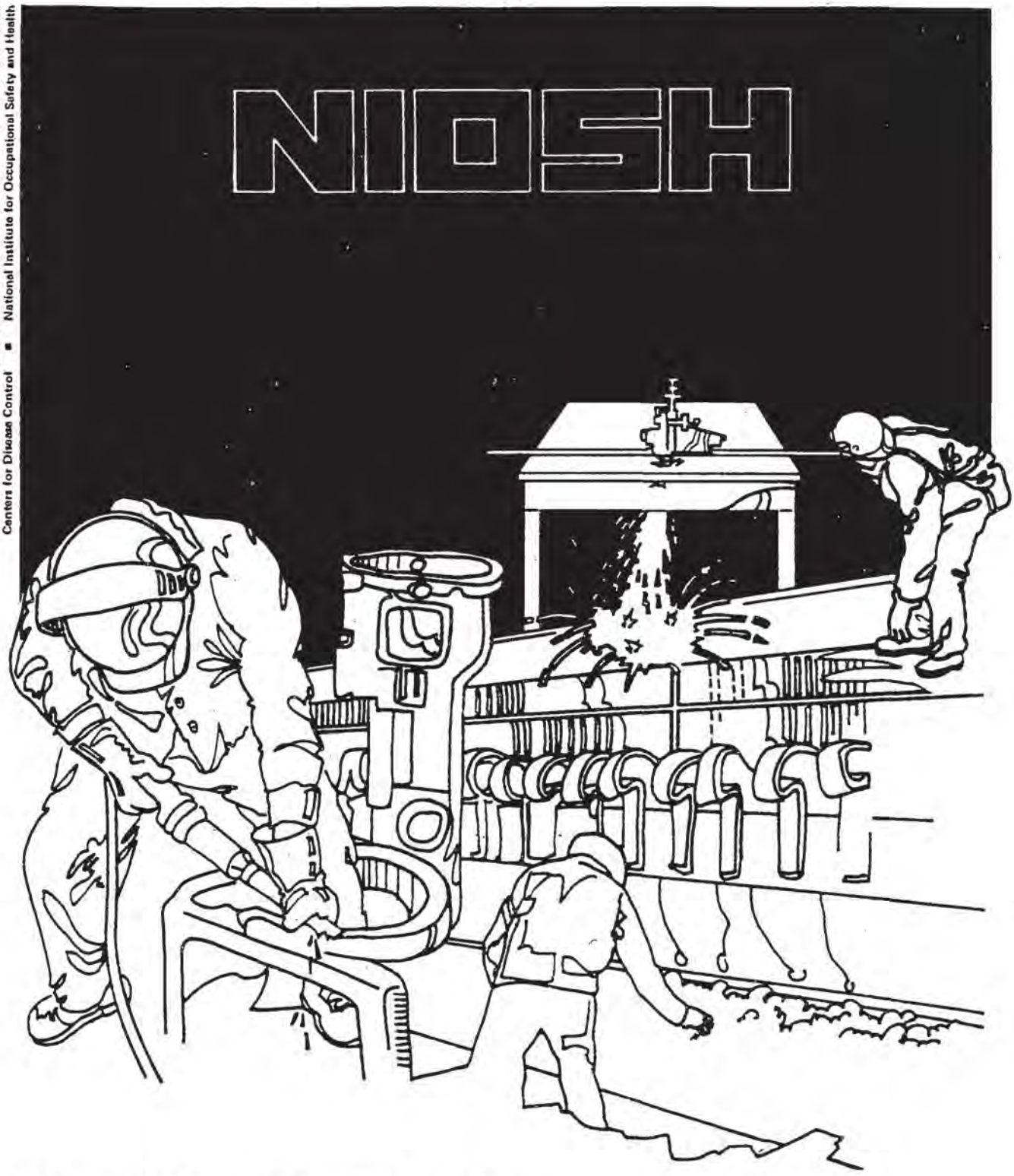

Health Hazard Evaluation Report

HETA 82-032-1384
BADISCHE CORPORATION
KEARNY, NEW JERSEY

PREFACE

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

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial nygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

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I. SUMMARY

In November 1981, the National Institute for Occupational Safety and Health (NIOSH) received a request to evaluate occupational exposures to chemicals, particularly di(2-ethylhexyl)phthalate (DEHP) at Badische Corporation, Kearny, New Jersey. In January 1982, a NIOSH survey team conducted an initial environmental/medical survey; we conducted a follow-up environmental/medical survey in March 1983.

During the initial survey, environmental monitoring was conducted to measure airborne exposures to DEHP and phthalic anhydride (PA). Of three full-shift samples collected for PA from operators, only one was above the analytical limit of detection (7 microgram (ug)/sample), reported at 0.055 milligram/cubic meter of air (mg/m^3); OSHA standard 12 mg/m^3. Of 13 samples collected for DEHP (four general area and nine personal) only two area samples were above the analytical limit of detection (0.01 mg/sample) with both results below 0.2 mg/m^3; OSHA standard 5 mg/m^3. During the follow-up evaluation, environmental monitoring was again conducted for DEHP and PA, plus 2-ethylhexanol and dibutylphthalate (DBP). DEHP and PA samples were obtained over five shifts, primarily from the breathing zone of workers in most existing job categories. Of 50 samples obtained for DEHP, six were reported above the analytical limit of detection (0.01 mg/sample), with exposure concentrations ranging from 0.02 to 4.11 mg/m^3, averaging 0.71. Of 50 samples obtained for PA, 33 were reported above the analytical limit of detection (0.0015 mg/sample), with exposures ranging from 0.01 to 0.20 mg/m^3, averaging 0.04. Results of eight samples collected for 2-ethylhexanol from operators ranged from 0.24 to 18.1 mg/m^3, averaging 4.1 mg/m^3. All DBP sample results in the Batch Ester Plant were below the 0.01 mg/sample limit of detection.

NIOSH investigators interviewed 66 of 73 hourly workers and 29 salaried workers, and collected pre- and post-shift urine samples for measurement of total phthalate content and serum samples for evaluation of liver function. Of the non-administrative personnel, 48 were considered to be in jobs at higher risk and 27 in jobs at lower risk for exposure to DEHP. There were no significant differences between these groups in the prevalence of reported gastrointestinal/constitutional or nervous system symptoms or symptoms of mucous membrane irritation. There were no significant differences between the two groups in the prevalence of abnormal liver function tests. The mean (+ S.D.) pre- to post-shift change in urine phthalate concentrations in the higher exposure group (increase of 3.37 + 9.4 nanomoles/ml) was significantly greater than that (decrease of 0.44 + 5.8 nanomoles/ml) in the lower exposure group ($p=0.04$). The mean change in the higher exposure group was significantly different from zero ($p=0.02$). Substantial increases (doubling) in urine phthalate over the shift were observed in eight of 33 chemical operators and in none of 36 non-operators ($p=0.02$). The increased urinary phthalate likely resulted from absorbed phthalic acid rather than from DEHP.

