March 31, 2010

Lisa P. Jackson, Administrator
United States Environmental Protection Agency
Ariel Rios Building
1200 Pennsylvania Avenue, NW
Mail Code 1101A
Washington, DC 20460

Re: Petition to Suspend and Cancel All Registrations for the Soil Fumigant Iodomethane (Methyl Iodide)

Dear Ms. Jackson:

On behalf of Pesticide Action Network North America, Pesticide Watch, Californians for Pesticide Reform, Farmworkers Association of Florida, Toxic Free North Carolina, Oregon Toxics Alliance, California Rural Legal Assistance Foundation, Migrant Clinicians Network, Pineros y Campesinos Unidos del Noroeste, Farmworker Justice, and the United Farm Workers (collectively, “Petitioners”), we are writing to petition the United States Environmental Protection Agency (“EPA”) to exercise its authority under Section 6 of the Federal Insecticide Fungicide and Rodenticide Act (“FIFRA”), 7 U.S.C. § 136d, to suspend and cancel all registrations for the pesticide iodomethane. This petition is filed pursuant the Administrative Procedure Act, 5 U.S.C. § 551 et seq.

INTRODUCTION

On January 6, 2006, EPA proposed designating iodomethane, or methyl iodide, as a non-food use pesticide when used as a pre-plant soil fumigant in agricultural fields to be planted with tomatoes, strawberries, peppers, certain ornamentals, turf, and replanted trees and vines. On October 5, 2007, EPA approved a time-limited, one-year conditional registration of methyl iodide. On September 29, 2008, EPA issued a new registration notice converting the time-limited conditional registration to a time-unlimited conditional registration, leaving as conditions only the requirements that the registrant provide a product training/stewardship program and that it follow the data and label submission requirements of other soil fumigants.

Methyl iodide was developed as an alternative to the fumigant methyl bromide, a notorious ozone-depletor. While methyl iodide’s impact on the ozone layer is unquestionably far less than that of methyl bromide, its toxicity is now known to be significantly greater than assumed by EPA at the time of registration, as is its potential to contaminate sources of drinking water. In light of this new science, the use of methyl iodide should be re-considered based on the current data. Nothing in this petition should be construed to countenance any delay in the existing legally-mandated phase-out of methyl bromide.
FIFRA SPECIFIES THAT THE REGISTRATION OF A PESTICIDE FOUND TO BE UNREASONABLY HARMFUL MAY BE SUSPENDED AND CANCELLED.

FIFRA is “a comprehensive federal statute which regulates pesticide use, sales, and labeling, and grants enforcement authority to EPA.” Headwaters v. Talent Irrigation Dist., 243 F.3d 526, 530 (9th Cir. 2001). The statute prohibits the distribution or sale of any pesticide that is not “registered” by the EPA. 7 U.S.C. 136(a). FIFRA sets forth a detailed process whereby a manufacturer can apply to have a pesticide registered. 7 U.S.C. § 136a(c). The statute provides that EPA may register a pesticide if:

(A) its composition is such as to warrant the proposed claims for it;
(B) its labeling and other material required to be submitted comply with the requirements of this subchapter;
(C) it will perform its intended function without unreasonable adverse effects on the environment; and
(D) when used in accordance with widespread and commonly recognized practice, it will not generally cause unreasonable adverse effects on the environment.

7 U.S.C. § 136a(c)(5). The phrase “unreasonable adverse effects on the environment” is defined to mean “(1) any unreasonable risk to man or the environment, taking into account the economic, social and environmental costs and benefits of the use of any pesticide, or (2) a human dietary risk from residues that result from a use of a pesticide in or on any food inconsistent with the standard under [the Federal Food, Drug and Cosmetic Act].” 7 U.S.C. § 136(bb). Thus, “FIFRA utilizes a cost-benefit analysis to ensure that there is no unreasonable risk created for people or the environment from a pesticide.” Washington Toxics Coalition v. United States Environmental Prot. Agency, 413 F.3d 1024, 1032 (9th Cir. 2005).

