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Animal and Plant Health Inspection Service

Pale Cyst Nematode in Bingham and Bonneville Counties, Idaho

Supplemental Environmental Assessment
March 2017

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I. Purpose and Need

The U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), is conducting a treatment program to eradicate the pale cyst nematode (PCN) (formerly referred to as potato cyst nematode), *Globodera pallida*, in areas of Bingham and Bonneville Counties, Idaho. PCN is a devastating soil-borne pest to potato crops with the potential to impact related agricultural and nonagricultural plant species (appendix A). Damage varies from small patches of affected plants to complete crop failure. Infestations generally start out as isolated patches which become larger in subsequent years. If untreated, PCN can cause up to 80-percent yield loss in potato fields. The nematode is primarily spread through the transport of soil via seed potatoes, nursery stock, flower bulbs, farm equipment, or soil-bearing surfaces. Natural dispersion in soil is limited.

PCN was first detected in Idaho during a Cooperative Agricultural Pest Survey in mid-April 2006. In June and July of 2006, two fields were confirmed positive for PCN. On August 29, 2006, APHIS and the Idaho State Department of Agriculture (ISDA) announced the establishment of a regulatory area covering approximately 10,000 acres near Shelley, Idaho. Five new fields tested positive after additional testing within the regulatory area. Surveys of seed potatoes yielded no positive detections of PCN in the state. No additional PCN detections were found in surveys conducted throughout other potato growing states in a 2006-2007 National Survey.

Today, a total of 9,520 acres are currently regulated for PCN in Bonneville and Bingham Counties, Idaho, of which 3,047 acres are infested with PCN (figure 1). APHIS regulates infested fields in addition to other fields that may have been exposed to PCN-infested soil in the past, typically through sharing of farming equipment that may have resulted in soil transfer between fields. APHIS continues to find fields infested with PCN in the area. See the APHIS Pale Cyst Nematode webpage for more information about regulation of PCN.

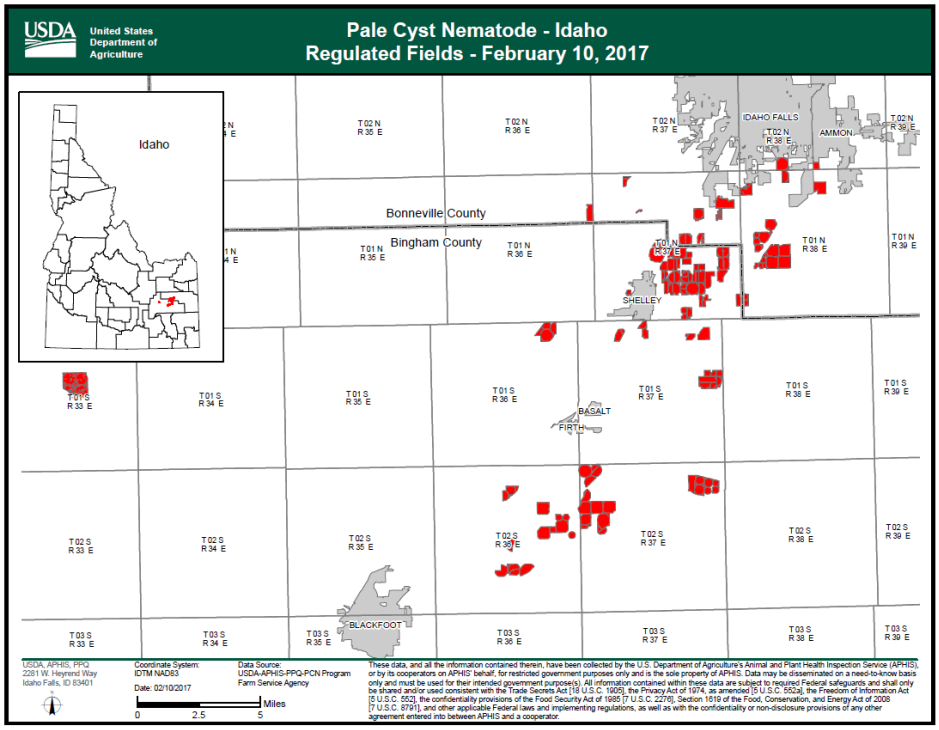

Figure 1. Pale cyst nematode regulated fields (in red) in Bingham and Bonneville Counties, Idaho as of February 10, 2017.

APHIS has the responsibility for taking actions to exclude, eradicate, and/or control plant pests under the Plant Protection Act (7 U.S.C. §7701 et seq.). It is important that APHIS take the steps necessary to eradicate PCN from areas in Idaho to prevent spread to other host crops in the United States. APHIS, in cooperation with the ISDA, is currently conducting a program to eradicate PCN from the infested fields in Idaho.