Although no health effects attributable to exposures were observed, this health hazard evaluation demonstrated exposure of employees at Badische Corporation to DEHP, a suspect carcinogen. Efforts to minimize exposure should continue. Recommendations are contained in Section VIII of this report.

KEYWORDS: SIC 2869 (Industrial organic chemicals, Not Elsewhere Classified), plasticizers, di(2-ethylhexyl)phthalate (DEHP), 2-ethylhexanol, phthalates, liver disease

II. INTRODUCTION

On November 2, 1981, the National Institute for Occupational Safety and Health (NIOSH) received a request from the United Rubber, Cork, Linoleum and Plastic Workers of America, International Union, to evaluate employee exposures to chemicals during the production of phthalates at Badische Corporation, Kearny, New Jersey. In particular, employees were concerned about di(2-ethylhexyl)phthalate (DEHP).

NIOSH conducted an initial survey at Badische Corporation in January 1982, consisting of an opening conference and subsequent walk-through survey and initial combined environmental/medical evaluation. An interim letter containing the analysis of the company's medical testing was issued in November 1982. NIOSH conducted a follow-up environmental/medical survey on March 7-11, 1983. Individuals were notified of their blood and urine results on July 15, 1983.

III. BACKGROUND

A. Process

The phthalic anhydride and DEHP production areas (PA/DOP Plant) are located out of doors, with a common, enclosed control room. The phthalic anhydride (PA) production process reacts ortho-xylene with air in four reactors at 400 to 500°C in the presence of a catalyst. The PA is purified by passing through desublimators and two distillation columns. The light contaminants are distilled off in the first distillation column and include maleic anhydride and benzoic acid, which are incinerated. The second column removes the heavy, or crude contaminants, which are also incinerated. The plant is capable of producing 100 million lbs./yr, which is approximately 10%-15% of the current U.S. production of PA.

The production of DEHP is a continous-flow operation. The production process involves the esterification of 2-ethylhexanol with PA in the presence of acatalyst. Capacity is 180 million lbs/yr.

In 1981, the Batch Ester Plant (BEP) came on-line, with the ability to produce esters such as dibutylphthalate, diisodecyl phthalate, butyl octyl phthalate, trioctyl trimaletate, dioctyl adipate, and DEHP. The Plant's total nameplate capacity is 58,000,000 lbs./yr., with the market dictating which of the chemicals are to be produced. The basic chemical processes are very similar to the DEHP plant where an acid, alcohol and catalyst are reacted to form an ester. This differs solely in configuration, i.e. batch nature vs continuous.

In addition to the shift supervisor, 4 operators work in the PA/DOP plant each shift, two on the DOP side, and 2 on the PA side. A shift supervisor and two operators work in the BEP on each shift. Process operators are classified as "AA", "A", or "B", in both the PA/DOP and

BEP plants. "B" operators spend greater amounts of time in the actual production areas (approximately 90%) relative to the other operators. A typical shift on the DOP "side" would consist of four cycles; each cycle involves sample acquisition from process streams and transport of the samples to QC or Control Room laboratories where titrations, refractive index, or other analyses are conducted. Other duties include logging of process meter readings and general surveillance of the process. Sample acquisition probably represents the greatest potential for employee exposure, especially in the PA/DOP plant where sample ports are opened, and flushed, and sample containers are then filled by hand. At the BEP plant, samples are obtained via a "sample bomb" method, which greatly reduces the potential for employee exposure. For the PA "B" Operator, duties with the highest potential for exposure would include sample acquisition and occasional involvement with dumping of the PA sublimator boxes.

"A" operators reportedly spend approximately 50% of the shift in the control room, while "AA" Operators spend 90% of the shift in the control room monitoring the electronic surveillance systems of both processes.

Tank Farm operators are primarily concerned with loading tank trucks with DEHP and PA. This operation covers all shifts, with three Operators on the first shift, two on the second, and one on the third. The use of respiratory protection is required, and the loading ports are locally exhausted.

Half-face dual cartridge respirators (GMC-H) are available for the PA/DOP operators, although their use is not required. The use of these respirators is required, however, for truck or rail car loading of PA, and for removing PA from the sublimator boxes. At the BEP process, "Ultra-twin" full-face respirators (GMC-H) are required for charging of trimellitic anhydride and for removing PA from the sublimator boxes. The above discussion of respirators mentions those that are routinely used by operations personnel. The Kearny Site maintains an inventory of additional respirators to provide adequate protection for personnel during any situation.

B. Sequence of Events

Production of phthalic acid and di(2-ethylhexyl)phthalate (PA/DEHP) began at Badische (then BASF Wyandotte) in 1971. In 1975, a screening program was carried out by the company; it was aimed at detecting early liver disease in workers exposed to one of the products then being made, vinylidene chloride (VDC). This program revealed abnormalities in approximately one-third of this subgroup of employees. Those with abnormal tests were subsequently studied in more detail by the New Jersey College of Medicine and Dentistry; liver biopsies in selected individuals did not reveal consistent morphological changes.

The United Rubber Workers (URW) subsequently submitted a request to NIOSH for a Health Hazard Evaluation (HHE). NIOSH conducted an evaluation of the entire workforce in 1976 (the production of VDC had been discontinued prior to the study) and found that the prevalence of one or more abnormal results of liver function tests was statistically significantly greater in employees in the PA/DEHP department than that in the various other departments. An attempt by NIOSH at follow-up of those with abnormalities was unsuccessful. In 1978, NIOSH recommended conducting liver profiles at the start of employment and periodic evaluation directed at detecting liver dysfunction, as well as environmental monitoring for PA/DEHP.[1] The company has instituted these recommendations, although comprehensive epidemiologic analysis had not been undertaken.

In recent years, attention has been focused on the toxicity of DEHP. In 1980, a draft of the carcinogenesis bioassay report on DEHP from the National Toxicology Program was released. In October 1981, NIOSH received the current request from the URW for a follow-up HHE of workers at the plant. In March 1982, the final report of the DEHP carcinogen bioassay was published;[2] the study found that under the conditions of the bioassay, DEHP was carcinogenic for F344 rats and B6C3F1 mice, causing increased incidences of female rats and male and female mice with hepatocellular carcinomas and inducing an increased incidence of male rats with either hepatocellular carcinomas or neoplastic nodules. This study, however, has been criticized.[3]

At the time of the final NIOSH visit, the plant had 163 employees, of which 76 were hourly (all male); 87 were non-hourly (70 males, 17 females). Of these, three were on disability (2 hourly) and one (hourly) was on suspension.

IV. EVALUATION DESIGN AND METHODS

A. Environmental

During the initial site visit, environmental monitoring was conducted during the day shift of January 26, 1982, and during the evening shift of the 27th, to determine airborne concentrations of DEHP and phthalic anhydride. For "breathing zone" samples, calibrated personal sampling pumps attached to the employee's belt were connected to the device containing the sampling medium, which was attached to the worker's collar. General area samples were collected in the general work area.

