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U. S. ENVIRONMENTAL PROTECTION AGENCY
Washington, D.C. 20460

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OFFICE OF
PREVENTION, PESTICIDES
AND TOXIC SUBSTANCES

July 30, 1999

MEMORANDUM

SUBJECT: Final EFED RED Chapter for **Methyl Parathion**
PC Code No. **053501**

RECEIVED

OPP DOCKET

TO: Dennis Deziel, Chemical Review Manager
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RB III, Special Review and Reregistration Division (7508W)

FROM: Kevin Costello, Geologist, Task Leader
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Kevin J Costello 7/30/99

THROUGH: Denise Keehner, Acting Division Director
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Attached is the revised Environmental Fate and Effects Division RED chapter for methyl parathion. This document was prepared in response to comments received from Cheminova, the principal registrant of methyl parathion, and from other interested parties who submitted comments during the public comment period. This RED chapter incorporates some revisions in response to these comments.

However, the conclusions of the methyl parathion risk assessment remain essentially the same as they were described in the draft RED chapter. One exception is the **expected reduced risk to bees associated with the voluntary cancellation of tree fruit uses of methyl parathion.** Most changes made in response to Cheminova's comments were clarifications to the risk assessment, or corrections of internal inconsistencies in the document. The original risk assessment was based on scientifically-sound data submitted to the Agency to meet registration requirements for methyl parathion, and was well-supported by incident data and the open literature.

INTRODUCTION AND USE CHARACTERIZATION

Methyl parathion is an insecticide and acaricide used to control boll weevils and many biting or sucking insect pests of agricultural crops. Methyl parathion is in the organophosphate class of insecticides and kills insects by contact, stomach and respiratory action.

Methyl parathion has been registered for agricultural use since 1954. It has been classified as a Restricted Use Pesticide (RUP) since 1978 based upon its acute toxicity to humans and birds. Therefore, it can only be sold or distributed to, and used by, Certified Pesticide Applicators or persons under their direct supervision. Methyl parathion is registered for outdoor, agricultural uses only.

There are two main registrants for methyl parathion. Cheminova Agro AS produces most of the technical methyl parathion sold in the United States (Griffin Corporation was granted a registration for technical methyl parathion in 1998). Cheminova also produces a 4 lb ai/acre emulsifiable concentrate formulation, and a 6/3 EC mixture with their insecticide ethyl parathion. Elf Atochem North America is the registrant of the Penncap-M formulation, which has been registered in the United States since 1974. Penncap-M is formulated into microcapsules which range in size from approximately 5 to 50 microns (about the size of dust or pollen particles).

The registrants are supporting the use of methyl parathion on 26 crops. More than two-thirds of the estimated 4,000,000 pounds of methyl parathion used annually is on cotton and corn. The cotton market accounts for more than half of the usage in the United States, and is dominated by Cheminova's EC formulation.

Because cotton accounts for a majority of methyl parathion sales, use of methyl parathion is heaviest in the southern United States and California. Cotton production is most concentrated in five regions of widely varying climate and hydrogeology: the Mississippi Delta, the High Plains and southern tip of Texas, California's Southern Valley, and southwest Arizona. However, although cotton is the most important market for methyl parathion, data provided by Cheminova indicates that this chemical is used in almost every state in the Union. Penncap-M accounts for most of the use of methyl parathion on corn, and corn is consistently the largest market for this formulation.

With the reregistration of methyl parathion, the registrants will no longer support the use on a large number of crops. Included among the uses that will be voluntarily canceled are tree fruits such as apples, peaches, pears, plums and cherries, vine crops such as grapes, all berry uses, a number of field crops (including sorghum and many vegetables), and non-food uses such as for public health, wastelands, ornamentals and grass grown for seed. A full list of the crops that will no longer be supported is included later in the chapter.

Since Cheminova has decided not to support these uses with tolerances, they will not be included in EFED's methyl parathion risk assessment.

Organophosphate insecticides such as methyl parathion are generally highly toxic compounds which work "primarily by phosphorylation of the acetylcholinesterase enzyme at nerve endings." Acetylcholinesterase inhibition interferes with "normal transmission of nerve fibers to innervated tissues" (Morgan, 1976). Organophosphate poisoning can be fatal to non-target organisms, often through depression of respiration, or by causing a variety of sublethal effects which may adversely affect survival.

The current label includes language warning of the hazards this chemical can pose to human health, birds, bees, aquatic invertebrates and other wildlife. In response to problems related to product misuse, Cheminova has agreed to several mitigative measures for the EC formulation in addition to methyl parathion's RUP classification. These include the addition of a stenching agent to allow detection of methyl parathion and to discourage indoor use, the sole packaging of the chemical in containers 15 gallons and larger, unique tracking numbers on each returnable, refillable container, and the limitation that no formulation contain more than 5 pounds of the active ingredient per gallon. Cheminova has also developed an education and product stewardship program to promote safe and proper use.

The cumulative risk from other organophosphates must be considered along with methyl parathion under the requirements of the Food Quality Protection Act. Since label warnings and mitigation measures have already been implemented for methyl parathion, there are fewer options still available for mitigation of potential human health or ecological concerns. Given that either organophosphate and carbamate pesticides are applied to 70% of the acres treated with insecticides in the United States (Gianessi, 1997), it is imperative that mitigation measures be developed to reduce human health and ecological risks to acceptable levels. Possible mitigation measures are recommended in the Risk Characterization.

EXPOSURE ASSESSMENT

Environmental Fate Assessment

The environmental fate assessment for methyl parathion is based on acceptable and supplemental data. A common problem in the metabolism studies was the inability to identify all degradation products of methyl parathion. Since methyl paraoxon is a toxicologically significant degradate, EFED is concerned that methyl paraoxon may be an unidentified degradation product in the metabolism studies. Although the weight of evidence from supplemental data and open literature suggest that methyl paraoxon is not formed in aerobic soil environments, EFED believes that additional aerobic soil metabolism studies are needed to confirm that methyl paraoxon is not formed.

The major routes of dissipation for methyl parathion are microbial degradation, aqueous photolysis, hydrolysis, and incorporation into soil organic matter. Methyl parathion degrades rapidly ($t_{1/2}$< 5 days) in soil and water. It also is expected to photodegrade ($t_{1/2}$=49 hours) in aquatic environments. Other degradation processes appear to be less important routes of methyl parathion dissipation. Methyl parathion slowly hydrolyzed ($t_{1/2}$=68 days at pH 5, $t_{1/2}$=40 days at pH 7, $t_{1/2}$=33 days at pH 9) in buffer solutions and slowly photodegraded ($t_{1/2}$=61 days) on soil surfaces.

The major (>10% of applied) degradation product of methyl parathion is 4-nitrophenol. This degradate is formed through the hydrolytic cleavage of nitrophenyl C-O-P bond. Other minor degradates (<10% of applied) that have been found in laboratory studies include methyl paraoxon, monodesmethyl parathion, phosphorothioic acid, O,S-dimethyl o-(4-nitrophenyl)ester, nitrophenyl phosphoric acid, mono (4-nitrophenyl) ester and CO_2. Of these, only methyl paraoxon is included in HED's tolerance expression. Methyl paraoxon has only been detected (2.1% of applied) in the anaerobic aquatic metabolism study. This degradate is formed through a desulfonation (P=S to P=O) reaction. It should be noted, however, that the amount of methyl paraoxon derived by aerobic soil metabolism is not clear at this time. In addition, analyses for methyl paraoxon in two field dissipation studies are questionable because of storage stability issues.

Methyl parathion is mobile to relatively mobile in soil and thus runoff and leaching could be potential routes of dissipation. However, the low persistence of methyl parathion is expected to limit the extent off-site movement. Supplemental data on parent methyl parathion indicate that it is very mobile to somewhat mobile [K_{oc}s =230-to-670 l/kg] in mineral soils. Since the soils used in the batch equilibrium experiment were sterilized by autoclaving, confirmatory batch equilibrium data are needed. Another route of dissipation is the secondary movement through volatilization of methyl parathion from soil and leaf surfaces. Although laboratory studies indicate that methyl parathion volatilization is not a major route of dissipation, methyl parathion has been detected in air and rain samples across the United States. These detections appear to be correlated to use on cotton, soybeans, wheat, and tobacco. EFED notes that methyl parathion will no longer be supported for use on tobacco.

Methyl parathion, formulated as EC, dissipated rapidly (<1 day) in a field dissipation study performed in a cotton field in California. Methyl parathion was not detected below 4 inches. Acceptable field studies have not been performed using the microencapsulated formulation Penncap-M.

Status of Environmental Fate Data

The current status of environmental fate data requirements for support of registration of methyl parathion is detailed below.

(1) <u>Satisfied:</u>

161-1. **Hydrolysis (Satisfied)-** MRID #0013275,40784501

Phenyl ring-labeled [^{14}C]methyl parathion (radiochemical purity >99%), at 3.87-3.95 mg/L, hydrolyzed with half-lives of 68 days at pH 5, 40 days at pH 7, and 33 days at pH 9 in sterile aqueous buffered solutions at 25 C. Major hydrolysis degradates (10% of applied) of methyl parathion are monodesmethylparathion-methyl and 4-nitrophenol. Impurities and "unknowns" comprised a maximum of 2% of the applied during the 30-day study. In an earlier unacceptable study, methyl parathion hydrolyzed in unbuffered distilled water containing 0.1% acetone. Methyl paraoxon was not detected in abiotic hydrolysis studies.

161-2. **Photodegradation in Water (Satisfied)** MRID #40809701.
161-3. **Photodegradation on Soil (Satisfied)** MRID #00061200,00072377,40809702.

[^{14}C]Methyl parathion (radiochemical purity >99%), at 4.71 mg/L, photodegraded with a half-life of 49 hours in sterile aqueous pH 5 buffered solutions that were irradiated continuously for 212 hours with a xenon arc lamp at 25 C. In the dark control solutions (incubation conditions not described), methyl parathion was relatively stable. Major photodegradation products (8-13%) were 4-nitrophenol and monodesmethylparathion-methyl. Unidentified degradates (fractions "A" and "B", which each contained more than one compound) each comprised up to 38% of the recovered radioactivity, and radioactivity designated as "remainder", which included paraoxon-methyl, comprised a maximum of 16% of the recovered. $^{14}CO_2$ accounted for 18.4-30.9% of the applied radioactivity at 212 hours posttreatment, and organic volatiles comprised a maximum of 3.0-5.3% of the applied.

In two photodegradation studies on soils under artificial light, [^{14}C]methyl parathion (radiochemical purity >99%), at approximately 14 μg/cm^2, degraded with a biphasic half-life of an initial half-lives of 3.9 to 4.5 days and a secondary half-lives of 8.6 to 24 days on sandy loam soil when irradiated continuously for 281 hours with a xenon arc lamp at 25-28°C. Methyl parathion was stable ($t_{1/2}$=29 to 54 days) in dark controls.

In a photodegradation study on soil under natural light, [^{14}C]methyl parathion (radiochemical purity >99%), at >14 μg/cm^2, degraded with a dark control corrected half-life of 61 days on sandy loam soil. The soil was irradiated with sunlight outdoors for 22 days at approximately 25 C at Monheim, Germany, beginning July, 1987. Methyl parathion was relatively stable ($t_{1/2}$ = 106 days) in dark control treatments. The major photodegradate was 4-nitrophenol. However, unidentified radioactivity reached a maximum of 17.8% of the recovered radioactivity. Unextracted methyl parathion residues comprised a maximum of 20.1 to 41% of the applied radioactivity. At 281 hours posttreatment, $^{14}CO_2$ totaled 2.0 to 16.1% of the applied radioactivity, and organic volatiles were <0.1%.

162-1. **Aerobic Soil Metabolism (Upgradable Supplemental)**-MRID #41735901.

Ring-labeled [^{14}C]methyl parathion (radiochemical purity 97.2%) degraded with a registrant calculated half-life of 4.7 days in sandy loam soil that was incubated in the dark at 25 C. Since methyl parathion degradation appears to be biphasic, EFED recalculated a half-life of 3.75 days for methyl parathion using non-linear fitting techniques of the first-order degradation kinetic model to non-transformed data. Minor degradates (<10% of applied) were 4-nitrophenol and O,O-bis(4-nitrophenyl)-O-methyl phosphorothioate. Unidentified degradates ("solvent front") each comprised up to 4.97% of the applied radioactivity. Unextracted radioactivity in the soil was a maximum of 38.72% of the applied at 1 month posttreatment. Unextracted methyl parathion was predominately detected in the fulvic acid (31.9-15.7%) and humin fraction (38.5 to 45.1%). At 6 months posttreatment, volatilized $^{14}CO_2$ totaled 62.72% of the applied, and organic volatiles totaled 1.37% of the applied.

162-2. **Anaerobic Soil Metabolism**; not required if Anaerobic Aquatic Metabolism is made acceptable by the submission of supplemental data.

162-3. **Anaerobic Aquatic Metabolism (Not Satisfied)-** MRID #41768901.

Uniformly ring-labeled [^{14}C]methyl parathion (radiochemical purity 95%), at a nominal concentration of 10 µg/g, degraded with a half-life of 12.2 hours in flooded sandy loam soil (10 g soil:20 mL water) that was incubated under anaerobic conditions in the dark at 25 $\pm$ 1 C. Methyl parathion (50% EC, Metacid), at 25 ppm, degraded with an observed half-life of 1-2 days in flooded alluvial soil incubated at 28 $\pm$ 4 C for 12 days. The major degradate of methyl parathion was p-nitrophenol. Minor degradates (< 10% of applied) of methyl parathion are S-methyl parathion; O,O-bis-(4-nitrophenol)-O-methyl-phosphorothioate; methyl paraoxon; amino-methyl parathion; and S-phenyl-methyl parathion. Five unidentified degradates (Unknowns 2-6) were detected at maximum concentrations of 1.2-14.4% of the initial radioactivity. At 12 months posttreatment, unextracted [^{14}C]residues in the soil totaled 75.2% and $^{14}CO_2$ totaled 2.74% of the initial radioactivity. Unextracted [^{14}C]residues in the 14-day and 9-month samples were predominately detected in the fulvic acid (13.2-15.3%) and humin (20.1-20.2%) organic matter fraction. No organic volatiles were detected (detection limit not reported).

162-4. **Aerobic Aquatic Metabolism (Satisfied)-**MRID# 0013361, 00128789, 42069601

Radiolabeled methyl parathion degraded with a half-life of approximately 4.1 days in sandy loam soil that was flooded with water incubated for 30 days in the dark at 25°C (MRID 42069601). Methyl parathion was primarily associated with the soil fraction; it was not detected in the flood waters after 2 days posttreatment. The only degradate identified was paranitrophenol.

163-1. **Leaching and Adsorption/Desorption (Not Satisfied-Supplemental)**-MRID 40999001

Based on batch equilibrium experiments conducted using autoclaved soils, [^{14}C]methyl parathion (radiochemical purity 98.8%), at 1.86-19.1 ug/mL, is expected to be very mobile in sand and sandy loam soil:0.01 N calcium chloride solution slurries and mobile in silt loam and clay loam soil:solution slurries (3:10 for sand and sandy loam soils, 1:10 for silt loam and clay loam soils) that were equilibrated for 24 hours at 25 C. Freundlich K_{ads} and exponential (1/n) values were 0.574 (1/n=0.96) for the sand soil, 1.82 (1/n=0.909) for the sandy loam soil, 7.09 (1/n=0.917) for the silt loam soil, and 8.71(1/n=0.961) for the clay loam soil. Since there is a correlation of methyl parathion sorption and soil organic matter content, it is appropriate to use the K_{oc} model for describing methyl parathion sorption (Sanchez-Martin and Sanchez-Camazano, 1991). K_{oc} values were 230 for the sand soil, 456 for the sandy loam soil, 591 for the silt loam soil, and 670 for the clay loam soil. Following desorption in pesticide-free calcium chloride solution for 24 hours, 43.12-54.26% of the radioactivity that had been adsorbed to the soils was desorbed from the silt loam and clay loam soils, 57.23-67.84% was desorbed from the sandy loam soil, and 98.62-112.35% was desorbed from the sand soil.

In earlier supplemental soil column studies, methyl parathion was mobile in sand and relatively immobile in sandy loam, silty clay loam, and silt loam through 30 cm soil columns eluted with 15.7 inches of water (MRID 00071198). Methyl parathion was only detected in the leachate of the sand soil. Open literature data indicate that methyl parathion sorption on soil is correlated to soil organic matter content (Sanchez-Martin and Sanchez-Camazano, 1991). Methyl parathion had an average K_{oc} of 697 ml/g across 8 mineral soils. In contrast, methyl paraoxon sorption was correlated to clay content. Methyl paraoxon had distribution coefficients (K_ds) ranging from 1.77 to 14.3 ml/g in 8 mineral soils..

163-2. **Laboratory Volatility (Satisfied)-** MRID #42264201, 41194001

Methyl parathion, formulated as 4 lb ai/gallon EC, volatilized slightly (<0.51% of applied) from a Sesquatchie sandy clay loam soil that had been moistened to 50 or 75% at 1/3 of field capacity and then incubated in the dark at 25°C for 9 days. The maximum air concentration and volatility rate of methyl parathion was 55.88 $\mu g/m^3$ and 0.0128 $\mu g/cm^2$/hour, respectively, when incubated at 75% of the soil water holding capacity and 300 mL/minute air exchange rate.

163-3. **Field Volatility-(Not Satisfied)-**MRID 41194001

Methyl parathion, applied at 1 lb ai/A either as EC or MCAP formulations (concentration of methyl parathion in the formulations not specified) to tobacco plots (soil not characterized) near Raleigh, North Carolina, volatilized with maximum mean air concentrations (110-cm sampling level immediately posttreatment) of 7400 and 3800 ng/m^3 for the EC and MCAP formulations, respectively.

In a USGS review, methyl parathion has been detected in air samples in Alabama, Florida, and Mississippi at concentrations ranging from 5.4 to 129 ng/m^3 (Majewski and Capel, 1995). Methyl parathion in air also was detected (0.4 to 42 ng/m^3) throughout the southeastern United States. Methyl parathion has also been detected (1.60 $\mu g/L$) in Iowa precipitation. The USGS suggested the methyl parathion concentrations in air tend to correspond with methyl parathion use areas associated with cotton, soybeans, wheat, and tobacco production. Of these crops, Cheminova will no longer be supporting the use of methyl parathion on tobacco.

164-1. **Terrestrial Field Dissipation (Partially Satisfied)- MRID 41481001, 41752501, 41481002, 41752502**

Methyl parathion rapidly dissipated with a half-life of approximately 1 day from plots of sandy loam soil located in California following the last of six applications of methyl parathion (4 lb/gal EC) to cotton at 1 lb ai/A/application (total application 6 lb ai/A). Supplemental field dissipation data indicate that methyl parathion (4 lb ai/gal EC), applied at six weekly applications at 1 lb ai/A/application (total 6 lb ai/A) to cotton on plots of loam soil located near Steele, Missouri, beginning July 28, 1988, decreased from an average of 0.052 ppm immediately following the last treatment to below the detection limit (0.05 ppm) by 1 day following the last treatment in the surface 4 inches of soil. Methyl parathion was not detected in the soil by 7 days posttreatment. Methyl parathion did not appear to accumulate or move into the soil as a result of repeated applications.

164-2. **Aquatic Sediment Dissipation (Satisfied)**-MRID #41481003 and 41752503.

Methyl parathion dissipated from irrigation water with an observed half-life of approximately 1 day following the last of six weekly treatments of methyl parathion (4 lb ai/gal EC) at 0.75 lb ai/A/application (total 4.5 lb ai/A) to plots of irrigated (6-inch depth) sandy loam soil that was planted to rice and located near Madera, California; methyl parathion had totally dissipated from the irrigation water by 7 days post-treatment. Methyl parathion dissipated from irrigation water with an observed half-life of <7 days following the last of six weekly treatments of methyl parathion (4 lb ai/gal EC) at 0.75 lb ai/A/application (total 4.5 lb ai/A) to plots of irrigated (3-inch depth) loam soil planted to rice that were located near Steele, Missouri. Methyl parathion did not accumulate in the water as a result of repeated applications. The degradate p-nitrophenol was isolated in the irrigation water.

165-4 **Accumulation in Fish (Satisfied)**-MRID #41001901.

Bluegill sunfish exposed to radiolabeled methyl parathion at 0.104 mg/L had steady-state bioaccumulation factors of 39X in edible tissues, 108X in nonedible tissues, and 71X in whole body over a 28 day accumulation period. Steady-state conditions were obtained within 3 days. Radiolabeled residues in whole fish tissues were identified as 0,0-dimethyl-0-4-nitrophenyl phosphorothioate (methyl parathion 22.6%), 0-methyl-0-4-nitrophenyl phosphorothioate (46.3%), 0-methyl-0-4-nitrophenylphosphate (5.7%), 4-nitrophenol (18.1%), and 4-NP-gluconuride (1.2%). Unextracted residues represented 6.1%.

WATER RESOURCE ASSESSMENT

First-Tier Water Assessment for Methyl Parathion

SURFACE WATER ASSESSMENT FOR METHYL PARATHION:

EFED uses the GENEEC screening model to estimate surface water concentrations for first-tier exposure assessments. GENEEC is a screening model designed by the Environmental Fate and Effects Division (EFED) to estimate the concentrations found in surface water for use in ecological risk assessment. As such, it provides upper-bound values on the concentrations that might be found in ecologically sensitive environments because of the use of a pesticide. It was designed to be simple and require data which is typically available early in the pesticide registration process. GENEEC is a single event model (one runoff event), but can account for spray drift from multiple applications. GENEEC is hardwired to represent a 10-hectare field immediately adjacent to a 1-hectare pond that is 2 meters deep with no outlet. The pond receives a spray drift event from each application plus one runoff event. The runoff event moves a maximum of 10% of the applied pesticide into the pond. This amount can be reduced due to degradation on the field and the effects of soil binding in the field. Spray drift is equal to 1 and 5% of the applied rate for ground and aerial spray application, respectively.

Modeling results indicate that methyl parathion has the potential to move into surface waters. This estimate is based on the maximum application rate for cotton, which represents the highest application rate for any crop used to support residue tolerances. Coincidentally, cotton also accounts for the majority of methyl parathion use in the United States, according to data provided by Cheminova. EFED notes that higher use rates are reported on product labels but the registrant has stated they will not support rates greater than those defined in crop residue studies. Based on the inputs shown in Table 1 the peak GENEEC estimated environmental concentration (EEC) of methyl parathion in surface water is 452 ppb (Table 2). This was the value recommended to HED as the highly conservative Tier I estimate of *acute* drinking-water exposure for their human health risk assessment. EFED recommended a highly conservative Tier I *chronic* drinking-water exposure estimate of 50 ppb, based on the 56 day average GENEEC value obtained with the highest use-rate for methyl parathion.

