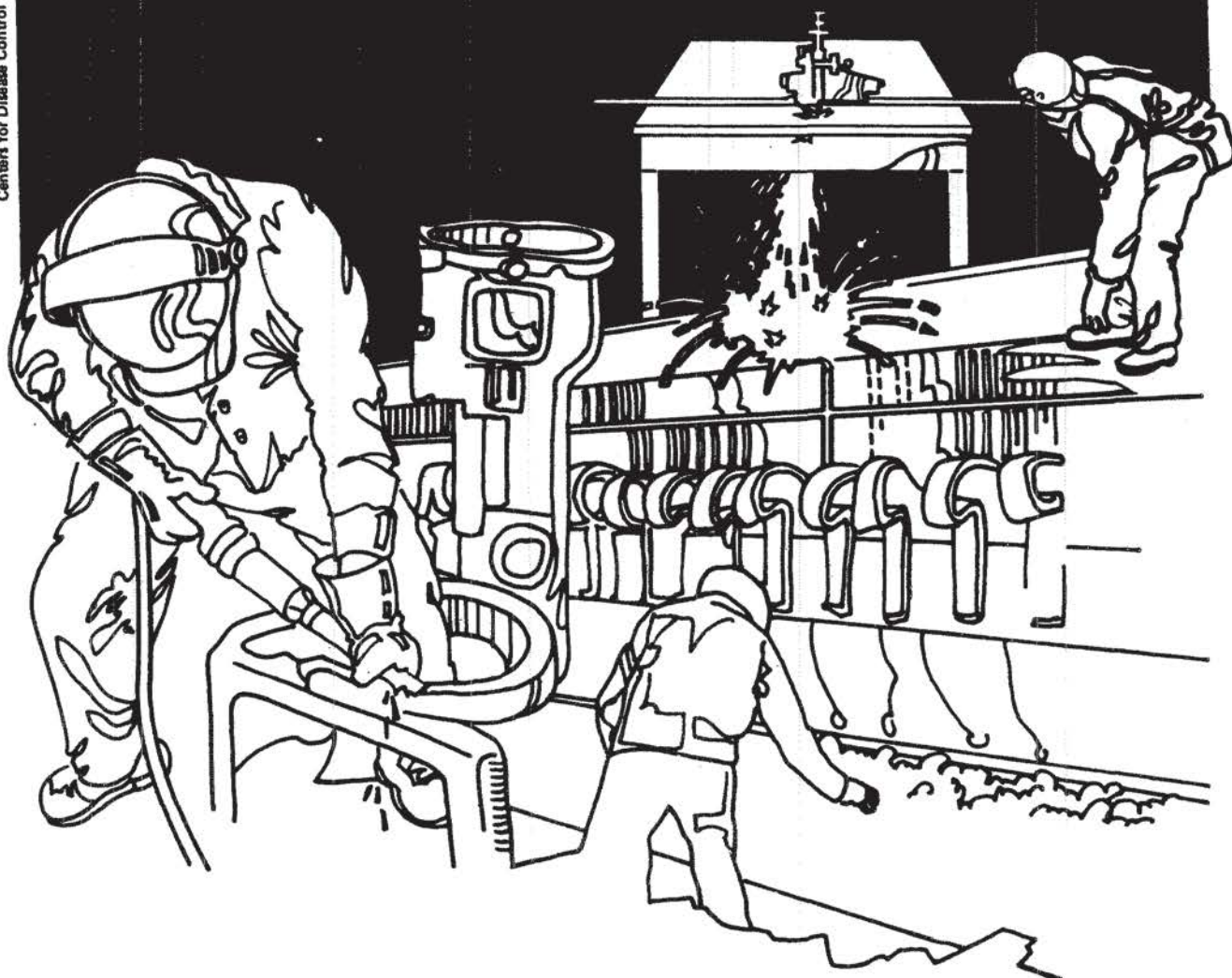


NIOSH



Health Hazard Evaluation Report

HETA 82-091-1176
TODD MEADOWS GIN
ST. CLAIR, ALABAMA

PREFACE

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

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

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

HETA 82-091-1176
September 1982
Todd Meadows Gin
St. Clair, Alabama

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I. SUMMARY

On January 18, 1982 the National Institute for Occupational Safety and Health received a request for a health hazard evaluation at the Todd Meadows Gin in St. Clair, Alabama. The request stated that a ginner complained of respiratory distress, unconsciousness, chills, lapse of memory and double vision which he believed to be caused by chemical sprays used on cotton plants prior to ginning of the cotton.

An on-site survey of the facility was conducted February 4, 1982. The ginning and baling of the cotton in this operation normally employs five persons; one ginner, two balers and two gin loaders. The gin was not operating, so the description of operations and estimates of expected dust exposure are based on judgment and information provided by the ginner.

