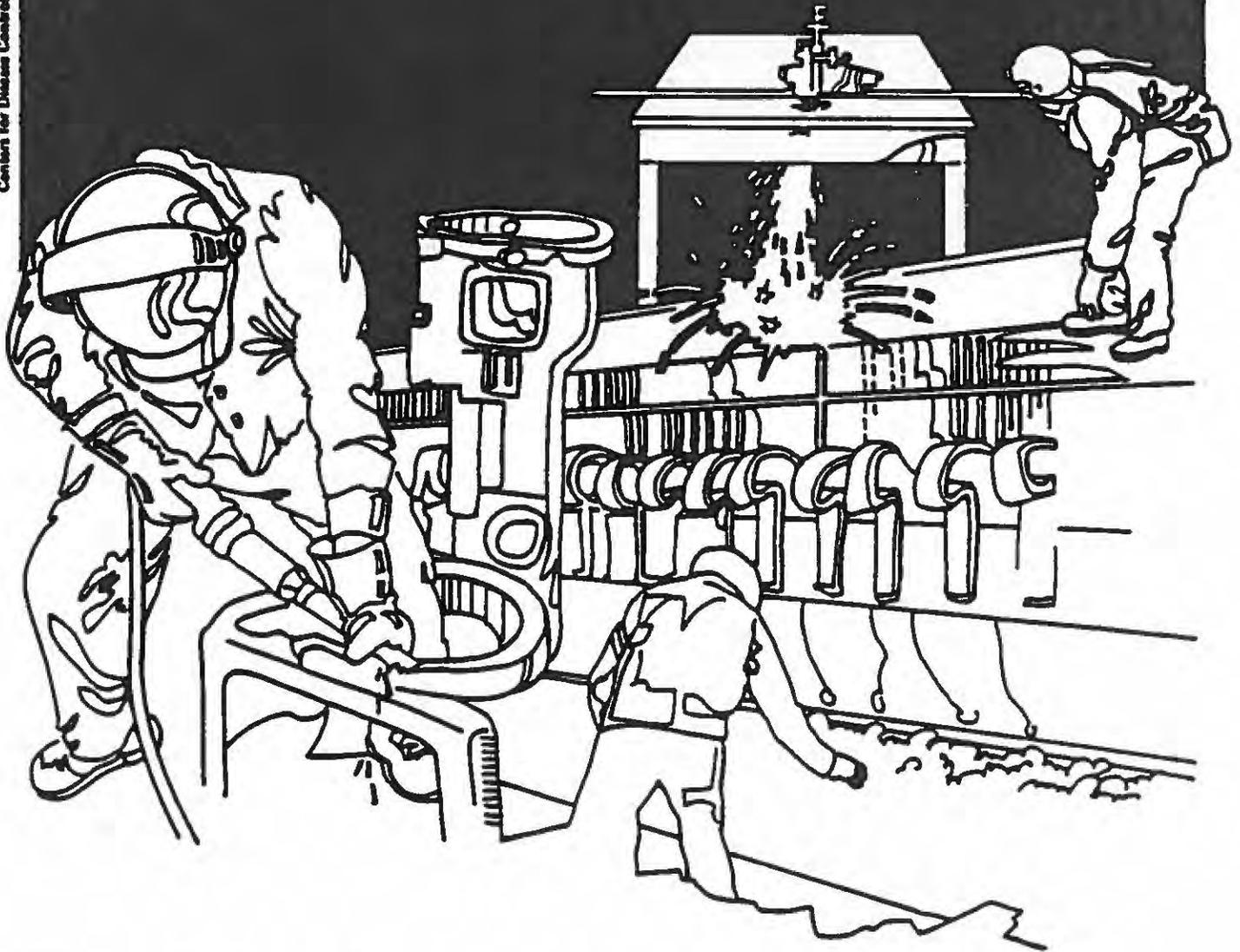


NIOSH



Health Hazard Evaluation Report

HETA 81-301-1467
UNITED FARM WORKERS
OF AMERICA, AFL-CIO
SALINAS, CALIFORNIA

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 81-301-1467
MAY 1984
UNITED FARM WORKERS OF AMERICA, AFL-CIO
SALINAS, CALIFORNIA

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I. SUMMARY

On April 28, 1981, the National Institute for Occupational Safety and Health (NIOSH) received a request for a Health Hazard Evaluation from the United Farm Workers of America, AFL-CIO in Salinas, California. The requestor reported the recent poisoning of a group of fieldworkers by the pesticide mevinphos, and requested assistance in evaluating the use and interpretation of cholinesterase test results.