Notwithstanding the above, FIFRA provides that a pesticide may be “conditionally registered” in any of three “special circumstances.” 7 U.S.C. § 136a(c)(7). See also Syngenta Crop Prot. v. United States Environmental Prot. Agency, 44 F. Supp. 2d 435, 439 (M.D.N.C. 2006) (contrasting conditional and unconditional registration under FIFRA). One of these circumstances allows that EPA “may conditionally register a pesticide containing an active ingredient not contained in any currently registered pesticide for a period reasonably sufficient for the generation and submission of required data . . . on the condition that by the end of such period the Administrator receives such data and the data do not meet or exceed risk criteria enumerated in regulations issued under this subchapter, and on such other conditions as [EPA] may prescribe.” 7 U.S.C. § 136a(c)(7)(C). However, FIFRA provides that a conditional

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1 If a pesticide will not generally cause unreasonable adverse effects on the environment when applied in accordance with its directions for use, its warnings and cautions, and the uses for which it is registered, EPA classifies the pesticide “for general use.” 7 U.S.C. § 136a(d)(1)(B). EPA classifies a pesticide “for restricted use” if normal usage may cause unreasonable adverse effects on the environment, including injury to the applicator, without additional regulatory restrictions. 7 U.S.C. § 136a(d)(1)(C).
registration under this subparagraph “shall be granted only if [EPA] determines that use of the pesticide during such period will not cause any unreasonable adverse effect on the environment, and that use of the pesticide is in the public interest.” *Id.*

If it appears that a registered pesticide, “when used in accordance with widespread and commonly recognized practice, generally causes unreasonable adverse effects on the environment,” FIFRA provides that EPA issue a notice of intent “to cancel its registration.” 7 U.S.C. § 136d(b).

In addition, if immediate action is necessary “to prevent an imminent hazard during the time required for cancellation,” FIFRA provides that EPA may “by order, suspend the registration of the pesticide immediately.” 7 U.S.C. § 136d(c). An “imminent hazard” is defined by FIFRA to include “a situation which exists when the continued use of a pesticide during the time required for cancellation proceeding would be likely to result in unreasonable adverse effects on the environment.” 7 U.S.C. § 136(l).

As the D.C. Circuit has explained, “[T]he ‘imminent hazard’ requisite for suspension is not limited to a concept of crisis: ‘It is enough if there is substantial likelihood that serious harm will be experienced during the year or two required in any realistic projection of the administrative process.’” *Envt’l Defense Fund, Inc. v. EPA*, 548 F.2d 998, 1005 (D.C. Cir. 1976) (quoting *Envt’l Defense Fund v. EPA*, 510 F.2d 1292, 1297 (D.C. Cir. 1975). Thus, the standard weighs the risks against the benefits, and is limited in scope to only the period of time during which the cancellation proceedings are taking place.

To determine whether there is a substantial likelihood of serious harm during the administrative suspension processing period, courts have directed EPA to look at five factors: (1) the seriousness of the threatened harm; (2) the immediacy of the threatened harm; (3) the probability that the threatened harm would result; (4) the benefits to the public of the continued use of the pesticides in question during the suspension process; and (5) the nature and extent of the information before the Administrator at the time he makes his decision. *See Dow Chemical Co. v. Blum*, 469 F. Supp. 892, 902 (E.D. Mich. 1979); *see also Love v. Thomas*, 858 F.2d 1347, 1357 n.16 (9th Cir. 1988). Once the first three factors have established a risk, it is the responsibility of the proponents of continued registration to demonstrate that the benefits outweigh the risks. *Dow Chemical*, 469 F. Supp. at 906; *see also EDF*, 510 F. 2d at 1302.

As outlined below, in the case of methyl iodide, current evidence demonstrates that the potential harms from the use of this fumigant are serious, immediate, and of unacceptably high probability. Industry cannot justify its continued use in the face of this evidence, especially when there are readily available, less-toxic alternatives. We therefore request that EPA immediately suspend the use of methyl iodide and begin the process to cancel its registration permanently.
THE USE OF METHYL IODIDE CAUSES UNREASONABLE ADVERSE EFFECTS ON HUMAN HEALTH AND THE ENVIRONMENT.

A. Exposure to this Highly Toxic Fumigant is Inevitable.

The risk assessments conducted by US EPA, along with those of the California Department of Pesticide Regulation staff scientists and an independent expert panel initiated by CDPR, discussed infra (or below), highlight the serious risks associated with the use of methyl iodide. This pesticide is a developmental toxicant, interfering with the thyroid hormones essential for proper fetal development. Late-term fetal death (miscarriage) is the most sensitive endpoint, and exposures that can reasonably be anticipated from the approved use patterns are sufficient to cause this endpoint.