At the end of the last cotton ginning season, the ginner sent, in plastic bags, parts of his clothing, his dust mask and dust from the floor of the building to the Alabama Department of Agriculture and Industries, Pesticide Residue Laboratory Division, Auburn, Alabama, for analysis of pesticide content. Galecron, an insecticide, was detected in concentrations of 0.4 to 5.8 parts per million in the apparel, and 0.3 in the floor dust. Other pesticides were not detected. The ginner was interviewed by the investigation team's physician, who also interviewed the ginner's personal physician.

No specific material or environmental condition was identified as being the direct cause of the health problem reported. From the ginner's history it may be concluded that he has a combination of bronchitis symptoms related to heavy smoking and an atypical byssinosis-like syndrome related to cotton dust exposure at the gin house. The possibility of an idiosyncratic reaction from exposure to residuals of chemicals sprayed in raw cotton in the field prior to picking and processing seems an unlikely explanation for his episodes of tremor, double vision, and syncope. However, there is the potential for exposure to sprayed chemicals for field workers, gin operators and baler operators.

Further studies need to be made at the gin house during ginning. Detailed discussion of a follow-up survey and recommendations are contained in the body of the full report.

Key words: SIC 0724, cotton ginning, respiratory illness, insecticides, defoliants.

II. INTRODUCTION

On January 18, 1982 an employee requested a health hazard evaluation at the Todd Meadows Gin in St. Clair, Alabama. The request stated that an employee, the ginner, complained of respiratory distress, unconsciousness, chills, lapse of memory and double vision which he believed to be caused by chemical sprays used on cotton plants prior to ginning of the cotton.

An on-site survey of the facility was conducted February 4, 1982, by two industrial hygienists and a physician. The goals of the evaluation were to interview and record the medical history of the employee and evaluate the environmental conditions where possible. The ginning season was over, and the gin was not expected to be operated again until Fall 1982.

III. BACKGROUND

The health hazard evaluation was begun at the Todd Meadows Gin by the Occupational Health Studies Group, University of North Carolina, Chapel Hill, North Carolina, under a cooperative agreement with, and as a representative of, NIOSH. A current employee who operates the cotton gin, the ginner, met the investigators and took them on a step-by-step inspection of the cotton ginning process. The ginner had been operating and maintaining the gin each ginning season (September-December) since 1975; however, his complaints of health problems are more recent. His concern is that the cotton processed more recently may have been contaminated with chemicals, possibly defoliants, and that exposure to one or more of these chemicals was the cause of his acute symptoms.

The ginning and baling of cotton in this operation normally employs five persons; one operator (the ginner), two balers and two gin loaders. The gin was not operating, so the following description of operations and estimate of expected dust exposure are based on judgment and information provided by the ginner. The gin loaders work outside the building, operating the input vacuum hoppers and transporting trucks to and from the point of unloading at the gin building. Their exposure to airborne cotton dust and physical contact with the cotton is expected to be slight. The two balers, who work inside the gin building near one end of the gin, probably have some exposure to cotton dust and fibers. The ginner has perhaps the greatest exposure to cotton, both the dust and the fiber, due to the closeness of the operating console to the process machinery, and his constant overseeing of the operation.

At the end of the last cotton ginning season, the ginner sent, in plastic bags, parts of his clothing, his dust mask and dust from the floor of the building to the Alabama Department of Agriculture and Industries, Pesticide Residue Laboratory Division, Auburn, Alabama, for analysis of pesticide content. The ginner requested the Pesticide Residue Laboratory to analyze for common pesticides used in the county during the cotton growing and harvesting season. At the time of the evaluation survey, the results from the laboratory on the samples sent had not been received by the ginner; they were supplied later to the survey group.

IV. METHODS AND MATERIALS

A. Environmental

Environmental evaluation consisted of an interview with the gin operator (the ginner) about environmental conditions during the ginning seasons, a walk-through industrial hygiene survey and a request for a copy of the pesticide data from the bulk samples of clothing, face masks and dust from the floor, analyzed by the Alabama Department of Agriculture and Industries, Pesticide Residue Laboratory Division. Other employees were not available for interview or comment. Environmental sampling was not undertaken because the gin was not operating at the time of the survey.

B. Medical

Medical evaluation consisted of an interview with the ginner to obtain his medical history and information on the nature and extent of current and former exposures to cotton field sprays, cotton dust and fibers. The employee's personal physician was contacted by telephone to obtain information about his medical findings on the employee.