On April 23, 1981, a group of 44 fieldworkers were accidentally exposed to the organophosphate pesticide mevinphos (Phosdrin R) in a lettuce field in the Salinas Valley. None of the workers had pre-exposure baseline cholinesterase values. Twenty-nine of them were subsequently seen at the Natividad Medical Center, and followed in a cooperative study by the Department of Family Medicine and NIOSH medical staff. Twenty-seven workers were followed for at least 8 weeks, with periodic determinations of both plasma and erythrocyte (RBC) cholinesterase levels in a local laboratory and with split samples analyzed by a NIOSH laboratory. Initial symptoms at the time of exposure were abstracted from the medical records, and cholinesterase levels were obtained on a control group of fieldworkers with no known acute exposure to organophosphate pesticides.

The predominant symptoms at the time of exposure included irritation, headache, visual disturbances, dizziness, nausea, fatigue, chest pain or shortness of breath, and skin irritation; 76% of the workers reported 3 or more of these symptoms. Regeneration of cholinesterase was essentially complete by the end of the second week after exposure. Inhibition, calculated as $[100 - (\text{initial value} / \text{value at end of regeneration})]$ and expressed as a percent, was 15.6% for plasma cholinesterase and 5.6% for RBC. After this period there was a decrease in plasma values of 7.3%, while RBC values showed no significant change.

Based on this pattern of cholinesterase regeneration, it is recommended in cases of fieldworkers who have been exposed to organophosphate pesticides, and who present with moderate symptoms of organophosphate poisoning but without baseline cholinesterase values, that subsequent cholinesterase values be followed to confirm cholinesterase inhibition and to determine the appropriate date for return to work.

Based on the symptoms reported by exposed workers, and on their initial and subsequent cholinesterase values, it was determined that excessive exposure to the pesticide mevinphos had occurred in re-entry of a lettuce field on April 23, 1981. Recommendations are made for clinical confirmation of the diagnosis of moderate organophosphate poisoning, and for determination of an appropriate date for return to work.

KEYWORDS: SIC 0161 (Vegetables and Melons) organophosphates, mevinphos, Phosdrin, cholinesterase, pesticides

II. INTRODUCTION

At 7 a.m., April 23, 1981, a group of 44 field workers entered an iceberg lettuce field in Salinas, California to harvest the crop. By 9 a.m., many of the workers had begun to experience symptoms including dizziness, visual disturbances, headache, nausea and eye irritation. Some of the workers complained to the field supervisor, and several of the workers were not able to continue working because of their symptoms. Neither the supervisor nor the workers had been notified that pesticides had recently been applied to the field. At 11 a.m., a foreman arrived to notify the supervisor that the field had been sprayed with mevinphos at 5 a.m. that morning. A cancellation order sent the previous day had not been relayed to the pesticide applicator, and the field had been aerially sprayed with a formulation of mevinphos and water.

Thirty-one of the workers, together with three agricultural officials who had been inspecting the harvested lettuce, were taken to a local community hospital for evaluation and treatment. None of the workers had had previous baseline cholinesterase determinations. Plasma cholinesterase determinations were done, and two workers exhibited levels below the lower limit of the laboratory normal range. Two workers were hospitalized overnight for observation and treatment of respiratory difficulties. The other workers were disrobed and hosed down with water, sent home and told to wash their clothes at home. None was restricted from returning to work the next day.

The following day, a number of workers were unable to return to work because of symptoms. Their union representative arranged for them to be seen again at a second hospital, Natividad Medical Center, where 29 of them were examined and one worker was hospitalized for observation. Twenty-two (76%) of the workers reported 3 or more symptoms associated with organophosphate exposure. Because of previous differences among medical practitioners in the interpretation of post-exposure cholinesterase values in cases with no previously established baseline values, and because of similar differences in return to work policies, the United Farm Workers of America, AFL-CIO requested NIOSH assistance in evaluating these problems.

III. BACKGROUND

Pesticides are used extensively in the Salinas Valley of California. Local health officials receive more than 100 physician reports of pesticide poisoning a year. Many of these cases are fieldworkers exposed to organophosphate pesticide residues on the foliage of ground crops such as cauliflower and lettuce.(1)

In cases of acute cholinergic crisis, initial diagnosis and treatment are appropriately handled in most agricultural areas. In cases of pesticide applicator exposure to organophosphates, plasma and RBC cholinesterase activities are ordinarily compared to pre-exposure baseline values (required by California Food and Agriculture Department regulations), both to confirm the diagnosis and to decide when to release the patient for work involving potential re-exposure to cholinesterase inhibitors. Other agricultural workers rarely have baseline cholinesterase tests, however. Since there is wide variability among individuals' normal cholinesterase activities, the absence of baseline data makes the interpretation of single post-exposure values difficult.