Table 1: GENEEC Environmental Fate Input Parameters for Methyl Parathion

DATA INPUT	INPUT VALUE	DATA ASSESSMENT	SOURCE
Application Rate	3.0 lbs ai/A		Cheminova
Maximum Number of Applications	10		Cheminova
Application Interval	3 days		Cheminova
Batch Equilibrium (Koc)	230 mL/g*	Acceptable	MRID 40999001
Aerobic Soil Metabolism	$t_{1/2}$ = 11.25 days**	Supplemental	MRID 41735901
Solubility	60 ppm	Acceptable	Reported by registrant
Aerobic Aquatic Metabolism	$t_{1/2}$ = 4.1 days	Acceptable	MRID 41768901
Photolysis	$t_{1/2}$ = 49 hours	Acceptable	MRID 40809701

* The smallest K_{oc} value was used in order to produce the highest (most conservative) exposure

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Table 4: Agronomic Input Parameters for Corn				
Crop	Emergence Date	Harvest Dates	Application Dates	Application Method
Corn	May 16	October 11	Sept. 1 to 11	Aerial

This PRZM simulation reflects the maximum label rate (1.0 lb ai/a), number of applications (6/year) and application interval (2 days) sought by the registrants for methyl parathion on corn. In their QUA+ response, Atochem states that application is made from July to August at rates of 0.25 to 0.5 lb ai/a. For sweet corn, typical use is 0.5 to 1.0 lb ai/a later in the season, with one or two applications being typical. Food processor Del Monte reports that they use 0.5 to 0.75 lb ai/a only once per season on 10% of their crop, while competitors use 0.5 to 1.0 lb ai/a 1 to 4 times a year, on 50% of their crop.

Alfalfa

This input file was adapted from EFED's standard PRZM scenario for alfalfa grown on the Fury silty clay loam in Oregon, dated January 15, 1998. Thirty-six years (1948-83) of weather data from MLRA 23 are used for this simulation. Application dates used in this simulation reflect the average pre-harvest interval (15 days) reported to EPA by Elf Atochem, registrant of Penncap-M. Emergence, maturation and harvest dates were provided to EFED by Dr. Ben Simko, Extension Entomologist with the Malheur County, OR Cooperative Extension (Table 5).

Table 5: Agronomic Input Parameters for Alfalfa				
Crop	Planting Date	Harvest Date	Application Dates	Application Method
Alfalfa	March 22	September 7	April 19 to August 23	Aerial

This PRZM simulation reflects the maximum label rate (1.0 lb ai/a), number of applications (4/year) and application interval (42 days) sought by the registrants for methyl parathion on alfalfa. Atochem notes in their response for BEAD's QUA+ that one application each of 0.75 lb ai/a is made at the first and second cuttings. Usage is primarily on western alfalfa grown for seed.

Potato

This input file was adapted from EFED's standard PRZM scenario for potatoes grown on the Conant silt loam in Maine, dated February 13, 1998. Thirty-six years (1948-83) of weather data from MLRA 143 are used for this simulation. Application dates used in this simulation were provided by Dr. Jim Dwyer of the Aroostook County Office of the University of Maine

Cooperative Extension Service. Emergence, maturation and harvest dates used in the simulation were confirmed by Dr. Matthew Kleinhenz, also from the Aroostook extension office (Table 7).

Table 7: Agronomic Input Parameters for Potatoes				
Crop (Surrogates)	Planting Date	Harvest Date	Application Dates	Application Method
Potato (Cabbage)	May 5	September 18	July 1 to Aug. 5	Aerial

This PRZM simulation reflects the maximum label rate (1.5 lb ai/a), number of applications (6/year) and application interval (7 days) sought by the registrants for methyl parathion on potatoes. However, Dr. Kleinhenz reported that methyl parathion is not commonly used in Maine on potatoes. Atochem reports that use in the East is limited due to resistance in the Colorado potato beetle.

Pecans

This input file was adapted from EFED's standard PRZM scenario for pecans grown on the Williston loamy sand in Georgia, dated January 21, 1998. Thirty-six years (1948-83) of weather data from MLRA 138 are used for this simulation. Application dates used in this simulation were provided by Dr. Jim Dutcher of the University of Georgia Department of Entomology (Table 8). Dr. Dutcher indicated that harvest is 25% complete by Thanksgiving, and completed by Christmas.

Table 8: Agronomic Input Parameters for Pecans				
Crop (Surrogates)	"Emergence" Date	Harvest Date	Application Dates	Application Method
Pecans (Almonds)	May 11	October 25	July 9 to Oct. 1	Air Blast

This PRZM simulation reflects the maximum label rate (2.0 lb ai/a), number of applications (8/year) and application interval (14 days) sought by the registrants for methyl parathion on pecans. However, Dr. Dutcher explained that it is unlikely that growers could get around to make that many applications in a season, given the size of the orchards. He reported that two applications of 1 to 2 lb ai/a methyl parathion might be made for stinkbug control. The first would occur about two weeks after shell hardening, around the 20th of August. A second might be made two weeks after that. In order to accommodate 6 applications, the 14-day application interval, and the 30-day pre-harvest interval, applications are simulated in the model before and after these dates.

Atochem confirms that the main use is for stinkbug during nut development, but states that Penncap should not be used when flowering weeds are on the orchard floor. The National Pecan Shellers Association reports that 85% of methyl parathion use is at 0.5 lb ai/a, and the rest at 0.75 lb ai/a. They estimate that Penncap-M has 30 to 40% of the pecan market for stinkbug control.

Sweet Potatoes

This input file was adapted from EFED's standard PRZM scenario for sweet potatoes grown on the Calhoun silt loam in Louisiana, dated January 19, 1998. Thirty-six years (1948-83) of weather data from MLRA 133b are used for this simulation. Planting and harvest dates were provided by Dr. Donald LaBonte, of the Louisiana State University Agricultural Center (Table 11).

Table 11. Agronomic Input Parameters for Sweet Potatoes				
Crop	Planting Dates	Harvest Date	Application Dates	Application Method
Sweet Potatoes	May- June 15 (used May 25)	110 days after planting (9/13)	July 1 to Aug. 19	Aerial

This PRZM simulation reflects the maximum label rate (0.75 lb ai/a), number of applications (8/year) and application interval (7 days) sought by the registrants for methyl parathion on sweet potatoes. Dr. Abner Hammond of the LSU Ag. Center confirmed these dates as realistic, stating that methyl parathion might be applied from July 4 until October 1. Atochem suggests that Penncap-M is used typically at 0.38 lb ai/a 3 to 5 times a year. The 24C approvals are for use in Louisiana, Mississippi. Alabama, and Arkansas, with another pending for Texas.

Results

The Tier II EECs for methyl parathion are listed in Table 12. The EECs have been calculated so that in any given year, there is a 10% probability that the maximum average concentration of that duration in that year will equal or exceed the EEC at the site.

Table 12: Tier II upper tenth percentile EEC's for Methyl Parathion for simulated crops.						
Crop	Maximum ($\mu g \cdot L^{-1}$)	4 Day ($\mu g \cdot L^{-1}$)	21 Day ($\mu g \cdot L^{-1}$)	60 Day ($\mu g \cdot L^{-1}$)	90 Day ($\mu g \cdot L^{-1}$)	Long-term Mean ($\mu g \cdot L^{-1}$)
Cotton	254.40	174.20	70.63	32.76	23.19	6.55
Corn	39.45	27.28	12.225	5.35	3.60	.97
Alfalfa	4.33	2.9	1.432	.77	.61	.29
Potato	21.3	14.3	6.71	3.69	2.49	.69
Pecan	12.30	9.38	6.012	3.74	3.25	1.1
Sweet Potato	36.39	24.76	10.766	5.69	4.2	1.2

Limitations of this Analysis

The use of simulation models to estimate possible drinking-water exposure introduces several degrees of uncertainty to a human health or ecological risk assessment. The greatest of these may be the conservative assumptions of the modeling that are intended to ensure the maximum protection for human health. The scenario simulated by both GENEEC and PRZM-EXAMS is a single 10-hectare field draining to a 1-hectare pond with no outlet. This represents a highly conservative assumption, since this scenario does not accurately reflect the dynamics in a watershed large enough to support a drinking water facility.

Additional assumptions ensure that the resulting Tier 2 EEC's are sufficiently conservative to protect human health and the environment:

- Sites simulated in Tier 2 modeling are chosen by best professional judgement to be among the most vulnerable for each crop to which the pesticide is applied.

- The 10-hectare field is assumed to be planted completely to the crop in question;

- The entire annual application of the pesticide is assumed to occur over the 10 hectares within one day; and

- The application rates and timing for each crop are the maximum allowed on the product label.

A watershed large enough to support a drinking-water facility would rarely be planted completely to a single crop, and treated uniformly with the same pesticide at the maximum label rate.

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These conservative assumptions are intentionally chosen, in part, to account for other sources of uncertainty associated with the use of simulation models in risk assessment. The first of these is the quality of the input data used in the simulations, which is detailed to some extent above. For instance, data from invalidated environmental fate studies calls the input parameters derived from the studies to question. In addition, the precipitation data used is limited to a maximum of 36 years, with no irrigation simulated in any year. Finally, direct deposit to the pond by spray drift is simulated to be 1% and 5% of the application rate for ground and aerial applications, respectively. Outstanding data from the Spray Drift Task Force may require that these numbers be revised for future assessments.

Finally, the models themselves are a source of uncertainty in the assessments. While the models are some of the best environmental fate estimation tools available, they have significant limitations in their ability to represent some processes. Several of the algorithms (volume of runoff water, eroded sediment mass) are well validated and well understood, but no adequate validation has yet been made of PRZM 3.1 for the amount of pesticide transported in runoff events. Other limitations of the models used include the inability to handle spatial variability within the simulated 10-hectare field, a lack of crop-growth algorithms, and a simplistic soil water transport algorithm (the "tipping bucket" method).

Therefore, given these limitations, a Tier II EEC should be considered a reasonable upper bound estimate of the concentration that could be found in drinking water, and not a prediction of concentrations that would commonly be detected. Risk assessment using Tier II values can be used as refined screens to demonstrate that the risk to human health or the environment is below a level of concern. When Tier II EEC values are above levels of concern, additional data or proactive mitigation measures may be necessary, depending on the magnitude of the LOC exceedence.

Surface Water Monitoring

Direct drinking-water data for methyl parathion are not readily available, and it is not likely that many such data have been collected. Public drinking-water supply systems must periodically analyze drinking water for contaminants that either: 1) have a Maximum Contaminant Level (MCL) established by the Office of Water, or 2) are included on the Unregulated Contaminant Monitoring List (UCML). While the Office of Water has established a lifetime health advisory (HA) of 2 ppb, methyl parathion does not have an established MCL, and is not included on the UCML. Therefore, few public drinking water supply systems are likely to have analyzed for methyl parathion.

One exception is the Jefferson Parish, Louisiana CWS, which did test for methyl parathion in 1994 at two intakes on the Mississippi River. In this study, continuous raw water samples were collected with a peristaltic pump into a 5-gallon carbuoy. Composite samples so collected were analyzed weekly for a year. Methyl parathion was detected in 18 of 52 samples from the one bank of the river, and 21 of 52 from the other bank. The average concentration of the detections was

0.009 ppb, the level of detection. The highest concentration detected was 0.041 ppb.

While the samples analyzed in the Jefferson Parish study only reflect conditions of a single year, they are representative of possible chronic drinking-water contamination from a very large surface-water source. The low concentrations provide one piece of evidence that methyl parathion might not pose a chronic risk to drinking water supplies taken from large surface water sources. However, they are not useful in predicting possible acute exposure to methyl parathion.

Methyl parathion has been included as an analyte in several national-scale surface-water (non-drinking-water) monitoring studies since the early 1970's. Methyl parathion was detected in 2% or fewer of the samples taken in these studies, with a maximum concentration of 1 ppb in the USGS western streams study of 1968 to 1971 (Larsen, et al., 1997). In a recent example, Goolsby and Battaglin's Mississippi River and tributary study of the early 1990's, methyl parathion was detected at a maximum concentration of 0.008 ppb in 316 samples[4].

Methyl parathion is among the analytes included in the United States Geological Survey's National Water Quality Assessment Program (NAWQA). Low levels of methyl parathion were reported in preliminary results from samples collected from 1991-1995 from 20 major watersheds around the country[5]. The maximum concentrations detected are in Table 13.

Table 13: Surface Water Results, 1991-1995, USGS NAWQA Program

Type of Stream	# of Streams	# of Samples	Maximum Conc. (ppb)
Agricultural	37	1530	0.3
Urban	11	603	0.072
"Integrator"	14	555	0.028

The concentrations in the studies cited above are below those predicted by the GENEEC screening model. It should be noted that the analytical recoveries for methyl parathion in the NAWQA study is 46% (SD=13%). Such low recoveries limit extensive quantitative interpretation of the monitoring data. However, the monitoring data are expected to be lower than GENEEC because of the conservative assumptions used in the model for a first-tier assessment. Just as significant, however, is the fact that the Mississippi River and NAWQA programs were *non-targeted* monitoring surveys. These studies were designed to study the effects of agricultural runoff, but methyl parathion is only one of a suite of many pesticides included in the water analyses. There is no guarantee of how well samples taken in these programs correspond to times or locations of actual methyl parathion use.

A few reports are available that detail more targeted monitoring for methyl parathion. The California Environmental Protection Agency Department of Pesticide Regulation (CDPR) has a continuing, 10-year study of rice pesticides in surface water, which includes methyl parathion.

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CDPR samples the Colusa Basin Drain, an agricultural discharge channel that collects outflow from rice fields from about 20 to 100 miles north of Sacramento, and west of the Sacramento River. This area is used for many continuous miles of rice monoculture on heavy clay soils.

According to the CDPR, methyl parathion was detected at concentrations of up to 6 ppb in 1989. CDPR was concerned with surface water contamination by a suite of rice pesticides. By the late 1980s, CDPR had instituted a control program to reduce the surface water impacts of rice herbicides. In the early 1990s, the CDPR expanded the program to include rice insecticides.

The program includes both irrigation and application controls to reduce direct input of pesticides to the Colusa Basin Drain, which drains to the Sacramento River. Rice farmers are required to hold water on flooded rice fields for prescribed periods of time before releasing it to the drainage system, periods which depend on the pesticides applied. The holding time for methyl parathion is 24 days, but it is held longer if applied concurrently with another pesticide that must be held longer. Application controls include requirements such as positive shutoff systems for aircraft nozzles, use of drift control agents, and a 300-foot buffer from water bodies for aerial applications.

CDPR has seen measurable improvements in the samples they have taken each year from early or mid-April to mid-June. For instance, the peak concentration of methyl parathion detected in 1996, the last year for which a report has been prepared, was 0.12 ppb. A maximum concentration of 0.107 ppb was detected in 32 samples taken in 1997. **The results of this targeted study present data that are more realistic, but less conservative, than Tier I and Tier II estimates.** These data reflect successful mitigation, and also a reduction in methyl parathion use in the area over 15 years.

The surface-water database maintained by the CDPR includes 14 positive detections out of 1034 samples taken since 1991. Eleven of those detections were 1995-97 data from the Colusa Basin Drain study cited above. Two other detections connected with rice culture were collected from the Butte Flue in Yolo County; measured concentrations were 0.19 and 0.07 ppb. The only other detection in the database to date is from the San Joaquin Valley, a detection of 0.02 ppb in 1991, where methyl parathion is used in fruit production.

EFED has obtained more recent (after 1995), targeted surface-water monitoring data taken by the USGS NAWQA program from rivers in the Mississippi Embayment cotton-growing region. Samples were taken from five rivers in 1996 and 1997, and methyl parathion was detected in all five. Detected concentrations ranged from 0.015 to **0.422 ppb**. The site with the highest frequency of detections in this study had **8 detections in 17 samples during water year (WY)1996, and 8 detections in 37 samples during WY1997.**

In another 1996 monitoring program in the Mississippi Embayment, the USGS detected methyl parathion in 18% of the 60 samples it collected from tributaries of the Mississippi River. The highest concentration detected was about 0.12 ppb, and the 50th percentile concentration was about 0.05 ppb. Methyl paraoxon and 4-nitrophenol were not included as analytes for these samples, nor for the samples taken as part of the NAWQA program.

Heath, et al. (1993) cites data from a study that reported mean methyl parathion detections of 0.66 ppb in water from the Colusa Basin Drain in central California. This agricultural drain, which flows into the Sacramento River, accepts drainage from rice fields which are often treated with methyl parathion. The San Francisco Estuary Institute has reported as-yet unquantified detections of methyl parathion in regular (24 stations, 3 times yearly) sampling. A database maintained by Spectrum Laboratories reports that 15 ppb of methyl parathion was detected in storm water runoff following a foliar application. However, until a citation can be provided for this data, it must be considered anecdotal.

GROUND WATER ASSESSMENT FOR METHYL PARATHION

SCI-GROW is a screening level model developed by Dr. Michael Barrett (U.S. EPA/OPP/EFED) to estimate the "maximum" groundwater concentration from the application of a pesticide to crops. SCI-GROW is based on the fate properties of the pesticide, the application rate, and the existing body of data from small-scale groundwater monitoring studies[6]. The model assumes that the pesticide is applied at its maximum rate in areas where the groundwater is particularly vulnerable to contamination. In most cases , a considerable portion of any use area will have ground water that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimates. As such, the estimated "maximum" concentration derived using SCI-GROW should be considered a high-end to bounding estimate of drinking-water exposure from a ground-water source. If the risk associated with this estimate is exceeded, either at the acute or chronic end-points, refinement of the exposure estimate will be necessary to better characterize actual exposures. Table 14 provides the EEC for groundwater using the SCI-GROW model.

Table 14: Ground-Water Results for Methyl Parathion

CROP	App. Rate (lbs/ac)	# Apps./Yr	SCI-GROW Acute EEC (ppb)
Cotton	3.0	10	0.60

Ground-Water Monitoring

Methyl parathion has been detected in ground water, but these detections have been at low concentrations. The Pesticides in Ground Water Database (PGWDB) includes data from 3,357 wells, of which 20 showed positive detections of methyl parathion. The highest ground-water concentration reported from these wells was 0.256 ppb, from a well in Mississippi, although 13 wells in a 1987 Virginia study had detections below a 5 ppb level of quantification. The PGWDB reports that methyl paraoxon was not detected in samples taken from 125 wells in two states.

Methyl parathion was detected in 53 of 65 samples reported in a USGS study performed in Berkeley County, WV[7]. However, all of the detections were at levels below the quantification limit of 0.01 ppb. Berkeley County is an area underlain by karst geology, which can be considered as highly vulnerable to ground-water contamination. The samples in this study were taken from wells and springs.

In addition, methyl parathion was detected in ground water in samples taken from the NAWQA program. The maximum concentration detected from 1130 samples collected between 1991-1995 was 0.062 µg/L. **As with the surface-water monitoring, it should be noted that the analytical recoveries for methyl parathion in the NAWQA study is 46% (SD=13%). Such low recoveries limit extensive quantitative interpretation of the monitoring data.** Additionally, the NAWQA ground-water monitoring study was not specifically targeted for times and areas of methyl parathion use.

Methyl parathion was included, but not detected in the 1995 USGS Midcontinent Pesticide Study. The investigators analyzed 94 samples for methyl parathion, with an analytical reporting limit of 0.008 ppb. This study was not targeted specifically to methyl parathion, but did occur in corn and soybean growing areas.

This study included an analysis of the "age" of the ground water collected, measuring radioactive tracers to determine when the water recharged from the surface. Tritium levels in the water give an indication of whether the ground-water recharged from the surface before or after 1953, which marks the advent of atmospheric nuclear weapon testing. The year 1953 predates the registration of most current pesticides, including methyl parathion.

Analysis indicated that 19% of the samples collected were water that recharged prior to 1953. This water was more likely to occur in near-surface bedrock aquifers (50% of samples) than in near surface unconsolidated aquifers (9.1%). Pesticides were much less likely to be detected in pre-1953 water (16%) than in post-1953 water (70.3% of samples). The cause of the detections (atrazine at 3 to 9 ppt) in three "pre-1953" samples was likely the result of mixing with a small amount of post-1953 water in the aquifer.

The results of these analyses have important implications for ground-water derived drinking-water assessments. Large public drinking-water supply wells are often drilled deep into bedrock aquifers, and may represent water that recharged from the surface long before the advent of many modern pesticides. However, as indicated by the "pre-1953" water with atrazine detections described above, pesticides can persist in ground-water for lengths of time not consistent with laboratory degradation studies. Ground-water "age" data is rarely included with ground-water monitoring studies.

Methyl Parathion Degradates in Drinking Water

Degradate 4-nitrophenol, which is a degradate common to both methyl parathion and ethyl

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parathion, has been detected in drinking water. The EPA's National Pesticide Survey (NPS) reported that 4-nitrophenol was found in four samples, of which two were community water supply systems, and two private rural drinking-water wells. However, the study said that the analytical method used to detect 4-nitrophenol (GC/MS with electron capture) could not reliably quantify the concentration of the degradate in water.

It is important to note that 4-nitrophenol can be introduced into the environment by other pathways in addition to being a degradate of methyl parathion and ethyl parathion. This chemical is released in wastewater during the production of methyl parathion, ethyl parathion, and N-acetyl-p-aminophenol (pain-killer acetaminophen). 4-nitrophenol is also produced by photochemical reactions in the air connected with vehicular exhaust gas, and found on suspended particulate matter in the atmosphere.

Although 4-nitrophenol has been found in drinking water, the Health Effects Division has indicated that methyl paraoxon is the only degradate of methyl parathion included in the tolerance expression for methyl parathion. Degradate 4-nitrophenol is toxic to humans, but it has a different mode of action and toxic endpoint than methyl parathion and methyl paraoxon. The endpoint of concern for 4-nitrophenol is children under 3 months old, due to concerns about methemoglobin anemia. The EPA Office of Water has established one-day, ten-day and longer term Health Advisory levels (HA) for 4-nitrophenol of 800 ppb for a 10-kg child.

Therefore, some assessment of the potential of 4-nitrophenol to contaminate drinking water is warranted, in spite of the fact that it does not share a common mode of action with methyl parathion and methyl paraoxon. The uncertainty of such an assessment is significant, because EFED has not required that a full suite of environmental fate studies be performed for this chemical. Since 4-nitrophenol is produced in its own right as a fungicide used in the treatment of leather and cork insulation, EPA issued a RED for 4-nitrophenol in 1991. However, since 4-nitrophenol is only registered for indoor uses, the only environmental fate study that EFED requested be performed was the hydrolysis study. There is no indication that this study was ever submitted by registrant Monsanto.