V. EVALUATION CRITERIA

Criteria for evaluation of the health status of the employee and evaluation of the environmental conditions during ginning operations, and assessment of any relationship between the two, was primarily based on the judgments of the physician and the industrial hygienists. Results from the Pesticide Residue Laboratory also aided the hygienists to judge the extent of exposure to pesticides.

VI. RESULTS AND DISCUSSION

Results

A. Environmental

The employee interviewed previously worked as a farm machinery mechanic. He began work in cotton gins in 1972, doing machinery maintenance and providing relief time to gin operators. In 1975, he began his present position as operator of the Todd Meadows Gin. In this work, he operates a gin control console positioned to the side and center of the gin building. He is required to remove accumulations of trash and lint from various parts of the cotton cleaning apparatus. He must be stationed within the gin house constantly during operations. Four other workers are employed during ginning operations, two of them work outside loading unprocessed cotton from the field into the vacuum hopper which feeds the gin, and two work inside but near the exit doors of the gin building as balers and loaders of the ginned cotton. It appears that these workers are not exposed to as high concentrations of cotton dust and fiber as the gin operator, and none have experienced similar symptoms according to the employee interviewed.

Observations by the investigating team of various processes put into operation by the ginner during the walk-through survey indicated potentially highly dusty work areas. Even in areas where the process was not put into operation large accumulations of trash and lint were observed on the floor, exhaust fans, machinery surfaces and building rafters and side supports. Reportedly the ginner on occasions wears a dust mask when performing maintenance and cleaning while the gin is in operation. At the gin console where he spends most of his time during operation the dust mask is not worn. Other workers do not wear dust masks while performing their duties.

During the 1981 ginning season the ginner accumulated a list of spray chemicals used on cotton fields of farms which grow cotton processed at the Todd Meadows Gin. The names of the spray chemicals were gathered from barrels and bags used by the farmers or consultants hired to spray the cotton fields. The names of these chemicals with restriction information are shown in Table I. They are primarily insecticides. However, later in the year other chemicals not on this list may have been used, such as plant growth regulators, defoliants and desiccants.

Data received from the Alabama Department of Agriculture and Industries, Pesticide Residue Laboratory Division on insecticide and defoliant content in the clothing and face mask worn by the ginner and in bulk floor dust samples are listed in Table II.

These samples were received by the Pesticide Residue Laboratory on December 3, 1981, the end of the ginning season, and the results were reported back to the ginner February 25, 1982. Environmental samples were not taken by the survey team because of the lapse in time since operation and lack of information on scraps of cotton remaining in the gin building.

According to information gathered during the evaluation, most of the pesticides being used to spray cotton during the growing season through harvest are either restricted or proposed for restriction by EPA. Pesticide formulations bearing the labeling RESTRICTED USE PESTICIDE may be sold only by licensed dealers and purchased or used only by licensed commercial applicators, public operators and consultants, certified or licensed structural pest control applicators and certified private pesticide applicators or by persons working under their direct supervision. The survey team had no way of learning whether use restrictions were observed during application of pesticides to cotton ginned at this gin.

While strict regulations and guidelines govern the purchase and application of pesticides, workers who are responsible for checking the effectiveness of the chemical sprays, harvesting, hauling and processing the cotton have few or no guidelines for reducing exposure to sprayed pesticides. Workers may enter fields soon after spraying to harvest and deliver cotton to the gin, where gin house workers may also be exposed to residual pesticides on the cotton and cotton trash.

B. Medical

Four episodes of acute symptoms were reported by the employee interviewed, the ginner. The first occurred on or about October 1, 1981, a working day at the gin. He reported that, while at work, he experienced no coughing, shortness of breath or other untoward symptoms. However, one or two hours after returning home for the evening, he began to experience tremors and double vision and stated that he passed out in his chair. These symptoms were apparently quite transient. For the two days previous to this episode, he noted dryness of mouth and throat and prolonged but nonproductive cough. No further similar symptoms were reported until October 17, 1981 when, while working at the control console in the gin house, he experienced lightheadedness and double vision. Feeling very weak, he sat on the floor and apparently passed out or became unaware of his surroundings for one hour. He stated that he then was able to return to his work at the console for four or five more hours. A third and similar episode occurred on October 31, 1981, a work day. Symptoms began at home, after work, and again consisted of tremors and double vision. On this occasion he was hospitalized by his private physician. The fourth and last episode occurred on November 13, 1981, the day after his release from the

hospital. On November 13th, he went to the cotton gin, worked for three to four hours and returned home. At 8:00 p.m. he experienced tremors, chills, shortness of breath, and apparent wheezing. He was re-hospitalized about midnight of the same day. Since this fourth episode, he has not worked in the gin, and he states he has had no recurrences of these acute symptoms.