Major problems in the present usage of cholinesterase testing for clinical diagnosis and management of moderate organophosphate poisoning, for identification of exposures requiring field investigation and changes in work practices, and for release to work include the following:

1. The variability among individual cholinesterase values reduces the sensitivity and specificity of comparisons to laboratory normal values in detecting moderate inhibition.

2. The correlation of cholinesterase inhibition with even the most specific cholinergic symptoms of acute organophosphate poisoning is complicated by factors such as the chronicity of the exposure and the velocity of inhibition. Most physicians in agricultural areas are aware of this and do not rely solely upon cholinesterase levels to make a diagnosis. The non-specific central nervous system symptoms associated with minimal cholinesterase inhibition such as headache, nausea and malaise, however, are frequently dismissed in the absence of a marked cholinesterase inhibition. Although they are non-specific to organophosphate poisoning, these symptoms may significantly affect the worker's well-being and ability to continue working, and may also indicate that the individual is at greater risk of becoming symptomatic if re-exposed after return to work.

3. The lack of research on rates of regeneration of plasma and RBC cholinesterases, including the extent of a 'rebound' effect and potential re-exposure to low levels of cholinesterase inhibitors upon return to work, has made it difficult to standardize practices in the clinical confirmation of mild to moderate organophosphate poisoning and in the determination of release to work.

4. Physicians in agricultural areas frequently report complaints by workers of persistent symptoms after moderate organophosphate exposures, but the lack of research following such groups of workers has made clinical evaluation of these cases difficult. This issue is being addressed in a separate study by the Family Medicine Department of the Natividad Medical Center, and in other NIOSH research.

IV. EVALUATION DESIGN AND METHODS

A. Symptom Assessment

Because the workers were initially seen in the emergency room of a small community hospital for very brief examinations, the assessment of reported symptoms was based on full examinations and histories taken at the Natividad Medical Center on the second day after exposure. These histories included questions on the occurrence and timing of symptoms beginning from the time of exposure on.

Symptoms were abstracted from the charts by a NIOSH medical officer. Frequencies of symptoms and symptoms clusters were determined, and stepwise regression was used to establish the predictive value of the symptom clusters for cholinesterase inhibition. Symptom clusters studied were:

- eye and skin irritation
- nausea, vomiting and diarrhea
- headache, visual disturbance, dizziness and weakness
- eyelid and forearm fasciculations
- sweating
- dyspnea

B. Cholinesterase Measurements

Cholinesterase levels drawn at the community hospital on April 23, the day of the exposure, did not include RBC cholinesterase levels and were processed at a different laboratory from that used by the Natividad Medical Center to follow patients from April 24 forward. The local laboratory cholinesterase measurements reported in this evaluation therefore come from the laboratory utilized by the Natividad Medical Center, the Salinas Clinical Laboratories, which began the day following exposure, on April 24. The method of cholinesterase determination used at the Salinas Clinical Laboratories is the California State approved delta pH (Michel) method. Periodic examinations, including cholinesterase determinations, were conducted over the following 12 weeks. All exposed workers were invited to return on a weekly basis until June 18, and then at two week intervals until July 18.

Split samples were provided to NIOSH for testing on seven of the thirteen testing dates, and the correlation between these results was evaluated. Specimens provided to NIOSH were also analyzed by the delta pH (Michel) method.

The patterns of regeneration were studied in both plasma and RBC cholinesterases. Initial cholinesterase inhibition was estimated as percent of value at two weeks (recovery phase), and as percent of a final estimated baseline level, defined as the average of the last two values for each patient occurring after the May 28

testing date. Regeneration was expressed as the increase per day as percent of the plasma and RBC cholinesterase levels at the end of the initial recovery phase (second week).

In addition, a control group of fieldworkers with no known recent exposure to organophosphate pesticides was obtained; their cholinesterase levels were compared by analysis of variance with the initial and plateau levels for the exposed group.

