IV. ENVIRONMENTAL DATA AND BIOLOGIC EVALUATION

Air Sampling

Sampling for airborne methyl parathion requires equipment suitable for simultaneously collecting vapors, airborne solids, and airborne droplets, since formulations in common use include emulsifiable liquids, wettable powders, and dusts. [9] No sampling method was found which has been adequately investigated for its accuracy in collecting all three forms.

The midget impinger is recommended as the device for sampling methyl parathion because of its small size which imparts greater adaptability to the air sampling procedure without sacrificing precision. Although studies have not been reported on the efficiency of midget impingers for collecting airborne methyl parathion, Roberts and McKee [81] studied the efficiency of midget impingers for collecting ammonia (1 ppm) with distilled water as the collection medium. Efficiency was highest (nearly 90% trapped by the first impinger) at the flow rate recommended for collection of particulates: 0.1 cu ft/min (2.8 liters/min).

During development of this document, NIOSH was informed (RH Hill Jr, written communication, March 1976) that gaps existed in the performance specifications for the sampling and analytical techniques recommended for methyl parathion. In addition, the Environmental Protection Agency has withdrawn the method utilizing a midget impinger charged with ethylene glycol, since "a controversy concerning the reliability of the data" has arisen using this method (RH Hill Jr, written communication, March 1976).

NIOSH currently recommends in the NIOSH Manual of Analytical Methods

[82] that an ethylene glycol-charged midget impinger be used for parathion sampling and that hexane be used to extract the trapped pesticide. Details of sampling and air-flow calibration procedures for methyl parathion are given in Appendix I. Other air sampling methods of equivalent or superior efficiency may be substituted for the recommended method.

Analysis of Air Samples

Because of the industrial practice of formulating mixed active ingredient products, [12] the analytical method must be able to differentiate methyl parathion from other pesticides. Common pesticide vehicles either must not interfere or must be readily extractable prior to analysis.

Although UV absorption [83] and colorimetric [84] methods have been reported for parathion analysis, no reports concerning the use of these methods for analysis of methyl parathion were found.

Phosphorus-specific detectors were introduced in 1964 and 1965 and have made gas-liquid chromatography (GLC) the analytical method of choice for organophosphorus compounds. [85,86] Both the alkali flame ionization (thermionic emission) detector introduced by Giuffrida [85] and the flame photometric detector developed by Brody and Chaney [86] exhibit relatively high sensitivity. Giuffrida [85] indicated that 24 ng of methyl parathion could be detected by a gas chromatograph with an alkali flame ionization detector. The linear working range of this system was reported to be 0.05-1.0 ng/injection volume. [85] Brody and Chaney [86] reported that the flame photometric detector, equipped with a narrow band-pass interference filter for isolating phosphorus emissions at 526 nm, was sensitive to

0.25 ng of parathion or malathion, with a linear response range of 0.0063-63.0 ppm. The method was reported to be highly specific for phosphorus compounds, thereby excluding interference by pesticide vehicles or chlorinated hydrocarbon coingredients.

The NIOSH Manual of Analytical Methods [82] recommends the use of GLC with a phosphorus flame photometric detector for analysis of hexane-extracted parathion air samples. Since different retention times are indicated in this method for methyl parathion, methyl paraoxon, parathion, and paraoxon, the method appears suitable for methyl parathion analysis. Precision and accuracy of the method were indicated as unknown.

Gas-liquid chromatography utilizing a phosphorus flame photometric detector is recommended as a suitable analytical technique for determining methyl parathion in hexane-extracted samples. The recommended method detailed in Appendix II identifies four separate chromatographic columns for obtaining discrete peaks when interfering phosphorus compounds are present. Also presented are data on additional columns suitable for analysis of samples containing methyl parathion. [87,88] Analytical techniques of equivalent or greater precision, accuracy, and sensitivity may be substituted for the recommended method.

