Public Information and Records Integrity Branch (PIRIB) (7502C)
Office of Pesticide Programs
Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20461-0001
Attn: Docket ID Number OPP-2003-0237

13 August 2004

RE: Interim Reregistration Eligibility Decision for methyl parathion Docket ID# OPP-2003-0237

Dear Office of Pesticide Programs:

Thank you for the opportunity to comment on the Interim Reregistration Eligibility Decision (IRED) for methyl parathion and for allowing us additional time to finalize our comments on this important issue.

EPA's Decision

According to the document, EPA has assessed the risks of methyl parathion and reached in Interim Reregistration Eligibility Decision for this organophosphate pesticide and has concluded that "Provided that risk mitigation measures are adopted, methyl parathion fits into its own risk cup – its individual, aggregate risks are within acceptable levels." EPA also "determined that products containing methyl parathion are eligible for reregistration except for use on the following crops: cabbage, dried beans, dried peas, hops, lentils, pecans and sugar beets" and "that the use of eligible methyl parathion products in accordance with labeling specified in this IRED will not pose unreasonable adverse effects to humans or the environment."

Introductory Comments and Our Position

We have carefully reviewed the IRED and related documents and are extremely disappointed with EPA's decision to allow continued registration of methyl parathion. We strongly disagree with the EPA's decision on this important issue and simply cannot believe that the EPA is completely ignoring its own risk assessment for methyl parathion. Methyl parathion, which was first used in the United State fifty one years ago, is one of the most toxic chemicals available for agricultural use today. EPA has been aware of its extreme toxicity since at least 1978 when it determined that methyl parathion was so hazardous to humans and birds that it was designated as a "restricted use" only pesticide. More recently, in late 1998, the EPA conducted a "preliminary" risk assessment of methyl parathion and concluded that it posed an "unacceptable risk" as currently used. Furthermore, a similar compound, ethyl parathion, was cancelled by EPA in October 2000 (used until October 2003).

Despite EPA's awareness of the toxicity of methyl parathion and the cancellation of a closely related compound, EPA has allowed for its continued use and it remains one of the most widely used insecticides in the U.S., with an estimated annual use of 2-4 million pounds of active ingredient annually (Donaldson et al. 2002). It is inconceivable that in 2004, the EPA is proposing to approve its continued use on at least 20 crops, including on such major acreage crops as corn, cotton, wheat, soybeans and rice. Granted, in 1999, the EPA and the registrants developed some mitigation measures, including the cancellation of uses of certain fruits and vegetables consumed frequently by children, and modification of product labels in an attempt to increase worker protection but overall, these cancellations made up only a miniscule fraction of the total acreage in which methyl parathion is used in the U.S.

In effect, EPA will allow methyl parathion to be continued to be used on a vast majority of the acreage that it was being used on prior to the 1999 action with the addition of a few weak mitigation measures. We question EPA's rationale for such a decision and believe that the agency is failing miserably to protect humans and the environment from the harmful impacts of this deadly compound. The EPA failed to initiate a phase out of methyl parathion in 1999 and we are extremely disappointed that the agency has missed yet another opportunity in 2004 to cancel all uses of this outdated and overly hazardous chemical.

Specific Issues:

1) The risk assessments are flawed and fail to protect human health

The risk assessments (human, occupational, ecological) presented in this IRED completely fail to accurately quantify or even realistically consider the real health implications to humans and the environment from the continued registration of methyl parathion. Further, the mitigation measures proposed by this document generally are weak and will prove to be ineffective towards decreasing the overall hazard presented by methyl parathion in the environment. For example, the ecological risk assessment found that methyl parathion exceeds the Agency's level of concern (LOC) for all aquatic and terrestrial species considered. This indicates a significantly hazardous pesticide, yet on page 9, the document proposes that, to address ecological risks, the EPA will lower the maximum number of applications for several crops. Another example on page 9, where the EPA states that, as a further means of reducing human and environmental risks, many uses of methyl parathion were cancelled in 1999. Many food and non-food uses were in fact deleted in 1999; however, one look at the lists of these uses and it is obvious that all of these uses were relatively minor. So, there may be many less registered uses; however, the total acreage in which methyl parathion is being applied has not been significantly decreased. Overall, these "mitigation measures" are such minimal, token measures that they will do virtually nothing to address human and ecological risks posed by methyl parathion. There are many other examples (too numerous to list) of this contained in the document.