To measure airborne levels of DEHP and phthalic anhydride, air was drawn through Millipore "AA" filters using portable sampling pumps pre-calibrated at 1.0 liters per minute (lpm). Laboratory analysis of phthalic anhydride was done according the the NIOSH Method S-179. The limit of detection for was 1.5 micrograms (ug)/sample. DEHP samples

were analyzed according to NIOSH Method S-40. The analytical limit of detection was 10 ug/sample. Sampling media was replaced at mid-shift to avoid the possibility of over-loading.

During the follow-up evaluation, sampling was again conducted for PA and DEHP in a manner similar to that previously described, except that filters for phthalates were not replaced at mid-shift. Limited environmental sampling was also conducted for 2-ethylhexanol. These samples were collected on porous polymer sampling tubes at a flow rate of 0.05 lpm, and analyzed via gas chromatography equipped with a flame ionization detector. The analytical limit of detection was 2 ug/sample. Since dibutylphthalate was produced in the BEP plant during a portion of the environmental survey, limited environmental sampling (five personal samples collected from BEP operators) was conducted for this substance. Sampling and analytical methodologies for dibutylphthalate were identical to those used for DEHP.

B. Medical

During the initial visit, the medical officer reviewed medical records and briefly interviewed two shifts of employees from the PA/DEHP department. Subsequently, NIOSH obtained the medical data generated by the company for the years 1978-1981 inclusive. Data for department, age, alcohol consumption, date of blood testing and results of four biochemical tests were abstracted and analyzed.

During the follow-up study, a NIOSH medical evaluation was made available to all male employees at the plant. The evaluation included administration of a questionnaire, collection of blood specimens for liver function tests (LFTs) and urine specimens for determination of total phthalate concentration. In an attempt to assess accumulation or absorption of phthalate over the workshift, hourly workers were evaluated, where possible, on the first day back at work, that is, following a period of non-exposure. This was accomplished for the maintenance department and three of the four shifts of production workers. Most personnel air sampling was performed to coincide with these evaluations. The liver function tests determined included the enzymes: serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), gamma-glutamyl transpeptidase (GGT) and alkaline phosphatase (AP). All of these are commonly used tests of "liver function". These tests were performed by the same laboratory that had performed the analyses for the 1976 study. Serum bile acids (SBA) also reflect liver function, but their role as indicators of liver dysfunction are not well-established. One SBA, sulfolithocholylglycine (SLCG) was determined using a radioimmunoassay (Abbott Laboratories, Chicago, Illinois).

Random pre- and post-shift urine samples were collected and shipped frozen; phthalate concentrations were determined at the National Institute of Environmental Health Sciences, by a method previously described.[4] Historically, the lowest concentration determined at this laboratory was 0.4 nanomoles/ml. The highest was 250 nanomoles/ml in leukemia patients receiving platelet transfusions. A number of samples were also assayed for specific phthalate/DEHP metabolites. Specific gravity of the urine samples was determined. A system for the classification of jobs and departments based on the relative ranking of exposure to DEHP was developed by consensus during joint discussions involving the NIOSH medical officer, the local union president, the Badische industrial hygienist and the Manager of Environmental Protection and Safety. This initial classification was then modified by taking into consideration the industrial hygiene data derived during the study. The results for a small group felt to be highest exposed were also analyzed separately.

V. EVALUATION CRITERIA

A. Environmental Criteria

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. Both NIOSH recommendations and ACGIH TLV's usually are based on more recent information than are the 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 primarily 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 only 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.

Following are the evaluation criteria for sampled substances.

	Evaluation Criteria (mg/m^3)		
Substance	NIOSH	OSHA	ACGIH
DEHP	none	5	5
Phthalic Anhydride	none	12	6
2-ethylhexanol	none	none	none
Dibutylphthalate	none	5	5

B. Physiologic/ Toxicologic Effects

Di(2-ethylhexyl)phthalate: DEHP is one of a family of phthalate esters. By far the largest use for phthalate esters (annual production in U.S. estimated at 2,000,000 tons in 1975[6]) in general, and DEHP in particular, is as plasticizing agents for materials such as polyvinyl chloride (PVC). These plasticizers give the polymer the desired flexibility and softness.[5] DEHP and its isomer, dioctyl phthalate (DOP) are probably the most widely used plasticizers.[5] The main non-plasticizer use for DEHP is as a replacement for polychlorinated biphenyl in dielectric fluids for electric capacitors.[14] Phthalate

esters are also used as defoaming agents in the manufacture of paper, in cosmetic products as a vehicle for perfumes, in lubricating oils, as a solvent in erasable ink, as an inert ingredient in pesticides, and until recently, for quantitative fit testing of respirators.

Several reviews on the phthalate esters[5,15] and on DEHP[6,14] have discussed the low order of acute toxicity of these compounds. However, in recent decades, increasing amounts of phthalate esters have been incorporated in PVC blood storage bags and other plastic devices with medical applications including flexible tubing, heart valves, vascular grafting materials, dialyzing units, blood transfusion sets and disposable syringes.[6] In 1970, DEHP was detected in tissues and organs of two deceased patients who had previously received transfusions.[7] Attention was soon focused on the possible chronic effects of phthalates leached into blood stored in these containers. DEHP has been detected in concentrations of 5 to 8 mg/100 ml blood in blood stored in PVC bags for 21 days at 4°C and also in other blood products[8] and can thus be transferred to patients during transfusion. The significance of this finding is not well known. Early reports in the 1940s and 1950s on the subacute and chronic toxicity of DEHP (summarized in reference 6) had indicated growth retardation and testicular atrophy, increased liver and kidney weights with no significant histopathologic findings.

Effects on the liver: More recently, a study of the effects on rhesus monkeys undergoing chronic transfusion (6 or 12 months) with DEHP-containing blood products (plasma or platelet-rich plasma), found abnormal liver-spleen scans in four of seven, abnormal bromsulphthalein clearance in four of seven and abnormal liver histopathology in six. Nontransfused monkeys, and monkeys transfused with platelets processed in polyethylene containers were normal.[9,10] Other recent studies of chronic effects on the liver suggested that liver enlargement and some morphological changes can be detected in animals treated orally with DEHP but that these changes are consistently noted only at high doses and after administration for long periods.[6] DEHP administered orally for 14 months to ferrets results in weight loss, marked liver enlargement with morphological changes including liver cell enlargement, lysosomal changes, and dilatation of the endoplasmic reticulum.[18] The authors concluded that DEHP is hepatotoxic.