Biologic Monitoring Methods

Two general approaches may be taken in evaluating occupational exposure to cholinesterase-inhibiting insecticides. Direct methods identify the amount of insecticide in the blood, [89-91] or of the insecticide, [90] its metabolites or its degradation products [92] in urine. Indirect methods measure cholinesterase activity in erythrocytes,

plasma, serum, or whole blood. [29,93-111]

Monitoring of methyl parathion, paranitrophenol, or alkylphosphates in urine, or of methyl parathion in blood, is unsatisfactory for determining the hazards to workers by exposure to methyl parathion. Neither method considers the increased susceptibility to intoxication by methyl parathion faced by workers whose cholinesterase activity has been lowered by recent or simultaneous exposure to other organophosphorus or carbamate pesticides. In addition, these methods are nonspecific; for example, the appearance of paranitrophenol in urine may be due to the degradation of methyl parathion, parathion, or EPN.

Reports cited in Chapter III indicate that symptomatic methyl parathion intoxication in humans, irrespective of dose or route of entry, causes profound inhibition of circulating cholinesterases. The validity of monitoring blood cholinesterases for determining the efficacy of engineering controls, personal protective equipment, and work practices depends on the extent to which the activity of one or both of these enzymes is predictive of the relationship of total anticholinesterase exposure to increased susceptibility to systemic poisoning upon continued exposure. This correlation has not been firmly established for methyl parathion in humans. However, a correlation between circulating serum cholinesterase activity in parathion-poisoned workers and severity of systemic poisoning has been compiled by Namba et al [78] based on several single-dose parathion poisoning incidents. Symptomatic intoxication was associated with inhibition of more than 50% of baseline serum cholinesterase activity. The level of serum cholinesterase activity paralleled the severity of manifestations: 21-50% of normal in mild poisoning, 11-20% of normal in

moderately severe poisoning, and 0-10% of normal in severe poisoning cases.

Grob et al [6] studied the rate of restoration of plasma and erythrocyte cholinesterase activity in 67 persons poisoned as follows: 18 by parathion, 35 by diisopropyl fluorophosphate (DFP), and 14 by tetraethyl pyrophosphate (TEPP). Initial rates of return (during the first 3 days following exposure) for all three pesticides were comparatively rapid. For parathion, erythrocyte cholinesterase activity increased by 10% over this interval, while for the plasma enzyme the figure was 27%. While the initial rate of return of activity differed for the three organophosphorus insecticides, restoration rates were identical from day 4 on. Rate of return for erythrocyte cholinesterase activity averaged 1-2%/day after day 3. For plasma cholinesterase, the average rate of return was 5%/day for days 4-10 and 3%/day for days 10-20. If this steeper rate of return of plasma cholinesterase activity applies to methyl parathion and exceeds the rate of recovery of functional (neuroeffector) cholinesterase, the use of plasma values for monitoring purposes could fail as an index of cumulative functional inhibition of neuroeffector cholinesterase. The divergence between plasma and erythrocyte cholinesterase activity would be especially significant where inhibition is due to extended low-level exposures to methyl parathion or other anticholinesterase compounds.

Several attempts have been made to determine a range of normal human cholinesterase activities for nonexposed persons. [93,112-114] Laboratories deriving their own normal values have been able to compare their results with those produced in these studies to validate their analytical methods. Laboratory norms are particularly useful when preexposure cholinesterase baselines are not available for a worker.

Asymptomatic employees (whose cholinesterase activities may already be inhibited) may thus be assigned a "working baseline" value: the arithmetic mean of a laboratory's normal values or the employee's current cholinesterase activity, whichever is higher. Values from four studies of cholinesterase levels in nonexposed human populations appear in Table XVI-8. [93,112-114]

Interpretation of cholinesterase assays should take into account the known nonoccupational sources of inhibition of cholinesterase activity. Such possibilities include liver disease, [115] pregnancy, [116] malignant neoplasia, [117,118] and tuberculosis. [118] Familial reduction in plasma cholinesterase activity was reported by Lehmann and Ryan [119] and by Kalow [120] and was subsequently found by Kalow and Genest [121] to be related to the presence of an atypical gene. About 1 in 3,000 individuals tested in a healthy Canadian population was homozygotic for this atypical gene [122] and thus could be expected to have a genetically determined deficiency in serum cholinesterase activity. The activity of erythrocyte cholinesterase has been found to be inhibited in certain pulmonary and extrapulmonary cancers [118] and in paroxysmal nocturnal hemoglobinuria. [123] Familial asymptomatic reduction in erythrocyte cholinesterase activity also has been reported. [124] The importance of these observations to susceptibility to organophosphorus insectide poisoning has not been determined.