Methyl iodide is an alkylating agent that reacts directly with DNA, causing mutations that lead to cancers. In fact, the state of California has listed methyl iodide as a carcinogen under Proposition 65. In animal studies, malignant thyroid tumors, brain tumors, and cervical tumors were observed, as well as benign tumors. It is also a severe neurotoxicant, causing permanent neurological damage in those exposed. In addition, it is toxic to the thyroid, disrupting the normal production of thyroid hormones that control metabolism, immune functions, and growth and development in infants and children.

Mixtures of methyl iodide with chloropicrin, currently marketed as “Midas” products, are likely to be even more problematic than methyl iodide alone. Chloropicrin is not only also carcinogenic, but is a severe respiratory irritant, causing permanent lung damage. The toxicity of the mixture of methyl iodide and chloropicrin is unknown, since no studies have been conducted on the mixture, but it is certain that it will be at least as toxic and probably much more toxic than either pesticide alone.

The use of methyl iodide in the chemical industry takes place in closed systems to prevent human exposure to this extremely toxic chemical. In contrast, the currently approved use patterns of methyl iodide, involving injection or dripping this chemical into the soil, ensure that exposure of those in the vicinity of an application will occur. The plastic tarps spread over the field after the application to reduce that risk serve only to slow down the release of gaseous methyl iodide from the soil. Data from the risk assessments and from actual air monitoring studies indicate that plumes of drifting methyl iodide travel with the prevailing winds into homes, neighborhoods, parks, and adjacent fields, where residents and/or unprotected farm workers may be working. The minimum label-required buffer zone – 25 feet for fumigation of a five-acre parcel – offers scant protection for neighbors and workers in adjacent fields. There is no method for preventing airborne drift at concentrations that can reasonably be anticipated to cause harm. During inversion conditions, airborne exposures will be even higher, as more of the methyl iodide vapors are trapped near the ground.
Methyl iodide in the soil can readily be transported to shallow aquifers by rainwater or irrigation water, where it will persist as methyl iodide for as long as year and then degrade to methanol and iodide ions, with iodide concentrations predicted to be far above the minimum drinking water standard that was set by EPA itself to prevent thyroid damage from excess iodide.

When a pesticide is registered nationally, it is automatically registered for use in any state that does not have its own independent scientific review process (most states do not have such a process). To date, only California, Washington, and New York have not registered the chemical. Florida registered methyl iodide in 2008 following EPA’s approval. Arysta LifeScience North America, the manufacturer of methyl iodide, has indicated that the chemical has been used on 15,500 acres in the states of Florida, North Carolina, South Carolina, Georgia, Alabama, Tennessee, Virginia, West Virginia, Michigan, Maine, New Jersey and Oregon. Treatment of 15,500 acres represents approximately 2.7 million pounds of methyl iodide used over 12 states, assuming the label-recommended application rate of 175 lb/acre. There are no data indicating the proximity of methyl iodide-treated fields to inhabited areas, but many fields in the highly populated agricultural states of Florida, North Carolina, and New Jersey are near residences where exposure is likely. Arysta claims that no poisoning incidents have been reported, but there is no tracking system in place to detect such incidents. More significantly, no system is in place to detect adverse effects that are not acute poisonings, such as miscarriages, thyroid disease, and respiratory illness. The state of Florida mandated a groundwater monitoring program in areas of methyl iodide use, but to date, no data have been collected.

To put the current use of methyl iodide in perspective, 15,500 acres represents approximately 8–9% of the total acreage treated with soil fumigants in California alone in 2008, the most recent year for which data are available. If methyl iodide is registered in California, its use is likely to replace the use of substantial amounts of other fumigants, potentially as high as 16–23 million pounds per year on 90,000 to 130,000 acres in this state alone. Nationwide, methyl iodide use could rise to 49–57 million pounds, if it is phased in to replace methyl bromide.