The EFED chapter for 4-nitrophenol notes an aerobic soil metabolism half-life of 16 days, and a Koc value of 214. No details are given on the sources of these data, nor the conditions under which these values were derived. A better source of peer-reviewed data comes from the National Library of Medicine, which has prepared a review of open literature studies on the chemical properties of 4-nitrophenol. EFED performed a first-tier drinking water assessment for 4-nitrophenol using the data cited in that review:

Table 15. GENEEC Environmental Fate Input Parameters for 4-Nitrophenol		
DATA INPUT	**INPUT VALUE**	**SOURCE**
Effective Application Rate	0.52 lb ai/A (from methyl parathion 0.13 lbs ai/A (from ethyl parathion)	Label rates adjusted* for % of degradate and difference in molecular weight
Maximum Number of Applications	10 (m-parathion) 6 (e-parathion)	Cheminova
Application Interval	3 days (methyl-parathion) 7 days (ethyl-parathion	Cheminova
Batch Equilibrium (Koc)	55 ml/g	National Lib. Of Medicine
Aerobic Soil Metabolism	$t_{1/2}$ = 1.2 days**	National Lib. Of Medicine
Solubility	16000 ppm	National Lib. Of Medicine
Aerobic Aquatic Metabolism	stable	N/A
Hydrolysis	stable	N/A
Photolysis	$t_{1/2}$ = 6.7 days	National Lib. Of Medicine

* Maximum application rate of parent compounds multiplied by the maximum amount of 4-nitrophenol detected (as % of applied parent) in any laboratory study submitted by the registrant, multiplied by a molecular weight correction factor (i.e. MW of 4-nitrophenol/MW of parent)
** Half-life is from agricultural top soil experiment

Table 16. Surface Water Results for 4-Nitrophenol						
Use	**App. Rate of Parent (lbs/acre)**	**Adjusted app. rate for degradate (lbs/acre)**	**# Apps/year**	**App. Int. (days)**	**GENEEC Peak EEC (ppb)**	**GENEEC 56 Day EEC (ppb)**
Cotton	3.0 (MP)	0.52	10	3	42.42	40.66
Cotton	1.0 (EP)	0.13	6	7	8.02	7.69
Total	-------	-------	-------	-------	50.44	48.35

The values above include several conservative assumptions beyond those inherent in the GENEEC screening model itself:

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1) The application rates used for 4-nitrophenol can be derived from the maximum rates at which parents methyl parathion and ethyl parathion are applied. These maximum rates were multiplied by the highest percentage of 4-nitrophenol found in any of the laboratory studies cited above and then multiplied by the molecular weight correction factor (i.e. M.wt.of 4-nitrophenol/M.wt of parent) . The maximum 4-nitrophenol derived from methyl parathion was 33%, from the anaerobic aquatic metabolism study. The maximum amount derived from ethyl parathion was 27%, from the aerobic aquatic metabolism study. Using these percentages to calculate an effective application rate assumes that other degradative processes are not occurring to degradate 4-nitrophenol as it is produced by the aquatic metabolism processes above. This is a *very* conservative assumption which should be considered when evaluating the results of this first-tier screen.

2) Since aerobic aquatic metabolism data is not readily available for 4-nitrophenol, this degradate was assumed to be stable to that process;

3) Since hydrolysis data is not readily available for 4-nitrophenol, this degradate was assumed to be stable to that process;

4) The additive risk from 4-nitrophenol derived from methyl parathion and ethyl parathion assumes that the uses of the parent compounds chosen are occurring in the same area for the GENEEC simulation. This is also quite a conservative assumption.

5) No other potential sources of 4-nitrophenol in drinking water are considered in this assessment. EFED is not aware of the magnitude of discharge of 4-nitrophenol in wastewater, or potential deposition in rainwater. It is possible that these sources might result in a more significant contamination of drinking water by 4-nitrophenol than the degradation of methyl parathion and ethyl parathion. No attempt to quantify the risk posed by other sources of 4-nitrophenol is attempted here.

In spite of the conservative assumption detailed above, the estimated concentrations of 4-nitrophenol in drinking water do not approach the 800 ppb HA for a 10-kg child. These values do not exceed OW's lifetime HA for a 70-kg adult of 60 ppb, and HED has indicated that adults are not an endpoint of concern for this chemical, in any case.

Ground-Water Assessment for 4-Nitrophenol

Results of a SCI-GROW assessment for 4-nitrophenol are shown below. The assumptions made and chemical properties used to perform this assessment are the same as for the GENEEC run, with one exception. The aerobic soil metabolism half-life used in this assessment is 40 days, which was cited by the National Library of Medicine literature review as the half-life measured in subsoil samples. Using this half-life assumes that 4-nitrophenol quickly leaches to the subsoil, before degradation can occur in the top soil at the shorter half-life cited above.

Table 17. Ground-water results for 4-Nitrophenol				
Crop	**App. Rate of Parent (lbs/acre)**	**Adjusted app. Rate (lbs/acre)**	**# Apps./Year**	**SCI-GROW Acute EEC (ppb)**
Cotton	3.0 (MP)	0.52	10	3.70
Cotton	1.0 (EP)	0.13	6	0.55
Total	-------	------	------	4.25

The PGWDB reports that 4-nitrophenol was detected in 3 of 263 wells sampled in Mississippi from 1982 to 1990, at concentrations ranging from 0.004 to 0.02 ppb. No detections were reported in 81 wells sampled in Washington in 1988. EFED recommends that a concentration of 4.25 ppb be used for a first-tier assessment of drinking water derived from a ground-water source.

ECOLOGICAL HAZARD ASSESSMENT

The toxicity of a pesticide is determined through laboratory testing of representative surrogate species. For instance, two surrogate species each are used in toxicity testing to represent all freshwater fish (>2000 species) and birds (>680 species) in the United States. Acute mammalian studies are usually performed using the laboratory strain of the Norway rat or the house mouse as surrogate species. Estuarine/marine testing is limited to a crustacean, mollusk, and fish. Reptiles and amphibians are not tested. Avian toxicity studies are used as surrogates for reptilian toxicity assessments. Fish toxicity studies are used as surrogates for addressing the risk to amphibians, assuming that the tadpole stage has the same sensitivity as a fish.

The tabular data below present the results of selected studies for surrogate and most sensitive species of those tested for each endpoint. This in no way represents the extensive number of studies which have been reviewed or conducted with methyl parathion. A full tabular summary of ecotoxicological data is presented in Appendix 1. Open literature studies on the ecological effects of methyl parathion, as well as incident reports that show these effects, are included in the risk assessment.

a. Toxicity to Terrestrial Animals

I. Birds and Reptiles, Acute and Subacute

An acute oral toxicity study using the technical grade of the active ingredient (TGAI) is required to establish the toxicity of methyl parathion to birds and reptiles. The preferred test species is either mallard duck (a waterfowl) or bobwhite quail (an upland game bird). Results of this test requirement are tabulated below. Also shown are results for American Kestrel which was the most sensitive species tested.

Avian Acute Oral Toxicity

Species	% ai	LD50 (mg/kg)	Toxicity Category [1]	MRID No. Author/Year	Study Classification[2]
Mallard duck *Anas platyrhynchos*	80	6.6 (4.42-9.88)	"very highly toxic"	00160000 Hudson/ 1984	Core
Northern bobwhite quail *(Colinus virginianus)*	80	7.56(5.7-10)	"very highly toxic"	00160000 Hudson/ 1984	Core
American Kestrel (*Falco sparverius*)	98.2% Technical	3.08(2.29-4.14)	"very highly toxic"	44371701 Rattner/1983	Supplemental

[1] "Very highly toxic" designates chemicals whose LD_{50} is <10 mg/kg."Highly toxic" designates chemicals whose LD50 is between 10 and 50 mg/kg. "Moderately toxic" designates chemicals whose LD50 is between 51 and 500 mg/kg (Brooks (1973).

[2] Core (study satisfies guideline). Supplemental (study is scientifically sound, but does not satisfy guideline)

Because the lowest LD_{50} is less than 10 mg/kg, methyl parathion is "very highly toxic" to avian species on an acute oral basis. The guideline (71-1) is fulfilled (MRID 00160000).

Dermal studies were performed by dosing test birds with methyl parathion on their feet or under their wings. Available dermal studies are listed below.

Avian Acute Dermal Toxicity

Species	%a.i.	LD50 mg/kg	Toxicity Category	MRID No. Author/Year	Study Classification
Bobwhite Quail (*Colinus virginianus*)	45.42 EC	2.9 (2.3-3.7)	"very highly toxic"	71200/ Beavers/1980	Supplemental
Bobwhite Quail (*Colinus virginianus*)	22.0 Penncap-M	9.127	"very highly toxic"	83103/ Beavers/1980	Supplemental
Mallard duck (Anas platyrhynchos)	80.00	53.6 (39.3-72.9) Feet exposed	"Moderately toxic"	00160000 Hudson/1984	Supplemental

Two subacute dietary studies using the TGAI are required to establish the toxicity of methyl parathion to birds. The preferred test species are mallard duck and bobwhite quail. It appears that dermal toxicity values are nearly the same as the acute oral study values. Hence, we assign the same toxicity category of "very highly toxic." More species are likely to suffer adverse effects because of the dermal toxicity. Dermal poisoning does not require preference for contaminated food, but only that a bird walk through a contaminated area.

Results of these tests are tabulated below.

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Avian Subacute Dietary Toxicity

Species	% ai	5-Day LC50 (ppm)[1]	Toxicity Category[2]	MRID No. Author/Year	Study Classification
Northern bobwhite quail *(Colinus virginianus)*	Tech	28.2(22-35.3)	"very highly toxic"	102329 Pennwalt/ 1972	Supplemental
Mallard duck *(Anas platyrhynchos)*	80	336(269-413)	"highly toxic"	00022923 Hill/1975	Core
Ring-necked Pheasant *(Phasianus colchicus)*	80	91(77-107)	"highly toxic"	00022923 Hill/1975	Core

[1] "Very highly toxic" designates chemicals whose LD_{50} is <10 mg/kg."Highly toxic" designates chemicals whose LD50 is between 10 and 50 mg/kg. "Moderately toxic designates chemicals whose LD50 is between 51 and 500 mg/kg (Brooks (1973).

Methyl parathion is "very highly toxic" to avian species on a subacute dietary basis. The guideline (71-2) is fulfilled (MRID # 00022923).

ii. Birds and Reptiles, Chronic

Avian reproduction studies using the TGAI are required for methyl parathion because the following conditions are met: (1) birds may be subject to repeated or continuous exposure to the pesticide, especially preceding or during the breeding season, and (2) information derived from mammalian reproduction studies indicates reproduction in terrestrial vertebrates may be adversely affected by the anticipated use of the product. The preferred test species are mallard duck and bobwhite quail. Results of these tests are tabulated below.

Avian Reproduction

Species/ Study Duration	% ai	NOEC (ppm)	LOEC (ppm)	LOEC Endpoints	MRID No. Author/Year	Study Classification
Northern bobwhite quail *(Colinus virginianus)*	Tech	6.27	15.5	Number of eggs laid; eggs set/hen; adult female bodyweight	41179302 Beavers/1988	Core
Mallard duck *(Anas platyrhynchos)*	Tech	14.7	>14.7	No effects at highest conc.	41179301 Beavers/1988	Supplemental

The mallard duck study (44179301) is supplemental because it did not determine an effect level. Since the bobwhite quail study shows that the quail is more sensitive, a new mallard study is not required. Risk quotients (RQs) were determined using the lowest value. The guideline (71-4) is considered fulfilled (MRID 41179302).

iii. Mammals, Acute and Chronic

The mammalian toxicity values shown below were obtained from the Agency's Health Effects Division (HED):

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Mammalian Toxicity

Species/ Study Duration	% ai	Test Type	Toxicity Value	Affected Endpoints	MRID No.
laboratory rat 96 hours	80	Oral LD50	3.6 (1.63-7.92) mg/kg ♂ 23.0 (13.7-38.6) mg/kg ♀	Mortality	243414
Laboratory rat	NR	Dermal LD50	6 mg/kg (NR)	Mortality	(HED chapter)
Laboratory rat	NR	Inhalation LC50	<0.163 mg/L	Mortality	256961
Feeding-3 month rat	Technical	Feeding	NOEL=2.5 ppm (converts to 0.25 mg/kg) LEL=25 ppm (2.5 mg/kg) -	Clinical changes (lowered hemacrit; elevated SAP & urine specific gravity; depressed RBC, brain & plasma ChE.)	74299
Rat 2 generation	95.8	Repro-duction	Reproduction NOEL =5 ppm; Mat. NOEL=5 ppm	Significant decreased pup survival Reduced bodyweight during lactation	00119087

Methyl parathion is "very highly toxic" (NOEL <10 mg/kg) to small mammals on an acute oral basis (MRID No. 243414), and "highly toxic" to small mammals on an acute dietary basis (MRID No. 43961101). The feeding 3 month NOEL was very low at 2.5 ppm (MRID No. 74299) and the reproduction NOEL is 5 ppm (MRID No. 00119087).

iv. Insects

A honey bee acute contact study using the TGAI is required for methyl parathion because its use on flowering crops will result in honey bee exposure. Results of this test are tabulated below:

Nontarget Insect Toxicity

Species	% ai	Results	MRID No. Author/ Year	Study Classification
Honey bee *(Apis mellifera)*	—	LD50 0.111 μg/bee	44038201 Atkins/ 1981	Core
Honey bee *(Apis mellifera)*	Penncap-M	LD50 0.214 μg/bee	44038201 Atkin/ 1981	Core

Nontarget Insect Toxicity

Species	% ai	Results	MRID No. Author/ Year	Study Classification
Honey bee *(Apis mellifera)*	Penncap-M	"The average mortality of the adult honey bees was from 29 to 72 times higher than normal the first 48 hours after pollen containing Penncap -M, stored 13.5 and 14.5 months in the cells of wax combs, was introduced into nucleus colonies. After 1 week adult mortality was still 4 to 10 times higher than normal. After 4 weeks, mortality was nearly normal again. . .. Chemical analysis of the stored pollen showed 26 ppm methyl parathion."	160948 Rhodes/ 1980	Supplemental

Methyl parathion is very highly toxic to bees on acute contact basis and suggest strongly that mortality will occur under fields conditions. Additional evidence from the open literature is cited in the risk assessment. Field reports of bee kills are provided Appendix 2. Also, several studies have shown that methyl parathion is toxic to bees exposed to foliar residues (Atkins and Kellum, 1980, MRID 00074486, Waller, 1983 MRID 138663). Atkins and Kellum (1980) reported that residues of methyl parathion on alfalfa foliage were highly toxic to honeybees at application rates ranging from 0.03125 to 0.5 lb ai/acre. At the higher rates (0.25 and 0.5 lb ai/acre), the toxicity persisted from 4 to 6 days. The guideline requirements 141-1 and 141-2 are fulfilled by the cited studies.

b. Toxicity to Freshwater Aquatic Animals

I. Freshwater Fish and Amphibian Acute Toxicity

Two freshwater studies using the TGAI are required to establish the toxicity of methyl parathion to fish. The preferred test species are rainbow trout (a coldwater fish) and bluegill sunfish (a warm water fish). Results of tests on selected surrogate and other sensitive species are tabulated below.

Freshwater Fish and Amphibian Acute Toxicity

Species/	% ai	96-hour LC50 (ppm)	Toxicity Category	MRID No. Author/Year	Study Classification
Rainbow trout *(Oncorhynchus mykiss)*	43.2	2.2(1.5-2.7)	"moderately toxic"	40932101 Surprenant/1988	Core
Bluegill sunfish *(Lepomis macrochirus)*	77	1.0(0.6-1.6)	"highly toxic"	40098001 Mayer/1986	Core
Channel catfish *(Ictalurus punctatus)*	90	5.24(4.27-6.44)	"moderately toxic"	40094602 Johnson/1980	Core
Chorus frog *(Pseudacris triseriata)*	90	3.7(N.R.)	"moderately toxic"	40098001 Mayer/1986	Supplemental
Cutthroat trout (Oncorrhychus clark)	90	1.85 (1.39-2.47)	"moderately toxic"	40094602 Johnson/1980	Core

[1] Brooks (et al.,1973) toxicity classification indicates that LC50 values >1 to 10 ppm are "moderately toxic".

Because these LC_{50}s fall in the range of >1 to 10 ppm, methyl parathion is "moderately to highly toxic" to freshwater fish on an acute basis. The guideline (72-1) is fulfilled (MRID 40932101, 40098001, and 40094602). Methyl parathion is also moderately toxic to larval stages of developing frogs and possibly other amphibian species.

ii. Freshwater Fish, Chronic

A freshwater fish early life-stage test using the TGAI is required because residues may reach surface water. Also, the PRZM-EXAMS EEC for cotton is three-tenths of the early life-stage NOEC which exceeds the trigger that the EEC is equal to or greater than one-tenth of the NOEC for the early life-stage. The results for fathead minnow and rainbow trout are shown below. The guideline (72-4) is fulfilled (MRID No. 233438)

Freshwater Fish Early Life-Stage Toxicity Under Flow-through Conditions

Species/ Study Duration	% ai	NOEC/LOEC (ppm)	Endpoints Affected	MRID No. Author/Year	Study Classification
Fathead Minnow (*Pimephales promelas*)	80	0.31/0.38	Weight	233438 Jarvinen/1988	Core
Rainbow trout (*Oncorhynchus mykiss*)	Tech-nical 75.1	ND/<0.08	Length and weight	250628 Bailey/1983	Supplemental

Methyl parathion causes chronic effects in fish at concentrations less than 80 ppb.

iii. Freshwater Invertebrates, Acute

A freshwater aquatic invertebrate toxicity test using the TGAI is required to establish the toxicity of methyl parathion to aquatic invertebrates. The preferred test species is *Daphnia magna*. Results of selected tests with *Daphnia* and crayfish are tabulated below.

Freshwater Invertebrate Acute Toxicity

Species	% ai	48-hour LC50/ EC50 (ppb)	Toxicity Category	MRID No. Author/Year	Study Classification
Waterflea (*Daphnia magna*)	90	0.14(0.09-0.2)	"very highly toxic"	40094602 Johnson/1980	Core
Crayfish (*Orconectes nais*)	90	15(N.R.)	"very highly toxic"	40094602 Johnson/1980	Supplemental

[1] Brooks (et al., 1973) classification indicates the LC50 of 0.1 to 1 ppm are in the "highly toxic" range and those greater than 1 to 10 ppm are in the "moderately toxic" range.

Because the LC_{50}/EC_{50} is < 100 ppb, methyl parathion is in the "very highly toxic" range for aquatic invertebrates on an acute basis. The guideline (72-2) is fulfilled (MRID No. 40094602).

iv. Freshwater Invertebrate, Chronic

A freshwater aquatic invertebrate life-cycle test using the TGAI is required for methyl parathion because: 1) the rice use and multiple applications to turf (see EEC) are expected to result in contamination of natural water, (2) the aquatic acute EC_{50} is less than 1 mg/L, and (3) the EEC in water is equal to or greater than the 0.01 of the acute EC_{50}.

Freshwater Aquatic Invertebrate Life-Cycle Toxicity

Species/ Flow-through)	% ai	21-day NOEC/LOEC (ppb)	Endpoints Affected	MRID No. Author/Year	Study Classification
Waterflea (*Daphnia magna*)	96	0.178/0.562	Survival, growth, and offspring/parent Daphnia	41506801 Heimbach/1987	Supplemental
Waterflea (*Daphnia magna)*	80%	0.02/0.25	Neonates produced, survival, growth (length)	44371716 Fernandez-Casalderrey	Supplemental
Waterflea (*Daphnia magna)*	75.1 Technical	0.16/2.51	Young produced/ reproductive day and average No. of young produced	250628 Bailey/1983	Core

The guideline (72-4) is fulfilled (MRID No.250628).

Methyl parathion causes chronic effects in *Daphnia magna* at concentrations of <0.25 ppb.

c. Toxicity to Estuarine and Marine Animals

I. Estuarine and Marine Fish, Acute

Acute toxicity testing with estuarine/marine fish using the TGAI is required for methyl parathion because the active ingredient is expected to reach the estuarine/marine environment because of its use in coastal counties. The preferred test species is sheepshead minnow. Results of sheepshead minnow and other more sensitive species are tabulated below.

Estuarine/Marine Fish Acute Toxicity

Species	% ai	96-hour LC50 ppm	Toxicity Category	MRID No. Author/Year	Study Classification
Spot (*Leiostmous xanthurus*)	99	0.059 (0.045-0.074)	" very highly toxic"	40228401 Mayer/1986	Supplemental

Estuarine/Marine Fish Acute Toxicity

Species	% ai	96-hour LC50 ppm	Toxicity Category	MRID No. Author/Year	Study Classification
Striped bass (*Morone saxatilis*	80	0.79 (0.17-1.4)	"highly toxic"	05000819 Korn/1974	Core
Sheepshead minnow (*Cyprinodon variegatus*)	43.2	3.4 (2.8-4.1) a.i., not product	"moderately toxic"	40932103 Surprenant/1988	Core

[1] Brooks (et al.,1973) classification indicates that LC50s greater than 1 to 10 ppm are "moderately toxic".

Methyl parathion is "moderately to very highly toxic" to estuarine/marine fish on an acute basis. The guideline (72-3a) is fulfilled (MRID 40932103 and 05000819).

ii. Estuarine and Marine Fish, Chronic

Because the acute LC_{50} is less than 1 ppm, and the pesticide is expected to be transported to water, an *estuarine/marine fish early life-stage toxicity test using the TGAI is required.* Since freshwater fish are significantly more tolerant to methyl parathion exposure, the freshwater fish study cannot be used as a surrogate study to fulfill this guideline requirement.

iii. Estuarine and Marine Invertebrates, Acute

Acute toxicity testing with estuarine/marine invertebrates using the TGAI is required for methyl parathion because the active ingredient is expected to reach the estuarine/marine environment because of its use in coastal counties. The preferred test species are mysid and eastern oyster. Results of selected tests are tabulated below.

Estuarine/Marine Invertebrate Acute Toxicity

Species/Static or Flow-through	% ai.	96-hour LC50/EC50 (ppb) (measured)	Toxicity Category[1]	MRID No. Author/Year	Study Classification
Eastern oyster (*Crassostrea virginica)*	99	12000 (10000-16000)	"slightly toxic"	40228401 Mayer/1986	Core
Mysid (*Americamysis bahia)*	43.2	0.35 (0.31-0.39) a.i., not product	"very highly toxic"	40932104 Surprenant/1988	Core*
Mysid (Americamysis bahia)	99	0.78 (0.58- 1.1)	"very highly toxic"	40228401 Mayer/1986	Core

[1] Based on Brook's (et al. 1973) toxicity categories indicate that chemicals with an LC50 < 0.1 ppm are "very highly toxic" and those between 10 and 100 ppm are "slightly toxic". *Indicates core only for the formulated product.

Because the methyl parathion LC_{50}/EC_{50}s fall in the range of >0.1-1 ppm, methyl parathion is "highly toxic" to estuarine/marine invertebrates on an acute basis. The guideline (72-3b and 72-

3c) is fulfilled (MRID 40228401, 40932104).

iv. Estuarine and Marine Invertebrate, Chronic

An estuarine/marine invertebrate life-cycle toxicity test using the TGAI is required for methyl parathion. Methyl parathion meets the following criteria for requiring this test: (1)The end-use product may be expected to be transported to the estuarine/marine environment from the intended use sites. Methyl parathion has been found in estuarine environments as a result of its use on rice; (2) the aquatic acute EC_{50} is less than 1 mg/L; (3) the EEC in water is equal to or greater than the 0.01 of the acute EC_{50}, and (4) methyl parathion may persist with a half-life greater than 4 days. The preferred test species is mysid. Results of this test are tabulated below:

Estuarine/Marine Invertebrate Life-Cycle Toxicity

Species/(Static Renewal or Flow-through)	% ai	21-day NOEC/LOEC (ppb)	MATC[1] (ppm)	Endpoints Affected	MRID No. Author/Year	Study Classification
Mysid *(Americamysis bahia)*		0.11/0.37	0.20	Survival and Number of offspring/♀	66341 Lowe/1981	Core

[1] defined as the geometric mean of the NOEC and LOEC.