Plasma and Erythrocyte Cholinesterase Analyses

Methods for determining plasma and erythrocyte cholinesterase activity may be classified as electrometric, manometric, colorimetric,

spectrophotometric, radiometric, titrimetric, or chromatographic.

Michel [29] developed a widely used electrometric method for cholinesterase analysis in 1949. The method depended on measurement of the quantity of acetic acid liberated during a fixed period of time (1-1.5 hours) by the action of cholinesterase on acetylcholine. Enzymatic activity was thus measured in terms of the change in pH of a buffered solution of sample plus substrate/unit time. The pH was determined with a glass electrode. Both erythrocyte and plasma cholinesterase activities were measurable. Correction tables were provided to adjust for nonenzymatic hydrolysis and to correct for variations in the rate of pH change with decreasing pH. The author compared the electrometric and the manometric methods by performing parallel analyses on 31 independent samples of plasma and 27 independent samples of erythrocytes from 12 persons. The standard deviations of the differences in cholinesterase activities determined by the two methods were reported to be 5.49% for plasma cholinesterase and 5.50% for that of the erythrocytes. Michel [29] stated that the electrometric pH method was preferable to the manometric one (discussed below) because of its simplicity, the minimum of required equipment, and its suitability for doing a large number of determinations in a relatively short time. Both the initial and the final pH readings required for each sample took 1 minute to perform.

Wolfsie and Winter [93] developed a micromodification of the Michel method [29] to adapt it to fingertip blood samples. The analytical procedure of Wolfsie and Winter, [93] however, is identical with that of Michel. [29] Witter et al [94] presented another modification of the Michel procedure. This method eliminated the initial pH reading, used

distilled water instead of a solution of saponin to hemolyze the erythrocytes, started the enzyme reaction by adding a mixture of buffer and acetylcholine to the diluted sample, and shortened the period of incubation from 90 to 60 minutes. The results were nearly identical with those obtained by the Michel method, but twice as many samples could be analyzed in the same period of time.

Witter [95] stated that the manometric technique was one of the most accurate and precise (to within $\pm$1%) for the determination of plasma, erythrocyte, or whole blood cholinesterase activities. This method is based on the measurement of the amount of carbon dioxide liberated when acetic acid, produced by the enzymatic hydrolysis of acetylcholine, reacts with sodium bicarbonate. The author noted, however, that manometric techniques require 20-30 minutes to obtain a rate curve [95] and are thus too time-consuming for routine analysis.

Limperos and Ranta [96] described a rapid screening test for whole blood cholinesterase activity that could be used in the field without specialized equipment. In this visual colorimetric method, the change in pH resulting from the liberation of acetic acid from an acetylcholine-iodide substrate was estimated by the change in color of an indicator, bromthymol blue, after a 20-minute incubation with a drop of fingertip blood. Adjustments for temperature were not included but were subsequently proposed by Davies and Nicholls. [97] Separate determinations of erythrocyte and plasma cholinesterase activities were introduced in a modification by Fleisher et al [98] using different substrates specific for erythrocyte or plasma cholinesterase. Gerarde et al [99] adapted the basic visual colorimetric screening test by providing premeasured quantities of

stabilized reagents in capillary tubes. Forsyth and Rashid [125] used a field kit based on the colorimetric method of Edson [126] for the determination of the activity of cholinesterases in whole blood. WHO has sponsored field tests of this method. [127] While better adapted for field use than other methods, visual colorimetric techniques are either cumbersome, because of the requirement that the time necessary for a standard color change must be recorded for each sample, [98] or inadequate to determine separately the plasma and erythrocyte cholinesterases. [97,99]

Several investigators have described spectrophotometric methods for determining plasma, serum, whole blood, and erythrocyte cholinesterase activities. [100-104,112,128] Each method involves the hydrolysis of a substrate by plasma or erythrocyte cholinesterase, with measurement of the rate of loss of substrate [112,128] or the rate of appearance of hydrolytic products subsequently bound to indicators. [100-104] None of the spectrophotometric methods are as simple or as rapid as the electrometric methods, and few have been widely employed in laboratories.