The NTP bioassay referred to above[2] was a chronic feeding study of 50 male and 50 female F344 rats (6,000 or 12,000 ppm) and 50 male and 50 female B6C3F1 mice (3,000 or 6,000 ppm) for 103 weeks. Controls consisted of 50 untreated rats and 50 untreated mice of either sex. In addition to the significantly increased incidences of hepatocellular carcinoma, male rats had a statistically significant increased incidence of either hepatocellular carcinoma or neoplastic nodules. Degeneration of the seminiferous tubules and hypertrophy of cells in the anterior pituitary in increased incidences were also found in the high-dose male rats. A related compound, di(2-ethylhexyl) adipate (DEHA) was also considered carcinogenic by bioassay.

Studies in humans: Few studies of humans exposed to DEHP are available. A Russian study[11] examined the health status of 147 workers exposed to phthalate plasticizers in the manufacture of artificial leather and films based on PVC resins. This study reflects users of the compound, not producers as in the current NIOSH study. The workforce included 60 primers, 28 calender and mill operators, 35 mixing-apparatus operators and 24 inspectors. The ambient levels of vapors or aerosols of the plasticizers (mixed esters) ranged from 10 to 66 mg/m^3. The calender and mill operators had similar exposures. The mixture preparation section had exposures of 1.7 to 40 mg/m^3. Details of analytic methods and collection are not given. The authors found evidence of toxic polyneuritis thought to be occupationally-related, the frequency and degree of which increased with the length of stay on the job.

Theiss et al[12] performed a morbidity study of 101 workers employed in a DEHP production plant for an average 12 years (range 4 months to 35 years). DEHP had been produced at the plant since 1940. Atmospheric concentrations of DEHP ranged from 0.0006 to 0.01 ppm. Data on the workers were compared with those obtained from two in-house control groups with possible exposure to styrene and dimethylcarbiminic acid chloride (DMCC). No unusual findings were noted on general examination. Six workers with >20 years exposure, subjected to a "particularly thorough neurologic examination", revealed no indication of any neurologic disease or toxic nerve damage. Results of laboratory findings were generally comparable among the groups. However, among the DEHP, styrene and DMCC exposed, the following percent of findings "divergent from the norm" were reported: bilirubin, 4.0%, 1.1%, 1.9%, respectively; SGOT, 13.9%, 9.7%, 6.0%, respectively; SGPT, 14.9%, 22.6%, 10.0%, respectively; GGT, 25.7%, 21.5%, 26.0%, respectively; AP, 0.99%, 1.1%, not given, respectively. These differences were not statistically significant, although the DEHP group had the highest prevalence for two parameters and second highest for two others. An attempt was made in six exposed workers and three nonexposed personnel to detect DEHP and possible metabolites in blood or plasma and 24-hour urine. In plasma,

DEHP concentrations ranged from 0.5 to 7.0 mg/L in exposed workers and 0.5 to 10.0 mg/L in controls. Urine samples were processed by gas chromatography by the method of Albro. DEHP ranged from 0.2 (limit of detection) to 7.5 mg/L in exposed workers and from 0.2 to 1.5 mg/L in controls. Values for the monoester (the major metabolite in primates) were 61.6 amd 34.3 ng/day in exposed workers and 57.7 and 42.4 ng/day in controls. Questioning employees about premature births or miscarriages revealed that all 58 births since beginning employment at the DEHP plant were normal; one wife had had a miscarriage and later had a healthy child. In a chromosomal study by these authors[12] on selected workers, the frequency of aberrant metaphases in the exposed and control groups were similar. However, other researchers have found DEHP to be mutagenic in animals.[13]

Phthalic anhydride: Phthalic anhydride is a chemical reagent used in the manufacturing process of a variety of industrial products including plastics, epoxy resins and paints. It can be used as a curing agent for epoxies or, as in this investigation, as a starting material in the manufacture of DEHP and other phthalates. As well as a direct irritative effect on the bronchi and respiratory tract (in higher concentrations), sensitization and occupational asthma associated with this compound have also been described.[16] Symptoms of rhinorrhea, lacrimation and wheezing in addition to asthma were present in this worker, who also demonstrated positive skin and tests and higher serum titer of specific IgE antibody by the radioallergosorbent test (RAST). In fact, the first conclusive evidence that low molecular-weight substances in the workplace can be immunogenic was this description of IgE antibody against a conjugate of phthalic anhydride.[17]

2-ethylhexanol: The National Occupational Hazard Survey done by NIOSH estimated that 45,000 workers are potentially exposed to 2-ethylhexanol or products containing it. In 1978, the estimated U.S. consumption was 4.0×10^{11} gallons. Its primary use (78%) is as a plasticizer intermediate (70% for DEHP) but it is also used in perfumes and other toiletries; as a solvent for nitrocellulose, urea resins, enamels and alkyd varnishes and lacquers; as a wetting agent; in ceramics; and in paper coatings.[20,21] It is one of two initial metabolites of DEHP (MEHP is the other). This alcohol is apparently widespread in the environment and is discharged in plant effluents. No occupational standard for exposure to 2-ethylhexanol has been established by OSHA. No toxicological data in humans[20] or epidemiological studies or case reports examining the possible role of the alcohol in human cancer were found in the literature.[21] On mutagenicity testing, 2-ethylhexanol was negative in the Salmonella assay both with and without microsomal activation in tests conducted by NIOSH and the NTP.[20] However, concern exists that the 2-ethylhexyl moiety may play a role in the carcinogenic effects of DEHP and DEHA. Thus, this compound was nominated by EPA for carcinogenicity testing. A decision regarding

whether to test this compound for carcinogenicity is expected by NTP later this year (Canter D. NTP. Personal communication). A related compound, sodium 2-ethylhexyl sulfate, which is used primarily as a wetting and dispersing agent in the textile industry, has been tested in a carcinogenesis bioassay (feed study) by NTP. A draft of the NTP technical report on this compound, issued in August 1982,[19] concluded that under the conditions of the bioassay, the compound was not carcinogenic for F344/N rats or for male B6C3F$_1$ mice. There was an increased incidence of hepatocellular carcinoma in female mice which may have been associated with administration of sodium 2-ethylhexyl sulfate. The summary minutes from the peer review of this draft (September 22, 1982), agreed with these conclusions. The final report is in preparation.

VI. RESULTS

A. Environmental

During the initial survey in January 1982, environmental monitoring was conducted during the day shift of the 26th and the evening shift of the 27th, to determine airborne concentrations of DEHP and PA. Of three full-shift samples collected for PA from operators, only one was above the analytical limit of detection (7 ug/sample), which correlates to an air concentration of 0.05 mg/m^3. Expressed as a time-weighted average, this exposure is somewhat lower due to the initial of the two consecutive samples being reported as below the analytical limit of detection. This sample was collected from a "B" Operator who was involved with collection of process samples from "still tops" and day tanks. Aproximately 65% of this worker's time was spent in the contol room.

No personal samples collected for DEHP from the breathing zones of operators were above the 0.01 mg/sample analytical limit of detection. Two general area samples, collected from the third esterifier sample port and the carbon dump chute, were reported at 0.171 mg/m^3 and 0.154 mg/m^3, respectively. It should be noted that equipment malfunction caused a product overflow at the dump chute, which was probably responsible for the measurable concentrations of DEHP at this location. This occurrence is reportedly infrequent.