2) Methyl parathion has been banned around the world

Methyl parathion is so highly toxic and hazardous to humans, wildlife and the environment that

it was one of 27 chemicals included in the initial list of hazardous pesticides listed in the Rotterdam Convention on Prior Informed Consent Treaty, which requires exporters of these hazardous chemicals to notify importing countries that they are on this list of hazardous chemicals. The Director General of the United Nation's Food and Agriculture Organization, Jacques Diouf, referred to pesticides on this list as "inappropriate pesticides" because they pose such great hazard to developing countries that supposedly don't know any better. Well, we supposedly do know better, so why is it that the federal agency that we trust to protect our environment continues to register this inappropriate pesticide in this country? Of the 22 pesticides on the initial list, methyl parathion, lindane and methamidophos are all still registered in the United States.

Further, many countries around the world (at least 27 that we are aware of) have banned the use of methyl parathion outright (or never allowed its use in the first place), including developing countries such as Indonesia, Sri Lanka, and Tanzania, as well as developed nations such as Canada, the United Kingdom, and Denmark. Many countries, such as Iraq, United Arab Emirates, Ecuador, Peru, Jamaica, Trinidad and Tobago, and many of the countries in the European Union, Asia, and Africa, as well as Canada are countries listed as "no consent to import," meaning that they refuse to import (use) methyl parathion (PANNA 2004). We find it incredibly ironic that Denmark, the country that is home to Cheminova, the largest manufacturer of methyl parathion sold in the United States, has banned the use of methyl parathion in their own country because it is too hazardous.

3) Persistence

The EPA contends that methyl parathion has a short environmental persistence. Our review of the scientific literature found that the half-life for methyl parathion in aquatic media is 175 days and its half-life in soil is anywhere from 10 days to two months (PAN-UK 1998). These half-lives are not "short" and result in what we believe to be significant opportunities for exposure in humans, fish and wildlife and the environment given that the acute toxicity of methyl parathion is so high, its uptake is so efficient (oral, dermal, and inhalation or any combination thereof) and that in many instances it only takes one exposure to kill an organism. All factors that the EPA has chosen to accurately assess.

In addition, EPA also ignores the fact that methyl parathion breaks down in the environment and the initial degradation (and metabolic) product is methyl paraoxon, a compound that is considered at least three orders of magnitude **MORE TOXIC** than the parent compound methyl parathion due to its incredible cholinesterase-inhibiting abilities. The argument that methyl parathion is not persistent in the environment used by EPA to rationalize continued use of methyl parathion is incorrect because it fails to consider the continued toxicity of any of its degradation/metabolic products, including methyl paraoxon, its most toxic degradation/metabolic product.

Methyl parathion is widely used throughout the United States, applied to at least 5 million acres each year. Because it is aerially applied to many of these acres, drift can be significant. Further, methyl parathion is relatively mobile to mobile in soil and therefore runoff and leaching are potential routes of dissipation as well. Thus, substantially more than 5 million acres each year are (and would

continue to be) impacted by methyl parathion. Not surprisingly, methyl parathion and its degradation products are found in groundwater, surface waters, precipitation, fog, and air in the United States.

4) High toxicity concerns

Methyl parathion has high acute, chronic, and sublethal toxicity by all routes of exposure (oral, dermal, inhalation), is readily taken up through all routes of exposure (oral, dermal, inhalation), and although not known as a carcinogen, is a probable mutagen and teratogen, and is embryotoxic (Tanimura et al. 1967). Human fatalities have been caused through all three routes of exposure. The World Health Organization (WHO) classifies methyl parathion as an "extremely hazardous" pesticide. Currently, there are 15 products registered by the EPA with methyl parathion as the active ingredient (PANNA 2004). Of note is that fact that 14 of the 15 products are listed as Toxicity Class 1, which is classified in the "Danger" category. Clearly, this organophosphorus insecticide is so highly toxic in so many ways (acute, chronic, sublethal) that its continued use poses a significant hazard to any human or any other animal with a nervous system unfortunate enough to be exposed to it.