Cholinesterase assays have also been performed using a 14C-labeled substrate, [105-107] a liquid membrane electrode highly selective for acetylcholine, [108] GLC, [109] titrimetric techniques, [95,110] or substrate-impregnated indicator papers. [111] The GLC, titrimetric, and liquid membrane electrode methods require more expertise than the electrometric methods. The radiometric micromethod developed by Winteringham and Disney [105-107] operates with very small samples and involves a simple counting technique to measure the appearance of labeled acetic acid but requires a counter and a supply of labeled acetylcholine.

Based on the foregoing discussion, the electrometric method of Michel [29] is recommended as a suitable method for plasma and erythrocyte cholinesterase analyses. This selection is made because it is the most widely documented method and is sufficiently precise. It requires ordinary laboratory equipment that is relatively inexpensive and simple to use. The Wolfsie and Winter [93] micromodification, when used in conjunction with the original electrometric method of Michel, [29] will provide sufficient precision in analysis without excessive bloodletting. This method has been automated. Details of the recommended method are presented in Appendix IV. Laboratories performing numerous or frequent cholinesterase assays may prefer to use a modified Ellman method in conjunction with an autoanalyzer. The pH-stat method is also considered an acceptable analytical method.

Environmental Levels

Few studies were found in which the skin and respiratory exposures of workers handling methyl parathion were measured. A study by Trefilov et al [77] was the only one found to report air levels of methyl parathion in manufacturing plants. Most samples were found to be below 0.1 mg/cu m, and very few reached 0.2 mg/cu m. Of the many opportunities for exposure during the performance of custom applications of methyl parathion, only cotton checking has been studied directly. [129-131] Indirect methods involving the monitoring of urinary metabolites have been used to assess the comparative exposures of different occupational groups to parathion. [92] The ranking of exposure potential derived from the latter study probably can be applied also to workers exposed to methyl parathion.

Three occupational exposure studies of cotton checkers were found.

[129-131] Cotton checkers, or scouts, typically enter recently sprayed fields to determine the effectiveness of an insecticide application in destroying target insects. This occupational group was selected because of its allegedly high exposure to dislodgeable foliar residues [129] and because more than 80% of the methyl parathion used domestically is applied to cotton. [9] According to Ware et al, [129] checkers move through sprayed foliage for up to 10 hours daily.

Quinby et al [131] examined methyl parathion exposures among cotton checkers in 1958. Methyl parathion was applied at the rate of 0.5 lb/acre with ground equipment under conditions of "high" relative humidity and temperatures ranging from 59 to 97 F. Exposure was determined for two test subjects during periods of 5-65 minutes, 1.5-3.5 hours, and 4-5 hours after the application. Gloves and cotton shirt sleeves showed the greatest contamination. Respirator pads showed no detectable quantities of methyl parathion by the analytical method used, which was sensitive to 20 μg for 1-hour samples and 10 μg for 2-hour samples. Contamination levels for leg and foot coverings were not reported, although much work by the checkers was done on hands and knees. One-hour exposures beginning 5 minutes after application resulted in an average retention of 2.69 mg/hour on gloves. For exposures during the periods 1.5-3.5 hours and 4-5 hours after application, the average rates of accumulation of methyl parathion on the gloves were 2.22 and 0.56 mg/hour, respectively. In trials performed during each postapplication interval, residues on gloves accounted for more than 80% of the total potential exposure reported. The authors stated that "blood cholinesterase" activity measured 24 hours after exposure of the checkers showed no significant inhibition. However, none should have been

expected, since opportunities for absorption were minimized (1) by the gloves, respirator, and clothing, (2) by the immediate change of clothing after the test exposure, (3) by the short exposure periods, which never exceeded 2 hours, and (4) by the absence of repeated exposures which could have resulted in cumulative inhibition of blood cholinesterase activities in checkers. The average total weight of methyl parathion extracted from garments worn by the checkers (gloves, shirtsleeves, T-shirts) declined with time. The total weights for the first, second, and third postapplication intervals averaged 3.2, 2.6, and 0.7 mg/hr, respectively. This decline was presumably due to both the absorption of methyl parathion by the foliage and the decay of the nonabsorbed fraction. Quantitative conclusions are difficult to draw from this study for two reasons. First, significant methyl parathion may have been lost from clothing during travel to the laboratory. The authors [131] were able to demonstrate such losses from artificially exposed garments saturated with saline to simulate sweat after incubation for 2-3 days, the approximate time that checker garments were in the mail. Second, it does not appear that the analytical method, the spectrophotometric technique of Averell and Norris, [84] measured methyl paraoxon separately from methyl parathion. Use of a method that measures the combined quantity of methyl parathion and methyl paraoxon would not be an accurate measure of the toxicologic hazard of methyl parathion exposure since the oxon form is the more toxic one to mammals. The half-life of dislodgeable foliar residues of methyl parathion was calculated by the authors to be "less than one hour." The significance of this half-life estimate for methyl parathion on cotton foliage is obscured, however, by the apparent failure to differentiate between residues of