In summary, methyl iodide is highly toxic to many different systems of the human body. Moreover, its high volatility and water solubility means broad use of this chemical in agriculture will guarantee substantial releases to air, surface waters, and groundwater and will result in many people being exposed. Widespread and increasing use can reasonably be anticipated when methyl bromide is fully phased out. Therefore, methyl iodide poses an extremely adverse and,

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3 California Pesticide Use Reporting data, 2008, California Department of Pesticide Regulation, [http://www.cdpr.ca.gov/docs/por/pormain.htm](http://www.cdpr.ca.gov/docs/por/pormain.htm).

4 Because methyl iodide is being put forward as a “drop-in” replacement for methyl bromide, this estimate is based on the total methyl bromide use in California before the methyl bromide phase-out.

indeed, imminent hazard to human health and the environment and its further distribution, sale, and use should be suspended immediately.


“Food and Agriculture Code sections 12824 and 12825 establish the basic requirements for California registration.” Syngenta Crop Prot. v. Helliker, 138 Cal. App. 4th 1135, 1155 (2006). These sections require that before a substance is registered as a pesticide for the first time, it must undergo a “thorough and timely evaluation” by CDPR. Cal. Food & Agric. Code § 12824. The statute sets forth numerous criteria for registration, and the implementing regulations specify additional factors to which CDPR must give “special attention,” including “[a]cute health effects, such as oral toxicity, dermal toxicity, inhalation toxicity” and “[e]vidence of chronic health effects, such as carcinogenicity . . . mutagenicity, fetal toxicity, delayed neurotoxicity,” and “availability of feasible alternatives.” Cal. Food & Agric. Code § 12825; Cal. Code Regs., tit. 3, § 6158. “If any of these factors are anticipated to result in significant adverse impacts which cannot be avoided or adequately mitigated,” California’s regulations provide that “registration will not be granted unless [CDPR] makes a written finding that anticipated benefits of registration clearly outweigh the risks.” Id.

Pursuant to this process, CDPR promulgated a draft risk assessment in March 2009 and a revised risk assessment in August 2009. Its final risk assessment was issued on February 10, 2010, following the release five days earlier of the results from a special external scientific peer review requested by CDPR (discussed infra).

CDPR’s final risk assessment reveals some significant flaws in the EPA risk assessment and updates the summary of the science. First, the rabbit developmental toxicity studies by Nemec (2002, 2009)6 clearly established a reduction in viable fetuses at 10 parts per million (“ppm”) (11% dead fetuses per litter). EPA’s risk assessment erroneously chose 10 ppm as a No Observable Adverse Effect Level (“NOAEL”). Second, EPA erroneously concluded that the cancer mode of action is not relevant to humans despite demonstrated accumulation of the radio-labeled methyl group in the same tissue (thyroid) where cancers are observed (MRID 45641401).

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6 Full references to the cited studies are in the DPR risk assessment unless otherwise specified.
This finding, combined with the known mutagenicity of methyl iodide, should compel the presumption of a mutagenic mode of action that is relevant to humans.

CDPR’s assessment concludes that exposures to workers, bystanders, and residents from use of methyl iodide in California would result in highly significant acute health risks, one of the regulatory factors to be given “special attention.” Specifically, for bystanders and residents, acute margins of exposure (“MOE”) range from 0.1-11, compared to target MOEs of 300, indicating that exposures could be as high as 3,000 times the dose determined to be “acceptable” by CDPR. In some cases (i.e., fetal death among bystanders and residents), the MOE is less than unity, implying that those populations would be exposed at levels above the NOAEL (human equivalent). CDPR noted:

*This risk assessment recommends the acute MOEs evaluated with a benchmark MOE of 300, an additional uncertainty factor of 10-fold applied to the conventional value of 30, because of concerns about Mel causing potential pre- and post-natal developmental neurotoxicity and adequacy of neurotoxicity testing. For some acute exposure scenarios, the calculated MOEs of these endpoints are well below this benchmark, indicating that significant reduction of exposure, up to 3,000 fold, is needed.*

Cancer risk levels are also of significant concern, with risk levels in the range of 90 cancers per million for residents and workers; a cancer risk in this range is 90-fold above the level of concern EPA uses under the Food Quality Protection Act. In addition to the above issues, DPR identified a significant potential threat to shallow groundwater aquifers from iodide contamination. California, Florida, and other states rely on shallow groundwater aquifers in many areas and will rely even more on these aquifers in the future.