The guideline (72-4) is fulfilled (MRID No. 66341).

d. Toxicity to Plants

I. Terrestrial

Terrestrial plant testing (122-1 a and b) is required for pesticides other than herbicides if data from the literature indicate that a pesticide is phytotoxic. Environmental Health Criteria 145 from the World Health Organization (WHO) 1993 reports that phytotoxic effects of methyl parathion have been observed in cotton and lettuce and that methyl parathion has been shown to cause a reduction of growth in sorghum.. Given its widespread use on a variety of important crops, terrestrial plant data for methyl parathion are required.

ii. Aquatic Plants

Aquatic plant testing is required for insecticides applied to aquatic food, aquatic nonfood, and forestry sites. In these cases aquatic plant testing is required (122-2) on *Kirschneria subcapitatum*, *Lemna*, *Skeletonema costatum*, *Anabaena flos-aquae*, and a freshwater diatom. The following test was found in Mayer, 1986 (MRID 48228401). It indicates that methyl parathion is "moderately toxic" to marine diatoms.

Nontarget Aquatic Plant Toxicity (Tier II)

Species	% ai	EC50/ (ppm)	MRID No. Author/Year	Study Classification
Nonvascular Plants				
Marine diatom (*Skeletonema costatum*)	99	5.3 (4.3-5.7)	Lowe 66341/1981	Supplemental

Aquatic species testing (122-2, aquatic plant growth) using a marine diatom (Skeletonema costatum) and a freshwater diatom (Navicula pelliculosa) is not fulfilled. However, this requirement will be reserved pending the results of terrestrial plant toxicity testing required above.

ECOLOGICAL RISK ASSESSMENT

Unsupported Uses

Although the uses shown below appear on current methyl parathion labels, the registrant has informed SRRD that these uses will no longer be supported by tolerances. These uses, which are not included in this risk assessment, will be removed from the label. If any potential registrant requests that use on these crops be resumed, a new risk assessment will be needed.

Treefruit/Nut/Vine Crops:
Apples, apricots, avocados, cherries, dates, figs, grapes, guavas, mangoes, nectarines, olives, peaches, pears, pineapple, plums, quinces.

Small Fruits/Berries:
Blackberries, blueberries, boysenberries, cranberries, currants, dewberries, gooseberries, loganberries, raspberries, strawberries, youngberries.

Field Crops:
Artichokes, birdsfoot trefoil, broccoli, brussel sprouts, carrots, cauliflower, celery, clover, collards, cucumbers, eggplant, endive, garden beets, garlic, kale, kohlrabi, lettuce, melons, mustard greens, okra, parsnips, peppers, pumpkins, radishes, rutabagas, safflower seed, sorghum, spinach, squash, succulent beans, succulent peas, summer squash, swiss chard, tomatoes, turnips, vetch.

Non-Food/Feed Uses:
Chrysanthemums, daisies, field grown ornamentals, flowering plants, grasses grown for seed, guayule, jojoba, marigolds, any mosquito larvicide use, nursery stock, non-agricultural land, roadside areas, wasteland.

The registration of additional uses beyond those currently supported by the registrants would require a new risk assessment.

3/8/65

Risk Quotients and Levels of Concern

EFED uses an indexing method of risk assessment which considers exposure and toxicity components. Risk quotients (RQs) are calculated by dividing exposure estimates by toxicity values, both acute and chronic.

RQ = EXPOSURE/TOXICITY

The resultant quotient is then compared to predetermined levels of concern (LOCs). This quotient is used as a screen to show relative risk.

The LOC criteria are defined as follows:

(1) acute high - potential for acute risk is high; regulatory action may be warranted in addition to restricted use classification;

(2) acute restricted use - the potential for acute risk is high, but this may be mitigated through restricted use classification;

(3) acute endangered species - the potential for acute risk to endangered species is high regulatory action may be warranted; and

(4) chronic risk - the potential for chronic risk is high regulatory action may be warranted.

Currently, EFED does not perform similar assessments for chronic risk to plants, acute or chronic risks to nontarget insects, or chronic risk from granular/bait formulations to mammalian or avian species.

Risk presumptions, along with the corresponding RQs and LOCs, are tabulated below.

Risk Presumptions for Terrestrial Animals

Risk Presumption	RQ	LOC
Birds and Mammals		
Acute High Risk	EEC[1]/LC50 or LD50/sq ft or LD50/day[3]	0.5
Acute Restricted Use	EEC/LC50 or LD50/sq ft or LD50/day (or LD50 < 50 mg/kg)	0.2
Acute Endangered Species	EEC/LC50 or LD50/sq ft or LD50/day	0.1
Chronic Risk	EEC/NOEC	1

[1] abbreviation for Estimated Environmental Concentration (ppm) on avian/mammalian food items

[2] $\frac{mg/ft^2}{LD50 * wt.\ of\ bird}$ [3] $\frac{mg\ of\ toxicant\ consumed/day}{LD50 * wt.\ of\ bird}$

Risk Presumptions for Aquatic Animals

Risk Presumption	RQ	LOC
Acute High Risk	EEC[1]/LC50 or EC50	0.5
Acute Restricted Use	EEC/LC50 or EC50	0.1
Acute Endangered Species	EEC/LC50 or EC50	0.05
Chronic Risk	EEC/MATC or NOEC	1

[1] EEC = (ppm or ppb) in water

Risk Assessment for Nontarget Terrestrial Animals

For pesticides applied as liquids, the estimated environmental concentrations (EECs) on food items following product application are compared to LC50 values to assess risk. The predicted 0-day maximum residues of a pesticide that may be expected to occur on selected avian or mammalian food items immediately following a direct single application at 1 lb ai/A are tabulated below.

Estimated Environmental Concentrations (EECs) on Avian and Mammalian Food Items (ppm) Following a Single Application at 1 lb ai/A)

Food Items	EEC (ppm)[1]
Short grass	240
Tall grass	110
Broadleaf/forage plants, and small insects	135
Fruits, pods, seeds, and large insects	15

[1] Maximum EEC are for a 1 lb ai/A application rate and are based on Fletcher *et al.* (1994).

EECs resulting from multiple applications are calculated from the maximum number of applications, minimum application interval, and foliar half-life data. Willis and McDowell (1987) reported a number of methyl parathion foliar half-lives ranging from 0.1 to 13.5 days, with most values being <2 days.. This assessment uses a foliar half-life of 2.4 days which is the upper 90th percentile confidence limit of the mean value.

It is important to note that foliar dissipation considers only the degradation of the parent compound and does not account for the formation of toxic degradates. Methyl paraoxon, which is highly toxic, may form on plant foliage after the parent degrades. This analysis may underestimate avian risk because it does not consider potential avian exposure methyl paraoxon.

These EEC estimates consider the effect and timing of multiple applications by assuming first-order decay of parent using a foliar half-life of 2.4 days.

Avian Risk Assessment

The major uses of methyl parathion are likely to pose significant risk to birds. EFED has summarized potential risk from use on 10 major crops in the table below. In addition to mortality, a number of sublethal effects has been documented in avian species. These include adverse reproduction effects, negative impacts on nesting birds and their young, damage to food resources, reduced feeding and detrimental behavioral changes, and greater vulnerability to predation and environmental stress. For some crops, RQs exceed LOCs by more than two orders of magnitude.

The acute and chronic RQs for broadcast applications of liquid products tabulated below are based on a bobwhite quail (LC50 = 28.2 ppm; reproduction NOEC = 6.27 ppm).

Avian Acute and Reproduction Risk Quotients for Single and Multiple Applications for Major Use Crops

				Single Application		Multiple Application	
Site[1] (# Apps, App. Interval in days)	App.Rate (lbs ai/A)	Food Items	Maximum EEC (ppm)	Acute RQ (EEC/ LC50)	Reproduction RQ (EEC/ NOEC)	Acute RQ (EEC/ LC50)	Reproduction RQ (EEC/ NOEC)
Rice, Grasses (6,3)	0.79	Short grass	190	6.74	30.30	40.44	181.80
		Tall grass	87	3.09	13.88	18.54	83.28
		Broadleaf plants/Insects	107	3.79	17.07	22.74	102.42
		Seeds	12	0.43	1.91	2.58	11.46
Sunflower (3,5)	1	Short grass	240	8.51	38.28	25.53	114.84
		Tall grass	110	3.90	17.54	11.70	52.62
		Broadleaf plants/Insects	135	4.79	21.53	14.37	64.59
		Seeds	15	0.53	2.39	1.59	7.17
Soybean, (6,3) Corn (all) (6,2)	1	Short grass	240	8.51	38.28	51.06	229.68
		Tall grass	110	3.90	17.54	23.40	105.24
		Broadleaf plants/Insects	135	4.79	21.53	28.74	129.18
		Seeds	15	0.53	2.39	3.18	14.34
Alfalfa (4,42)	1	Short grass	240	8.51	38.28	34.04	153.12
		Tall grass	110	3.90	17.54	15.60	70.16

Avian Acute and Reproduction Risk Quotients for Single and Multiple Applications for Major Use Crops

				Single Application		Multiple Application	
Site[1] (# Apps, App. Interval in days)	App.Rate (lbs ai/A)	Food Items	Maximum EEC (ppm)	Acute RQ (EEC/ LC50)	Reproduction RQ (EEC/ NOEC)	Acute RQ (EEC/ LC50)	Reproduction RQ (EEC/ NOEC)
		Broadleaf plants/Insects	135	4.79	21.53	19.16	86.12
		Seeds	15	0.53	2.39	2.12	9.56
Cotton (10,3)	3	Short grass	720	25.53	114.83	255.30	1,148.30
		Tall grass	330	11.70	52.63	117.00	526.30
		Broadleaf plants/Insects	405	14.36	64.59	143.60	645.90
		Seeds	45	1.60	7.18	16.00	71.80

The single and multiple application scenarios estimate that all methyl parathion applications will result in endangered species, restricted use, and avian acute high risk LOC exceedences. The avian reproduction LOC is exceeded at all application rates.

Dermal exposure to methyl parathion is hazardous to birds. In two studies, bobwhite quail were exposed to methyl parathion under their wings. The resulting LD50 values of 2.9 and 9.127 mg/kg indicate that methyl parathion is "very highly toxic" by dermal exposure. Another study, in which mallard ducks' feet were exposed to methyl parathion for 24 hours, resulted in an LD50 of 53.6 mg/kg. This would place methyl parathion in the "moderately toxic" category.

Driver, et al., 1991 (MRID 44357804) also investigated the importance of other routes of exposure. In wind-tunnel experiments, "routes of uptake in order of contribution to toxicologic response from 8 to 48 h post-spray were dermal> preening ≥ oral>inhalation." Since poisoning can occur by multiple routes of exposure, RQ index values may underestimate the risk, since they consider only dietary exposure.

Acute Effects

Acute oral LD50s are available for mallard duck, northern bobwhite quail, ring-necked pheasant, kestrel, and grackle. All but the grackle are in the highest category - "very highly toxic" - with grackle in the second highest toxicity category - "highly toxic."

Pen studies using northern bobwhite quail and incident reports document methyl parathion's acute toxicity to birds (see table below). Shellenberger (1970) reported 40% mortality (8 birds) of caged, 12-week-old northern bobwhite quail exposed to eight weekly sprays of 1 lb ai/A methyl parathion EC. Another study reported mortality rates of 8 to 67% and increases in stress in bobwhite quail exposed to microencapsulated (Penncap-M) and EC formulations of methyl parathion (Pennwalt 1980; MRID 00061213). Edwards (1968; MRID 00090488) observed mortality rates of 5 and 20% for caged quail and pheasants, respectively, in an alfalfa hayfield treated with 0.5 lb/acre methyl parathion. Another study of 42 penned pheasants reported 11 deaths and sickness in half of birds treated with three applications of methyl parathion at 3 lb ai/A (Smith, 1987). Another study with caged bobwhites showed potentially lethal levels of acetylcholinesterase (AChE) inhibition (55.3% and 59.9%), respectively for both Penncap-M and Technical methyl parathion when sprayed at 1 lb ai/A (Knittle, 1973; MRID 093632). ACHE inhibition of ≥50% may cause death (Ludke et al. 1975). The relevance of pen studies is supported by White, et al. (1990; MRID 44357806) who reported that free bobwhites spent 60% of the time they were observed in or within 100 m of a Georgia sorghum and cotton fields treated with methyl parathion.

Tipton et al (1980; MRID 44378603), working with computer simulations to estimate mortality using laboratory and field data from Smithson and Sanders (1978; MRID 44378606), predicted bird mortality of up to 99% mortality after 6 weekly methyl parathion applications.

Adverse Sublethal Effects

Lethargy

Lethargy, a potentially hazardous behavioral effect of acute methyl parathion intoxication, is likely to increase a bird's susceptibility to predation. Hyperglycemia may explain the lethargy commonly associated with AChE inhibitors (Mineau, 1991). Mineau (1991) reports of a study where, "... northern bobwhite quail were given one of three oral doses of methyl parathion. Average brain AChE inhibition in quail from each treatment group and a control (corn oil only) were subjected to predation by a domestic cat following 30 minutes of acclimation to the test arena. Quail that were captured had greater brain ChE inhibition (mean =33%) and spent more time being still than quail that avoided capture (mean AChE inhibition=17%)."

Reproduction Effects

Studies show that successful bird reproduction is very sensitive to methyl parathion exposure. Exposure periods of 8 and 21 days can cause the same reproductive effects as longer exposure periods (Bennett and Bennett, 1990; MRID 44371701; Bennett et al., 1990; MRIDs 44371601 and 44371602). Methyl parathion avian reproduction results provided levels almost identical to the acute values. The acute dietary LC50 is 28 ppm. The surrogate study with bobwhite quail showed effects (number of eggs laid and survival of offspring) at 15.5 ppm (LOEC). The reproductive LOC is exceeded by the risk quotient (EEC/NOEC) for all crops.

Bennett, et al. 1990 (MRID 44371608) showed that nesting success in mallards may be impacted by short dietary exposures to methyl parathion, particularly during early incubation. The number of hatchlings at several stages in the nesting cycle for dosed birds (400 ppm) was only 43 to 61% of the number in the control group. This report noted that "except for the numbers of adult mortalities, all dose-related effects observed in the long-term exposure test also were observed in the short-term test."

Effects on Young Birds

Young birds display additional stress behavior and reduced survival when raised in or near methyl parathion treated fields. Brewer et al. (1988; MRID 44271604) found that fewer ducklings (16%) survived in a treated field than in the control (58%). Because of the additional stress of surviving in the wild, young birds died when exposed to lower concentrations than in the laboratory (Christensen. 1971; MRID 44342001). Skin penetration, probably due to the lack of feathers on young birds, is a major route of exposure. (Driver et al. 1991; MRID 44357804).

Young birds, like adult birds, may demonstrate behavioral effects from a sublethal dose. Fairbrother et al. (1988; MRID 44371601) reported that dosed duckling "preened and loafed" on the land while their siblings fed and swam. Mineau (1991) reports that two-week old northern bobwhite quail did not discriminate between untreated food and diets containing 45 or 90 ppm methyl parathion, and initially (0-24 hour post-dose) chose treated over untreated food. This indicates that there will be little avoidance of treated food sources.

Effects of Reduced Food Supply

Methyl parathion is "very highly toxic" to aquatic and terrestrial invertebrates, with RQs of up to 1500 (see aquatic risk assessment). It may therefore have effects on birds by killing invertebrates and reducing food supply (USDI, 1951; Martin et al. 1951). Several authors made the following points concerning the effects of reduced food supply on ducklings in the prairie-pothole region of the U.S.:

1. Grue et al. (1988; MRID 44357080) noted that ducklings of dabbling ducks are dependent on emerging insects during their first few days of life.

2. Krapu (1979), Swanson et al. (1979), and Swanson et al. (1985) reported that during egg-laying, female waterfowl are also dependent of aquatic invertebrates as source of protein and calcium.

3. Nest losses (e.g., due to predation) force many females to re-nest one or more times during the breeding season, thereby increasing the amount or time that females require high-protein invertebrate diets to meet the nutrient demands (1988; MRID 44357080)

4. Reduced food availability may lengthen the pre-fledgling period, increasing the period of maximum vulnerability of ducklings to predation (Brown and Hunter 1984, 1985)

5. Overland movement of females and their broods in search of adequate food may increase losses to predation (Ball et al. 1975, Talent et al. 1983)

Effects on Maternal Behavior

Various studies report adverse changes in maternal behavior due to methyl parathion exposure. Such behavioral changes are expected to increase juvenile mortality through increased exposure to predation. Brewer et al. (1988; MRID 44371604) reported brood abandonment and mortality among wood duck and teal hens in a field treated with 1.25 lb ai/acre methyl parathion, but not in a control field. Two-thirds of the nesting hens from the treated field had significantly depressed brain cholinesterase levels. Mortality among ducklings in the treated field (84%) was greater than that in the control field (42%) by 22 days post-spray.

Buerger et al. (1991; MRID 44371606) reported that the higher mortality due to increased predation of northern bobwhites in treated fields than in untreated fields may be due to negative effects on covey integrity caused by methyl parathion exposure.

Kendall, et al. (1984; MRID 44413601) reported a 39% increase in mortality among nesting starlings in a treated field. Since this effect did not correlate with ChE depression, the authors surmised that changes in maternal behavior or depressed food abundance might have been to blame. This same study reported nest abandonment by mallards and teals adjacent to a field treated at 0.6 lbs ai/A. Therefore, intoxication of mother birds may result in increased juvenile mortality due to insufficient care and increased predation.

Anorexia

In addition to environmental stresses, the loss of appetite in the wild can be life threatening. Food is not always readily available and animals need a minimum number of calories to survive. Two studies show these effects. Grue (1982; MRID 44371606) studied the behavioral and physiological responses of common grackles to ingestion of methyl parathion and three other organophosphates. The study showed that mortality was largely due to pesticide-induced anorexia that lasted as long as 12 hours after exposure. Grackles that died lost an average of 28 to 36% of their body weight. Edwards (1968; MRID 00090488) noted that birds sprayed with 0.5 lb ai/A of methyl parathion suffered a 20% weight loss shortly after the spraying, but recovery was rapid. Based on the availability of food, amount of stored calories, and energy needs, a bird may not survive anorexia. Also, a higher dose may be lengthen the effect or exposure and add additional poisonous effects. Therefore, birds exposed to methyl parathion experiencing the stresses of living in the wild may not consume sufficient calories to survive.

Increased Toxicity from Environmental Stress

Environmental stress increases the susceptibility of birds to methyl parathion. Rattner and Franson (1983; MRID 44371701) reported that cold was found to enhance methyl parathion

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toxicity in kestrels, as a dose considered sublethal at thermoneutral temperature resulted in 60% mortality at -5°C." Also, Fairbrother et al. (1988; MRID 44342007) observed that 40% of 5-day-old mallards given a sublethal oral dose (based on laboratory studies) of methyl parathion died within the first hour after the broods were placed on outdoor ponds in cold weather. Methyl parathion is unlikely to be applied when the temperature outdoors is below freezing. However, these studies suggest that environmental stresses may reduce the amount of methyl parathion needed to cause intoxication or mortality below concentrations indicated by laboratory studies.

Mammalian Risk Assessment

Methyl parathion is "very highly toxic" to mammals on an acute basis (LD50 = 3.6 mg/kg for laboratory rat). The acute herbivores/insectivores RQs for the lowest application rate (0.1 lb ai/A) range between 1 and 6.33. All mammalian acute LOCs are exceeded.

In the animal, hydrolysis of the sulfur/phosphate bond creates methyl paraoxon which is more toxic than methyl parathion. HED's mammalian studies therefore account for methyl paraoxon. Feeding and reproduction studies also show effects at low dietary concentrations (2.5-5 ppm). Hence, the RQs are high and exceed the chronic LOC of 1. RQs for short grass, which has the highest expected concentration of methyl parathion for any of the food items listed, ranged from 6.4 to 320.

RQs for reproduction were as high as 641 for multiple applications. The feeding study showed stomach lesions, reduced brain cholinesterase, and reduced hematocrit for a laboratory rat. The reproduction study showed decreased pup survival for mice. These effects are expected to cause reduced reproduction and increased mortality due to the inability to efficiently gather or catch food and avoid predators. Also, predators may be indirectly affected by reduced food supply because of lower numbers of small herbivores and insectivores.

Mammals are also very sensitive to dermal exposure (rat dermal LC50 = 6 mg/L; HED tox category I) and to inhalation of methyl parathion (LC50 = 0.163 mg/L; tox category I). Unlike birds, mammals are less able to readily escape treated fields, and hence are very sensitive to the multiple routes of exposure.

Estimating the potential for adverse effects to wild mammals is based upon EFED's draft 1995 SOP of mammalian risk assessments and methods used by Fletcher *et al.* (1994). The concentration of methyl parathion in the diet that is expected to be acutely lethal to 50% of the test population (LC_{50}) is determined by dividing the LD_{50} value (usually rat LD_{50}) by the percent body weight consumed. There is, however, uncertainty associated with this estimation. A risk quotient is then determined by dividing the EEC by the derived LC_{50} value. RQs are calculated for three separate weight classes of mammals (15, 35, and 1000 g), each presumed to consume four different kinds of food (grass, forage, insects, and seeds). The following RQ tables for liquid applications are based on a rat LD50 of 3.6 mg/kg.

Mammalian (Herbivore/Insectivore) Acute Risk Quotients for Single Broadcast of Liquid Products

Site/ Rate in lbs ai/A	% Body Weight Consumed	EEC (ppm) Short Grass	EEC (ppm) Forage & Small Insects	EEC (ppm) Large Insects	Acute RQ[1] Short Grass	Acute RQ Forage & Small Insects	Acute RQ Large Insects
Rice	95	190	107	12.00	50.14	28.24	3.17
0.79	66				34.83	19.62	2.20
	15				7.92	4.46	0.50
Corn Field Sweet	95	240	135	15.00	50.67	35.63	3.96
	66				35.20	24.75	2.75
Soybean Sunflower 1.0	15				8.00	5.63	0.63
Alfalfa Barley	95	300	169	18.75	79.17	44.60	4.95
Oats Rye	66				55.00	30.98	3.44
Wheat 1.25	15				12.50	7.04	0.78
Cotton 3	95	720	405	45.00	190.00	106.88	11.88
	66				132.00	74.25	8.25
	15				30.00	16.88	1.88

[1] RQ = $\frac{\text{EEC (ppm)}}{\text{LD50 (mg/kg)/\% Body Weight Consumed}}$

Mammalian (Granivore) Acute Risk Quotients for Single Application

Site/ /Rate in lbs ai/A	% Body Wt Consumed	EEC (ppm) Seeds	Acute RQ[1] Seeds
Grasses Rice	21	11.85	0.69
0.79	15		0.06
	3		0.01
Corn - field, sweet	21	15.00	0.88
Soybean Sunflower	15		0.63
1.0	3		0.13
Alfalfa Barley	21	18.75	1.09
Oats Rye	15		0.78
Wheat 1.25	3		0.16

Mammalian (Granivore) Acute Risk Quotients for Single Application

Site/ /Rate in lbs ai/A	% Body Wt Consumed	EEC (ppm) Seeds	Acute RQ[1] Seeds
Almond 2.0	21	30.00	1.75
	15		1.25
	3		0.25
Soybean 2.5	21	37.50	2.19
	15		1.56
	3		0.31
Cotton 3	21	45.00	2.63
	15		1.88
	3		0.38

[1] The three percent bodyweight consumed values (21, 15, and 3) represent three sized animals 15, 35, and 1000 gram animals.