methyl parathion and of methyl paraoxon.

Ware et al [129] examined the exposure of cotton scouts (checkers) to methyl parathion and to a mixture of parathion and methyl parathion in field studies. In each experiment, the plasma and erythrocyte cholinesterase activities of two volunteers were measured immediately before they entered sprayed fields of maturing cotton. The first experiment involved application of 1.0 lb of methyl parathion plus 4.0 lb of toxaphene in 5 gallons of spray mixture/acre. Canopy (top) foliage was dense and overlapping. Twelve aluminum foil sheets were placed in random horizontal positions on top of the canopy to determine the quantity of spray reaching the foliage. Urine was collected from the checkers at the time of entry into the sprayed fields and for 24 hours thereafter. Checkers entered the fields wearing fresh T-shirts and bluejeans at 3 minutes and at 4 hours after pesticide application. Portable air samplers were positioned just below the chin. Gauze patches were taped to the trousers at shin, thigh, and hip levels and to unprotected forearms. While in the field for 30 minutes, the subjects took four 100-leaf samples of cotton canopy and four 100-leaf bottom samples for subsequent foliar residue analysis. The report did not state whether checkers performed their usual task of opening cotton blossom squares to check for pests. Quinby et al [131] asserted that this latter task probably accounted for most of a checker's exposure. Hands of subjects were washed in hexane after each 30-minute test period. [129] Gauze patches and clothing were removed and sent to the laboratory. Analysis by GLC, using a flame photometric detector sensitive to phosphorus-containing substances, showed deposition of methyl parathion on the foil sheets of 0.17-1.99 lb/acre,

plus a "trace" of methyl paraoxon. Methyl parathion was most heavily deposited on the trousers and the hands of the checkers. When the subjects took canopy leaf samples, methyl parathion on the trousers averaged 15.9 mg, almost three times the amounts detected when samples of bottom leaves were collected. Entry into the fields 4 hours after spraying produced contaminations of hands and trousers during the collection of samples, but higher ones when bottom leaves were sampled. Of the gauze patches examined, those on thighs and hips held the highest amounts of methyl parathion. The airborne concentrations of methyl parathion were low during both the canopy and bottom leaf samplings at 3 minutes and 4 hours after application. For these trials, the highest value measured was 1.77 μg/cu m.

In a second experiment, Ware et al [129] used an aerial application of 0.5 lb of methyl parathion, 0.5 lb of parathion, and 2 lb of toxaphene in 5 gallons of spray/acre of mature cotton. The use of both parathion and methyl parathion in mixed active ingredient applications is commonplace. [10] After cholinesterase, paranitrophenol, and serum methyl parathion pretests, the same two test subjects who had taken part in the earlier experiments entered the fields for 30-minute periods beginning 10 minutes or 12, 24, 48, or 72 hours after application. [129] Neither gauze pads nor air samplers were worn in this experiment. Since the application was performed in the morning, test subjects were not in the fields during the hours of highest ambient temperature (high temperatures for the test days were 92-98 F, lows 62-68 F).