A comparison of the toxicity of methyl iodide and methyl bromide suggests that methyl iodide is approximately two orders of magnitude more acutely toxic and has about ten-fold greater subchronic toxicity, but may have slightly lower chronic toxicity (Table 1). These findings are not surprising, because methyl iodide is more reactive with biomolecules than methyl bromide, thereby enhancing the toxicity of the chemical. Although there is evidence for carcinogenic effects from both methyl bromide and methyl iodide, the latter is significantly more mutagenic, and the cancer data are considerably stronger.

<table>
<thead>
<tr>
<th></th>
<th>Methyl Bromide</th>
<th>Methyl Iodide</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute</strong></td>
<td>21 ppm</td>
<td>0.24 ppm</td>
</tr>
<tr>
<td>Developmental tox, rabbit</td>
<td></td>
<td>Fetal death, rabbit</td>
</tr>
</tbody>
</table>

Table 1: Methyl Bromide vs. Methyl Iodide Toxic Potency – Human Equivalent Concentrations (HECs)
Acute

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>25 ppm</td>
<td>3.4 ppm</td>
</tr>
<tr>
<td></td>
<td>Neurotox, dog</td>
<td>Neurotox, rat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.2 ppm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Olfactory epithelium degen, rat</td>
</tr>
</tbody>
</table>

Subchronic

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12 ppm, adult</td>
<td>1.4 ppm, adult</td>
</tr>
<tr>
<td></td>
<td>Neurotox, rabbit</td>
<td>Developmental delay, rat</td>
</tr>
</tbody>
</table>

Chronic

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>0.1 ppm, child</td>
<td>0.6 ppm, child (0.5 ppm, infant)</td>
</tr>
<tr>
<td></td>
<td>Nasal epithelial hyperplasia, rat</td>
<td>Salivary gland metaplasia, rat</td>
</tr>
</tbody>
</table>

Cancer

|        | None calculated | 1.61 x 10^{-2} mg/kg-day |


C. The California-Appointed Extra-Agency Scientific Review Committee Concludes that Even the California Risk Assessment Understates Risks of Methyl Iodide.

CDPR convened a special Scientific Review Committee (“SRC”) of independent experts to review its methyl iodide health risk assessment as well as EPA’s 2007 assessment. On February 5, 2010, the SRC reported on its findings (Attachment 1). It identified significant concerns about the toxicity of methyl iodide, gaps in data that would result in CDPR underestimating risk, and unrealistic mitigation measures.

First, based on the data that are available, the SRC determined that methyl iodide is “highly toxic” and its anticipated uses “would result in exposures to a large number of the public,” leading to the report’s conclusion that methyl iodide has a “significant adverse impact on the public health.” In fact, the SRC had “little doubt that the compound possesses significant toxicity.”

Second, the SRC determined that gaps in the toxicity data raise “serious doubts about the adequacy of any risk assessment to fully estimate the risks that would be associated with the introduction of methyl iodide into the general environment.” In fact, the data gaps “could make the upside to all of the risk calculations even higher” by decreasing the margins of exposures considered in the report. Furthermore, the SRC was “convinced,” based on the toxicology of the chemical, that more sensitive studies could reasonably be expected to find health effects at levels below those that cause fetal death. Specifically, the chemistry of this chemical predicts that methyl iodide is “a potent developmental neurotoxicant at exposures well below those required for overt signs of acute exposure . . .”

Finally, the SRC also found that the protective measures proposed in the CDPR health risk assessment to mitigate worker and population exposure would be “difficult, if not

7 Attached to this petition. Also available at http://www.cdpr.ca.gov/docs/risk/methyliodide.htm.
impossible, to achieve in practice.” Fumigations are difficult applications to perform correctly, and methyl iodide applications require even greater attention to detail. Respiratory protection for workers often malfunctions or is not fitted properly, resulting in high worker exposure; weather predictions are not always accurate, which could result in neighborhoods being exposed during temperature inversion conditions; applicators using methyl iodide are not required to warn adjacent landowners or workers in adjacent fields, which could result in significant exposures for these people. It should be noted that even under the best of conditions, CDPR’s predicted exposures exceed the NOAEL for fetal death.

The high toxicity associated with methyl iodide combined with the near impossibility of controlling human exposure strongly indicates that unreasonable adverse effects can be anticipated with the use of methyl iodide; therefore, this chemical should not be registered for use.