[2] $RQ = \frac{\text{EEC (ppm)}}{\text{LD50 (mg/kg)/\% Body Weight Consumed}}$

The following table shows mammalian RQs for multiple applications of methyl parathion. Since all herbivore and insectivore LOCs are exceeded for single applications, they are not included in this table. Multiple application RQs for granivores are shown only for those uses for which RQs do not exceed LOCs after a single application.

Mammalian (Granivores) Acute Risk Quotients for Multiple Applications of Liquid Products (Broadcast) that Do Not Exceed LOCs from a Single Application

#Apps() Site (Interval)	Rate in lbs ai/A	Body Weight (g)	% Body Weight Consumed	Rat LD50 (mg/kg)	EEC (ppm) Seeds	Acute RQ Seeds
Rape or Canola (4)(3)	0.5	15	21	3.6	15	0.9
		35	15			0.6
		1000	3			0.1
Lentils (6)(3)	0.5	15	21	3.6	19	1.1
		35	15			0.8
		1000	3			0.2

The lowest application rate, 0.5 lb ai/A, exceeds all three LOCs.

[1] $RQ = \frac{\text{EEC (ppm)}}{\text{LD50 (mg/kg)/\% Body Weight Consumed}}$

The chronic RQs below for broadcast applications of liquid products are based on a mouse NOEC of 2.5 ppm in a feeding study and a rat NOEC of 5 ppm in a reproduction study.

Mammalian Chronic Risk Quotients for Single Applications

Site (# of Apps) (Interval App)	App. Rate Lbs a.i./A	Food Items	Maximum EEC[1] (ppm)	Chronic Feeding RQ (EEC/ NOEC)	Repro-ductive RQ (EEC/ NOEC)
Rape or Canola (8)(14) Lentils Onion, green Onion, bulb	0.5	Short grass	120	48.0	24.0
		Tall grass	55	22.0	11.0
		Broadleaf plants/ Insects	68	27.2	13.6
		Seeds	8	3.2	1.6

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Mammalian Chronic Risk Quotients for Single Applications

Site (# of Apps) (Interval App)	App. Rate Lbs a.i./A	Food Items	Maximum EEC[1] (ppm)	Chronic Feeding RQ (EEC/ NOEC)	Repro- ductive RQ (EEC/ NOEC)
Rice, Grasses (6,3)	0.79	Short grass	190	76.0	38.0
		Tall grass	87	34.8	17.4
		Broadleaf plants/ Insects	107	42.8	21.4
		Seeds	12	4.8	2.4
Sunflower (3,5) Soybean (6,3) Corn (6,2) Alfalfa (4,42)	1	Short grass	240	96.0	48.0
		Tall grass	110	44.0	22.0
		Broadleaf plants/ Insects	135	54.0	27.0
		Seeds	15	6.0	3.0
Barley Oat Rye Wheat (6,3)	1.25	Short grass	300	120.0	60.0
		Tall grass	138	55.2	27.6
		Broadleaf plants/ Insects	169	67.6	33.8
		Seeds	19	7.6	3.8
Cotton (10, 3)	3	Short grass	720	288.0	144.0
		Tall grass	330	132.0	66.0

Mammalian Chronic Risk Quotients for Single Applications

Site (# of Apps) (Interval App)	App. Rate Lbs a.i./A	Food Items	Maximum EEC[1] (ppm)	Chronic Feeding RQ (EEC/ NOEC)	Repro-ductive RQ (EEC/ NOEC)
		Broadleaf plants/ Insects	405	162.0	81.0
		Seeds	45	18.0	9.0

All three LOCs have been exceeded by all single application rate scenarios. Since estimated EECs for multiple application are higher than single application scenarios, all multiple treatments would also exceed the LOCs. Therefore, calculation of RQs for chronic effects from multiple applications are not necessary.

Risk to Pollinating Insects

Methyl parathion is very highly toxic to bees and other similar insects. The effect of methyl parathion exposure on bees has been of concern for many years to EPA, State regulators, and beekeepers, among others. Methyl parathion has caused very serious damage to colonies across the country, and continues to do so in spite of concerted efforts to mitigate the problem. The bee contact LD_{50} study indicates that the methyl parathion is "very highly toxic" to bees at rates ranging from 0.03 to 0.5 lb ai/acre. It may not be possible to eliminate the risk of methyl parathion use to bees. However, the removal of tree fruit uses of methyl parathion should significantly reduce the overall risk to bees.

Pollinators (bees, wasps, bumble bees. etc) fill an important ecological niche. They help transfer pollen between plants to ensure fruit and vegetable growth and seed viability. Pollinators can be very specialized. For example, the alkali bee is especially apt at opening the alfalfa flower and extracting pollen. Therefore, loss of specific pollinators can change ecological relationships which can reduce yield of a given crop, or in the case of wild plants reduce viability. Reduced viability would reduce the success of a given plant and make unintended changes in flora. Changes in the flora may also affect the animal population which relies on the plants for cover, feeding, etc.

EPA documented its concern for methyl parathion effects on bees in a 1979 HED position paper. This paper, and subsequent studies in the open literature, document the following risk to bees from Penncap-M, the microencapsulated formulation of methyl parathion:

1. Bees forage microcapsules and transport contaminated pollen back to the hive, leading to decreased viability or complete mortality of the colony. (Burgett and Fischer, 1977; Johansen and Kious, 1978; Russell, et al., 1998)

2. The tendency of the microcapsules to adhere to bees is much greater than with standard powder formulations (Johansen and Kious, 1978, Barker et al., 1979).

3. Because of its special formulations, Penncap-M residues on crops may remain toxic for days, rather than hours (Johnansen and Kious, 1978).This increases the length of time the microcapsules remain toxic to foraging bees.

4. Foragers returning to the hive bearing Penncap-M contaminated pollen loads can enter the hive unchallenged by the guard bees (Stoner et al., 1978).

5. The encapsulated methyl parathion formulation may remain toxic in stored pollen from one season to the next (Johansen and Kious, 1978), or as long as 19 months (Barker et al., 1979).

6. Although Penncap-M causes a lower initial knockdown than other insecticides, it causes a delayed-action break in honeybee brood cycles about two weeks after an application is made (Johansen & Kious, 1978) The lower initial knockdown may result in a greater mass of methyl parathion being transported to the hive by a greater number of bees (Mason, 1986) .

Both formulations of methyl parathion have killed bees. Anderson and Glowa (1984) and Anderson and Wojtas (1986) reported that non-encapsulated methyl parathion can be returned and incorporated into a beehive. However, the microencapsulated formulation extends the persistence of methyl parathion toxicity to bees in the field. The State of Washington (Mayer, et al.,1996), for instance, informs beekeepers to avoid fields treated with Penncap-M for 5 to 8 days; beekeepers are told to avoid fields treated with the EC formulation for at least a day.

Honey Bee Mortality Incidents

The risk to honeybees reported in the studies above is well illustrated by two decades of bee kills. When Penncap-M was first marketed in the 1970's large bee kills were reported and EPA required more restrictive labeling. In 1989, when Elf Atochem began marketing Penncap-M in new areas, including fruit orchards and corn, another wave of bee kills occurred. For instance, the Washington State Department of Agriculture reported that 12,500 honey bee colonies were poisoned by insecticides in 1992, half by Penncap-M. Millions of dollars were lost in both production and fruit crops that suffered from inadequate pollination. North Carolina had a similar outbreak of apple orchard-related bee kills in the years of 1993-1995. A more detailed table of known methyl parathion bee kill incidents is attached. **It is significant that the great majority of the incidents included in the table are related to orchard uses of methyl parathion.** The removal of tree fruit uses should significantly reduce the number of bee kills reported in the future.

In response to bee kills in the 1990s, some states have instituted bee-protection programs, such as educational programs, hive registration and notification systems (farmer informs beekeeper of spray plans), and even funding to help a beekeeper move hives when spraying is planned. The States of Washington and California have imposed regulations more restrictive than EPA's regarding Penncap-M use. For instance, Washington farmers cannot spray Penncap-M on corn when it is shedding pollen. The North Carolina Department of Agriculture and Consumer Services is funding a project to reduce bee kills through training and outreach to both apple growers and beekeepers.

There is evidence that Penncap–M has continued to cause bee kills since 1992, and as a result, States have taken action. For instance, the State of New Jersey collected dead bees from several sites throughout the state in 1995. Although more than 100 pesticides were included in the analysis, only methyl parathion was detected. The 52 samples collected consisted either of pollen stripped from hive frames or returning foragers, or dead honeybees. Twenty-two of these samples (42%) had detections of methyl parathion. The State did not determine whether the formulation of methyl parathion had been the microencapsulate of the emulsifiable concentrate, because "standard methods do not distinguish between encapsulated and non-encapsulated formulations." The State of New Jersey now has state-specific label language for methyl parathion on a 24(c) local needs label.

The State of New Jersey requires applicators to notify beekeepers within a mile when a chemical that is toxic to bees is going to be applied. However, based on this study of bee kills from use of methyl parathion in orchards, the authors concluded that even this precaution was insufficient to protect bees. However, the voluntary cancellation of tree fruit uses of methyl parathion should be sufficient to mitigate this concern.

The State of Nebraska also has reported methyl parathion bee kills since 1992. For instance, Nebraska collected samples of "dead bees, pollen, wax, honey, hive surfaces and vegetation" in 1995 and 1996 in response to 14 suspected pesticide bee kills from use on corn. The results of the analyses indicated that 75% of the samples, "which consisted entirely of dead bees or pollen, contained measurable residues of methyl parathion." The analytical suite for these investigations also included carbaryl, carbofuran, lambda-cyhalothrin and permethrin, but these pesticides were not detected. Although there has been a reduction in the number of reported bee kills in recent years in Nebraska, the State indicated to EPA that it wasn't clear whether this was the result of successful attempts to reduce beekills, or a reflection of beekeepers' frustration that label violations could not be pursued against applicators based on current label language.

The Washington Professional Beekeepers Association provided information on beekills reported to the Washington State Department of Agriculture between 1992 and 1996. The WSDA received reports of 114 incidents during this period, mostly from Central Washington. Forty-seven of these kills are attributed in whole or in part to methyl parathion.

<u>Beekeeper Survey</u>

The American Beekeeping Federation, Inc. did a survey of its members to determine the extent of damage to bee colonies due to pesticide exposure. This survey was compiled through June 16, 1997. Sixty beekeepers, operating 127,950 colonies in 22 states, reported that bee losses from pesticides are a significant issue in their operations. By comparison, 26 beekeepers, operating 16,439 colonies, did not believe that pesticide losses were significant to their operations. The following table is a state-by-state breakdown of respondents who considered damage from pesticides to be a significant issue in their operations

Resident Beekeepers Responding	State	Colonies in Operation	Colonies Damaged	
			Year 95	Year 96
1	Arizona	5,000	1,000	1,000
0	*Arkansas	0	200	300
19	California	47,059	9,950	13,432
4	Colorado	7,650	2,100	2,050
0	*Delaware	0	100	110
3	Florida	3,350	2,150	2,070
2	Georgia	425	46	62
4	Idaho	16,612	3,102	3,003
1	Illinois	1,200	0	0
1	Maryland	1,400	600	650
5	Minnesota	5,800	603	450
1	Missouri	500	150	30
3	Nebraska	5,000	3,300	2,500
2	New Jersey	4,000	4,000	2,700
5	New York	4,800	1,495	1,115
0	*North Dakota	0	350	300
1	Oregon	350	3,104	2,250
3	South Dakota	7,800	1,400	1,600
2	Texas	8,000	820	1,270

1	Washington	5,000	300	500
1	Wisconsin	1,204	0	0
1	Wyoming	2,800	1,200	800
Total 60		127,950	35,970	36,192

* Migratory beekeepers reported losses in some states where no resident beekeepers responded.

The survey also listed the pesticides in order according to number of bee kill responses as follows: Ferritin, Penncap-M, Sevin, and Parathion (ethyl). Based it appears second on this survey, it appears that Penncap-M bee kills were occurring as late as 1996. Although the use of encapsulated methyl parathion on field crops will continue to pose a risk to bees, the removal of tree fruit uses will significantly reduce the overall risk of exposure to methyl parathion.

Other Insects

Brown, et al. (1978) demonstrated that predators of a cereal aphid were highly susceptible to methyl parathion.

b. Exposure and Risk to Nontarget Freshwater Aquatic Animals

EFED calculates acute and chronic EECs for aquatic organisms using predicted surface water concentrations from the GENEEC screening model, which is described in the Drinking Water assessment, above. Acute risk assessments are performed using peak EEC values for single and multiple applications. Chronic risk assessments are performed using the 21-day EECs for invertebrates and 56-day EECs for fish. A representative subset of EECs derived from GENEEC model predictions are tabulated below.

GENEEC Estimated Environmental Concentrations (EECs) For Aquatic Exposure

Site	Application Method Simulated	Application Rate (lbs ai/A)	# of Apps.; Interval Between Apps. (days)	Initial (PEAK) EEC (ppb)	21-day average EEC (ppb)	56-day average EEC (ppb)
Rice, Grasses	Aerial	0.79	6;3	95.90	27.64	10.63
Sunflower	Aerial	1.00	3;5	69.80	20.23	7.78
Soybean	Aerial	1.00	6;3	120.80	34.98	13.45

GENEEC Estimated Environmental Concentrations (EECs) For Aquatic Exposure

Site	Application Method Simulated	Application Rate (lbs ai/A)	# of Apps.; Interval Between Apps. (days)	Initial (PEAK) EEC (ppb)	21-day average EEC (ppb)	56-day average EEC (ppb)
Corn	Aerial	1.00	6;2	137.90	39.95	15.37
Alfalfa	Aerial	1.00	4;42	33.70	9.80	3.77
Barley, Oat Rye, Wheat	Aerial	1.25	6;3	151.00	43.73	16.82
Cotton	Aerial	3.00	10;3	452.05	130.74	50.28

GENEEC exposure estimates are used in EFED's first-tier assessment of risk to aquatic organisms. If EEC's from GENEEC simulations exceed LOCs, the assessment is refined using EFED's second-tier exposure model, PRZM-EXAMS. As indicated below, GENEEC-derived EEC's for methyl parathion exceed LOC's for many aquatic organisms. Therefore, a refined assessment was performed, using PRZM-EXAMS to simulate methyl parathion application to major crops.

PRZM-EXAMS Estimated Environmental Concentrations (EECs) For Aquatic Exposure

Site	Application Method Simulated	Application Rate (lbs ai/A)	# of Apps.; Interval Between Apps.	Initial (PEAK) EEC (ppb)	21-day average EEC (ppb)	60-day average EEC (ppb)
Corn	Aerial	1.00	6;2	39.45	12.23	5.35
Alfalfa	Aerial	1.00	4;42	4.32	1.43	0.77
Cotton	Aerial	3.00	10;3	254.20	70.62	32.76

ii. Freshwater Fish and Amphibians

Laboratory studies suggest that freshwater fish are not as sensitive to methyl parathion as other aquatic organisms. The high acute risk LOC and chronic LOC were not exceeded for any methyl parathion application scenario. The only exceedences were for the endangered species and restricted use LOCs for use on cotton. However, open literature studies suggest that indirect effects to fish may occur as a result of methyl parathion use.

Acute and chronic RQs tabulated below are based on a bluegill sunfish LC50 of 1.0 (0.6-1.6) ppm and a rainbow trout NOEC of <80 ppb. Note that an NOEC was not determined for rainbow trout because the lowest level tested showed effects.

Risk Quotients for Freshwater Fish and Amphibians

Site/ Rate in lbs ai/A (No. of Apps.) (App. Interval)	EEC Initial/Peak (ppb)	EEC 56-Day Ave. (ppb)	Acute RQ (EEC/LC50)	Chronic RQ (EEC/NOEC)
Rice, Grasses 0.79 (6,3)	95.44	10.63	0.10	0.11
Sunflower 1.0 (3,5)	69.79	7.78	0.07	0.08
Soybean 1.0 (6,3)	120.81	13.45	0.12	0.13
PRZM-EXAMS Corn 1.0 (6,2)	39.45	5.35	0.04	0.05
Corn 1.0 (6,2)	137.87	15.37	0.14	0.15
PRZM-EXAMS Alfalfa 1.0 (4,42)	4.324	0.77	0.00	0.01
Alfalfa 1.0 (4,42)	33.73	3.77	0.03	0.04
Barley, Oat Rye, Wheat 1.25 (6,3)	151.01	16.82	0.15	0.17
PRZM-EXAM Cotton 3.0 (10,3)	254.40	32.76	0.25	0.33
Cotton 3.0 (10,3)	452.05	50.28	0.45	0.50

Ecological and Sublethal Effects to Aquatic Organisms

Although submitted studies indicate that methyl parathion is only moderately toxic to freshwater fish, studies in the open literature indicate that methyl parathion can cause sublethal and ecological effects in aquatic environments:

Rossland (1984; MRID 44371714) found that growth of rainbow trout was affected when parathion was added to three outdoor ponds. He also discovered a secondary effect which would not have been seen in laboratory studies: "An increase in populations of *Diaptomus* in treated ponds was probably caused by mortality of predators and competitors. A bloom of filamentous algae which then collapsed, leading to severe depletion of dissolved oxygen and fish deaths, may have been triggered by mortality of herbivorous mayflies and daphnids." Rossland (1988; MRID 44371712) performed another small pond study with three ponds which showed growth reduction in rainbow trout. After three weeks, control fish had grown 6.3% per day, whereas growth was 4.3% per day in the pond treated with 10 μg/L methyl parathion, and 3.7% per day in the 40μg/L-treated pond. These growth reductions were apparently caused by damage to the invertebrate food supply. These are concentrations well below estimates from PRZM-EXAMS.

Henry et al.(1984) reported that exposure to methyl parathion resulted in an involuntary whole body flinch (which moved sequentially from head to tail), rapid and repeated "S-jerks" and fin flicks. These involuntary spasms increased with methyl parathion concentration in the water, but occurred at concentrations as low as 3 ppb. The most dominant and submissive individuals suffered these effects "more pronouncedly" than "intermediately ranked fish". Such disruptions to the social hierarchy could affect reproduction and ultimately the survival of an exposed bluegill population "if associated courtship territoriality, aggression, feeding and comfort movements are disrupted."

In addition, several other studies reported subacute effects at concentrations well below the LC50 value. Chakraborty, et al. (1989; MRID 44378601) studied the effect of methyl parathion on brain and olfactory organ acetylcholinesterase activity (AChE) of the fish, *Heteropneustes fossilis*. The brain AChE activity depleted significantly (up to 95.39% in olfactory organ) during 2-4 hours at 0.025 to 0.20 ppm of the pesticide. Rastogi, et al. (1990; MRID 44371715) reported that sublethal doses of methyl parathion caused severe damage to ovaries of the carp minnow *Rasbora daniconius*, and caused damage and size reduction in oocytes. These effects increased with the length of exposure. The ovarian damage caused by methyl parathion was greater than that caused by carbofuran and endosulfan. Rao, et al., 1985 (MRID 44371713) report that sublethal levels of methyl parathion have a profound effect on the rate of oxygen consumption by the fish *Tilapia mossambica* over a 48-hour study, based on results from whole-fish and specific tissue sampling.

Based on these observations the RQ analysis may underestimate the total effect on freshwater fish and amphibians.

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ii. Freshwater Invertebrates

Laboratory studies submitted to EPA indicate that methyl parathion will cause adverse affects in freshwater invertebrates under all labeled methyl parathion use scenarios. The freshwater invertebrate acute and chronic RQs tabulated below are based on a *Daphnia magna* EC50 of 0.14 ppb and a *Daphnia magna* NOEC of 0.02 ppb. All RQs listed below (for major use scenarios) exceed all freshwater invertebrate LOCs.

Risk Quotients for Freshwater Invertebrates

Site/ Application Method/ Rate in lbs ai/A (No. of Apps.)	EEC Initial/Peak (ppb)	EEC 21-Day Average	Acute RQ (EEC/LC50)	Chronic RQ (EEC/NOEC or MATC)
Rice, Grasses 0.79 (6,3)	95.44	27.64	681.71	1,382.00
Sunflower 1.0 (3,5)	69.79	20.23	498.50	1,011.50
Soybean 1.0 (6,3)	120.81	34.98	862.93	1,749.00
PRZM-EXAMS Corn 1.0 (6,2)	39.45	12.23	281.77	611.50
Corn 1.0 (6,2)	137.87	39.95	984.79	1,997.50
PRZM-EXAMS Alfalfa 1.0 (4,42)	4.324	1.43	30.89	71.50
Alfalfa 1.0 (4,42)	33.73	9.8	240.93	490.00
Barley, Oat Rye, Wheat 1.25 (6,3)	151.01	43.73	1,078.64	2,186.50
PRZM-EXAMS Cotton 3.0 (10,3)	254.40	70.62	1,817.14	3,531.00
Cotton 3.0 (10,3)	452.05	130.74	3,228.93	6,537.00

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Estuarine and Marine Animals

Acute Risk

The RQs calculated with the PRZM-EXAMS model exceeded endangered species LOCs for all crops simulated. Acute estuarine and marine species RQs exceed all LOCs for four crops: corn (1.0 lbs/A), potato (1.5 lbs/A) and cotton (3.0 lbs/A). Restricted use and endangered species LOCs were also exceeded by the pecan (2.0 lbs/A) use scenario.

Risk Quotients for Estuarine/Marine Fish Based on a Spot LC50 of 59 ppb

Site/Appl Method Rate-ai/A(no. appl,interval)	EEC Initial/ Peak (ppb)	Acute RQ (EEC/LC50)
Rice, Grasses 0.79 (6,3)	95.44	1.62
Soybean 1.0 (6,3)	120.81	2.05
PRZM-EXAMS Corn 1.0 (6,2)	39.45	0.67
Corn 1.0 (6,2)	137.87	2.34
PRZM-EXAMS Alfalfa 1.0 (4,42)	4.324	0.07
Alfalfa 1.0 (4,42)	33.73	0.57
Barley, Wheat 1.25 (6,3)	151.01	2.56
PRZM-EXAMS Cotton 3.0 (10,3)	254.40	4.31
Cotton 3.0 (10,3)	452.05	7.66

Effects of methyl parathion exposure on estuarine and marine fish species include behavioral changes, growth reduction from damage to the food supply, and indirect mortality. The toxicity tests for estuarine and marine fish indicate that they are more sensitive to methyl parathion than freshwater species. The most sensitive freshwater species has an LC50 of 1.0 mg/L (bluegill sunfish). In comparison, the LC50 for the estuarine spot is 0.059 mg/L.