V. EVALUATION CRITERIA AND HEALTH EFFECTS

A. TOXICOLOGY

Mevinphos is 0,0-dimethyl 1-carbomethoxy-1-propen-2-yl phosphate (CAS registry no. 7786-34-7). It is a compound of high toxicity both orally and dermally, with an oral LD/50 of 6.1 mg/kg (rat) and a dermal LD/50 of 4.7 mg/kg (rat)(2). The California State Department of Food and Agriculture has established a 24 hour (1 day) re-entry period after treatment of a field with mevinphos. The Federal (EPA) mandated label states that any worker exposed to mevinphos and manifesting symptoms should be observed carefully for adverse health effects for 48 hours after exposure, and that because exposure to cholinesterase inhibitors may cause prolonged susceptibility to further exposure at even small doses, no such further exposures should be allowed until blood tests give ample proof of cholinesterase regeneration.

Organophosphate insecticides produce their effect by inhibition of acetylcholinesterase at cholinergic synapses, resulting in an exaggeration of the muscarinic, nicotinic and central nervous system actions of acetylcholine. Diagnosis of acute organophosphate poisoning is made by a history of exposure and clinical signs and symptoms, and may be confirmed by response to a test dose of atropine; treatment should not await laboratory confirmation. For moderate exposure, symptoms include headache, dizziness, weakness, nausea and vomiting, eyelid and skin fasciculations, miosis and blurred vision, and sweating. More toxic exposures may cause abdominal cramps, muscular tremors, dyspnea, and ultimately death from respiratory paralysis.

Confirmation by laboratory analysis depends upon demonstration of depressed levels of plasma or erythrocyte (red blood cell, RBC) cholinesterase activity. Plasma (serum) cholinesterase is more labile than RBC cholinesterase; it is generated in the liver and therefore may be affected by any factor or disease process which interferes with liver function. RBC cholinesterase, because it is analogous to the enzyme active in nerve tissue, is the preferred index of toxicologic effect.(3)

Chronic exposure to organophosphates over a prolonged time period may result in extreme inhibition of cholinesterases in the absence of symptoms; on the other hand, a lesser but more rapid inhibition may provoke moderate but disabling symptoms. Because

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Chronic exposure to organophosphates over a prolonged time period may result in extreme inhibition of cholinesterases in the absence of symptoms; on the other hand, a lesser but more rapid inhibition may provoke moderate but disabling symptoms. Because

of the intra-individual variability in cholinesterase values, a second cholinesterase value must vary from the first (pre-exposure) value by at least 20% (plasma) or 15% (RBC) to achieve significance; symptoms, however, may occur in the absence of this degree of inhibition (2). Workers who are re-exposed to organophosphates before cholinesterase regeneration is complete are at greater risk of poisoning because their threshold is depressed.

B. MEDICAL

Criteria for the finding of organophosphate-induced illness were documentation of exposure to an organophosphate insecticide, symptoms consistent with cholinesterase inhibition, and observation of the pattern of cholinesterase levels obtained during the weeks after exposure.

This evaluation studied the use and interpretation of cholinesterase tests, in addition to the initial exposure and associated acute illness. In the case of cholinesterase inhibition, the pattern of regeneration of the enzyme is important in evaluating the use of the test to set release to work dates.

VI. RESULTS AND DISCUSSION

A. Study Group

All fieldworkers working in the treated field on April 23 were invited to participate in this evaluation. Participants included 29 male fieldworkers (age range 22 - 46), three female family members who handled and washed the workers' clothes, and one male worker who helped to disrobe the affected workers immediately after the exposure. The family members and this worker presented in the Natividad clinic with symptoms of cholinesterase inhibition at the time of the initial examination for the fieldworkers. Results for the study, including symptoms and cholinesterase levels, are for the 29 fieldworkers only unless otherwise mentioned. Twenty-seven workers were seen at least twice from the fifth week onward, and the average of their last two values was used to estimate their baseline cholinesterase values. Twenty-two workers were seen at the final examination on July 16, 12 weeks after the exposure. The number of subjects represented in cholinesterase data reported for specific dates may vary according to the number of workers seen on that date. Almost all workers returned to the fields within two weeks after the incident, in some cases against medical advice.

A control group of fieldworkers was invited to contribute samples for plasma and RBC cholinesterase testing in the NIOSH laboratories on May 25, 1981; 49 male Hispanic fieldworkers (age range 18 - 53) participated.

B. Symptoms

The predominant symptoms reported by the workers at the onset of symptoms were eye irritation, headache, visual disturbances, dizziness, nausea, fatigue, chest pain or shortness of breath, skin irritation, and eyelid fasciculation, arm fasciculation, excessive sweating, and diarrhea. (See Table I) Twenty-two of the workers (76%) reported three or more of these symptoms.