During the follow-up evaluation, environmental monitoring was again conducted for DEHP and PA, plus 2-ethylhexanol and dibutylphthalate (DBP). DEHP and PA samples were obtained over five shifts, primarily from the breathing zone of workers in most existing job catagories. Tables I through IV present results of environmental monitoring for these substances, grouped by job category. Of 50 samples obtained for

DEHP, six were reported above the analytical limit of detection (0.01 mg/sample), with exposure concentrations ranging from 0.02 to 4.11 mg/m^3, averaging 0.71. The measurable exposures were obtained from one maintenance worker and five process operators. The maintenance worker was engaged in repairing the DEHP esterifier and reportedly used respiratory protection. Samples obtained from "B" and "AA" operators over a single shift at the BEP plant were both reported at 0.026 mg/m^3. These operators did not use respiratory protection. All samples obtained from operators on the "DOP" side of the PA/DOP plant during a single shift were above measurable concentrations. The highest, reported at 4.11 mg/m^3, was obtained from the "B" operator who spent approximately six hours of the shift outside the control room, near the process, with no respiratory protection. The "A" operator also spent roughly six hours outside the control room and reportedly used respiratory protection for at least part of time. This worker's exposure was reported at 0.021 mg/m^3. The "AA" operator, who was exposed to an average DEHP concentration of 0.022 mg/m^3, spent approximately two hours outside the control room, with no respiratory protection.

Exposures to measurable quantities of phthalic anhydride were much more widespread. As indicated in Tables I through IV, exposures ranged from 0.01 to 0.20 mg/m^3 and were found in at least a portion of all manufacturing processes monitored. 2-ethylhexanol exposures ranged from 0.24 to 18.12 mg/m^3. These samples were obtained from PA, DOP, and BEP process operators in all job catagories ("AA", "A", and "B"). The highest reported result (18.12 mg/m^3) was obtained from the "B" operator who had the highest DEHP exposure (approximately 4 mg/m^3).

Of sixteen personal samples for DEHP collected from tank farm and PA/DOP operators by Badische from 1978 to 1981, three were above the analytical limit of detection, with results ranging from 0.0002 to 0.004 mg/m^3. Of seventeen general area samples for DEHP collected during this same time period, five were above the analytical limit of detection, with results ranging from 0.64 to 12.1 mg/m^3, averaging 1.1 mg/m^3. The personal exposure for the employee working in the area where the 12.1 mg/m^3 DEHP concentration was obtained (railcar manway) was less than 0.7 mg/m^3, which probably reflects the relatively short period of time spent in this area by the employee.

B. Medical

Initial Survey: During a review of medical records of PA/DEHP employees, company-generated surveillance data (1978-1981) was available for 14 of 33 of these employees assessed during the 1976 survey. Six of the 14 had been categorized as "abnormal" in 1976. Of these, three had had abnormal tests of liver function at least once using the laboratory's age-specific reference ranges, while 5 (87%) had had abnormalities using the reference ranges used by NIOSH in 1976. Thus the possibility of persistent abnormalities was raised; however, no epidemiologic analysis of the company's data had been performed. To this end, the data generated by the company was analyzed. The results of this analysis were communicated by letter to the company and union in November 1982. Briefly, in comparisons between four groups/departments (Maintenance, PA/DEHP, Palanil, and other), where significant differences in mean values for the enzymes SGOT, SGPT and AP were found, the PA/DEHP department tended to have the highest or second highest means. No significant differences between departments were found for the proportion of employees with abnormal liver profiles. This data was limited by a number of factors including missing identification of medications, poor participation leading to small numbers for analysis, no data for date of change of jobs, etc.

Follow-up survey

A total of 96 employees participated of whom one was on sick leave and is not included in further analyses. The remaining 95 included 66 from the bargaining unit (representing 90% participation) and 29 from salaried personnel (representng 42% participation). All employees had been in their current job for at least 0.5 years.

1. Exposure classification

As shown in Table V, the job titles were divided into Group 1 (Higher exposure), which included all chemical operators, tank farm operators, and mechanics spending most of their time in PA/DEHP; Group 2 (Lower exposure, non-administrative), which included all other hourly employees plus instrument technicians who had been in the bargaining unit until a few years previously) and first-line supervisors in maintenance and BEP; and Group 3 (No exposure: salaried/administrative). Although not all operators in Group 1 are equally exposed, and those in the Batch Ester plant (BEP) only produced DEHP a fraction of the time, NIOSH environmental sampling of workers in these job classifications revealed measurable DEHP exposure. A separate analysis of tank farm operators and DEHP "B" operators, the job classifications felt to be at highest risk for exposure to DEHP but too small a group for statistical comparisons, is also performed.

The employees evaluated were distributed in these exposure groups as follows: Group 1, 48; Group 2, 32; Group 3, 15. Of the 32 workers in the lower exposure group, five (16%) had held previous jobs at Badische for one year or longer that have been classified among the "higher" exposed ones. Because of our lack of knowledge concerning chronic effects due to past exposure to DEHP, there was uncertainty about whether they properly belonged in this group. Thus, for the inter-group comparisons that follow, these five individuals are not included in the lower exposure group.

2. Demographic data

The higher exposure group was comparable to the lower exposure group with respect to age and years of schooling (Table VI). Employees within the higher exposure group were more frequently non-white, and had significantly shorter mean duration of employment at current job (4 vs 7 years).

3. Questionnaire data

Before examining symptoms and liver function test results, Groups 1 and 2 were compared for possible confounding factors related to the liver (Table VII). There were no significant differences in any variables including distribution of current, ex- and never drinkers, and mean units/week of reported alcohol consumption (one "unit" of alcohol is 1 bottle or can of beer, 1 glass of wine, or 1 oz. of whiskey or other hard liquor).

The prevalence of reported gastrointestinal/constitutional symptoms, nervous system symptoms or symptoms of mucous membrane irritation in the higher and lower exposure groups was similar (Table VIII).

4. Liver function tests

LFT results are shown in Table IX. There were no significant differences in mean log-transformed values for SGOT, SGPT, or AP, between the higher and lower exposure groups. The mean log-transformed value for GGT was significantly greater in the higher exposure group ($t=2.45$, $p=0.0168$). The prevalence of abnormal LFT results was low and was similar in the two groups (Table IX). The comparisons were similar whether one applied the normal ranges shown (those reported by the laboratory) or the ranges used by NIOSH in the 1976 study.

There was no significant difference in mean log-transformed value for the serum bile acid, SLCG between the two groups (Table IX). The prevalence of abnormal SLCG values (> 1.5uM) was greater than for the enzyme tests but there was essentially the same in the two groups (Table IX).