Foe et al. (1991) and Heath, A.G.et al.(1993)(MRID No.44378602) investigated the effects of rice cultivation on the striped bass population in the San Francisco Bay and its tributaries. Foe et al.(1991) correlated the larval bass population in the delta between the Sacramento and San Joaquin Rivers with the pounds of methyl parathion applied to rice in that drainage basin. The following figures, 6a and b from Foe et al. (1991), show that methyl parathion use (lbs/A) correlates with the striped bass population decline in this portion of the estuary:

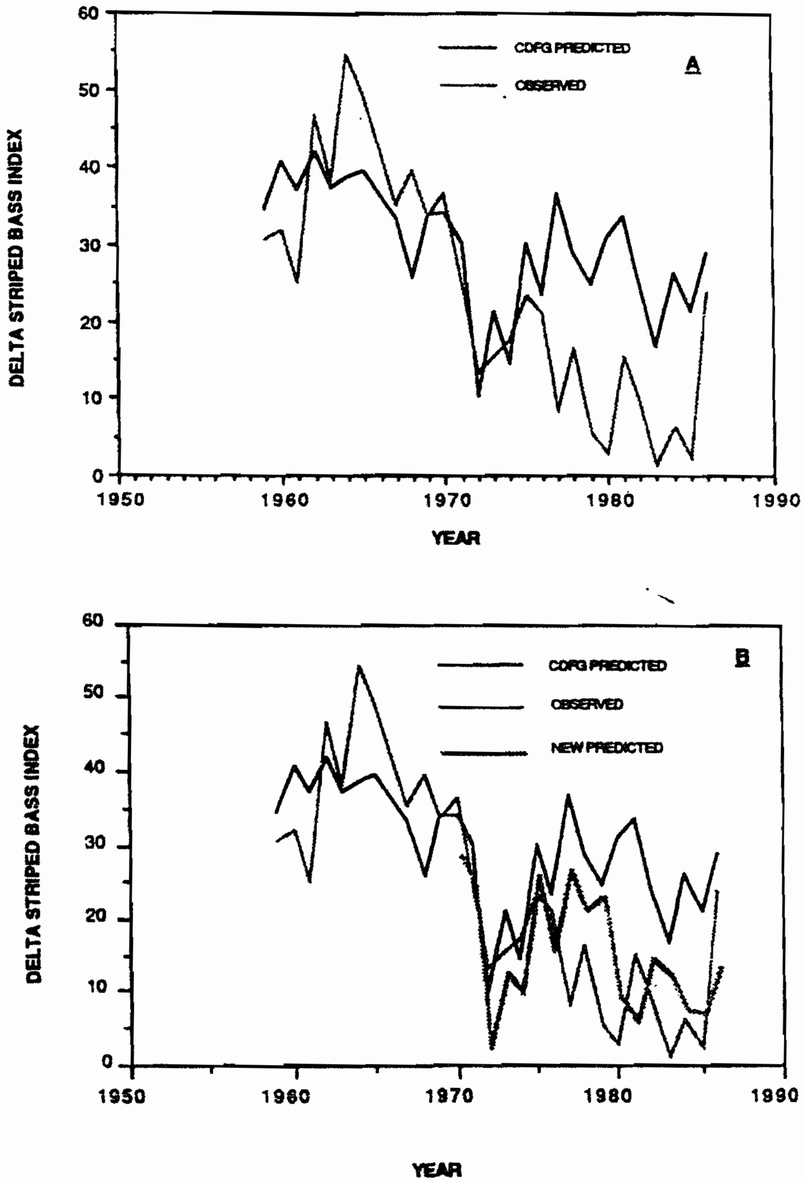

Figure 6a and b. (a) Relationship between the California Department of Fish and Game's Delta portion of the striped bass index and the actual number of fish observed in the Sacramento-San Joaquin Delta between 1959 and 1986. The index ceased to predict abundance well after 1977. Data provided by Mr. Lee Miller, CDFG, Stockton. (b) Same relationship as in 6a with the exception that the "new predicted" curve is the result of the inclusion of an additional term (- (3.34 (lbs methyl paration applied to rice fields/Sacramento River flow rate) + 2.83)) in the index. Th new equation improves the predictive power of the old index in all years after 1977 except for 1986.

Heath et al. (1993; MRID 44378602) studied the effects of methyl parathion at concentrations found in the San Francisco Bay estuary on newly hatched striped bass. In an attempt to simulate larvae exposed in the river which then float downstream away from the contamination, the larvae were exposed to methyl parathion for 4 days and observed for 10 days in uncontaminated water. The two most significant effects were abnormal swimming performance (swimming on their side) and increased AChE inhibition, especially if food was restricted. Spawning of striped bass occurs during May and early June in the Sacramento river between Colusa and Knights Landing, California. Methyl parathion is one of several rice insecticides used extensively in this area at the time of striped bass spawning (Cornacchia et al. 1984; Finlayson and Faggella 1986).

Heath et al. (1993) suggested that poorer swimming performance during times of food scarcity is significant because it can affect the ability of striped bass to avoid predation. This risk is compounded by the fact that adult fish require days or weeks to recover to normal ACHE activity levels, depending on the degree of cholinesterase inhibition caused by methyl parathion exposure. As indicated in the estuarine/marine invertebrate assessment below, methyl parathion contamination may affect their invertebrate food supply at concentrations reported in Heath, et al. (1993).

Unfortunately, this experiment was limited to only one estuary and one species. Acute toxicity studies submitted to EPA show that striped bass is not the most sensitive estuarine/marine fish species. While the striped bass LC50 is 0.79 ppm, the spot LC50 is 0.059 ppm, many times more sensitive than the striped bass. If we assume that the relationship between the sensitivity of striped bass and spot holds for subacute effects, then subacute effects in spot, and possibly other species, would be expected at much lower concentrations.

Eisler (1970; MRID 44378611) also showed toxicity increased by changes in environmental conditions, such as the length of exposure to methyl parathion, salinity and temperature. He found that extending the exposure period from 96 to 240 hours reduced the LC50 by a factor of 8.3 for mummichog, (*Fundulus heteroclitus*). In a second experiment, fish were moved to methyl parathion-free water after a 96 hour exposure and observed for 72 and 240 hours. The 72 hours observation period allowed time for mortality to increase 1.33 times over the mortality at the end of the treatment period. For the 240 hours observation period mortality increased 2 times. Eisler (1970) also indicated that mummichogs, "unlike other groups, were sluggish and refused to feed during the observation period." By increasing the temperature 5° C from 20 to 25°C the LC50 value became the LC100. Similarly, toxicity was seen to increase with salinity. The LC50 at 24‰ salinity was equal to the LC100 at 36‰. The observation period, temperature and salinity increases are expected to decrease the concentration of methyl parathion needed to cause mortality or sublethal effects.

Methyl parathion may reduce available food resources for estuarine and marine fish which feed on invertebrates. Both estuarine and marine aquatic freshwater invertebrates are highly sensitive to methyl parathion (see below). In addition, insects with an aquatic life stage can be killed by methyl parathion sprays while still in their terrestrial stage, and therefore not be available to produce larvae, a food source for fish.

Chronic Effects

No acceptable fish early-life stage study is available for estuarine /marine fish. Therefore, chronic risk to estuarine/marine fish cannot be evaluated at this time. The registrant must submit new studies to fulfill this guideline requirement.

Estuarine/ Marine Invertebrates

Methyl parathion is very highly toxic to estuarine/marine invertebrates, at concentrations that have been found in surface water. The *daphnia* (freshwater) EC50 is 0.14 ppb and the mysid (saltwater) EC50 is 0.35 ppb. Concentrations of methyl parathion in the Colusa Basin Drain study mentioned above (Heath et al, 1993; MRID 44378602) were as high as 0.66 ppb. GENEEC and PRZM-EXAMS RQs for all use scenarios exceed all LOCs. It should be noted, however, that GENEEC and PRZM-EXAMS do not simulate estuarine or marine scenarios.

Other open literature studies report effects of methyl parathion exposure on estuarine/marine invertebrates. Finlayson et al. (1993; MRID 44572901) reported methyl parathion toxicity to a mysid species (*Neomysis mercedis*) in a California estuary. The author reported that of three pesticides identified in the Colusa Basin Drain (carbofuran, malathion, and methyl parathion), methyl parathion was most likely responsible for observed effects on mysids, since survival was best correlated with the presence or absence of that contaminant. *Neomysis mercedis* is an important food source for juvenile striped bass, and an important component of both the pelagic and the epibenthic communities.

Lowe (1981; MRID 66341) showed that survival and number of offspring in *Mysidopsis bahia* were affected at concentrations between 110 and 370 ppt..

RQs for estuarine/marine invertebrates are based on a mysid EC50 of 0.35 ppb, and an NOEC of 0.11 ppb.

Risk Quotients for Estuarine/Marine Invertebrates

Site/ Application Method	EEC Initial/ Peak (ppb)	EEC 21-day Average	Acute RQ (EEC/LC50)	Chronic RQ (EEC/ NOEC)
Rice, Grasses 0.79 (6,3)	95.44	27.64	272.69	251.27
Soybean 1.0 (6,3)	120.81	34.98	345.17	318.00

5/[illegible]

Risk Quotients for Estuarine/Marine Invertebrates

Site/ Application Method	EEC Initial/ Peak (ppb)	EEC 21-day Average	Acute RQ (EEC/LC50)	Chronic RQ (EEC/ NOEC)
PRZM-EXAMS Corn 1.0 (6,2)	39.45	12.23	112.71	111.18
Corn 1.0 (6,2)	137.87	39.95	393.91	363.18
PRZM-EXAMS Alfalfa 1.0 (4,42)	4.324	1.43	12.35	13.00
Alfalfa 1.0 (4,42)	33.73	9.80	96.37	89.09
Barley, Wheat 1.25 (6,3)	151.01	43.73	431.46	397.55
PRZM-EXAMS Cotton 3.0 (10,3)	254.40	70.62	726.86	642.00
Cotton 3.0 (10,3)	452.05	130.74	1,291.57	1,188.55

All acute and chronic LOCs are greatly exceeded by Rqs for estuarine and marine invertebrates.

d. Exposure and Risk to Nontarget Plants

I. Terrestrial and Semi-aquatic

Terrestrial and semi-aquatic plant testing are required. Youngman, et al., (1989) suspected possible phytotoxic effects based on the phytotoxicity of ethyl parathion, and the chemical relationship of 4-nitrophenol to the herbicide DNOC (2-methyl-4, 6-dinitrophenol). Their subsequent study showed a nearly 50% dry-weight reduction in whole lettuce plants treated with methyl parathion.

Therefore, vegetative vigor (122-1) and seedling emergence (122-1) studies are required.

ii. Aquatic Plants

Exposure to nontarget aquatic plants may occur through runoff or spray drift from adjacent treated sites. An aquatic plant risk assessment for acute high risk is usually made for aquatic vascular plants from the surrogate duckweed *Lemna gibba*. Non-vascular acute high aquatic plant risk assessments are performed using either algae or a diatom, whichever is the most sensitive species. An acute aquatic plant risk assessment for endangered species is usually made for aquatic vascular plants from the surrogate duckweed *Lemna gibba*. Runoff and drift exposure is computed from GENEEC. The RQ is determined by dividing the pesticide's initial or peak concentration in water by the plant EC_{50} value.

Methyl parathion is "practically non-toxic" *to Skeletonema costatum*. However, data are lacking on other aquatic plants. These data are important because it is known that methyl parathion is very toxic to aquatic invertebrates, and any detrimental effects on aquatic plants could result in further damage to invertebrates which, in turn, could have significant effects on fish. Accordingly, testing of additional species (*Kirchneria subcapitatum*, *Lemna,* and *Anabaena flos-aquae*) for aquatic plant growth (122-2) is needed.

Endangered and Threatened Species

At currently proposed rates, endangered species LOCs are exceeded for all species groups except plants. The Agency has developed a program (the "Endangered Species Protection Program") to identify pesticides whose use may cause adverse impacts on endangered and threatened species, and to implement mitigation measures that will eliminate the adverse impacts. At present, the program is being implemented on an interim basis as described in a Federal Register notice (54 FR 27984-28008, July 3, 1989), and is providing information to pesticide users to help them protect these species on a voluntary basis. As currently planned, the final program will call for label modifications referring to required limitations on pesticide uses, typically as depicted in county-specific bulletins or by other site-specific mechanisms as specified by state partners. A final program, which may be altered from the interim program, will be described in a future Federal Register notice. The Agency is not imposing label modifications at this time through the RED. Rather, any requirements for product use modifications will occur in the future under the Endangered Species Protection Program. Currently available county specific information, maps and a downloadable version of the Endangered Species data base can be found on the Internet at the Agency's web site, http://www.epa.gov/ESPP.

RISK CHARACTERIZATION

EFED concludes with a great deal of certainty that the use of methyl parathion poses significant risk to nontarget organisms in terrestrial and aquatic environments. The toxicological and exposure data suggest strongly that acute and chronic effects on birds and mammals, acute effects on bees, and acute and chronic effects on aquatic invertebrates are likely to occur as a result of methyl parathion applications.

Monitoring data include detections of methyl parathion residues in ground and surface water, but suggest that the risk of drinking water exposure is less than that predicted by simulation models.

Drinking Water

Surface Water

Direct drinking-water data for methyl parathion are not readily available, and it is not likely that much of such data has been collected. While the Office of Water has established a lifetime health advisory (HA) of 2 ppb, methyl parathion does not have an established Maximum Contaminant Level, and is not included on the Unregulated Contaminant Monitoring List. Therefore, public drinking water supply systems are not required to analyze for methyl parathion. Consequently, EFED relied on simulation models and other surface- and ground-water monitoring data for this risk assessment.

Surface-water concentrations estimated from the PRZM-EXAMS screening model for human health risk assessments are quite high (acute- 254 ppb, chronic- 6.6 ppb). However, these screening estimates are significantly higher than the concentrations seen in monitoring studies. This can be attributed in part to the conservative nature of the models themselves. As detailed in the drinking water section above, the assumptions are intentionally conservative to ensure the maximum protection of human health. There is fairly high uncertainty in the assessment that acute and chronic exposure to methyl parathion in drinking water will occur at those concentrations.

Acute Risk

Data from targeted monitoring studies such as those in California and the Mississippi River basin may provide a better estimate of possible acute drinking water concentrations than the models. First, the scenario of a canal or river that drains a watershed which is extensively treated with methyl parathion is a more realistic scenario for predicting drinking-water contamination than the models' 10-hectare field draining to a 1-acre pond. In addition, the California data show the effects of mitigation on concentrations detected year-to-year in surface water. Previous to a mitigation program instituted by California EPA's Department of Pesticide Regulation (CDPR) in the early 1990's, peak concentrations of methyl parathion in the Colusa Basin Drain were as high as 6 ppb. Since the implementation of buffer zones, the requirement for applicators to use specific equipment to mitigate spray drift, and holding time requirements for water on rice fields, peak concentrations have been at the sub-ppb level.

Although these monitoring data are more realistic than modeling results, they don't necessarily reflect the use scenarios most vulnerable to contamination. For instance, the CDPR monitoring of the Colusa Basin Drain is targeted to methyl parathion use on rice. It includes sampling which coincides with times of application, but the maximum rate at which methyl parathion is applied to rice is one quarter of the maximum rate applied to cotton, with fewer applications annually. In addition, retention of water on treated fields is a mitigation measure relevant only to rice, and not other crops to which methyl parathion is applied.

EFED has obtained recent, targeted surface-water monitoring data taken by the USGS NAWQA program from rivers in the Mississippi Embayment cotton-growing region. Samples were taken from five rivers in 1996 and 1997, and methyl parathion was detected in all five. Detected concentrations ranged from 0.015 ppb up to **0.422 ppb**. The site with the highest frequency of detections in this study had **8 detections in 17 samples during water year (WY)1996, and 8 detections in 37 samples during WY1997.**

In another 1996 monitoring program in the Mississippi Embayment, the USGS detected methyl parathion in 18% of the 60 samples it collected from tributaries of the Mississippi River. The highest concentration detected was about 0.12 ppb, and the 50^{th} percentile concentration was about 0.05 ppb.

EFED attempted to determine whether data from the Mississippi Embayment NAWQA study could be directly related to potential drinking water supplies. Barbara Kleiss, head of the MS Embayment NAWQA study, indicated that **the state of Mississippi derives its drinking water almost exclusively from ground water**, and that of the five stations sampled for methyl parathion, only one was within 25 miles of a surface-water body used for drinking water. Neighboring Louisiana, however, does have a number of public supplies which derive drinking water form surface-water sources.

Mary Gentry of the Louisiana State Department of Environmental Quality indicated that of the surface water supplies in the state, only two had laboratory facilities which might allow them to analyze for methyl parathion in their water supply. In addition, these two CWS were the only supplies that might have activated carbon filtration systems. This is significant, as Dr. Thomas Speth of EPA's Cincinnati lab indicated that carbon filtration would be the method of choice for the removal of methyl parathion from drinking water. Based on raw and finished drinking water data for malathion, it seems possible that methyl parathion would be converted to the more toxic methyl paraoxon during routine oxidative water sterilization.

The two community water sysems (CWS), located in New Orleans and in East Jefferson Parish, both draw their water from the Mississippi. Mr. Marvin Russell of the New Orleans CWS indicated that their system had not included methyl parathion as an analyte in their contaminant testing.

The Jefferson Parish CWS did test for methyl parathion in 1994 at two intakes on the Mississippi River. In this study, continuous raw water samples were collected with a peristaltic pump into a 5-gallon carbuoy. Composite samples so collected were analyzed weekly for a year. Methyl parathion was detected in 18 of 52 samples from the one bank of the river, and 21 of 52 from the other bank. The average concentration of the detections was 0.009 ppb, the level of detection. The highest concentration detected was 0.041 ppb.

While the samples analyzed in the Jefferson Parish study only reflect conditions of a single year, they are representative of possible chronic drinking-water contamination from a very large surface-water source. The low concentrations are consistent with EFED's conclusion (detailed below) that methyl parathion should not pose a chronic risk to drinking water supplies. However, they are not useful in predicting possible acute exposure to methyl parathion.

Based on the data that are currently available, EFED believes that acute (peak) concentrations of methyl parathion in surface water can at least be periodically detected in the range of 0 to 6 ppb, based on CDPR data taken before mitigation measures were adopted in the early 1990's. It is likely that higher concentrations could result from uses that have higher application rates and numbers of annual applications. However, acute concentrations are unlikely to be as high as simulated by PRZM-EXAMS. Although the CDPR Colusa Basin Drain study only includes 10 years of data, the data are of high quality. Therefore, the concetration of 6 ppb detected in this study should be given greater weight than the peak concentration of 95 ppb simulated by GENEEC for rice.

EFED can not state with confidence that the detection of 6 ppb found in a targeted (rice) monitoring study represents the highest surface-water concentration that might occur in areas of methyl parathion use. Given the lack of direct drinking water data, and uncertainties related to the effects of water treatment on methyl parathion, EFED can neither state with certainty that concentrations of methyl parathion detected in surface water correspond to the concentrations that might be detected in drinking water derived from surface water. Therefore, EFED can only say that in evaluating drinking-water values for use in human-health risk assessment, there is greater confidence in targeted surface-water monitoring values such as the value of 6 ppb from rice-growing areas and 0.422 ppb from the Mississippi Embayment cotton-growing region than in the peak PRZM-EXAMS value for cotton of 254 ppb.

Chronic Risk

In addition to the single year of targeted, composited surface-water monitoring data from Louisiana described above, non-targeted surface-water survey studies performed over 30 years have not shown concentrations of methyl parathion at chronic levels predicted in modeling assessments. Concentrations from available studies were below the 2 ppb HA, with the highest reported at 1 ug/L. The results of the more recent studies in the Mississippi River Basin and NAWQA study areas resulted in lower concentrations. It should be noted, though, that these recent studies are not specifically targeted to methyl parathion use areas, and that the analytical recoveries for methyl parathion in the NAWQA study averaged only 46%. **Such low recoveries limit extensive quantitative interpretation of the monitoring data.** However, the monitoring data are expected to be lower than the modeling predictions because of the conservative assumptions used in the models.

Therefore, the consistent indication that methyl parathion is not a widespread contaminant in surface water adds greater uncertainty to the Tier I and Tier II chronic water exposure estimates. Although the available monitoring data do not allow a definitive assessment, EFED does not believe that chronic concentrations of methyl parathion in surface water will reach the 2 ppb HA.

Ground Water

Using the screening model SCI-GROW, EFED calculated a ground-water concentration of 0.6 ppb for first-tier human-health risk assessment. Data collected from a variety of sources did not identify any known instance in which a ground-water concentration higher than this was detected, although individual detections have been within the same order of magnitude. Therefore, EFED suggests that 0.6 ppb is a reasonable conservative estimate of possible acute concentrations of methyl parathion in drinking water derived from ground water.

Since methyl parathion has been detected in ground-water rarely in all studies evaluated, the concentration of 0.6 ppb does not seem appropriate for chronic risk assessments. For instance, methyl parathion was not found in the Mid-Continent Pesticide Study (from Barbash and Resek, 1996), and was found at a maximum of 0.062 ppb in 1130 samples taken between 1991 and 1995 in the USGS NAWQA study. Again, these studies were not specifically targeted to methyl parathion, and the uncertainty of the NAWQA results is increased because of analytical recovery problems. EFED does not have a tool for estimating second-tier ground water concentrations for dietary risk assessments. However, EFED concludes that methyl parathion does not pose a chronic concern for drinking water derived from ground water.

Ecological Effects

Avian Risk Characterization

EFED concludes with a high level of certainty that methyl parathion poses significant acute and chronic risk to birds. This certainty is founded on (1) the consistent toxicological data, (2) the potential for degradation products to be highly toxic, (3) the widespread use of the compound on many crops that are attractive to wildlife, and (4) field-observed effects during use.

There is very little uncertainty in the toxicology data because of the consistent results reported in registrant and open literature studies. Studies cited in this chapter indicate that a suite of effects occur with short exposure to methyl parathion. These include direct mortality, as well as acute sublethal effects such as:

- reproduction effects,
- changes in maternal care and viability of young birds,
- anorexia,
- increased susceptibility to predation, and
- greater sensitivity to environmental stress.

For several reasons, most of the uncertainty in this risk analysis is associated with the terrestrial exposure component. First, there were no direct field measurements of methyl parathion residues used in the avian risk assessment. Furthermore, while the application method and timing are such that one can reasonably assume exposure of birds each time methyl parathion is applied, there are little direct data (e.g. incidents) showing avian exposure.

Finally, the uncertainty in the environmental fate database for the highly toxic degradate methyl paraoxon may lead to an *underestimation* of avian and mammalian exposure to biologically active methyl parathion residues. The quantities of methyl paraoxon produced from parent on food items are not known. This point is particularly important because degradation of parent to methyl paraoxon on the surfaces of leaves and avian food items may result in a prolonged exposure to toxic residues which can result in acute and/or chronic effects to birds, mammals, and reptiles.