The workers entered the field at 7:00 AM, and the majority of affected workers reported the onset of symptoms occurring between 8:30 and 10:00 AM, or between 1 1/2 hours and 3 hours after exposure began.

C. Cholinesterase Measurements

1. Correlation of local laboratory and NIOSH laboratory results:

The Spearman correlation coefficients for cholinesterase values determined by the Salinas Clinical Laboratories (SCL) and the NIOSH laboratories (NL) on split samples are given in Table II.

Values reported below in establishing estimated baseline values and in estimating inhibition are based on the SCL data, unless specified. Laboratory normal range for the SCL was 0.58 - 1.05 (RBC) and 0.44 - 1.25 (plasma) Michel units, and that for the NIOSH laboratory was 0.44 - 1.09 (RBC) and 0.38 - 1.54 (plasma) Michel units.

2. Estimated Baseline Values

Two end points were used as baselines to estimate cholinesterase inhibition. The first was the end of observed regeneration for the study group, or the initial recovery phase, which ended on the 14th day after exposure. The second end point was the average of the last 2 cholinesterase values during the final weeks of testing (weeks 7-12), after most of the farmworkers had returned to work with potential low-level exposure to cholinesterase inhibitors but with no further reported poisonings.

3. Estimates of Inhibition

Comparison of the initial cholinesterase values to these endpoints resulted in two estimates of inhibition (see Table III and Figure I).

PLASMA

a) Initial Recovery Phase

The group mean for the 14th day after exposure was 15.1 Michel units greater than that for the initial determination. On paired T-test analysis, the 14th day values are significantly greater

than initial values ($p < .0001$). Differences ranged from -2 to 35 Michel units. 28 of 29 subjects had differences of 4 Michel units or greater. The estimated inhibition of plasma cholinesterase, in comparison with levels reached by 14 days after exposure, is 15.6%.

[Comparison of initial values to those on the 7th day after exposure found the group mean to be 5.1 Michel units greater on the 7th day. On paired T-test analysis, this difference is significantly greater ($p < .0001$). 19 subjects had differences of 4 units or greater.]

b. Final Averaged Baseline

On paired T-test analysis, both the final single values and the averaged estimated baselines are significantly greater than initial plasma values. The mean of the final values is only 5.7 Michel units greater than the initial values, however, and the mean of the baseline values is only 8.8 units greater. The estimate of plasma cholinesterase inhibition, in comparison with the averaged baseline at the end of the testing period, is 9.7%. Plasma cholinesterase values decreased by 7.3% from the end of the initial recovery phase to the final averaged baseline.

RBC

a. Initial Recovery Phase

The group mean for the 14th day after exposure was 4.6 Michel units greater than that for the initial determination. On paired T-test analysis, the 14th day values are significantly greater than initial values ($p < .0001$). This estimated inhibition of RBC cholinesterase, in comparison with levels reached by the 14th day after exposure, is 5.6%.

b. Final Averaged Baseline

On paired R-test analysis, both the final single values and the averaged baselines were significantly greater than initial RBC values. The mean of the baseline values is 4 Michel units greater than the initial values, and the estimate of RBC cholinesterase inhibition, in comparison with the averaged baseline at the end of the testing period, is 5.5%. RBC cholinesterase values did not change significantly from the end of the initial recovery phase to the final averaged baseline.

4. Regeneration Rates

The regeneration rates for plasma and RBC cholinesterase were calculated as per cent of change per day to the end of the initial recovery phase (14th day), that is, the inhibition estimated for the initial recovery phase divided by the number of days. (See Figure 1)

a) Plasma

The regeneration rate for plasma cholinesterase during the initial recovery phase was 1.20% / day.

b) RBC

The regeneration rate for RBC cholinesterase during the initial recovery phase was 0.43% / day.

5. Comparison with Control Group

Because the control group cholinesterase testing was done at the NIOSH laboratory only, data reported are for that laboratory. The control group values were compared by analysis of variance to the first NIOSH test of the exposed group, on 5/7, or 14 days after the initial exposure. At that time the exposed group's mean RBC cholinesterase (48.5 Michel units) was significantly less than that of the control group (58.3 units, $p = .0001$). The exposed group's mean plasma cholinesterase (85.4) was greater than that for the control group (74.6), although this difference was not significant.

6. Correlation of Symptoms with ChE Inhibition

In addition to determining the frequencies of symptoms, the association of symptom frequency with initial cholinesterase values was examined, and stepwise regression was used to establish the predictive value of several symptom clusters for cholinesterase inhibition.