The SRC noted differences between the CDPR’s risk assessment of methyl iodide with the one conducted by EPA. It concluded that each difference between the two risk assessments was due to a “more insightful and scientifically rigorous approach having been undertaken by the [California] DPR.” EPA’s own testimony before the SRC included the following statements:

> Depending on the outcome of California’s external peer review and final risk assessment, EPA may choose to initiate reevaluation of the methyl iodide registration. If the scientific review panel provides new information that would alter or change EPA's scientific analysis, we will include that information in this reevaluation decision.

Comments by Jeffrey L. Dawson, EPA OPP, Methyl Iodide External Peer Review Panel Workshop, September 25, 2009).

Because the SRC’s report contains “new information that would alter or change EPA’s scientific analysis,” Petitioners request that EPA promptly take action to suspend and cancel methyl iodide.

D. New York State Expressed Its Own Concern Over Risks of Methyl Iodide Use.

In December 2008, Arysta LifeScience North America withdrew its application to the New York State Department of Environmental Conservation to register methyl iodide for use in the state. This withdrawal followed the Department’s issuance of a notice of intent to deny the application, published a month earlier. While no final decision on the merits of the application was made, in its letter accepting withdrawal of the application, the Department enumerated the many concerns it had with the human health risks of methyl iodide use. See Letter from New York State to Arysta LifeScience North America, Jan. 14, 2009 (Attachment 2). These included the complexity of use restrictions and instructions, exposure to workers, bystanders, and nearby residents, toxicity of the coactive ingredient chloropicrin, and impacts to groundwater.
In its letter, the Department explained that “[t]he directions for use of these products are extremely complicated and restrictive,” which “could make it difficult to use these products in a manner that is compliant with the label.” The Department also specifically expressed concern about human exposure, stating:

iodomethane poses several toxicity concerns and the four application methods listed on the product labels (raised bed-shank injection, raised bed-drip application, broadcast/flat fume-shank injection and auger probe-deep injection) allow for significant worker exposure potential to the active ingredient. Also, [the New York State Department of Health] has concerns for exposure to bystanders and nearby residents from both routine applications and mishaps associated with the application of these products. While the label restriction states not to use the products within a quarter of a mile of any occupied sensitive site (e.g., schools, daycare centers, nursing homes etc.), the labels do not require such a buffer zone for residential properties. Given the potential exposure of nearby residents, some of who may be children, pregnant women, elderly or those with preexisting medical conditions, such a restriction seems warranted. This could be a particular problem if an accidental release were to occur as iodomethane is very volatile and hundreds of pounds of product are needed to treat even relatively small fields.

Further, because methyl iodide is very soluble in water, the Department raised a number of concerns regarding the chemical’s impact on New York’s ground- and drinking water, emphasizing:

Based on the environmental fate data, LEACHP simulations, and lack of cautionary label text prohibiting the slicing or removal of the tarpaulin if it is raining or if rain is expected within 48 hours, the potential for iodomethane to impact groundwater/drinking water resources in New York State cannot be discounted.

Finally, in addition to concerns over the acute toxicity of methyl iodide, the state also raised concerns over possible effects from the pesticide’s companion ingredient – chloropicrin – explaining that “[a]lthough [the New York State Department of Health] did not review the properties of the coactive ingredient..., chloropicrin, they note that this compound has significant toxicity and irritant properties of its own. . .”

E. A Comparison of Methyl Iodide To Other Pesticides Suspended Due to “Imminent Hazard” Indicates that It Should Join Their Ranks.

EPA has suspended other previously-registered pesticides under 7 U.S.C § 136d(c) due to the “imminent hazard” posed by their use. These pesticides include the soil and grain fumigant ethylene dibromide (EDB), the herbicide 2,4,5-T, the herbicide dinoseb, and the soil fumigant
dibromochloropropane (DBCP). In each case, registration was suspended and ultimately canceled.