The two job classifications felt to be at highest risk for exposure to DEHP, DEHP "B" operators and tank farm operators, were examined separately. Nine employees in these jobs participated in our study. The prevalence of abnormal LFTs in this group was as follows: SGOT 0/9; SGPT 0/9; AP 0/9; GGT 1/9.

5. Urine phthalate concentrations

The mean (log-transformed) pre-shift urine phthalate concentrations in the higher and lower exposure groups were very similar and were slightly higher in those with no exposure (salaried, administrative personnel) (Table X). The mean (log-transformed) post-shift phthalate concentration was higher but not statistically significantly so, in the higher exposure group. The mean (± S.D.) pre- to post-shift difference in the higher exposure group, an increase of 3.37 (± 9.4) nanomoles/ml, was significantly greater than that in the lower exposure group [mean decrease of 0.44 (± 5.8) nanomoles/ml] (p=0.04). The no exposure group showed (an even larger) mean decrease over the shift. Within Group 1, the mean increase of 3.37 nanomoles/ml was statistically significantly different from zero (n=45, p=0.02 by paired t-test, p=0.006 by signed rank test); in Group 2, the mean change (decrease of 0.44 nanomoles/ml), was not statistically significant (n=25, p=0.71 by paired t-test, p=0.35 by signed rank test); and in Group 3, the mean change (decrease of 0.76 nanomoles/ml), was not significant (n=14, p=0.46 by paired t-test, p=0.49 by signed rank test). A similar pattern of mean increase in Group 1 and mean decrease in the other two groups was observed when the concentrations were corrected to urine specific gravity of 1.024. However, the corrected post-shift (log-transformed) phthalate concentrations were significantly greater in the higher exposure than in the lower exposure group (p=0.04).

To further assess which job titles exhibited substantial increases over the shift, we arbitrarily defined a "substantial" increase as a post-shift value > 2 times the pre-shift value and post-shift value > 10 nanomoles/ml. Eight employees were identified who met this rule; all eight were chemical operators, but job titles varied (Table XI.) Thus, eight (24%) of 33 chemical operators with both pre- and post-shift urine specimens available displayed a substantial increase in urine phthalate concentrations over the shift while none of 36 other non-management, non-chemical operators

did (p=0.017, Fisher's exact test, 2-tailed). Of these eight chemical operators, personal exposure data on the day of urine collection was available in six. PA was detected in samples for four, ranging from 0.006 to 0.102 mg/m^3 and was below the limit of detection in the other two (less than 0.004 and 0.003 mg/m^3, respectively). PA concentrations did not appear correlated with urine phthalate concentrations.

The five highest post-shift phthalate concentrations (> 20 nanomoles/ml) occurred in four of these eight chemical operators (PA "A", DEHP "AA", BEP, DEHP "B") and in one salaried administrator who was unlikely to be near the reactors. The pre-shift phthalate concentration in this employee was actually higher than the post-shift.

The laboratory found little or no MEHP (a DEHP metabolite) in the urine samples. Thus the increased phthalates likely resulted from absorbed phthalic acid rather than DEHP.

VII. DISCUSSION

The environmental study found levels of airborne DEHP (and other substances) that were below existing standards. However, the development of these standards was not based on a consideration of possible carcinogenicity. As DEHP is considered by NTP to be a suspect carcinogen in animals and the International Agency for Research on Cancer (IARC) considered that there is sufficient evidence for the carcinogenicity of DEHP in mice and rats,[20] efforts should be made to keep occupational exposure as low as possible.

The medical study demonstrated no differences in the prevalence of reported symptoms, or of abnormal liver function test results, between employees considered at higher risk and those at lower risk of exposure to DEHP. There is little data available on groups occupationally exposed to phthalates. We have attempted to determine the utility of urinary phthalate as a tool for the biological monitoring of such groups. We have observed small but statistically significant mean increases in total urinary phthalate over the shift in higher exposed employees while decreases occurred in the other groups. This apparently indicates absorption over the shift resulting from occupational exposure, possibly by inhalation. The fact that only chemical operators displayed a substantial increase in urinary phthalate concentration suggests that the test is specific and increases the likelihood that the increases were related to occupational exposure. However, DEHP metabolites such as MEHP were not readily detected. Thus, rather than DEHP-derived phthalate, the increase may represent PA-associated phthalate, airborne levels of which were more frequently detected in this study.

In a cross-sectional study, exposure information is usually ascertained simultaneously with disease information. This was the case with this study. However it is important to assess the exposure with respect to the period of time that might be etiologically relevant to current disease (i.e. consideration of a meaningful induction period).[22] As the phthalates do not appear to have significant acute toxicity, it is likely that any associated hepatic disease would require some chronic period of exposure. In this study, all "higher exposure" employees had been in the current jobs for at least six months, with an average of 4 years. Those with previous, but not current, "higher exposure" were excluded from the comparison group.

The reasons why the current study did not demonstrate the association between work in the PA/DOP department and abnormal LFTs seen in the previous Hazard Evaluation at this facility are not clear. It is possible that the finding in the initial study was a chance occurrence or that error introduced into the design or analysis of the current investigation resulted in dampening of the association. Presumably, selection bias was largely prevented because knowledge of disease (i.e. abnormal liver function) was not known at the time of selection of entrants. The participation rate among the hourly workers, who largely composed the higher and lower exposure groups, was quite high. The 1976 study included all employees (including salaried/administrative personnel) in the groups being compared to production workers and these groups may not have been suitable for comparison.

The current classification of exposure was based on consensus discussion plus environmental sampling. The 1976 study simply made comparisons among departments without regard to jobs and no air sampling was done. However, exposure assignment was similar in the two studies; thus misclassification of exposure information is unlikely to have reduced any possible association. Without exposure data from the earlier study, one can only speculate that greater exposures may have occurred at that time and accounted for the disease found. The same parameters were used to determine disease information in the two studies. It is conceivable that misclassification of disease information occurred because of the inability of available tests to detect low grade, chronic liver dysfunction (which may, in reality, be more prevalent in DEHP-exposed workers) that does not cause necrosis with elevated serum enzyme levels.

Possible confounding factors (factors associated with both exposure and with disease) must be considered. It is possible that there were other hepatotoxic chemical exposures in the 1970s that are no longer present. For example, vinylidene chloride (VDC), the production of which had been discontinued just prior to the earlier study, may have resulted in residual liver disease if some VDC workers were in PA/DEHP at the time of that investigation. Other potentially confounding exposures were considered in this study and found not to be differentially distributed between the groups. Of note, the distribution of reported alcohol consumption status

was similar. The 1976 final report[1] did not address this factor although the data was collected. It is conceivable that differences in alcohol consumption may have contributed to the association found.

VIII. RECOMMENDATIONS

1. The company should continue the practice of providing pre-employment and periodic history and laboratory evaluation directed at detecting liver dysfunction. As well as proper follow-up of individual abnormalities, the data should be analyzed periodically in an epidemiologic manner to seek changes that might be attributable to workplace exposures.