Ecological/Wildlife Impacts

Lethal Impacts

In birds, a number of mass mortality events involving numerous avian species have been documented in the scientific literature. But even more troubling is the fact that **methyl parathion kills birds even at recommended label application rates!** This is very troubling to us and we would really like to see the EPA act to put an end to allowing pesticides to be used whose recommended label application rates routinely cause mortality and morbidity in fish and wildlife species as well as in humans. According to Mineau (2001), rates of methyl parathion use as low as 100-160 g a.i./ha (far below any registered use level) already carry a 1 in 10 chance of avian mortality. However, when you consider application rates in current use, the probability of avian mortality is likely in excess of fifty percent. **This exceedingly high probability of avian mortality with current application rates of methyl parathion is totally unacceptable and the estimated 80-90 million other people in the United States who count themselves in the ranks of "birders."**

On page 49 in the document, it is noted that there are relatively few bird and fish kill incidents that are strongly linked to methyl parathion use. The discussion continues with speculation on why there might be so few incident reports. First, we would remind the EPA that absence of evidence does not equate to evidence that there are no bird or fish (or other vertebrate) kills. Second, we wonder if the EPA made the effort to check any other ecological/wildlife mortality databases besides their own EIIS database? Just because there are relatively few incident reports for methyl parathion in EPA's EIIS database does not then mean that methyl parathion is not hazardous to everything in the environment that is exposed to it. There may be relatively few incident reports in

the EPA's EIIS database; however, there are many other databases for incident reports, at the state, regional, national and international levels, and we question whether or not any of these were ever consulted by the EPA. Third, after having conducted numerous searches of ecological/wildlife mortality databases ourselves, we suspect that some of the incidents listed for "parathion" may in fact be methyl parathion. There has been some confusion between the two parathion compounds, ethyl and methyl, over time, and this confusion may have translated into misclassification in the ecological/wildlife mortality databases.

Methyl parathion has been implicated in mass mortality events on at least four of the six continents in which it is used, including North America, South America, Africa, and Europe. In 1992, a massive avian mortality event occurred in Costa Rica following the aerial application of methyl parathion to a cotton field (PAN-UK 1998). Methyl parathion is also implicated in deaths of waterfowl in Spain, and the deaths of fish, birds, cattle, and wildlife in Sudan (PAN-UK 1998). Finally, parathion (methyl and/or ethyl) is commonly used in other countries (e.g., Venezuela) as an avicide to intentionally kill songbirds. The work of Basili and Temple (1999) has documented huge mass mortality events involving dickcissels (*Spiza americana*) that flock in agricultural areas. These international mortality incidents cannot be ignored and must be included in any ecological risk assessment of methyl parathion.

Finally, it should be further noted that methyl parathion is at least as effective on the many beneficial invertebrate species as it is on target pests, so every time methyl parathion is applied to a field, it causes a mass mortality event for beneficial invertebrate species in and around the sprayed area. This likely includes honey bees (*Apis mellifera*) and other pollinators, which continue to decline in the U.S. with our continued heavy reliance on organophosphorus insecticides. Extensive field incident data for almost 30 years indicate that methyl parathion poses a very high risk to bees and that bee mass mortalities continue to occur following application of both EC and microencapsulated (ME) formulations. The LD_{50}s for each formulation (0.111 µg/bee and 0.214 µg/bee, respectively) indicate very high toxicity to bees. The ecosystem service that bees and other pollinators provide to humans is invaluable and many of our most important food crops require pollination by bees and other organisms that continue to decline in the face of our continued heavy reliance on pesticides.

Sublethal Impacts

Numerous sublethal effects in an array of wildlife species have been documented for methyl parathion exposure. Of particular concern to us is the risk that methyl parathion poses to vertebrate species. In amphibians, Johnson and Prine (1976) found that exposure to only 25 µg/l methyl parathion over 24 hrs caused juvenile western toads (*Bufo boreas*) to lose their tolerance to temperature, a sublethal effect that likely becomes lethal rather rapidly. Methyl parathion (Folidol M50 formulation) contained in simulated rice paddy overflow water was found to cause mortality in giant toads (*Bufo marinus*) at levels as low as 280 µg/l (Calumpang et al. 1997). In another study, frogs exposed to 1.0 mg/l methyl parathion suffered changes in the composition of their connective tissue matrices, which resulted in malformations of the spinal column (scoliosis) and/or limbs (short and think long bones with the epiphyses grossly twisted; Alvarez et al. 1995). Further, a study by

Fleming et al. (1982) showed that parathion actually bioaccumulated in cricket frogs (*Acris crepitans*), thereby posing a serious secondary poisoning hazard to American kestrels (*Falco sparverius*). Studies by Hall and Kolbe (1980) and Hall (1990) further document bioaccumulation of parathion and other organophosphorus insecticides in amphibians, which is cause for great concern, particularly for amphibian predators that are likely to die from secondary poisoning.