In his past medical history, the employee born November 1937 reported good health, and that he has smoked 2-3 packs of cigarettes per day for many years. In 1958 he was treated for a bleeding peptic ulcer; in 1962 he was hospitalized for a severe influenza illness and in 1963 had an appendectomy. In September 1978 he suffered an episode of productive cough and sharp pains in his chest. He was hospitalized at that time and was found to have a "spot" on his lung. His diagnosis was chronic bronchitis. He has been treated with Aminophylline and Brethine, both medications being given as bronchodilators for treatment of chronic bronchitis, emphysema, and/or asthma. From October 1981 to January 1982 he was also treated with oral Prednisone at a dose of four 5mg tablets daily.

He denied Monday morning chest tightness, wheezing or cough, but did admit to intermittent shortness of breath, nonproductive cough and chest tightness occurring during the cotton ginning seasons since 1978. These symptoms were more pronounced toward the end of each work week rather than at the beginning. He also admitted to recurrent cough and sputum production throughout the year and averages one chest cold per year. He gave a history of hay fever since age 12, manifested by runny nose and sneezing. His oldest brother had asthma and two of his brother's daughters have asthma.

The employee's personal physician was contacted by telephone on February 4, 1982. He stated that the ginner has a diagnosis of chronic bronchitis. During the hospitalization of November 14-18, 1981, for an apparent syncopal episode, blood gases, blood chemistries, EEG and brain scan were negative. Pulmonary function tests were within normal limits. His doctor did not believe that his symptoms were clearly related to work exposure. He was discharged on bronchodilators and Prednisone.

Discussion

A. Walk-Through Survey

According to the employee interviewed and observations made by the investigating team, airborne particulates consisting of cotton fibers and dust (possibly with residual pesticides) would likely be in high concentrations in the gin house during ginning. This likelihood is reinforced by the dusty appearance of the structures and machinery within the gin house during the survey. No clean-up of the facility had been made after the last truckload of raw cotton was processed.

Due to the concern for pesticide exposure to workers, bulk samples were collected by the ginner and sent to a laboratory for pesticide analysis. The actual time from the date of gathering the samples to the date of analysis at the Pesticide Residue Laboratory is not known. However, from the date of gathering the samples to date of reporting is approximately 3 months. If several months did indeed elapse before analysis, decomposition of chemicals in the samples might have occurred. Also, transportation of the samples to the laboratory was not supervised, and there may have been contamination of or chemical loss from the samples.

B. NIOSH-HHE Reports

NIOSH final reports on other evaluations of gins in Colorado are not yet available. According to one of the NIOSH hygienists involved, environmental and medical samples were taken at these gins to determine chemical and noise exposure of the workers and the possible toxic effects of chemical exposures. Environmental evaluation included collection of air samples to determine exposure to cotton dust, chemically-laden dust materials, nuisance dust and noise. Medical evaluation consisted of determination of blood cholinesterase levels in employees. Exposure to noise and chemically-laden dust materials appeared to present the greatest health threats in these gins.

The survey showed that all of the noise levels in both area and personal tests exceeded the permissible exposure limit set by Occupational Safety and Health (OSHA), 90dBA.

Blood samples were taken from the gin workers and examined for red cell cholinesterase levels. The early and late season tests indicated that some gin workers were possibly affected by organophosphate residues in the raw cotton to the extent that their cholinesterase levels were lowered.

According to the NIOSH hygienist, an interim progress report concluded that based on the environmental chemical dust analyses and the cholinesterase levels obtained in the medical study, the cotton picking machine operators and the cotton trailer packing workers should also be evaluated for chemical exposure. DEF, a defoliant sprayed heavily in the last part of the season, was present in all material examined. It was concluded that exposure is affecting the cholinesterase level in field, transport and gin workers.

Studies of DEF and FOLEX in cotton fields and gins in Texas and California indicated air concentrations of both defoliants were detectable during operations^{2,3}. On the basis of current animal toxicity data, clinical effects from DEF exposure were judged to be unlikely at the dermal and inhalation exposure levels observed for cotton picking machine operators.³

C. General Toxicologic Effects of Pesticides

The following information about general toxicologic effects of pesticides is abstracted from the NIOSH criteria for a recommended standard...Occupational Exposure During the Manufacture and Formulation of Pesticides.⁴

Approximately 90% of all present-day pesticides are organic compounds. Insecticides consist primarily of organochlorine, organophosphorus, and carbamate compounds. Fumigants include halogenated hydrocarbons and inorganic gases. Herbicides include amides, arsenicals, carbamates and thiocarbamates, organophosphorus compounds, and substituted ureas. Fungicides include thiocarbamates, phthalimides, and organotin compounds. The production of these compounds involves many chemical processes including chlorination, alkylation, nitration, phosphorylation, sulfonation, and bromination.