2. Past and present personnel lists should be preserved to permit future assessment of morbidity and mortality.

3. Because DEHP is a suspect carcinogen, efforts should continue to keep exposures as low as possible.

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X. 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), 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. Badische Corporation, Kearny, New Jersey
2. United Rubber Workers, International Union
3. United Rubber Workers, Local 234
4. NIOSH, Region II
5. OSHA, Region II
6. Consumer Product Safety Commission

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

Di(2-ethylhexyl)phthalate and Phthalic Anhydride
Exposure Concentrations: PA/DOP

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Date	Duration	Location/Operation	Concentration(mg/m^3) DEHP	PA
03-07-83	07:45-15:51	PADOP/Maintenance	0.071	0.026
03-07-83	07:23-14:15	DOP/"B" Operator	<0.023	0.006
03-07-83	07:47-15:42	PADOP/Maintenance	<0.023	0.011
03-07-83	07:37-14:21	DOP/"AA" Operator	<0.026	0.066
03-07-83	07:08-14:29	PA/"A" Operator	<0.026	0.007
03-07-83	07:50-15:35	PADOP/Maintenance	<0.023	0.018
03-07-83	07:48-15:48	PADOP/Maintenance	<0.022	0.042
03-07-83	07:46-15:40	PADOP/Maintenance	<0.024	<0.003
03-08-83	14:53-22:25	PA/"A" Operator	<0.026	0.015
03-08-83	15:02-22:30	DOP/"AA" Operator	<0.026	0.008
03-08-83	15:04-22:25	DOP/"B" Operator	<0.025	0.006
03-09-83	22:35-06:25	PA/"A" Operator	<0.021	0.004
03-09-83	22:44-06:25	DOP/"AA" Operator	<0.021	<0.004
03-09-83	22:47-06:26	PA/"B" Operator	<0.024	0.187
03-09-83	22:50-06:30	PA/"AA" Operator	<0.024	<0.004
03-09-83	22:55-06:20	DOP/"B" Operator	<0.019	<0.003
03-10-83	07:15-11:00	PA/"B" Operator	<0.050	<0.007
03-10-83	08:43-14:56	PADOP/Inst. Technician	<0.026	<0.004
03-10-83	22:43-06:05	DOP/"B" Operator	4.106	0.022
03-10-83	23:25-06:06	DOP/"A" Operator	0.024	0.102
03-10-83	22:52-06:08	DOP/"AA" Operator	0.022	<0.044

TABLE II

Di(2-ethylhexyl)phthalate and Phthalic Anhydride
Exposure Concentrations: BEP

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Date	Duration	Location/Operation	Concentration (mg/m^3) DEHP	PA
03-07-83	08:04-15:29	BEP/Maintenance	<0.024	<0.004
03-07-83	08:03-15:29	BEP/Maintenance	<0.024	0.044
03-07-83	08:02-15:26	BEP/Maintenance	<0.022	0.021
03-08-83	15:17-22:26	BEP/Supervisor	<0.027	0.010
03-08-83	15:24-22:21	BEP/Helper	<0.028	0.009
03-08-83	15:28-22:21	BEP/"B" Operator	<0.026	0.007
03-08-83	15:54-22:21	BEP/"AA" Operator	<0.038	0.007
03-09-83	23:30-06:18	BEP/"B" Operator	<0.027	0.005
03-09-83	23:22-06:20	BEP/"B" Operator	<0.025	<0.004
03-10-83	07:27-14:24	BEP/"B" Operator	0.026	0.021
03-10-83	07:37-14:24	BEP/"AA" Operator	0.026	0.016
03-10-83	11:21-06:16	BEP/"B" Operator	<0.027	0.015
03-10-83	11:09-06:17	BEP/"AA" Operator	<0.025	0.005

TABLE III

Di(2-ethylhexyl)phthalate and Phthalic Anhydride
Exposure Concentrations: Tank Farm

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Date	Duration	Location/Operation	Concentration (mg/m^3) DEHP	PA
03-07-83	08:12-14:20	TF/Operator	<0.028	0.148
03-07-83	10:56-15:55	TF/Operator	<0.036	0.052
03-07-83	08:45-14:20	TF/Operator	<0.034	0.017
03-09-83	22:47-06:16	TF/Operator	<0.023	0.203
03-10-83	07:51-14:35	TF/Operator	<0.022	0.017
03-10-83	07:53-14:35	TF/Operator	<0.025	0.036

TABLE IV

Di(2-ethylhexyl)phthalate and Phthalic Anhydride
Exposure Concentrations: Controls

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Date	Duration	Location/Operation	Concentration (mg/m^3) DEHP	PA
03-07-83	11:17-16:00	Q.C. Lab/Technician	<0.041	0.019
03-07-83	08:25-13:06	Q.C. Lab/Technician	<0.038	0.011
03-08-83	15:09-22:25	Boiler Plant/Operator	<0.026	<0.004
03-08-83	15:40-22:13	Waste Water/Operator	<0.030	<0.004
03-10-83	07:57-14:43	Boiler Plant/Operator	<0.030	<0.004
03-10-83	08:02-14:43	Waste Water/Operator	<0.025	<0.004
03-10-83	08:11-15:50	Warehouse/Warehouseman	<0.021	<0.003
03-10-83	08:31-15:20	Office/Engineer	<0.024	<0.004
03-10-83	08:22-15:25	Office/Engineer	<0.022	<0.004
03-10-83	08:57-15:00	Varied/Inst. Technician	<0.026	<0.004

TABLE V

Classification of Job Titles into
Exposure Groups

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Group 1: Higher Exposure

Chemical Operators:
- DEHP "A" Operator
- DEHP "B" Operator
- PA "B" Operator
- PA "A" Operator
- PA "AA" Operator
- BEP "B" Operator
- BEP "AA" Operator

Mechanics spending most of time in PA/DEHP.
Tank farm operators.

Group 2: Lower Exposure

Mechanics spending most of time in BEP and rest of plant
Instrument technicians
Storekeepers
Wastewater operators
Boiler operators
R&D technical applicators, quality control
Supervisors (maintenance and BEP)

Group 3: No Exposure

Administrative, Safety
Engineering
Accounting

TABLE VI

Demographic Data

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

		Higher Exposure Group 1 (N=48)	Lower Exposure Group 2 (N=27)	No Exposure Group 3 (N=15)	t* (unless x^2 specified)	p value **
Age(yr):	Mean (S.D.)	46 (10.0)	46 (10.4)	38 (10.2)	0.12	0.90
	Range	28-66	24-64	25-59		
	Median	45	45	35		
Race (W/B/O)		10/30/8	10/8/9	13/1/1	X^2=7.5 df=2	0.025
School:	< Gr 11	17 (35%)	9 (33%)	0 (0%)		
	Gr 12	18 (38%)	11 (41%)	2 (13)	X^2=0.077	0.962
	> Gr 12	13 (27%)	7 (26%)	13 (87%)	df=2	
Years, current job:	Mean (S.D.)	3.9 (3.0)	6.9 (4.5)	3.8 (3.5)	3.07	0.0039
	Range	0.5-11.6	0.8-19	0.4-11.1		
	Median	2.4	7.6	2.8		
Years, total at company	Mean (S.D.)	11.8 (4.5)	9.9 (4.6)	6.2 (3.7)		
	Range	3-37	1-20	1-11	1.73	0.089
	Median	11	10	6		