In addition to the many different impacts on the nervous (target) system, methyl parathion is listed by the Illinois EPA as a probable endocrine disruptor, and by Benbrook (1996) as an endocrine disruptor. Further, it is a known immunotoxicant in mammals (Street and Sharma 1975). Both methyl parathion and its closely related chemical analog ethyl parathion have been found to cause reproductive, physiological, biochemical, and behavioral effects that ultimately lead to premature mortality in exposed organisms. In birds, reproductive effects such as decreased productivity (% eggs hatching/% nests successful) have been documented in songbirds exposed to parathion (ethyl/methyl). Physiological effects such as reduced daily food consumption (anorexia) and decreased body temperature (hypothermia) have been documented in songbirds and upland game birds exposed to parathion. Behavioral effects may be the most insidious and damaging of the sublethal effects. Decreased ability to avoid predators, decreased nest attentiveness during incubation and maternal care following hatching, and increased brood abandonment by females have been documented in upland gamebirds, gulls, and waterfowl exposed to parathion (King et al. 1984, White et al. 1979, 1983). Research from studies conducted with other OP insecticides suggests that many other debilitating sublethal effects may occur following exposure to methyl or ethyl parathion. As sublethal effects are generally difficult to document in the field, there is great concern for continued use of a pesticide such as methyl parathion that has so many documented cases of lethal and sublethal poisonings, both experimentally and in the field.

In mammals, exposure to the OP insecticide methyl parathion has been found to result in loss of motor coordination in bats (Clark 1986), reduced predator escape response (Galindo et al. 1985), altered hearing ability in squirrel monkeys (Reischl et al. 1975), a decreased ability to learn (Reiter et al. 1973), and suppression of the immune system (Street and Sharma 1975). In rats, Zhu et al. (2001) found that repeated dermal exposures of as little as 1 mg/kg/day methyl parathion resulted in sustained inhibition of cholinesterase activity and impairment of both motor function and memory. These subtle yet insidious sublethal effects are never monitored in wildlife species, so they almost always go undetected unless morbid animals are found by accident. It seems that we often miss these types of sublethal effects in humans even when we are looking hard for them, so it is clear that we are barely seeing the tip of the iceberg when it comes to negative impacts of methyl parathion on ourselves and our environment.

6) Impacts to Endangered Species

On page 49 of the document, it states that "the endangered species LOCs are exceeded for acute and chronic risks to birds, mammals, and freshwater and estuarine/marine invertebrates, fish, amphibians, reptiles and terrestrial invertebrates (including insects)." Therefore, it is clear that methyl parathion has the real potential for harming any listed species that is exposed. We are

extremely concerned about the discussion on page 49 regarding the Section 7 consultations under the ESA that the agency is engaged in at present. These consultations are used as justification for the agency's overall environmental effects mitigation strategy. It is our understanding that the ESA counterpart regulations are now in place, and we want to know if the EPA will be completing its consultations as stated in this document. Failure to complete these consultations negates the agency's overall environmental effects mitigation strategy and will lead to a lack of longterm protection measures to reduce the likelihood that endangered and threatened species may be exposed to methyl parathion at levels of concern.

Furthermore, EPA's own evaluations on threatened and endangered species are not sufficiently considered in the Interim Reregistration Eligibility Decision (IRED) for methyl parathion . The April 1, 2004 Methyl Parathion Analysis of Risk to Threatened and Endangered Salmon and Steelhead notes specific pesticide uses that are linked with may effect determinations yet, these uses are not mitigated in the IRED. For example, the Evolutionary Significant Unit for the Upper Willamette River steelhead has the potential for approximately 250,000 pounds of methyl parathion use in spawning, rearing and migratory areas, predominately on alfalfa and hay (Daughtry and Turner, 2004). Yet, neither of these two uses has been restricted, nor have other measures been suggested to eliminate potential for harm to this species listed under the Endangered Species Act.