Pesticides have caused diverse toxic effects on various human and animal organs and organ systems including the liver, kidneys, skin, lungs, brain, nervous system, and eyes. Certain pesticides appear to be carcinogenic in humans and others have produced tumors of vital organs in test animals. They have also caused structural and functional defects in unborn experimental animals and mutagenic changes in heredity characteristics in in vivo and in vitro test systems.

The many types of chemical compounds used as pesticides can be grouped on the basis of chemical structure into generic classes such as chlorinated hydrocarbons, organophosphorus, carbamates, and chlorophenoxy acid esters and salts. While there are significant variations in the toxic effects of the individual pesticides within each structural class, common effects have been observed. Table III lists some of the pesticides, with their generic class, used on cotton prior to being processed at the Todd Meadows Gin.

(a) Organochlorine Insecticides

Organochlorine (OC) compounds are all nonpolar substances and thus are soluble in lipids and organic solvents and are relatively resistant to metabolism or degradation. Consequently, these compounds have a strong tendency to penetrate cell membranes and to be stored in the body fat. Chronic, long-term exposure to these compounds usually presents a more serious problem than acute exposure.

OC pesticides primarily tend to damage the liver and kidneys. The hazards from skin absorption are small when the material is dry or in powdered form. On the other hand, when dissolved in oil or organic solvents, the materials are well absorbed through the skin and constitute a considerable hazard. Behavioral changes, disturbances of sensory and equilibratory functions, involuntary activity of skeletal muscles, and depression of vital centers have also been attributed to exposure to OC insecticides.

(b) Organophosphorus Insecticides

There are a large number of OP insecticides in use. They include phosphates, phosphonates, phosphoramidates, pyrophosphates, thio-pyrophosphates, and phosphorothioates.

In contrast to the OC insecticides, the OP compounds present a high hazard of acute intoxication which varies considerably from compound to compound. Parathion and fensulfothion are very toxic, with oral LD50's in rats of about 2 mg/kg. Malathion is one of the least toxic compounds, with an oral LD50 in rats of 1,400 mg/kg. These substances exert their toxic effects through their ability to inhibit cholinesterases (ChE's). OP compounds containing a P=S nucleus, such as parathion, must be metabolically activated by exchanging an oxygen atom for the sulfur. Animals, man, and insects all perform this activation. In mammals, the activation is done by microsomal oxidases of the intestinal wall and liver. Other OP compounds do not require metabolic activation. The inhibition of ChE by active forms is essentially irreversible and renders their toxic actions persistent until the inhibited enzymes are replaced by newly produced ones. Repetition of a small dose may finally result in serious intoxication even though each single dose may inhibit only a small percent of the ChE activity. The symptoms result from the accumulation of excessive quantities of acetylcholine at peripheral, ganglionic, and central nerve endings and from an elevated concentration of acetylcholine in the blood plasma and interstitial fluids. Poisoning with compounds for which the inhibition of ChE is reversible, such as tetraethyl diphosphate (TEPP), is naturally more amenable to therapy than is poisoning by compounds which cause irreversible inhibition.

Increased bronchial secretions, salivation, sweating, bradycardia, miosis, muscular weakness, hyperglycemia, low blood pressure, anxiety, headache, neurosis, slurred speech, disorientation, and convulsions are signs and symptoms that characterize poisoning by organophosphorous compounds. Respiratory failure is the most usual cause of death from a single, high dose. Such failure results from a combination of blockage of the respiratory tract from excessive secretion from glands of the mouth and respiratory tract, by possible bronchoconstriction, and by paralysis of the respiratory areas of the brain stem.

The degree of acute intoxication by most OP compounds may be gauged readily by the measurement of the extent of inhibition of acetylcholinesterase (AChE) in red blood cells (RBC's) or of the nonspecific ChE present in plasma. Some evidence has accumulated that the chronic depression of AChE activity by OP compounds may be associated with behavioral changes, but there is some doubt of the scientific validity of these conclusions. Based on analysis of available

human and animal data pertaining to behavioral changes attributed to OP pesticides, it appears that insufficient criteria exist for assessing the significance of relatively subtle, apparently reversible, alterations in brain function on the health of exposed workers. However, there is cause for concern and additional research is recommended in this area.