No significant depressions of plasma or erythrocyte cholinesterase activities were reported for subjects in either of the experiments, nor was

urinary PNP detected after exposure. [129] After the second 30-minute exposure in the first series of tests, serum parathion levels were 27 and 32 ppb for the two test subjects. Neither parathion nor methyl parathion was detected in serum following exposure in the second experiment. For the same reasons cited above, no significant inhibition of blood cholinesterase activity was expected under the circumstances. The authors estimated the total time in the field for cotton checkers in actual practice to be 5 hours/day and used this estimate to calculate 24- and 48-hour reentry exposure levels from the 30-minute exposure data. When entry was made 24 hours after application under these experimental conditions, it was estimated that 11.6 mg of the methyl parathion-parathion mixture had accumulated on hands and forearms, and 64.7 mg on clothing over a 5-hour exposure. The authors estimated that 3.6 μg of the mixture would be inhaled during this exposure. When the field was entered 48 hours after the application, estimated accumulations were 6.0 mg on hands and forearms and 45.2 mg on clothing, while approximately 1.8 μg of the parathion-methyl parathion mixture was inhaled. By assuming there was no absorption from the clothing and, because of the dryness of the residue from hands, forearms, and respiratory tract, the authors surmised that probably 5.8 mg are absorbed in a day with a 5-hour exposure (equivalent to 1 day of checking) when 24 hours elapsed between application and entry by the checker and 3.0 mg/day with an elapsed time of 48 hours. No data were reported to support the assumptions regarding the fraction of the insecticidal mixture absorbed by clothing, hands, forearms, and respiratory tract. It is unclear from the report how respiratory figures were calculated for the second series of trials, since no air sampling was

performed. No symptoms of systemic poisoning were reported in either series.

Ware et al [130] subsequently conducted a similar study in which four cotton checkers were exposed to methyl parathion for 5-hour working periods 24 hours after the last application. Methyl parathion was applied repeatedly to the test field at 5-day intervals for a total of four applications, each at the rate of 1 lb/acre, by high-clearance ground equipment. Subjects wore fresh T-shirts and blue jeans with an optional head covering. Portable air samplers, with the intake tubes attached to the upper front of the T-shirt of each subject, were used for 2.5 hours during the exposure period. The impingers were charged with ethylene glycol. The concentrations of methyl parathion in serum were determined before, during, and after the exposure period. Twenty-four-hour urinary paranitrophenol collections were performed before exposure and twice during the next 48 hours. Subjects simulated the activities normally performed by cotton checkers or entomologists, in addition to gathering leaf samples for residue analysis. Foliage was dense and was intertwined between rows, causing relatively high body contact and green stains on some parts of the clothing. No signs of systemic poisoning were observed. Physical activity at the prevailing environmental temperatures did cause sweating and dehydration. In tests immediately following the exposure period, no methyl parathion was detected in serum, nor were the cholinesterase activities of plasma or erythrocytes inhibited when measured by the Michel electrometric method. [29] The total amounts of paranitrophenol excreted in 48 hours by the four subjects were 0.15, 0.19, 0.44, and 1.20 mg, but the authors [130] cautioned that recoveries were inconsistent for the "lower" concentrations.

Again, the same type of data was absent in this report. When methyl parathion was recovered by hexane washes from clothing and from hands, the authors found the most methyl parathion (1.7 mg) and methyl paraoxon (39.0 μg) on the trousers. The discrepancy between these and the considerably greater quantities retained on clothing and hands in the earlier studies by Ware et al [129] were not discussed. Inhaled methyl parathion was estimated from the impinger data to be 1.2 μg over the 5-hour exposure, with an average pulmonary ventilation of 20 liters/minute assumed. The investigators concluded that 24 hours was a safe reentry interval for methyl parathion. This conclusion fails to recognize the many environmental variables and differences in the performance of applications that influence the decay rates of methyl parathion and methyl paraoxon on foliage and soil, as well as the amount which remains airborne. The data do, however, confirm the importance of wearing impervious gloves and fresh full-body clothing daily.

Arterberry et al [92] compared data from persons exposed to parathion in various job categories. These categories included mixing-plant personnel, part-time ground applicators, aerial application workers, commercial ground applicators, and workers in orchards. Erythrocyte and plasma cholinesterase and urinary paranitrophenol excretion were measured in a majority of these occupational groups. Generally, samples taken on the last day of mixing, heavy spraying, or other intensive contact with parathion were chosen to represent maximum exposure (period of exposure unspecified), and the results were compared with preexposure values. The ranges and the means of plasma cholinesterase activities for all job categories tested did not differ from the preexposure values. Similarly,