*Statistical comparisions are between higher exposure group (Group 1) vs lower exposure group (Group 2)

** two-tailed tests

TABLE VII

Background Data from Questionaire

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

	Higher Exposure (n=48)		Lower Exposure (n=27)		No Exposure (n=15)		P Value[a,b]
	#	(%)	#	(%)	#	(%)	
Travel out of country	22	(46)	17	(63)	14	(93)	0.154
Hobbies with chemicals	2	(4)	3	(11)	7	(47)	0.344
Previous liver disease	7	(15)	5	(19)	1	(7)	0.655
Blood transfusion	1	(2)	2	(7)	0	(0)	0.293
General anaesthesia	22	(47)	14	(52)	9	(60)	0.9
Malaria	2	(4)	1	(4)	0	(0)	1.000
Potentially hepatotoxic medications	20	(42)	11	(41)	10	(67)	0.938
Ever used needles or syringes for non-medical purposes	1	(2)	0	(0)	0	(0)	1.000
Tatoos	3	(6)	2	(7)	0	(0)	1.000
Pesticide exposure	0	(0)	0	(0)	2	(13)	--
Ever worked with Beryllium	0	(0)	1	(4)	0	(0)	0.36

Continuation of Table VII, Background Data from Questionnaire

Alcohol consumption				
Current drinker	33 (69)	18 (67)	12 (80)	0.474
Ex-drinker	8 (17)	7 (26)	2 (13)	
Never drinker	7 (14)	2 (7)	1 (7)	chi-square=1.49 df=2
Units/week [c] among Present Drinkers				
Mean (S.D.)	8.9 (8.8)	8.7 (12.9)	6.7 (6.3)	p=.936
Range	0.2-33	0.2-52	0-20	(t=0.073)
0-6 units/week	42%	56%	58%	p=0.36
$\geq$ 7 units/week	58%	44%	42%	(Chi-square=0.848)

a Chi-square or Fisher's Exact Test, 2-tail
b Statistical comparisions are between higher exposure group (Group 1) vs lower exposure group (Group 2)
c Units/week as explained in text

Table VIII

Prevalence of Reported Symptoms

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

	Higher Exposure (N=48)	Lower Exposure (N=27)	No Exposure (N=15)	p value b,c,d
	# (%)	# (%)	# (%)	
Gastrointestinal/ constitutional				
Poor appetite	4 (8)	3 (11)	0 (0)	0.70
Nausea	2 (4)	1 (4)	1 (7)	1.0
Easily tired	11 (23)	5 (19)	0 (0)	0.665c
Feeling down	9 (19)	6 (22)	0 (0)	0.772c
Nervous system symptoms				
Headaches	4 (8)	2 (7)	0 (0)	1.000
Lightheadness	2 (4)	2 (7)	1 (7)	0.6628
Mucous membrane irritation				
Eyes	18 (38)	5 (19)	2 (13)	0.097c
Nose	15 (31)	10 (37)	3 (20)	0.618c
Throat	9 (19)	6 (23)	5 (33)	0.665c

a. All questions refer to period "in the past month" except skin which was during past three months

b. All are Fisher's Exact Test, 2 tailed, except those marked "c"
c. Chi-square
d. Statistical comparisons are between higher exposure group (Group 1) vs lower exposure group (Group 2)

Table IX

Liver Function Test Results

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Test	Mean (S.D.), Log-Transformed				Test (Normal Range)	Abnormal Result			
	Higher Exposure (n=48)	Lower Exposure (n=27	No Exposure (n=15)	p[a,b]		Higher Exposure (n=48) # (%)	Lower Exposure (n=27) # (%)	No Exposure (n=15) # (%)	p[a,c]
SGOT	1.4 (0.2)	1.4 (0.1)	1.3 (0.2)	0.7553	SGOT (up to 70)	1 (2)	0 (0)	0 (0)	1.000
SGPT	1.4 (0.3)	1.4 (0.3)	1.4 (0.2)	0.4200	SGPT (up to 70)	2 (4)	1 (4)	0 (0)	1.0
AP	1.5 (0.3)	1.4 (0.2)	1.4 (0.2)	0.5701	AP (up to 50)	2 (4)	1 (4)	1 (7)	1.0
GGT	1.5 (0.3)	1.3 (0.3)	1.3 (0.4)	0.0168	GGT (up to 70)	6 (13)	1 (4)	1 (7)	0.41
SLCG	-0.09 (0.39)	-0.15 (0.37)	-0.09 (0.55)	0.5416	one or more tests abnormal	10 (21)	3 (11)	1 (7)	0.35
					two or more tests abnormal	3 (6)	1 (4)	1 (7)	--
					SLCG (up to 1.5 uM)	11 (23)	7 (26)	7 (47)	0.7776[c]

a Statistical comparisons are between higher exposure group (Group 1) and lower exposure group (Group 2)
b t-test, two-tailed
c chi-square

Table X

Urine Phthalate Concentrations

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

	Higher Exposure (46)	Lower Exposure (26)	No Exposure (15)	Pa,b
Pre-Shift (log) [Mean (S.D.)]	0.60 (0.34)	0.58 (0.35)	0.77 (0.34)	0.8884
Post-Shift (log)	0.78 (0.36)	0.69 (0.21) (only 25 samples)	0.72 (0.34) (only 14 samples)	0.1817
Difference	3.37 (9.4)	-0.44 (5.8)	-1.05 (5.2)	0.042

a Statistical comparisons are between higher exposure groups (Group 1) and lower exposure group (Group 2)

b t-test, 2-tailed

TABLE XI

Employees Showing Substantial Pre- to Post-shift Increases in Urine Phthalate Concentrations*

Badische Corporation
Kearny, New Jersey
HETA 82-032

March 7-11, 1983

Case No.	Pre-shift Phthalate Concentration (nanomoles/ml)	Post-shift Phthalate Concentration (nanomoles/ml)	Job Title
0002	4.7	26.7	PA "AA" operator
0040	4.9	44.3	DEHP "AA" operator
0041	6.9	14.4	DEHP "B" operator
0047	6.8	30.4	BEP "B" operator
0052	2.1	18.8	BEP "AA" operator
0055	4.9	22.6	DEHP "B" operator
0062	4.7	11.1	BEP "B" operator
0093	7.0	17.2	PA "A" operator

* Substantial increase: based on decision rule - post-shift concentration greater than twice (pre-shift concentration) and post-shift concentration greater than 10 nanomoles/ml

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