The use of methyl parathion is expected to coincide with the timing of waterfowl breeding. The major breeding grounds for waterfowl are in the prairie-pothole region of North America, with the greatest concentration of breeding ducks per square mile found in the Dakotas (see Appendix 3). Grue, et al. (1988) reported that about 75% of cultivated land in North Dakota is in the prairie-pothole region where important crops include spring wheat, barley and sunflowers; methyl parathion is used on each of these crops. Grue also reported effects of methyl parathion exposure to waterfowl and the freshwater invertebrates upon which they feed.

Cotton and rice use in Mississippi River watersheds and in California are expected to affect resident bird populations (non-migratory birds) with nests near treated fields. In addition to waterfowl, a large number of shorebirds such as gulls, cranes, herons, plovers, sandpipers, egrets, stilts, terns and others are found in and around aquatic resources that could be contaminated with methyl parathion.

Mortality and reproductive impairment of survivors pose important risk to the maintenance of viable populations of avian species. Because these species are representative of the more than 50 avian species known to occur in and around cotton fields, the potential for adverse population impacts to many avian species from methyl parathion exposure is great. The table below presents trends in breeding bird populations of several avian species relevant to this risk characterization. These data originate from National Biological Service (Sauer et al. 1997). All the species shown exhibit downward trends in population in three or more cotton states since 1966. Four species (white-eyed vireo, mourning dove, northern cardinal, and red-winged blackbird) showed population declines that were statistically significant ($p<0.05$) in three or more states. These data do not establish causality for population declines (a variety of factors are likely to contribute to population declines), but they do suggest that local populations of many bird species could be sensitive to the subacute or reproductive effects from exposure to methyl parathion detailed in the risk assessment.

Population Status of Important Bird Species in Cotton States

State	Trends in Breeding Bird populations 1966-1996					
	Carolina Wren	White-Eyed Vireo	Northern Cardinal	Blue Grossbeak	Mourning Dove	Red-Winged Blackbird
AL	negative	positive	negative	positive	negative	negative*
AR	negative	negative*	positive	positive	negative	positive*
AZ	no data	no data	negative	positive	negative	positive
CA	no data	no data	no data	positive	negative*	positive
FL	positive	negative	negative	positive	positive	negative*
GA	positive	negative	negative*	positive	negative	negative*
LA	positive	negative	negative	positive	positive	negative
MO	positive	negative	negative*	positive	negative*	positive
MS	positive	positive	negative	negative	negative	negative*
NC	positive	positive	negative	positive	negative	negative
NM	no data	no data	no data	positive	negative	negative
OK	positive	positive	positive	negative	negative*	positive
SC	negative	stable	negative*	positive	negative	negative*
TN	positive	negative*	negative*	positive	negative	positive
TX	positive	negative*	positive	negative	negative*	negative
VA	positive	positive	negative*	positive	negative	negative*

* denotes declines significant to $p<0.05$

Further avian exposure to methyl parathion is likely in the 80 million acres in the United States planted to corn which accounts for more than 11% of methyl parathion applied annually. As shown in Appendix 4, at least 200 bird species are found in and around corn, the majority of which is produced in three regions (the Corn Belt - Iowa, Missouri, Illinois, Indiana, Ohio; the Great Lakes states - Minnesota, Michigan, Wisconsin; and the northern plain states - North and South Dakota, Nebraska, Kansas, and Colorado). Methyl parathion applied to corn planted near prairie-potholes in the Great Lakes and northern plains regions would be expected to affect waterfowl. Application of methyl parathion to corn in states that border the Gulf of Mexico and the Atlantic and Pacific Oceans is also expected to result in exposure to waterfowl and water birds.

Aquatic Organisms

The uncertainty in the assessment of potential concentrations of methyl parathion in surface water (see above) has ramifications for risk assessments for aquatic organisms.

Freshwater Fish

Calculated EECs indicate that only use at the highest label rates might result in exposure to freshwater fish above acute LOCs. The PRZM-EXAMS RQ for cotton was 0.21, which exceeds the restricted use (0.1) and endangered species (0.05) LOCs. Given the uncertainty in the exposure estimates derived from PRZM-EXAMS, the level of certainty in these LOC exceedences is not high.

However, outside data indicate that methyl parathion exposure has detrimental effects on freshwater fish, including behavioral changes, growth reduction from damage to the food supply, and indirect mortality. Given that the cotton use area extends in the southern United States from California to Virginia, a large number of freshwater species could be affected by methyl parathion exposure. Therefore, although there is substantial uncertainty in the magnitude of the exposure calculated using simulation models, sublethal or indirect effects from exposure in the cotton use area seem likely.

Freshwater Aquatic Invertebrates

Laboratory studies submitted to EPA indicate that methyl parathion is likely to cause adverse effects in freshwater invertebrates under all labeled methyl parathion use scenarios. The PRZM-EXAMS cotton (3.0 lb ai/A) RQs are 1530 and 3503 for acute and chronic exposure, respectively. Use on rice, the use with the lowest application rate (0.79 lbs ai./A), yields RQs of 681 and 1382 for acute exposure and chronic exposure, respectively. Hence, all LOCs are exceeded by all application scenarios. The acute RQ values above exceed LOCs by at least an order of magnitude. Therefore, even considering the uncertainty of exposure estimates from PRZM-EXAMS, the certainty that methyl parathion will cause acute adverse effects in freshwater invertebrates is high.

Damage to populations of freshwater aquatic invertebrates can cause additional damage to the ecosystem, as discussed above. For instance, Crossland (MRID 44371714) reported that damage to freshwater invertebrates led to an algae bloom which caused a fish kill by depleting dissolved oxygen in treated ponds.

Although chronic data are not available for freshwater invertebrates, the magnitude of the acute RQs indicates that it is highly likely that toxic exposure will occur on a chronic basis as well.

Estuarine and Marine Fish

EFED concludes that methyl parathion poses significant acute risk to estuarine and marine fish. This assessment is founded on consistent toxicological data submitted by the registrants and in the open literature and the widespread use of the compound on many crops that may result in transport of methyl parathion to surface-water bodies.

The certainty in the acute toxicity analysis for estuarine and marine fish is limited by the use of PRZM-EXAMS for estimating estuarine and marine exposure. Given the wide range of depths and flushing rates of estuaries, for instance, EFED cannot be sure whether values predicted by PRZM/EXAMS are underpredictions or overpredictions of potential exposure. The RQs calculated with the PRZM-EXAMS model exceeded endangered species LOCs for all crops simulated. Acute estuarine and marine species RQS exceed all LOCs for four crops: corn (1.0 lbs/A), potato (1.5 lbs/A), peach (1.5 lb/A) and cotton (3.0 lbs/A). Restricted use and endangered species LOCs were also exceeded by the cherry (1.5 lbs/A), pecan (2.0 lbs/A), and grape (3.0 lb/A) use scenarios. EFED is not aware of estuarine or marine monitoring data that include detections of methyl parathion at concentrations equivalent to the 29.5 ug/l acute LOC used in the risk assessment for estuarine and marine fish.

However, open literature studies attest to adverse affects of methyl parathion exposure to estuarine and marine fish. For instance, a study of methyl parathion effects on striped bass spawn in the delta between the Sacramento and San Joaquin Rivers correlated declines in the larval bass population with the pounds of methyl parathion applied to rice in that drainage basin (Foe et al.,1991). Other studies have also reported acute sublethal effects on estuarine and marine fish, such as behavioral changes, cholinesterase inhibition, and ovarian damage.

As with freshwater fish, there is significant uncertainty associated with the likely magnitude of exposure to methyl parathion. As noted above, targeted monitoring data from the Colusa Basin Drain in California produced a peak surface-water concentration that was about an order-of-magnitude less than predicted for rice by GENEEC. However, the Colusa Basin Drain study reflected usage before mitigation measures were put into effect for methyl parathion application to rice. Furthermore, while the California study considered the use of methyl parathion on rice, higher application rates are used on a greater number of cotton acres in coastal areas of Texas, Louisiana and Alabama. A more detailed discussion of species that might be exposed to methyl parathion in cotton-growing areas can be found below.

An assessment of the **chronic** effects of methyl parathion use on estuarine species is complicated by the lack of chronic estuarine study data. The need for such data is evaluated by comparing expected exposure levels to 0.01 of the acute LC50, in this case 0.59 ppb. If this concentration is on the order of that expected to be found in surface water, then such studies are required. Based on surface-water monitoring results reported in this chapter, Cheminova should perform chronic estuarine studies to clarify the possible chronic risk to estuarine and marine fish. However, given the lack of data needed to derive the chronic LOC, it is not possible to evaluate the chronic risk of methyl parathion to estuarine/marine fish at this time.

Estuarine and Marine Invertebrates

As reported in the toxcity portion of this RED, estuarine/marine invertebrates are extremely sensitive to methyl parathion, with the exception of mollusks. The certainty of this toxicity is quite high; EC50s for species such as daphnia (0.14 ppb) and mysids (0.35 ppb) are at concentrations that have been detected in surface water such as in the NAWQA Mississippi Embayment Study.

Open literature studies show that use of methyl parathion under normal use conditions has contaminated the estuarine/marine environment and had an effect on estuarine invertebrate species. For instance, Finlayson (1993)(MRID 44572901) reported methyl parathion toxicity to a mysid species (*Neomysis mercedis*). However, the CDPR has performed *Ceriodaphnia dubia* bioassays concurrently with their surface water sampling, and reported no observable effects connected with methyl parathion concentrations since mitigation measures were instituted in response to a decline in striped bass populations.

The following mitigation methods have been applied to the use of methyl parathion on rice to control tadpole shrimp in California:

1. Planting the seed and quickly flooding fields so that the tadpole shrimp eggs do not mature in time to significantly damage the rice.
2. Holding contaminated water on the field longer so that the chemical has time to degrade.
3. Educating rice growers that overuse has caused resistance.
4. Prescribing specific equipment for aerial spraying;
5. Use of copper sulfate as an alternative;
6. Observing a 300 foot buffer zone from bodies of water for aerial sprays.

Mitigation measures instituted in California for rice may not be appropriate in other states. For instance, the use of copper sulfate and flooding to control tadpole shrimp are not appropriate for the Gulf States, because the tadpole shrimp is not a pest in that region. In addition, while rice in California is grown during the dry season, the Gulf states do not have a distinct dry season. Therefore, water held on a rice field in the Gulf States may flow off the field during rain events. Finally, mitigation measures such as holding water on a field are not applicable for crops such as cotton, soybeans, hay, and corn.

Therefore, in light of supporting open literature data, and the evidence of adverse effects in California before mitigation was instituted, the certainty in the overall risk to estuarine/marine invertebrates is high.

Estuarine/Marine Fish and Invertebrates Likely to Be Affected

In addition to California, where effects on estuarine species has been observed in connection with methyl parathion use on rice, the coastal areas of the Gulf States include a vast area of wetland habitats for estuarine species. For instance, Texas has over 300,000 acres of tidal flats, the most in the nation. Tidal flats are an important habitat and feeding ground for coastal shorebirds, fish and invertebrates such as crabs, oysters, clams, shrimp and mussels. Texas ranks second in the nation in total area of salt marshes, with about 480,000 acres, and third in the nation in freshwater marshes with approximately 530,300 acres. Freshwater marshes, which are located upstream along river valleys, support a variety of species of fish, birds, and fur-bearing animals, as well as shrimp and crayfish.

Game fish, shrimp and crabs will visit shallow water of these estuarine habitats in the late spring and summer when methyl parathion runoff is likely. Species such as red and black drum, sea trout and blue crabs spawn in estuaries or shallow bays, and male crabs remain there after breeding. Black drum thrive in water so shallow that their backs are exposed, and red drum feed in water shallow enough that their tails emerge from the water when they feed. Other important commercial species such as yellow flounder and brown, white and pink shrimp also spend a portion of their lives in estuaries. Therefore, runoff of methyl parathion into shallow aquatic areas is likely to cause hazardous exposure to many commercially important estuarine species.

Mammals

Acute and chronic exposure studies indicate that methyl parathion is very highly toxic to mammals. Calculated risk quotients exceed at least one LOC for all labeled application rates. There is uncertainty associated with Rs because they are based on LC50 values derived from LD50 data. Mammals are expected to be adversely affected by methyl parathion through oral, dermal, and inhalation exposure pathways.

Herbivores and insectivores are more likely than granivores to be adversely affected by **oral** methyl parathion exposure, because they must consume a greater amount of food in proportion to their body weight each day. All herbivore and insectivore LOCs are exceeded after a single application of methyl parathion at the lowest application rate (0.1 lb ai/A), except by the RQ for the large insect food source. The single-application LOCs for small (15 g) granivores are all exceeded at application rates equal to or greater than 0.75 lb ai/A. All LOCs for 35-gram granivores are exceeded for application rates at or above 1.0 lb ai/A. Therefore, both the corn and cotton uses will result in acute LOC exceedences for these mammals after a single application. All chronic and reproduction LOCs for grass, foliage and seed are exceeded after a single application of 0.5 lb ai/A.

The risk posed by exposure to methyl parathion is expected to increase with the number of applications. The minimum number of applications as recommended on the label is 2 and the maximum is 10. Acute, chronic, and reproductive RQs are greater for multiple applications. The risk assessment for multiple applications to cotton at the maximum application rate of 3.0 lb ai/A predicts the exceedence of every LOC for herbivores, insectivores and granivores of all sizes.

Dermal exposure to methyl parathion is highly likely for mammals. Small mammals, such as meadow voles or field mice, live in and around the treated fields and find it difficult to impossible to escape the treated area. In addition, mammals have bare skin showing on the nose and feet and must travel through treated crop or nearby edge of grass.

Young mammals are expected to be at greater risk than adults. The young of almost any species eat more than adults. In addition, very young mammals are hairless and may be susceptible to dermal exposure from a variety of sources including residue on the fur of the mother.

Effects on Bees and Beneficial Insects

The effects of methyl parathion exposure on bees has long been recognized, and is reflected in label language on the Penncap-M label. A large body of data submitted to EPA and found in the open literature documents bee mortality and colony destruction connected to methyl parathion exposure. Atkins and Kellum (1980) reported that residues of methyl parathion on alfalfa foliage were highly toxic to honeybees at application rates ranging from 0.03125 to 0.5 lb ai/acre. At the higher rates (0.25 and 0.5 lb ai/acre), the toxicity persisted from 4 to 6 days. In addition, microencapsulated methyl parathion may remain toxic in stored pollen from one season to the next (Johansen and Kious, 1978), or as long as 19 months (Barker et al., 1979). Therefore, the certainty in this assessment is very high.

There has long been concern about the effect of Penncap-M on bees, since microencapsulated methyl parathion is similar in size to pollen. The warning statement on the Penncap-M label warns against exposing blooming plants to the pesticide, whether directly or through drift. However, the bee-kill incidents detailed in this chapter indicate that current label language and mitigation measures have not sufficiently reduced the risk of methyl parathion use to honey bees.

In spite of efforts to strengthen label language, however, it is quite possible that the risks of methyl parathion exposure to bees cannot be mitigated below levels of concern. **However, the voluntary cancellation of tree fruit uses of methyl parathion will significantly reduce the overall risk of methyl parathion to bees by eliminating uses that have been associated with a large number of bee kill incidents.**

Recommendation for Endocrine Disruption Testing

Methyl parathion has been observed in the open literature to display metabolic effects which hinder successful reproduction and/or sexual development in birds, mammals, and fish. The observations included the following:

1. Damage to oocytes in fish (Rastogi and Kulrestha, 1990)

2. Interference with spermatogenesis in rats (Zlateva and Moleva, 1976)

3. Decreased testes weight and function in birds (Maitra and Sarkar, 1996)

4. Serum and pituitary gland gonadotropin hormone decreases in fish (Ghosh, et al., 1990)

5. Interference with glucose metabolism in rats, snails, prawns, and birds. (Lukaszewicz-Hussain, et al., 1985, Reddy and Rao, 1991 and Rambabu and Rao, 1994.)

6. Elicitation of strong estrogenic response in liver hepatocyte cells, possibly due to a metabolite (Petit, F., et al., 1997)

7. Disruption of eggshell formation in birds (Bennett and Bennett, 1990).

The amendments to the FQPA and the Safe Drinking Water Act (SDWA) mandate or support the development of a screening program that will determine whether pesticides and certain drinking water source contaminants "may have an effect in humans that is similar to an effect produced by a naturally-occurring estrogen, or other such endocrine effect as the Administrator may designate." Very early in its deliberations, EPA's Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) determined that there was both a strong scientific basis and feasibility, considering time and resource constraints, to expand the scope of the screening program to include the androgen- and thyroid hormone systems, and to include evaluations of the potential impact on wildlife as well as on human health. EPA agrees and is developing a screening program which incorporates these modifications.

Based on the adverse results observed in the above data, EFED recommends that when current protocols under consideration by the Agency's Endocrine Disruptor Screening Program (EDSP), methyl parathion be subjected to more definitive testing to better characterize endocrine disrupting effects.

Persistence of Toxicity

Risks from Methyl Parathion and Other Pesticides Due to Simultaneous and Sequential Applications

The concern attached to the use of methyl parathion is compounded by uses of other organophosphates, which share a common mode of action (cholinesterase inhibition). Under FQPA the risk posed by different pesticides with the same mode of action must be considered together. The EC combination with ethyl parathion is the most obvious example. Ethyl parathion is used extensively on cotton and on other crops on which methyl parathion is used. To the extent that different OPs are used in tank mixes, or in the same area as methyl parathion, the risk is compounded. EFED is currently working on REDs for other OPs which may be applied simultaneously with methyl parathion.

References

Alexander, M. 1994. Biiodegradation and Bioremediation. Academic (NewYork).

Anderson, J. and Glowa, W., 1984. Insecticidal poisoning of honey bees in Connecticut. Environmental Ent. v. 13. 5 p. 160952

Anderson, J.F. and Wojtas, M.A., 1986. Honey bees (Hymnoptera: Apidae) contaminated with pesticides and polychlorinated biphenyls. J. Econ. Ent. 79: 1200-1205.

Atkins, E.L., Kellum, D.,and Atkins, K.W. 1978. Encapsulated Methyl Parathion Formulation is Highly Hazardous to Honey Bees. American Bee Journal. pp. 483-485 (MRID No.: 44552702)

Atkins, E.L., 1981. *Can beekeepers live with pesticides? Can we live with microencapsulated insecticides?* From talk presented at 37th Annual Convention of American Bee Federation, January, 1981, Seattle WA.

Bailey, H. (1983) Chronic Toxicity of Penncap M to Daphnia magna:SRI Project LSC-4652. Rev. final rept. (Unpublished study received Jun 30, 1983 under 4581-293; prepared by SRI International, submitted by Agchem Div., Pennwalt Corp., Philadelphia, PA; CDL:250628-A) 128790

Bailey, H. (1983) Chronic Toxicity of Penncap M to Rainbow Trout (Salmo gairdnerii): SRI Project LSC-4652. Rev. final rept.(Unpublished study received Jun 30, 1983 under 4581-293; prepared by SRI International, submitted by Agchem Div., Pennwalt Corp., Philadelphia, PA; CDL:250628-B) 128791

Ball, I.J., Gilmer, D.S., Cowardin, L.M. and Riechmann, J.H., 1975. Survival of wood duck and mallard broods in north central Minnesota. J. Wildlife Management 39: 776-780.

Barbash, J.E. and Resek, E.A., 1996. Pesticides in Ground Water. Chelsea, MI: Ann Arbor Press, 584 pp.

Beavers, J.; Corbitt, A.; Jaber, M. (1988) Methyl Parathion: A One-generation Reproduction Study with the Bobwhite (*Colinus virginianus*): Laboratory Project No. 232-108. Unpublished study prepared by Wildlife International Ltd. 96 p. 41179302

Bennett, J.K. and R.S. Bennett. 1990. Effects of dietary methyl parathion on northern bobwhite egg production and eggshell quality. Env. Tox. Chem. 9: 1481-1485. MRID 44371710.

Bennett, R.S. et al. 1990. Effects of the Duration and Timing of Dietary Methyl Parathion Exposure on Bobwhite Reproduction. Environmental Toxicology and Chemistry, Vol. 9, pp.

1473-1480(MRID No.:44371608)

Bennett, R.S. and B. A. Williams. 1991. Effects of Dietary Exposure to Methyl Parathion on Egg Laying and Incubation in Mallards. Environmental Toxicology and Chemistry, Vol 10, pp.501-507.(MRID No.:44371602)

Brewer, L.W. et al.1988. Effects of Methyl Parathion in Ducks and Duck broods, Environmental Toxicology and Chemistry, Vol. 7., pp. 375-379. (MRID No.:44371604)

Brown, A. W. A. 1978. Ecology of Pesticides. Wiley-Interscience.6

Brown, P.W. and Hunter, Jr., M.L., 1984/85. Potential effects of insecticides on the survival of dabbling duck broods. J. Minnesota Academy of Science 50: 41-44.

Brooks, H.L. et al. 1973. Insecticides, Cooperative Extension Service, Kansas State University, Manhattan, Kansas.

Buerger, T.T. 1991. Effects of Methyl parathion on Northern Bobwhite Survivability. Environmental Toxicology and Chemistry, Vol. 10,527-532. (MRID No.: 44371606)

Buerger, T.T. et al. 1994. Metabolism and Acute Toxicity of Methyl Parathion in Pen-reared and Wild Northern Bobwhites. Environmental Toxicology and Chemistry, Vol. 13, No. 7, pp.1139-1143. (MRID No. 44342701)

Burgett, M. and G.C. Fisher. 1980. Recovery of Penncap-M from Foraging Honey Bees and Pollen Storage Cells. Environ. Entomo. 9:430-431.(MRID No.:44552704)

California Department of Environmental Protection, Department of Pesticide Regulation, 1995. "1995 Rice Pesticides Program".

California Department of Environmental Protection, Department of Pesticide Regulation, 1997. "Department of Pesticide Regulation Information on Rice Pesticides Submitted to the Central Valley Regional Water Quality Control Board". 32 p.

Chakraborty, P.S., Mallik, A., Dingal, D.K. and Banerjee, S., 1989. *Effect of methyl parathion on brain and olfactory organ cholinesterase activity of the fish* heteropneustes fossilis. Environment and Ecology **7(2):** 310-314.

Christensen, G.C. 1971. The Effects of Parathion Upon Pheasant Populations in Smith and Mason valleys, Nevada. Presented at the Fifty-First Annual Conference of the Western And Midwest Fish and Game Comissioners. (MRID No.: 44342001)

Cornacchia, J., Cohen, D., Bowes, G, Schnagl, R. and Montoya, B, 1984. *Rice herbicides:*

Molinate (Ordam) and thiobencarb (Bolero). Special Projects Report 84-45P. California State Water Resources Control Board, Sacramento, CA.

Creger, T., 1998. Letter from Nebraska Department of Agriculture. Subject: Local Experiences with Methyl Parathion and Bee Mortality. April 28.

Crossland, N.O., 1983. *Fate and biological effects of methyl parathion in outdoor ponds and laboratory aquaria.* Ecotox. And Env. Safety **8**, 482-495.