**Ethylene dibromide (“EDB”):** EDB’s use was suspended in the United States in 1983 when low-level residues were found in groundwater and stored grains that had been treated with this fumigant. EDB was used as a soil and post-harvest fumigant for crops and as a quarantine fumigant for citrus and tropical fruits and vegetables. It is highly acutely toxic (Toxicity Category I). Chronic exposure damages the liver, kidneys, and testes of rats. Reproductive effects include decreased sperm quality in both humans and animals. EDB is a carcinogen, with tumors of the mammary glands, spleen, adrenals, liver, and kidney observed in laboratory animals.8

**2,4,5-T:** Made infamous through its use in the compound Agent Orange, the defoliant used in the Vietnam war, registration of 2,4,5-T was suspended by the U.S. in 1979 when a study showed significantly higher incidence of miscarriages among women living in forest areas where the chemical was sprayed. 2,4,5-T was used for the selective control of weeds in cereal crops and lawns, nettles in pasture, woody weeds in forestry, particularly with conifers, and aquatic weeds. It has a moderate acute toxicity (Toxicity Category II). Chronic exposure results in destruction of hemoglobin. Reproductive effects include reduced neonatal survival of pups from treated dams and fetal death. Developmental toxicity was evident in laboratory animals in the form of increased incidence of cleft palate, cystic kidneys, absence of eyelids, and delayed head ossification. 2,4,5-T products were contaminated with 2,3,7,8-tetrachlordibenzo-p-dioxin (TCDD), a highly carcinogenic form of dioxin. There is some evidence for carcinogenicity in animals dosed with technical grade 2,4,5-T, although it is not clear whether this is a result of the dioxin contaminant or 2,4,5-T.9, 10, 11

**Dinoseb:** Registration of Dinoseb use was suspended by the U.S. in 1986 based on the potential risk of birth defects and other adverse health effects for applicators and other persons with substantial dinoseb exposure. This herbicide was used in soybeans, vegetables, fruits and nuts, citrus, and field crops for control of weeds, and also as an insecticide in grapes. It is highly acutely toxic (Toxicity Category I). Chronic exposure results in a decrease in the body’s ability to process glucose, which affects many organ systems. Dinoseb interferes with reproduction in laboratory animals and is considered to reduce fertility or cause sterility in humans. It is also a developmental toxicant, causing birth defects in laboratory animals. Chronic toxicity data indicate that dinoseb is not a carcinogen.12

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8 ExToxNet Pesticide Information Profile for Ethylene Dibromide. http://extoxnet.orst.edu/pips/edb.htm
Dibromochloropropane (“DBCP”): DBCP registration was suspended in the U.S. in 1977, except for use on pineapples in Hawaii; in 1985, EPA issued an intent to cancel all registrations for this purpose as well. DBCP was used as a soil fumigant on over 40 different crops in the U.S. It is moderately acutely toxic (Toxicity Category II). Chronic exposure results in adverse effects on the nasal cavity, spleen, adrenal gland, kidneys, stomach, and liver. Exposure to DBCP causes decreased sperm counts and reduced fertility in humans and laboratory animals. EPA has classified DBCP as a Group B2, probable human carcinogen.13

Methyl iodide: Methyl iodide was registered in 2007 over the protests of 55 members of the National Academy of Sciences because of its known toxicity. Methyl iodide is highly acutely toxic (Toxicity Category I). Chronic exposure results in degeneration of the olfactory epithelium, liver adrenal and thymus weight changes, and reduced body weights. Reproductive effects included increased primordial follicles and decreased implantation sites and live litter size. Developmental effects included decreased pup body weights and viability and delayed development. Late-term exposures result in increased incidence of fetal death. Methyl iodide has been listed as a carcinogen by the state of California on its Proposition 65 list. EPA classifies it as “[n]ot likely to be carcinogenic to humans at doses that do not alter rat thyroid hormone homeostasis,” based on the findings of thyroid tumors at doses that do alter rat thyroid hormone homeostasis. CDPR and the SRC disagreed with EPA’s cancer assessment, noting that brain and cervical tumors were also noted and that methyl iodide was mutagenic in most studies. Potential for groundwater contamination is evident from the physical properties of methyl iodide.

Table 2 highlights the toxicity and exposure potential associated with these chemicals, compared to methyl iodide.