Because 76% of the workers had reported three or more potentially organophosphate exposure-related symptoms, a more rigorous standard of four or more reported symptoms was used to investigate the association between symptom frequency and eadpasa and RBC inhibition. Although the extent of inhibition associated with 4 or more reported symptoms was greater for both RBC and plasma cholinesterase, this difference did not attain significance.

The symptom clusters enumerated in Section IV-A were evaluated by stepwise regression for their predictive power for cholinesterase inhibition, with no positive findings.

VII. DISCUSSION AND CONCLUSIONS

A. Symptoms, Cholinesterase Inhibition and Regeneration

Both the range of symptoms and the extent of cholinesterase inhibition encountered in the 29 fieldworkers suggest that this

was a moderate rather than severe organophosphate exposure. Although authoritative texts state that mild poisoning may occur in the presence of cholinesterase values within the laboratory normal range (2), it is also commonly stated that symptoms do not appear without inhibition of 30 - 50% (3). In this case, moderately severe symptoms were suffered by a group of workers whose plasma cholinesterase appears to have been inhibited by only 16% and RBC by 6%. Nevertheless these symptoms prevented a large number of the exposed from returning to work despite the fact that the local hospital emergency room in which they were initially seen released them for immediate return to work.

None of the initial plasma and RBC cholinesterase levels drawn at the Natividad Medical Center were below the lower limit of the laboratory normal range. Similarly, none of the plasma values analyzed at the NIOSH laboratories (i.e., at 14 days and after) were below the laboratory normal range; RBC values for two control and nine exposed workers were slightly below the laboratory normal range (subsequent values on the same individuals showed increases into the normal range). When the fieldworkers were compared to a control group on day 14, however, their mean RBC cholinesterase was significantly lower than that for the controls. The mean plasma value was slightly higher, perhaps reflecting the more rapid recovery of plasma cholinesterase, a rebound effect, or a combination of these factors.

Thus, because fieldworkers do not usually have baseline cholinesterase values recorded, initial cholinesterase values within the laboratory normal range do not rule out the possibility of moderate poisoning which may incapacitate the individual for return to work and may place that individual at greater risk upon re-exposure to organophosphates.

In cases of fieldworkers who report potentially organophosphate-related symptoms but have initial cholinesterase values within the laboratory normal range, the nature and extent of possible exposure is frequently unknown. Because the range of normal cholinesterase values is so wide, patients whose normal values are at the upper end of this range may lose half of their cholinesterase activity and still have values above the lower limit of the laboratory normal range. Thus it is frequently difficult for the attending physician to determine whether the symptoms are in fact due to cholinesterase inhibition, and whether and when to release the patient for work; if the workers in this incident had presented to separate physicians without a history of group poisoning, the diagnosis might well have been missed.

Plasma cholinesterase activity levels return to normal more rapidly than RBC levels after exposure to organophosphates. In this evaluation, the upward trend in plasma cholinesterase values was apparent within the first week after exposure, while the upward trend in RBC values was not apparent until the end of the second week. Thus sequential post-exposure plasma values may be

a feasible and useful alternative to reliance on the laboratory normal range as a means to confirm cholinesterase inhibition. Values drawn every second day after exposure should establish the existence or non-existence of an upward trend by the third consecutive value, or by the fourth day after exposure. A total of three values is needed to establish a trend, because of the 15% variation in plasma values for repeated tests on the same individual.

Such an approach may improve the accuracy of diagnosis, provide evidence of the work relatedness of the illness for compensation, and guide the physician in determining the patient's ability to work. Patients who have no baseline cholinesterase tests and present with a history of exposure, cholinergic symptoms, and values in the low normal range should be kept from work involving any additional exposure until retesting of their plasma cholinesterase fails to demonstrate an upward trend, or, if a trend is demonstrated, until RBC cholinesterase has reached a plateau or estimated baseline. RBC rather than plasma values are recommended as the end point because the former better reflect cholinesterase levels in nerve tissue, and therefore physiologic effect.

Patterns of cholinesterase regeneration for the 29 fieldworkers demonstrate that cholinesterase recovery was essentially complete within two weeks. It is probable that the regeneration rates and degree of cholinesterase inhibition reported are underestimates, because the initial cholinesterase values were not drawn on the day of exposure itself.