7) Resistance

Methyl parathion was first registered in the U.S. in 1954 and has been used fairly consistently on many of the same crops and same locations for the past 51 years. As a result, many pest species that methyl parathion is used to protect against have evolved a genetic resistance to methyl parathion. For example, this has been clearly documented by Parimi et al. (2003) for the western corn rootworm (*Diabrotica virgifera virgifera*) in the corn belt of the midwestern U.S. The time span of 51 years is more than enough time for **all** pest species targeted by methyl parathion to have evolved genetic resistance to it. Further, there is evidence suggesting that, as seen with organochlorine insecticides, there is cross-resistance with the organophosphorus insecticides as well, so this only compounds the resistance problem.

8) Costs to Humans

Methyl parathion has resulted in excessive costs to society – since 1994, in excess of 6,000 homes and businesses have been contaminated with methyl parathion in MS, LA, TN, AR, IL, OH, MI, and TX. Through their Superfund program, the EPA has been decontaminating homes and businesses to habitable condition at a response cost in excess of $65,000,000 As a result of misuse alone, the Agency has had to work extensively with the registrants, come up with a new, more aggressive enforcement strategy, and develop a national strategy for outreach to deal with the problems that methyl parathion has caused. The costs for all of this work over a period of years are likely staggering as well. Lastly, the affected individuals have had to bear the costs associated with their exposure to methyl parathion exposure. Such costs include funeral bills, hospital bills, lost

work hours, residual health effects, court/legal fees, etc. These costs are real and they are completely unnecessary. There is absolutely no reason that society should have to shoulder costs such as these for keeping this badly outdated product on the market.

9) Hazards to farmworkers and their families

From what is known about the hazards of methyl parathion to humans, particularly developing embryos, fetuses, infants, and children, continued registration of methyl parathion poses unacceptable risks. After reviewing the IRED, we feel that the Margins of Exposure (MOEs) are unacceptably low, that the magnitude of the health risks to handlers of methyl parathion must take into account the fact that handlers are exposed to other organophosphorus insecticides as well as methyl parathion in any given 30-day period, that the EPA improperly interpreted the incident data, that the EPA underestimates worker's exposure to methyl parathion by relying on faulty assumptions, that EPA should add an additional margin of safety to protect farmworker embryos, fetuses, infants, and children, and that the EPA underestimates the health risks from methyl parathion by failing to consider residential exposures (i.e., farmworkers bringing methyl parathion and other organophosphorus insecticides back home with them in their clothes and other items). When all of the potential exposure and possible effects are fully taken into account, it is clear to us that the unacceptable risks to farmworkers and their families far outweigh any potential benefit to growers, who have a number of less hazardous alternative products that are available to them.

10) EPA has inappropriately assessed the benefits of continued use of methyl parathion

On page 4 of the document, it says "Based on the use cancellation on tree fruits and vegetables, and considering the implementation of mitigation measures discussed above, the Agency has determined that pesticides containing methyl parathion generally will still present risk to humans and the environment. But there are significant benefits associated with the remaining uses which balance this risk." What exactly are these benefits and how did the EPA determine them? We contend that given the extreme toxicity of this compound that the risks posed by continued registration of methyl parathion far outweigh any perceived "benefits", particularly in light of the fact that there are much less hazardous alternatives to methyl parathion.

Conclusions

The current methyl parathion IRED, as well as the mitigation measures proposed within, are grossly insufficient. Further, it is clear to us that the EPA is refusing to recognize their very own risk assessment for methyl parathion that stated that methyl parathion posed unreasonable risks to humans and the environment. The risk assessments (human, occupational, ecological) presented in this IRED completely fail to accurately quantify or even realistically consider the real health implications to humans, fish and wildlife species, and the environment from the continued registration of methyl parathion. Further, the mitigation measures proposed to deal with the human

and environmental risks are generally weak and will not come close to being effective in mitigating the unacceptable adverse effects posed by continued registration of methyl parathion. We fail to see how the IRED will protect humans and the environment from this significantly hazardous pesticide, as there is no level of "benefits" that could ever come close to balancing out the unacceptable adverse effects posed by continued registration of methyl parathion. It is our assertion that the EPA should have taken the opportunity that they had in 1999 to cancel further uses of methyl parathion. We therefore request that the EPA rework their IRED for methyl parathion so that it cancels all current uses of methyl parathion. Thank you for the opportunity to provide comments on this document and on this very important issue in general.

Respectfully submitted,

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