Table 2: Toxicity Comparisons between Methyl Iodide and Other Canceled Pesticides

<table>
<thead>
<tr>
<th>Pesticide</th>
<th>Acute Toxicity</th>
<th>Carcinogen Effects</th>
<th>Developmental Effects</th>
<th>Reproductive Effects</th>
<th>Exposure Potential</th>
<th>Other Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methyl iodide</td>
<td>Cat. I</td>
<td>Yes</td>
<td>Yes. Fetal death.</td>
<td>Yes</td>
<td>High (air, groundwater)</td>
<td>Thyroid toxicant. Potential groundwater contaminant.</td>
</tr>
<tr>
<td>2,4,5-T</td>
<td>Cat. II</td>
<td>Unclear</td>
<td>Yes. Fetal death.</td>
<td>Yes</td>
<td>Moderate (drinking water, air) High (food residues)</td>
<td></td>
</tr>
<tr>
<td>Dinoseb</td>
<td>Cat. I</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>High (food residues)</td>
<td>Groundwater contaminant</td>
</tr>
<tr>
<td>DBCP</td>
<td>Cat. II</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>High (air, groundwater)</td>
<td>Groundwater contaminant</td>
</tr>
<tr>
<td>EDB</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
<td>High (air, food residues, water)</td>
<td></td>
</tr>
</tbody>
</table>

F. Non-Toxic Alternatives To Methyl Bromide Exist

Methyl iodide has been touted as a non-ozone depleting alternative to methyl bromide. However, there are safe and reduced-risk replacements for methyl bromide, obviating the need to allow the dangerous and continued use of this fumigant.

These alternatives are safer than methyl iodide and feasible. In 1995, the U.S. Secretary of Agriculture and the EPA Administrator formed the Methyl Bromide Alternatives Working Group to track and facilitate adoption of alternatives to methyl bromide. Among other things, the Department of Agriculture’s Agriculture Research Service (ARS) FY2004 Annual Report highlights the following non-hazardous replacements for methyl bromide pre-plant soil fumigation uses:

1) Biologically-based treatments incorporating nematode-resistant cover crops for nematode population suppression in tomato cropping systems provided tomato yields similar to, or greater than, treatments using methyl bromide. In addition, the biologically-based system had significantly lower productions costs. The net return per hectare over two years was $20,084 and $20,490 for methyl bromide and velvet bean cover crop treatments, respectively. ARS concluded that biologically-based production systems are an economically feasible alternative to production systems reliant on methyl bromide fumigation, are friendlier to the environment, and contribute significantly to soil fertility.

2) Use of low glucosinolate rapeseed meal as an orchard mulch could be a viable, organic alternative to pre-plant fumigation with methyl bromide for control of apple re-plant disease.

3) Soil solarization is also a feasible, affordable, safe replacement for pre-plant fumigation of strawberries and other fruit crops in areas with the appropriate climate. It is effective against many weed species, most nematodes, and most serious pathogens. The current recommendation for solarization in California is to leave the soil covered for 4-6 weeks with large plastic sheeting. While this technique may delay planting slightly, such delays are offset by the significantly reduced cost of solarization ($150-200/acre) compared with methyl bromide ($2000/acre) and metam sodium under plastic ($400-600/acre) according to the USDA-funded National Sustainable Agriculture Information Service.14

New research has also found substantial weed- and soil-borne disease control in California strawberry cultivation as a result of crop rotation with broccoli and cauliflower,15 as

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well as the use of steam and a combination of steam/solarization anaerobic disinestastion methods.\textsuperscript{16}

CONCLUSION

The body of science surrounding the toxicity of methyl iodide has grown significantly over the past few years and underscores the dangerous nature of the pesticide. An expert panel has concluded that this fumigant poses significant harms to public health that cannot be mitigated through limitations on exposures and, moreover, because it is already in use in some states, those harms are imminent. At the same time, methyl iodide is not the only feasible alternative available. Accordingly, continued use and registration of methyl iodide does not satisfy FIFRA, and therefore its registration should be suspended immediately while the procedures for permanent cancellation are initiated.

As a result of EPA’s registration of this fumigant, members of the organizations we represent and their children are being exposed to unsafe levels of methyl iodide and will continue to be thus exposed as long as the registration and use of methyl iodide remain in effect. Because of this imminent threat to our clients’ health and environment, we urge EPA to expedite its consideration of this petition for the suspension and cancellation of this dangerous pesticide.

Respectfully submitted,

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Gregory C. Loarie
Deborah S. Reames
Attorneys

Sarah Jackson
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Earthjustice

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