Although the two week period for plasma cholinesterase is consistent across many studies and case reports, the time required for RBC cholinesterase regeneration varies substantially, apparently requiring longer time periods when the extent of inhibition is greater. (In another group poisoning of 19 cauliflower fieldworkers exposed to mevinphos and phosphamidon, RBC inhibition averaged 35.5% and 66 days were required for regeneration to 95% of estimated baseline values. (1)). In determining when regeneration has been completed, consideration should be given to the fact that the RBC cholinesterase activity of an individual may vary by 10% upon retesting (4), and to the fact that little is known about the variability of or factors affecting RBC regeneration rates. Specifically, a steady 1% per day rate would not produce a rise greater than 10% for 10 days after an initial drawing. Thus it may not be possible to be certain when a true plateau has been reached; but if a rise greater than 10% is observed, this should strongly influence clinical management of the case.

Other factors which should be considered in deciding when to release a patient who has no baseline value for work include the likelihood of re-exposure to a cholinesterase inhibitor; the persistence of symptoms; whether values are at the lower limit of normal; and the economic impact on the patient of prolonged absence from work. Once the patient's symptoms have abated, the

physician may feel compelled to release the patient to work to minimize the impact of this loss of earnings, especially when the risk of re-exposure is small; the patient should be advised that additional exposure before recovery is complete will entail serious risks, and to seek medical care promptly should symptoms recur.

The decline in plasma cholinesterase after the regeneration period is consistent with reports in the literature. This is usually ascribed to a rebound effect, but may also be due in part to new low-level exposures on return to work.

The correlation between results from the local laboratory and the NIOSH laboratory indicates that the variability of local commercial laboratories following the California state approved methods for cholinesterase determinations should not preclude the use of test results reported from these laboratories in field research or for purposes of legal proof of cholinesterase inhibition.

B. Prevention of Future Accidental Exposures

Neither the fieldworkers nor the supervisors knew that the lettuce field had just been treated with a pesticide, and several hours elapsed between the first occurrence of symptoms and removal of the workers from the field. This problem can be prevented by a posting system, in which signs are posted in fields treated with potentially toxic pesticides. The signs should state the name of the pesticide, the date applied, and the date on which entry is permitted.

No water for emergency washing was available at the field where this incident occurred. Had it been available, immediate decontamination of the exposed workers would have been possible and of marked benefit, because most organophosphates are absorbed very rapidly through the skin.

VIII. RECOMMENDATIONS

1. Sequential post-exposure plasma values may be a feasible and useful alternative to reliance on the laboratory normal range as a means to confirm cholinesterase inhibition in persons without baseline cholinesterase values. Patients who have no baseline cholinesterase tests and present with a history of exposure, cholinergic symptoms, and values in the low normal range should be kept from work involving any additional exposure until retesting of their plasma cholinesterase fails to demonstrate an upward trend, or, if a trend is demonstrated, until RBC cholinesterase has reached a plateau or estimated baseline.

2. Fields to which potentially toxic pesticides have been applied should be posted with signs giving the name of the pesticide applied, date of application, and appropriate re-entry date.
3. Water should be made available for emergency washing purposes at the side of fields treated with pesticides when workers re-enter these fields.
4. The system of communication regarding pesticide applications and field re-entry periods between contracted pesticide applicators and field crew supervisors should be improved to avoid incidents of the kind evaluated in this report.
5. Education of field crews and supervisors in the recognition of pesticide-related symptoms and first aid should be improved.

X. REFERENCES

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XII. 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 90 days, the report will be available through the national Technical Information Service (NTIS) 5285 Port Royal Road, Springfield, Virginia. Information regarding its availability through NTIS can be obtained from the NIOSH Publications Office at the Cincinnati address.

Copies of this report have been sent to:

1. United Farm Workers, AFL-CIO
2. Natividad Medical Center, Salinas, CA
3. NIOSH -- Region IX
4. Cal-OSHA
5. Federal-OSHA

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

TABLE I: FREQUENCIES OF SYMPTOMS REPORTED BY EXPOSED WORKERS

Eye Irritation	////////////////////	75.9 %
Headache	////////////////	48.3
Visual Disturbances	////////////////	48.3
Dizziness	////////////////	41.4
Nausea	////////////////	37.9
Fatigue	////////	27.6
Chest Tightness, Shortness of Breath	/////	20.7
SKin Irritation	////	17.2
Vomiting	////	17.2
Eyelid Fasciculation	///	10.3
Excessive Sweating	//	6.9
Diarrhea	//	6.9
Arm Fasciculation	//	6.9