except for mixing-plant personnel, erythrocyte cholinesterase activities were lower than preexposure values. There was a 36% inhibition of erythrocyte cholinesterase activities in mixing-plant personnel, suggesting that low-level chronic exposures were responsible for this effect. The slower rate of erythrocyte cholinesterase reactivation [6,43] could have allowed its daily inhibition to exceed the rate of recovery, leading to a net reduction in erythrocyte cholinesterase activity. Paranitrophenol (a urinary degradation product of parathion, methyl parathion, fenitrothion, and EPN, and thus an index of exposure to these compounds) was found in five occupational groups. These, in order of decreasing paranitrophenol concentration in their urine, were commercial ground applicators, mixing-plant personnel, part-time ground applicators, aerial application workers, and workers in orchards. Results of this study were consistent with the findings of the California State Department of Food and Agriculture [132] which indicated that workers engaged in mixing, loading, and applying account for most of the reported severe occupational intoxications from pesticides.

Trefilov et al [77] studied clothing contamination and personal hygiene in a metaphos (methyl parathion) manufacturing plant. The authors found the highest degree of contamination of the special protective clothing of operators and mechanics in the chest area (16-190 mg/sq m/workday). For mechanics, trousers showed the highest contamination of the regular clothing, especially at the knees (640-720 mg/sq m/workday). Contamination of underwear ranged from 1 to 600 mg/sq m/workday. This latter finding is particularly significant for male workers because of the high absorbancy of parathion (and presumably methyl parathion) by scrotal

skin. [133] The authors also reported that showering with soap and water decreased the amount of methyl parathion in wrist washings from an initial range of 0.8-310 mg/sq m to 0.2 mg/sq m. Multiple skin washes from wrists, chest, forehead, and back of each operator and machinist confirmed the importance of dermal protection generally and of hand protection in particular. Dermal absorption was confirmed in the study by preexposure and postexposure erythrocyte and plasma cholinesterase tests on workers who wore gas masks.

Control of Exposure

Engineering controls and work practices for methyl parathion should have as their main objectives the control of vapor and aerosol concentrations, minimization of skin contact, and the prevention of fires. Closed systems and operator enclosures, properly operated and maintained, should be used where it is feasible to achieve all three of these objectives. Operations in which methyl parathion concentrates are poured or otherwise handled by workers should be eliminated, whenever possible, by transfer devices which minimize potential exposure. General room ventilation is necessary in methyl parathion-manufacturing and methyl parathion-formulating areas. Exhaust systems are needed at loaders, blenders, mixers, mills, packaging equipment, and at all other potential sources of vapor, spray, or dust containing methyl parathion. Liquid and dust exhaust systems must be designed so that neither the employees nor human and animal life in the surrounding area are endangered. Dust exhaust systems should be vented to a dust collector, not directly into the atmosphere. Exhaust air should not be recirculated. Detailed information

on the design and installation of methyl parathion vapor and dust exhaust systems should be sought from competent sources, such as ventilation engineers or industrial hygienists. Guidance for design can be found in Industrial Ventilation--A Manual of Recommended Practice, [134] or more recent revisions, and in ANSI Z9.2-1971. [135] Respiratory-protective equipment is not an acceptable substitute for feasible engineering controls but should be available for emergency purposes and for nonroutine maintenance and repair situations.

Methyl parathion is very unstable to heat [136] and may explode at 248 F (120 C). [136] The flashpoint of 80% methyl parathion in xylene is 115 F (46 C). [136] Structures in which methyl parathion is manufactured or stored should be designed to reduce the possibility of fire or the spread of fire. Overheated drums of technical product may rupture violently. [136] Heat and air currents will vaporize methyl parathion and cause contaminated particles to become airborne, producing highly toxic fumes and smoke. Heat also will promote conversion of methyl parathion to the corresponding oxon with an increase in toxicity. [136] Since water and chemicals used for firefighting may spread contamination over a wide area, efforts should be made to dike the run-off water, where possible, so as to prevent its entering sewers or streams. [136] Firefighting personnel should wear impervious gloves, hats, suits, and footwear, and use supplied-air respirators.

Firefighting procedures should be developed in advance, and local fire departments, as well as plant employees, should be informed of the hazards involved. Additional information on firefighting precautions appears in the Safety Guide for Warehousing Parathions. [136]