(c) Carbamate Insecticides

These insecticides, which include carbaryl, methomyl, permethrin and propoxur, have more recently come into wide use. They are also ChE inhibitors and produce symptoms in humans similar to that of the OP insecticides. Unlike some OP compounds, the carbamates do not require activation by microsomal enzymes to inhibit ChE. The inhibition of ChE by carbamates is more readily reversible than that produced by most OP compounds. Overexposure may result in local effects, such as constriction of the pupil of the eye, sweating on a localized area of skin, secretion of fluid by glandular mucosa, etc. After absorption into the blood, the compound will contact first the ChE of the plasma and the erythrocytes and will inhibit one or both of them. Detoxification and dissociation of the inhibitor from the enzyme begins promptly, and the concentration of active enzyme in the blood rapidly assumes normal values while ChE in the central nervous system (CNS) or in effector organs may still be depressed. In this case, measurement of blood ChE activities would yield normal values and might lead the physician to conclude falsely that the patient had not been poisoned by a ChE inhibitor. Even though a blood sample may be taken at a time when its ChE activity is still depressed, dissociation of a carbamate inhibitor from the enzyme will proceed by hydrolysis after the blood sample has been collected. When carbamates are the compounds of interest, it is important that blood samples be examined for ChE activity as soon after collection as possible and that a rapid sampling and analytic method be used involving no, or minimal, dilution of the blood. However, due to the rapid reversal of carbamate-induced ChE inhibition, NIOSH does not recommend routine monitoring for persons exposed only to carbamate insecticides.

(d) Chlorophenoxy Herbicides

The chlorophenoxy acids, salts, and esters are irritating to skin, eyes, and respiratory and gastrointestinal linings. They are absorbed through the gut wall, the lung, and the skin. These acids are not significantly fat storable, and excretion occurs within hours or at the most within days, primarily in the urine. They are regarded as being fairly nontoxic, although three cases of peripheral neuropathy were reported in workers after exposures to 2,4-D. In a few individuals, local depigmentation has apparently resulted from prolonged and repeated dermal contact with these substances. Some chlorophenoxy compounds have caused severe cases of dermatitis or

chloracne in workers, although in some cases contaminants were the responsible agents.

Chlorinated dibenzodioxins (TCDD) are contaminants of 2,4,5-T. Neurotoxic effects and chloracne have been found in workers exposed to TCDD-contaminated 2,4,5-T. Experimental animals exposed to TCDD may suffer teratogenic and mutagenic effects.

(e) Dipyridyls

Paraquat and diquat are the best known of this class of herbicides. The dipyridyl compounds can bind to and injure the epithelial tissues of the skin, nails, eyes, nose, mouth, and respiratory and gastrointestinal tracts. Concentrated solutions cause inflammation and sometimes necrosis and ulceration of mucosal linings.

Autopsy cases of accidental or suicidal poisonings from paraquat show evidence of lung, liver, and kidney damage. Some cases had myocarditis, and one case showed transient neurologic signs. Most striking was the widespread cellular proliferation in the lungs. Indications of diffuse toxic pneumonitis appear from 72 hours to 14 days after ingestion of paraquat. The pulmonary lesion has a complex histopathology, beginning with intra-alveolar edema and hemorrhage, followed by the proliferation of fibrous connective tissue. This fibrous connective tissue proliferation is often progressive and generalized and frequently results in death in 1-3 weeks.

(f) Dithiocarbamates

There are three main group in this class of fungicide. The first group contains the dimethyl derivatives including thiram, ziram, ferbam, and vapam. The second group is composed of the diethyl derivatives such as ethyl selenac, ethyl zimate, ethyl tellurac, and ethyl cadmate. And finally, there is the group of ethylene (bis) dithiocarbamate derivatives, which includes the pesticides zineb and maneb.

Many of the dimethyldithiocarbamate compounds are irritants and sensitizers. The toxicity of these compounds probably resembles that of disulfiram (Antabuse), which is used to condition individuals against beverage alcohol. They are metabolized in a manner similar to that of disulfiram. Disulfiram metabolites are powerful inhibitors of multiple sulfhydryl enzymes in the liver and the CNS. Animal experiments indicate that thiram is more toxic than medicinal disulfiram. Preliminary results reported by NIOSH indicate that a serious toxic synergism exists between disulfiram and ethylene dibromide (EDB). In rats fed 0.05% disulfiram in the diet, mortality was 3/48 for males and 3/48 for females. Rats exposed to 20 ppm

EDB by inhalation experienced mortality of 15/40 for males and 9/48 for females. However, rats exposed to both 0.05% disulfiram in the diet and 20 ppm EDB in air experienced mortality of 45/48 for males and 47/48 for females. All exposure periods were 13 months, and cause of death included an increased incidence of various tumors, including hemangiosarcomas of the liver, spleen, and kidney. Mortality for controls was 0/48 for males and 3/48 for females.