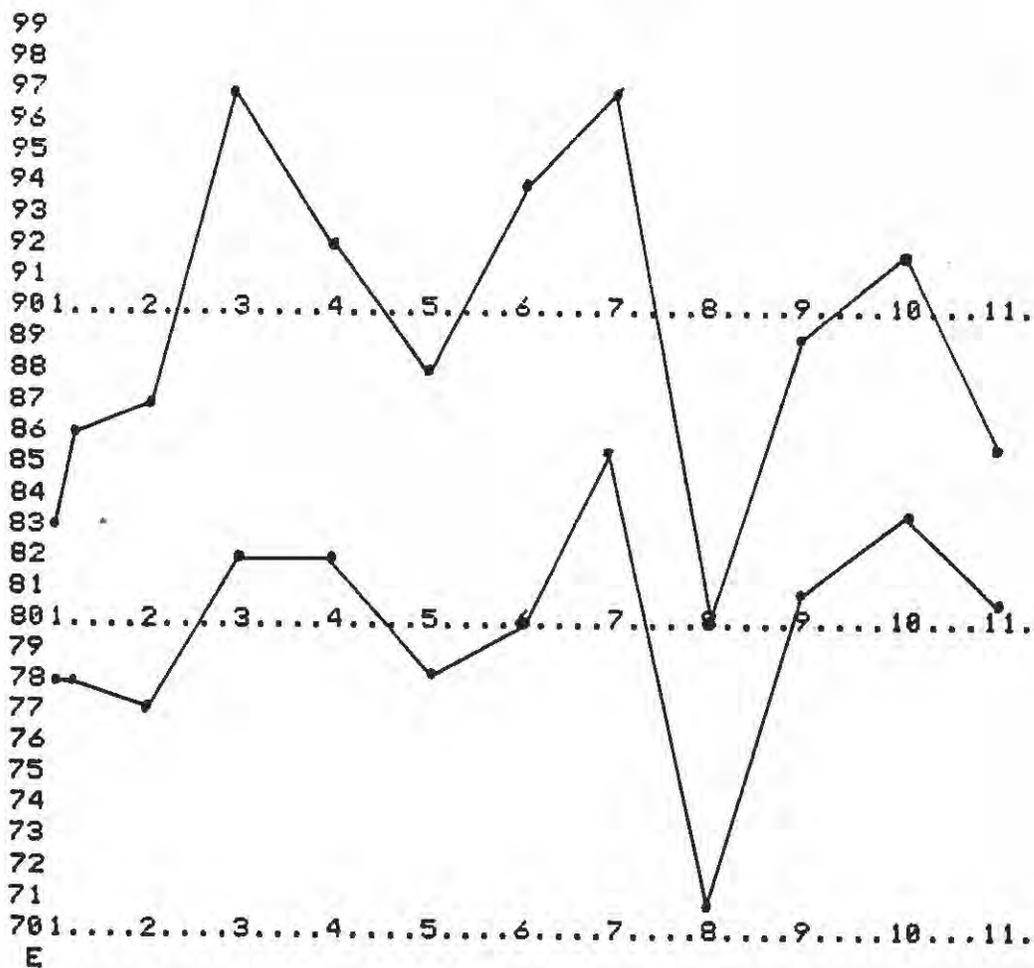
TABLE II: CORRELATION OF MEAN CHOLINESTERASE VALUES
AS DETERMINED BY SALINAS CLINICAL LABORATORIES (SCL)
AND NIOSH LABORATORIES (NL)*

Test	Mean	R	P
Initial NL RBC values	.46	.59	.0009
SCL values on same date	.81		
Initial NL plasma values	.99	.71	.0001
SCL values on same date	.95		
Estimated NL RBC baseline	.64	.39	.04
Estimated SCL RBC baseline	.82		
Estimated NL plasma baseline	.93	.51	.005
Estimated SCL plasma baseline	.88		

TABLE III: ESTIMATES OF CHOLINESTERASE INHIBITION

Test	Mean	N	SE	T	P
Initial Plasma 7th Day	82.5 87.615	26	1.034	4.948	.0001
Initial Plasma 14th Day	81.8 96.88	25	1.371	10.999	<.0001
Initial Plasma Final	81.524 87.238	21	1.729	3.304	.0038
Initial Plasma Estimated Baseline	81.56 90.32	25	1.734	5.053	.0001
Initial RBC 14th Day	77.56 82.12	25	.753	6.056	<.001
Initial RBC Final	77.714 81.429	21	.873	4.253	.0006
Initial RBC Estimated Baseline	78. 82.5	25	.825	5.457	.0001

FIGURE 1: MEAN PLASMA AND RED BLOOD CELL CHOLINESTERASE LEVELS FOR MEVINPHOS-EXPOSED WORKERS



E = exposure
1 - 11 = serial cholinesterase tests at one week intervals

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