Crossland, N. (1984) Fate and biological effects of methyl parathion in outdoor ponds and laboratory aquaria: II effects. Ecotoxicology and Environmental Safety 8:482-495. 44371714

Crossland, N. (1988) A method for evaluating effects of toxic chemicals on the productivity of freshwater ecosystems. Ecotoxicology and Environmental Safety 16:279-292. 44371712

Driver, C.J. et al. 1991. Environmental Toxicology: routes of Uptake and their Relative contribution to the toxicologic Response of Northern Bobwhite (*Colinus virginianus*) to an Organophosphate Pesticide. Environmental Toxicology and Chemistry, Vol. 10, pp.21-33 (MRID No.: 44357804)

EFED, 1996. Draft Corn Insecticide Cluster Analysis.

Eisler, R., 1970. *Factors affecting pesticide-induced toxicity in an estuarine fish.* United States Department of the Interior Technical Papers of the Bureau of Sport Fisheries and Wildlife no. 45. 19 pp.

Edwards, W.R. and R.R. Graber. 1968. Responses of Avians to Methyl Parathion in a Hayfield. 20TH Ill. Custom Spray Operators Training School -Summaries of Presentations, Urbana, Ill. Natural History Survey. (MRID No.: 00090488)

Fairbrother, A. et al. 1988. Changes in Mallard Hen and Brood Behaviors in Response to Methyl Parathion induced Illness of Ducklings. Short communicaton. Environmental Toxicology and Chemistry, Vol. 7, pp. 499-503 (MRID No.: 44371601)

Fernandez-Casalderrey, , A., Ferrando, M.D. and Andreu-Moliner, E., 1995. *Chronic toxicity of methylparathion to* daphnia magna: *Effects on survival, reproduction, and growth.* Bull. Environ. Contam. Toxicol. **54**: 43-49.

Dr. M.E. "Butch" Ferree, Professor, University of Georgia Department of Horticulture, personal communication.

Fink, R.; Beavers, J.B.; Brown, R.; et al. (1980) Final Report:Acute Dermal LD50--Bobwhite Quail: Project No. 110-124. (Unpublished study received Dec 4, 1980 under 4581-292; prepared

74 of 82

by Wildlife International Ltd. and Washington College, submitted by Pennwalt Corp., Philadelphia, Pa.; CDL:244308-O) 00071200

Fink, R.; Beavers, J.B.; Brown, R.; et al. (1980) Final Report:Acute Dermal LD50--Bobwhite Quail: Project No. 110-123. (Unpublished study received Dec 4, 1980 under 4581-292; prepared by Wildlife International Ltd. and Washington College, submitted by Pennwalt Corp., Philadelphia, Pa.; CDL:244308-N) 00083103

Finlayson, B.J. and Faggella, G., 1986. *Comparison of laboratory and field observations of fish exposed to the herbicides molinate and thiobencarb.* Trans. Am. Fish Soc. **115:** 882-890.

Finlayson, B.J., Harrington, J.A., Fujimura, R. and Isaac, G., 1993. *Identification of methyl parathion toxicity in Colusa Basin Drain water.* Env. Toxicol. And Chem. **12:** 291-303.

Fite, E. 1995. Draft of Mammalian Risk Assessments. EEB/EFED/U.S.EPA.

Fletcher, J.S., Nellessen, J.E., and Pfleeger, T.G. 1994. Literature Review and Evaluation of the EPA Food-chain (Kenaga) Nomogram, An Instrument for Estimating Pesticide Residues on Plants, Environmental Toxicology and Chemistry, Vol. 13, No.9, pp. 1383-1391.

Gianessi, L.P., 1997. The Uses and Benefits of Organophosphate and Carbamate Insecticides in U.S. Crop Production. Washington, D.C.:National Center for Food and Agricultural Policy, 48 pp.

Gianessi, L.P. and Marcelli, M.B., 1998 Draft: The Use of Organophosphate Insecticides in U.S. Crop Production- Information Sheets.

Ghosh, P., Bhattacharya, S. and Bhattacharya, S, 1990. Impairment of the regulation of gonadal function in *Channa punctatus* by metacid-50 and carbaryl under laboratory and field conditions. Biomed. And Environ. Sci. 3:106-112.

Goolsby, D.A. and Battaglin, W.A., 1993. Occurrence, distribution and transport of agricultural chemicals in surface waters of the midwestern United States. In Goolsby, D.A., Boyer, L.L. and Mallard, G.E., compilers. Selected papers on agricultural chemicals in water resources of the midcontinental United States: U.S. Geological Survey Open-File Report 93-418, pp. 1-25.

Grue, C.E. 1982. Response of Common Grackle to Dietary Concentrations for Four Organophosphate Pesticides. Arch. Environm. Contam. Toxicol. 11, 617-626. (MRID No.:00131892)

Grue, C.E. 1988. Agricultural Chemicals and the Quality of Prairie-pothole Wetlands for Adult and Juvenile Waterfowl - What are the concerns? Pages 55-64 in P.J. Stuber (Coord.) Proceedings National Symposium of Protection of Wetland from Agricultural Impacts. USDI, Fish and Wildlife Service Biological Report 88(16). 221 pp.(MRID No.:44357802)

Dr. Abner Hammond, Professor, Louisiana State University Department of Entomology, personal communication.

Heath, A.G., et al. 1993. Sublethal Effects of Methyl Parathion, Carbofuran, and Molinate on Larval Striped Bass. American Fisheries Society Symposium 14:17-28. (MRID No.:44378602)

Heimbach, F. (1987) Influence of Parathion-methyl on the Reproduction of Water Fleas (Daphnia magna): Lab Project No. HBF/RDM 02. Unpublished study prepared by Bayer AG. 32 p. 41506801

Henry, M.G., and G.J. Atchison. 1984. Behavorial Effects of Methyl Parathion on Social Groups of Bluegill (*Lepomis macrochirus)*. Environmental Toxicology and Chemistry, Vol.3 pp.399-408.(MRID No.:44371607)

Hill, E.F.; Heath, R.G.; Spann, J.W.; et al. (1975) Lethal Dietary Toxicities of Environmental Pollutants to Birds: Special Scientific Report--Wildlife No. 191. (U.S. Dept. of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center; unpublished report) 00022923

Reiner Honicke, San Francisco Estuary Institute, personal communication.

Hudson, R.H. et al. 1979. Acute Oral and Percutaneous Toxicity of Pesticides to Mallards: Correlations with Mammalian Toxicity Data. Toxicology and Applied Pharmacology 47, 451-460. (MRID No.:00139542)

Hudson, R.; Tucker, R.; Haegele, M. (1984) Handbook of toxicity of pesticides to wildlife: Second edition. US Fish and Wildlife Service: Resource Publication 153. 91 p. 00160000

Jarvinen, A.; Tanner, D.; Kline, E. (1988) Toxicity of Chlorpyrifos Endrin or Fenvalerate to Fathead Minnows Following Episodic or Continuous Exposure. Ecotoxicology and Environmental Safety 15:78-95. 41043903

Johansen, C.A., and Kious, C.W. 1978. Bee Poisoning characteristics of Microencapsulated Methyl Parathion. Gleaning in Bee Culture. pp. 382-385. (MRID No.: 44552706)

Johansen CA and Mayer, DF, 1990. Pollinator Protection: A Bee and Pesticide Handbook. Chesire, CT. Wicwas Press, 212 pp.

Johnson, C.R. 1978. The Effects of Sublethal Concentrations of Five Organophosphorus Insecticides on Temperature Tolerance, Reflexes and Orientation in *Gambusia affinis affinis* (Pisces:Poeciliidae) Zool. J. Linn. Soc. 64:63-70

Johnson, W.; Finley, M. (1980) Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates: Resource Publication 137. US Fish and Wildlife Service, Washington, D.C. 106 p. 40094602

Kendall, R. J. et al. 1984. The Effects of Methyl Parathion on Wild Avian Species on Wild Avian Species in Agricultural Areas of Skagit Valley, Washington. Final Report Submitted to: Corvallis Environmental Research Laboratory, United States Environmental Protection Agency. (MRID No.: 44413601)

Kendall, R. J. et al. 1985. The Effects of Methyl Parathion on Wild Avian Species on Wild Avian Species in Agricultural Areas of Skagit Valley, Washington. Final Report Submitted to: Corvallis Environmental Research Laboratory, United States Environmental Protection Agency. (MRID No.: 44413602)

Matthew Kleinhenz, crops specialist,,University of Maine, Cooperative Extension Service, Aroostook County, Presque Isle office, personal communication.

Kolpin, D.W., Goolsby, D.A. and Thurman, E.M., 1995. Pesticides in Near-Surface Aquifers: An Assessment Using Highly Sensitive Analytical Methods and Tritium. Journal of Environmental Quality, 24:6, November-December 1995, pp 1125-1132.

Korn, S.; Earnest, R. (1974) Acute toxicity of twenty insecticides to striped bass,~Morone saxatilis'~. California Fish and Game 60(3):128-131. 05000819

Krapu, G.L., 1979. Nutrition of female dabbling ducks during reproduction. In T.A. Bookhout (ed.) Waterfowl and wetlands- an integrated review. The Wildlife Society, Washington, Dc, pp 59-70.

Katherine Kuivila, United States Geological Survey-Sacramento, personal communication.
Dr. Donald LaBonte, Louisiana State Agricultural Center, personal communication.

Larson, S.J., Capel, P.D. and Majewski, M.S., 1997. Pesticides in Surface Waters. Chelsea, MI: Ann Arbor Press, 372 pp.

Ludke, J.; Hill, E.; Dieter, M. (1975) Cholinesterase (ChE) response and related mortality among birds Fed ChE inhibitors.Archives of Environmental Contamination and Toxicology 3(1):1-21. 44341101

Lukaszewicz-Hussain, A., Moniuszko-Jokaniuk, J. and Pawlowska, D., 1985. Blood glucose and insulin concentration in rats subjected to physical exercise in acute poisoning with parathion-methyl. Pol. J. Pharmacol. Pharm.. 37: 647-651.

Dr. Daniel Mahr, Professor, University of Wisconsin-Madison Department of Entomology, personal communication.

Maitra, S.K. and Sarkar, R., 1996. Influence of methyl parathion on gametogenic and acetylcholinesterase activity in the testis of whitethroated munia *(Lonchura malabrica)*. Arch. Environ. Toxicol. 30: 384-389.

Majewski, M.S. and Capel, P.D., 1995. Pesticides in the Atmosphere. Ann Arbor, MI: Ann Arbor Press

Marshall Lee, Associate Environmental Research Scientist, California Department of Environmental Protection, Department of Pesticide Regulation, personal communication.

Martin, A.C., Zim, H.S. and Nelson, A. L., 1951. American Wildlife and Plants. New York: Dover Publications.

Mason, C.E., 1986. Progression of knockdown and mortality of honey bees (Hymnoptera: Apidae) sprayed with insecticides mixed with Penncap-M. Env. Entomol. 15: 170-176.

Matsumura, F. 1975. Toxicology of Insecticides. Plenum Press (NewYork).

Mayer, D.F., Johansen, C.A., and Baird, C.R., 1996. How to Reduce Bee Poisoning from Pesticides. Washington State University Cooperative Extension, WREP 15. 14 pp.

Mayer, F., 1986. Acute Toxicity Handbook of Chemicals to Estuarine Organisms: EPA/600/X-86/231. USEPA Environmental Research Laboratory, Gulf Breeze, FL. 275 pp.

McCann, J.; Teeters, W.; Urban, D. et al. (1981) A short-term dietary toxicity test on small mammals. p. 132-142 of the Second Conference of Avian and Mammalian Wildlife Toxicology, Lamb, D.; Kenaga, E. Eds.; Published in American Society for Testing and Materials, ASTM STP 757; 1981. 43961101

Meyers, S, M, 1990. Effects of Methyl Parathion on Red-winged Blackbird (*Agelaius phoeniceus*) Incubation Behavior and Nesting Success. Environmental Toxicology and Chemistry, vol. 9, pp. 807-813 (MRID No.: 44371605)

Candace Miller, Associate Environmental Research Scientist, California Department of Environmental Protection, Department of Pesticide Regulation, personal communication.

Mineau, P. 1991. Cholinesterase-inhibiting Insecticides Their Impact on Wildlife and the Environment. Chemicals in Agriculture Volume 2. Elsevier. New York.

Morgan,.D.P., 1976. Recognition and Management of Pesticide Poisonings, Fourth Ed., U.S. Environmental Protection Agency, EPA-540/9-80-005.

Morgan,.D.P., 1982. Recognition and Management of Pesticide Poisonings, 3rd edition, U.S. EPA. EPA-540/9-80-005

Morgan, M.K.; Smith, S.; Arceo, R.J. (1976) Status Report to U.S. Borax Research Corporation: First Generation of a Three-Generation Reproduction Study with USB 3153 in Albino Rats: IBT No. 623-06981. (Unpublished study received Jun 29, 1976 under 1624-EX-19; prepared by Industrial Bio-Test Laboratories, Inc., submitted by United States Borax & Chemical corp., Los Angeles, Calif.; CDL:226084-C) 140767

(NCDA) North Carolina Department of Agriculture, Food and Drug Protection Division

(NDA) Nebraska Deptment of Agriculture

Niethammer, K.R. and T.S. Baskett. 1983. Cholinesterase Inhibition of Birds Inhabiting Wheat Fields Treated with Methyl Parathion and Toxaphene. Archives of Environmental Contamination and Toxicology. 12,471-475 (MRID No.: 44357806)

O'Brien, R. D. 1967. Pesticide: Action and Metabolism. Academic Press (New York).

O'Brien, R.D. 1960. Toxic Phosphorus Ester. Academic Press (New York).

Pennwalt Corp. (1972) Pencap M--LC50 for Bobwhite Quail. (Unpublished study received Mar 6, 1973 under 3F1361; CDL:093632-I) 00102329

Petit, F., LeGoff, P., Cravedi, J-P., Valotaire, Y. and Pakdel, F., 1997. Two complementary bioassays for screening the estrogenic potency of xenobiotics: recombinant yeast for trout estrogen receptor and trout hepatocyte cultures. J. Molec. Endocrin. 19: 321-335.

Rambabu, J.P. and Rao, M. B., 1994. Effect of an Organochlorine and three organophosphate pesticides on glucose, glycogen, lipid and protein contents in tissues of the freshwater snail *Bellamya dissimilis* (Muller). Bull. Environ. Contam. Toxicol. 53: 142-148.

Rao, K.S.P., Sahib, I.K.A.. and Rao, K.V.R., 1985. *Methyl parathion (o-o-dimethyl o-4-nitrophenyl thiophosphate) effects on whole-body adnd tissue respiration in the teleost,* tilapia mossambica (Peters). Ecotox. and Env. Safety **9(3)**: 339-345.

Rastogi, A. and Kulshreshta, S.K., 1990. *Effect of sublethal doses of three pesticides on the ovary of a carp minnow* rasbora daniconius. Bull. Environ. Contam. Toxicol. **45:**742-747.

Rattner, B.A. and J.C.Franson. 1984. Methyl Parathion and Fenvalerate Toxicity in American Kestrels; acute Physiological Responses and Effects of Cold. Can. J. Physiol. Pharmacol. 62:787-792. (MRID No.:44371701)

79712

Reddy, M.S. and Rao, K.V.R., 1991. Methylparathion induced alerations in the tissue carbohydrate catabolism of marine prawn, *Metapenaeus monoceros.* Bul Envir. Contam. Toxicol. 47: 925-932.

Rhodes, H.A., Wilson, W.T., Sonnet, P.E., and Stoner, A., 1980. *Honey bees dies from pollen contaminated fourteen months earlier with Penncap-M.* American Bee Journal, August 1980. 577-589.

Rhodes, H.A. et al. 1984. Honeybees die from pollen contaminated fourteen months earlier with Penncap -M. Amer. Bee J. 120(8):577-588. MRID No.: 160948)

Ross, B. et al. 1980. Chemical Detection of Penncap-M capsules in honeybee (*Apis mellifera L.*) Collected Pollen and Methyl Parathion Analyses of Honeybees and Bee Products from Five Apiaries in the Vicinity of Cotton (*Gossypium hirsutum* L.) Fields Treated with Four Applications of Penncap-M -Yuma, Arizona, 1980. (MRID No.: 138662)

Russel, D. et al. 1998. Potential Impact of Microencapsulated Pesticides on Jersey Apiaries. American Bee Journal. pp. 207-210 (MRID No.: 44552705)

Sanchez-Martin, M.J. and Sanchez-Camazano, M., 1991. Relationship Between the Structure of Organophosphorus Pesticides and Adsorption by Soil Components. Soil Science 152(4): 283-88.

Sauer, J.R., Hines, J.E., Gough, G., Thomas, I. and Peterjohn, B.G., 1997. The North American Breeding Bird Survey Results and Analyses, Version 96.4. Patuxent Wildlife Research Center, Laurel, MD.

Shellenberger, T. 1970. Letter sent or K. Nolan dated Dec. 30, 1970: A simulated field toxicity evaluation of Cygard-630 insecticide: GSRI Project No. NC-400. (Unpublished study received on unknown date under 241-EX-49; prepared by Gulf South Research Institute, submitted by American Cyanamid Co., Princeton, NJ; (MRID No.: 00102372)

Smith, G. (1987) Pesticide Use and Toxicology in Relation to Wildlife Organophosphorus and Carbamate Compounds. Resource Publication 170: 1-173 44585416

Smith,W.T. and G. Kritsky. 1982. Preliminary Report on the Effect of Penncap-M on Honeybees. Exhibit 7 in Information Relating to the Toxicity of Penncap-M to Bees. Submitted by Pennwalt Corp., (MRID No.: 138667)

Smithson, P.C. and Sanders, O.T., 1978. *Exposure of bobwhite quail and cottontail rabbits to methyl parathion.* Proc. Ann. Conf. S.E. Assoc. Fish & Wildl. Agencies. **32**: 326-334.

Solecki, R. et al. 1996. Effects of Methyl Parathion on Reproduction in the Japanese Quail. Bull. Envir.Contam. Toxicol. 57:902-908 (MRID No.: 44359601)

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Stoner, A. et al. Penncap-M Collection by Honey Bees. American Bee Journal. pp. 154-159. (MRID No.: 44552703)

Stoner, A., Howard, A.R., and W.T.Wilson. 1979. Case histories of the Effects of Microencapsulated Methyl Parathion (Penncap-M) Applied to Fields Near Honey Bee Colonies. American Bee Journal. pp. 648-654. (MRID No.: 44552701)

Stoner, A.., et al. 1984. Microencapsulated Methyl Parathion (Penncap-M): Effect of Long-term Feeding of Low Doses in Pollen on Honeybees in Standard-size Field Colonies. Unpublished draft. Exhibit 5 in Information Relating to the Toxicity of Pencap-M to Bees. Submitted by Pennwalt Corp. (MRID No.: 138665)

Surprenant, D. (1988) Acute Toxicity of Methyl Parathion TEP (Methyl Parathion Typical End-Use Product) to Rainbow Trout (Salmo gairdneri) Under Flow-through Conditions: SLS Report No. 88-7-2772; SLS Study No. 11704.0388.6113.108. Unpublished study prepared by Springborn Life Sciences, Inc. 39 p.

Swanson, G.A., 1988. Aquatic habitats of breeding waterfowl. In The ecology and management of wetlands: Vol 1, Ecology of wetlands. D.D. Hook, W.H. McKee, Jr., H.K. Smith, J. Gregory, V.G. Burrell, Jr., M.R. DeVoe, R.E. Sojka, S. Gilbert, R. Banks, L.H. Stolzy, C. Brooks, T.D. Matthews and T.H. Shear (eds). Timber Press: Portland, Oregon, pp 195-202.

Swanson, G.A., Krapu, G.L. and Serie, J.R., 1979. Foods of laying female dabbling ducks on the breeding grounds. In Waterfowl and wetlands- an integrated review. T.A. Bookhout (ed.). The Wildlife Society, Washington, DC, pp 47-57.

Talent, L.G., Jarvis, R.L. and Krapu, G.L., 1983. Survival of mallard broods in south-central North Dakota. Condor 85: 74-78.

Thurman, E.M., Zimmerman, L.R., Scribner, E.A. and Coupe, Jr., R.H., 1998. Occurrence of Cotton Pesticides in Surface Water of the Mississippi Embayment. USGS Fact Sheet FS-022-98, 4 pp.

Tipton, A. et al. 1980. A Model of the Impact of Methyl Parathion Spraying on a Quail Population. Bull. Environm. Contam. Toxicol. 25,586-593.(MRID No.:443778603)

United States Department of the Interior, 1951. Food of Game Ducks in the United States and Canada. Research Report 30. Reprint of USDA Technical Bulletin 634-1939.

USEPA. 1975. Substitute Chemical Program Initial Scientific and Minieconomic Review of Parathion. EPA-540/1-75-001

U.S. Environmental Protection Agency, Environmental Research Laboratory, 1981. Acephate, Aldicarb, Carbophenothion, DEF, EPN, Ethoprop, Methyl Parathion, and Phorate: Their Acute and Chronic Toxicity, Bioconcentration Potential, and Persistence as Related to Marine Environments: EPA-600/4-81-023. (Unpublished study) 00066341

U.S. Environmental Protection Agency, Office of Pesticide Programs, 1992. Pesticides in Ground Water Database. EPA 734-12-92-001.

USEPA Office of Ground Water and Drinking Water, 1998. Unregulated Contaminant Monitoring List. World Wide Web page: http://www.epa.gov/ogwdw000/ ncod/unregmonold.html

U.S. Environmental Protection Agency, Office of Water. *Drinking water regulations and health advisories.* EPA 822-R-96-001. February 1996

Waller, G.D. et al. 1984. A Comparison of Honeybee Losses with Two Formulations of Methyl Parathion Applied to Sunflowers. Unpublished draft. Exhibit 3 in Information Relating to the toxicity of Penncap-M to Bees. Submitted by Pennwalt. (MRID No.:138663)

Richard Weidman, Superintendent of Peninsula Agricultural Research Station, Door Co., Wisconsin, personal communication.

White, D.H., Seginak, J.T., and Simpson, R.C., 1990. *Survival of northern bobwhites in Georgia: Cropland use and pesticides.* Bull. Environ. Contam. Toxicol. **44:** 73-80.

Dr. Mike R. Williams, Extension Entomologist, Mississippi State University Cooperative Extension, personal communication.

Willis, G. H. and L.L. McDowell. 1987. Pesticide Persistence on Foliage. Reviews of Environmental Contamination and Toxicology, Vol. 100 pp.23-74

WHO (World Health Organization, UN). 1993. Environmental Health Criteria; 145. 244 p
Vaughan, A. and R. Lee. 1981. Memorandum to EEB Staff. Subject: Review of registration actions for bee hazard. USEPA\HED\EEB

Youngman, R.R., Toscano, N.C. and Gaston, L.K., 1989. *Degradation of methyl parathion to p-Nitrophenol on cotton and lettuce leaves and its effect on plant growth.* J. Econ. Entomol. **82(5):**1317-1322.

Lisa Zimmerman, United States Geological Survey- Kansas District (volunteer), personal communication.

Zlateva, M. and Moleva, E., 1976. Morphological changes in the testis of white rats under the conditions of chronic action of Vofatox 50. Eksper. Med. I Morf. 14(3): 127-132.

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