The toxic effects of these compounds can be categorized as those following absorption of the toxicant alone, and as those which result when the dithiocarbamate is followed by alcohol. Peripheral neuropathy and psychotic reactions have occurred in alcohol-abstinent individuals on high disulfiram regimens. Disulfiram followed by alcohol is characterized by flushing, excessive sweating, weakness, upper respiratory congestion, labored breathing, and in some cases, respiratory depression that has been life-threatening. High dietary intake of ferbam and zineb has produced functional and anatomical damage to the CNS in rats.

A number of these pesticides were tested in a screening study for their carcinogenic effects in mice. Elevated tumor incidences were observed in the mice fed ethyl selenac and bis (2-hydroxyethyl) dithiocarbamic acid potassium salt, whereas no significant increase in tumors was seen with zineb, maneb, ferbam, ethyl zimate, methyl zimate, methyl selenac, and ethyl cadmate. The authors also concluded that additional evaluation of ethyl tellurac and sodium diethyldithiocarbamate was needed. Ethylene thiourea (ETU) caused elevated tumor incidence when administered orally. ETU is an oxidation product of the ethylene bisdithiocarbamate fungicides. Many compounds of this class, including zineb and maneb, are skin irritants and have caused dermatitis.

D. Todd Meadows Gin

In the present evaluation, the 44 year old cotton operator gives a history of chronic bronchitis and heavy cigarette smoking, hay fever as a child, and more recently chest tightness, shortness of breath and possibly acute bronchospasm related to his working environment. It is the opinion of the investigators, based on this walk-through survey, that he is regularly exposed to a highly dusty environment at his work station in the cotton gin. The four episodes of tremors and double vision with syncope may have been a psychogenic reaction to bronchospasm. It is unlikely that chemicals sprayed on the cotton would produce such effects. In the face of his heavy cigarette smoking, it is difficult to ascribe his respiratory symptoms to occupational exposures. However, it does appear that exposure to cotton dust at the gin house is provoking a peculiar set of symptom responses that may be the result of bronchospasm and psychogenic reaction to the bronchospasm. He does not manifest a

classical case of byssinosis since he denies Monday morning chest tightness. But it does appear that, since 1978, he has been experiencing increased episodes of cough, chest tightness and possible wheezing during the cotton ginning season.

VII. CONCLUSIONS

No specific material or environmental condition was identified as being the direct cause of the health problem reported. However, when the survey was conducted the cotton ginning season was over and the gin was not operating. From the ginner's history it may be concluded that he has a combination of chronic bronchitis related to heavy smoking and an atypical byssinosis-like syndrome related to cotton dust exposure at the gin house. The possibility of an idiosyncratic reaction from exposure to residuals of chemicals sprayed on the raw cotton in the field prior to picking and processing seems an unlikely explanation for his episodes of tremor, double vision and syncope. However, there is the potential for exposure to sprayed chemicals for field workers, gin operators and baler operators.

VIII. RECOMMENDATIONS

The employee's possible reaction to cotton dust at the gin house needs further objective evaluation. If indeed he is experiencing byssinotic reactions, this could be documented with lung function tests administered before and at the end of a workshift during ginning operations. Cessation of smoking may reduce his sensitivity to cotton dust exposure. However, if lung function tests reveal a pronounced decline in lung function over the workshift, it is likely that he will be unable to tolerate further work as a cotton gin operator. Further studies need to be made at the gin house during ginning, for all employees. NIOSH reports have indicated spray chemicals and cotton dust exposure to other gin house and field workers. On a follow-up survey the following should be done:

1. Lung function tests should be performed for gin house employees.
2. A noise survey should be made of all work areas in and around the gin house, including personal noise measurements, to determine if there are potentially harmful noise exposures.
3. Individual "breathing zone samples" should be taken of the employees to determine cotton dust exposure and chemical exposure from pesticide residues in the dust. A list should be made of the chemicals sprayed on various cotton fields served by the Todd Meadows Gin, noting the date, formulation and method of spraying. After observation

of spraying technique and field handling of gathered raw cotton, medical and environmental monitoring may need to be expanded.

4. Blood samples should be taken from the employees before, during and after the ginning season to determine the cholinesterase level in the blood. Cholinesterase, produced by the body and essential to nerve functioning, is affected by certain chemicals in pesticides (organophosphate residue). A low cholinesterase level indicates interference by the pesticides and a health danger.

IX. REFERENCES

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X. AUTHORSHIP AND ACKNOWLEDGEMENTS

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XI. DISTRIBUTION AND AVAILABILITY

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

Copies of this report have been sent to:

- (a) Todd Meadows Gin, St. Clair, Alabama
- (b) U.S. Department of Labor, OSHA, Region IV
- (c) NIOSH Region IV
- (d) Alabama State Department of Health
- (e) Alabama Department of Labor

Table I - Chemicals Reportedly Used on Cotton Processed
at Todd Meadows Gin¹

<u>Common Name</u>	<u>Brand Name</u>	<u>Safety Code*</u>	<u>Criteria Influencing Restriction**</u>
<u>Insecticides:</u>			
Methomyl	Lannate, Lannate L Nudrin	D, D (R) D (R)	7, 8 -
Permethrin ^a	Ambush, Pounce Atroban, Ectibon, Pramex	W (R), D (R) C, C-W, W (R)	- -
Chlordimeform ^a	Fundal Galecron, Fundal SP	W (R) W (R)	- -
Monocrotophos	Azodin (> 19%) Azodin (> 55%)	D (R) D (R)	5, 8 2, 5, 8
Fenvalerate	Pydrin	D (R)	-
<u>Insecticide and Rodenticide:</u>			
Endrin ^b	Name varies	D (R)	2, 6
<u>Herbicide:</u>			
Paraquat	Ortho Paraquat CL, Others	D (R)	1, 2, 3 -

* Safety code: C = caution (slightly toxic); W = warning (moderately toxic); D = danger (highly toxic); R = restricted (at least some restricted uses that can be applied only by certified applicators or persons under their direct supervision).

** Key to Criteria Influencing Restriction:

- | | |
|---------------------------------|--|
| 1. Inhalation hazard | 6. Effects on nontarget species |
| 2. Dermal toxicity | 7. Accident history |
| 3. Oral toxicity | 8. Residue effects on
mammalian species |
| 4. Effects on aquatic organisms | 9. Toxicity to bees |
| 5. Effects on avian species | |

a. Pesticides restricted by means other than by regulation

b. Active ingredient - methyl parathion

Table II - Selected Pesticide Content of Floor Dust
and of Apparel Worn During Ginning

<u>Sample Description and Identification</u>	<u>Chemical</u>	<u>Amount (ppm)*</u>
Pants (VJ-1)	DEF	Not Detected
	Folex	" "
	Methyl Parathion	" "
	Parathion	" "
	Galecron	5.75
	Chlorophenoxy's	Not Detected
Shirt (VJ-2)	DEF	Not Detected
	Folex	" "
	Methyl Parathion	" "
	Parathion	" "
	Galecron	3.50
	Chlorophenoxy's	Not Detected
Face Mask (VJ-3)	DEF	Not Detected
	Folex	" "
	Methyl Parathion	" "
	Parathion	" "
	Galecron	0.40
Floor Dust (VJ-4)	DEF	Not Detected
	Folex	" "
	Methyl Parathion	" "
	Parathion	" "
	Galecron	0.29

* ppm = micrograms chemical per gram of sample

Defoliants: Folex = (tributyl phosphorotrithioite)
DEF = (s,s,s,tributyl phosphorotrithioate)

Insecticides: Methyl parathion = (o,o-Dimethyl o-(p-Nitrophenyl) Ester
Phosphorothioic Acid
Parathion = (o,o-Diethyl o-(p-Nitrophenyl) Ester Phosphorothioic
Acid
Galecron (chlordimeform) = N'-(4-Chloro-o-Tolyl)-N,N-Dimethyl
Formamidine
Chlorophenoxy's = (O-p-Chlorophenoxy)-Acetic Acid

Not detected = less than 0.01 ppm

Table III. Pesticides Grouped on the Basis of Chemical Structure into Generic Classes^{4,5}

	<u>Common Name</u>	<u>Chemical Structure</u>	<u>Generic Class</u>
1.	Methomyl	Thio-N-((Methylcarbamoyl)oxy)-, Acetimidic Acid, Methyl Ester	Carbamate
2.	Permethrin	3-(2,2-Dichlorovinyl)-2,2-Dimethyl-, 3-Phenoxybenzyl Ester (A (+)-, (cis, trans)- Cyclopropanecarboxylic Acid	Carbamate
3.	Chlorodimeform	N'-(4-Chloro-o-Tolyl)-N,N-Dimethyl Formamidine	Carbamate
4.	Monocrotophos	Dimethyl Ester, ester with (E)-3-Hydroxy-N-Methylcrotonamide Phosphoric Acid	Organophosphor
5.	Fenvalerate	2-(p-Chlorophenyl)-2-isopropyl-, Cyano (p-Phenoxyphenyl)Methyl Ester, Acetic Acid	Carbamate
6.	Endrin	1,2,3,4,10,10-Hexachloro-6, 7-Epoxy-1,4,4a,5,6,7,8,8a-Octahydro-,endo, endo- 1,4:5,8-Dimethanonaphthalene	Organochlorine
7.	Paraquat	1,1'-Dimethyl-,Dichloride, 4,4'-Bipyridinium	Dipyridyls