An environmental assessment (EA) was prepared in May 2007 to address the potential action of eradicating PCN where it had been detected near Shelley, ID. The EA was prepared consistent with the National Environmental Policy Act of 1969 (NEPA) and APHIS' NEPA implementing procedures (7 CFR, part 372) for the purpose of evaluating how the proposed action, if implemented, may affect the quality of the human environment.

In the May 2007 EA, the treatment alternative consisted of using one or a combination of fumigants. The fumigants proposed for use were methyl bromide (MeBr) and 1,3-dichloropropene (DCP). In the initial EA, DCP use was limited to one application per growing season applied at 177 pounds (lbs) of active ingredient (ai)/acre (ac). After further evaluation following the first treatment with MeBr in May 2007, there was a need to have the option to be able to apply DCP twice per year. In addition, higher

application rates were needed to ensure adequate efficacy during treatment. The pesticide label for DCP, sold as Telone II®, did not allow for two applications at a rate above 177 lbs ai/ac so a special local use need label, or Section 24(c) under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA), was prepared with the new use pattern that allowed for one or two DCP applications per season at a rate of 177 to 354 lbs ai/ac per application. A special local use need, or 24(c), is where a state registers additional uses to a federal registered use to meet site specific requirements. APHIS prepared an amended EA in July 2007 (USDA APHIS, 2007a) to discuss how the proposed changes would affect the quality of the human environment.

The purpose of this supplemental EA is to consider a potential PCN eradication program in all potato growing areas in Bingham and Bonneville Counties in the case that a new field or fields are found to be infested with PCN. This does not mean that all potato growing areas are being brought under APHIS regulation but that in the event an additional field is found to be infested, APHIS may not need to prepare another supplemental EA. Another purpose of this supplemental EA is to include updated information regarding current eradication program practices and activities. MeBr is not being considered for use in this supplemental EA under the preferred alternative. MeBr has not been used in the PCN program since May 2014 due to concerns raised by the public regarding its use to treat PCN infested fields. APHIS has taken this concern very seriously and the Agency immediately started to ascertain the facts and response related to this concern. Additionally, out of abundance of caution and in regard for those who raised the concern, APHIS decided in 2015 and 2016 not to use MeBr soil fumigation to treat PCN-infested fields. APHIS is working with the U.S. Environmental Protection Agency (USEPA) and others to investigate this concern before resuming the use of MeBrsoil fumigation in the PCN Eradication Program. MeBris registered for use to control PCN, however, going forward, the program will rely on DCP as the primary fumigant, along with trap cropping, which are discussed in this supplemental EA. MeBr use would be analyzed in a separate EA if it is proposed to be incorporated into any future PCN eradication activities.

A notice of availability for the supplemental EA was published on April 18 allowing public comments until May 28, 2016. Eight comments were received during and after the public comment period, with some of the comments providing support for the program and others raising concerns about various aspects of the program and the supplemental EA. A summary of comments and responses are included in Appendix B. In cases where similar comments were received from different commenters, the comment was summarized with one response. In response to comments APHIS has added an additional alternative that includes no

quarantine or treatment program for PCN in Idaho. Because of this new alternative and new information regarding the use of litchi tomato, APHIS is making the supplemental EA available for another 30-day public comment period.

A. Background

1. Biology of Pale Cyst Nematode

Nematodes are defined as members of the phylum Nematoda, and are elongated cylindrical worms parasitic in animals or plants, or are free-living in soil or water. PCN (*Globodera pallida*) is a plant parasitic nematode that affects agricultural crops.

Typical of most nematode life cycles, *G. pallida* has four distinct juvenile stages and an adult stage. The second-stage juvenile hatches from the egg which is contained within a cyst formed from the cuticle of an adult female. Three more molts will occur before reaching the adult stage. Upon hatching, the second-stage juvenile is considered the active phase because it is the life stage that actively seeks host plants. Hatching occurs based on appropriate environmental factors such as the presence of substances that diffuse from the roots of host plants. Extensive hatching will occur; however, some juveniles will always remain dormant, regardless of the conditions, to ensure population viability (Turner and Evans, 1998). In cases where a host plant is not present, infestations can persist up to 30 years because of delayed hatching of the 200–500 eggs per cyst and the ability of the eggs to remain dormant within the cuticle cyst of the female until hatching cues from hosts plants are detected (Turner, 1996; DEFRA, 1996). Once the second-stage juvenile female encounters a host, it will enter the root near the growing point, or a lateral root, and use the hypodermic needle-like stylet in its mouth to pierce a cell wall. The female then secretes proteins that cause changes to the host cells to include dissolving of cell walls, fusion of host cells, and a proliferation of host cellular machinery (such as nuclei and endoplasmic reticula). The syncytium facilitates and coordinates the passage of nutrients from the host plant to the juvenile female. Male juveniles do not set up feeding sites, but feed as endoparasites until the 4th larval stage. The males then emerge from the roots and molt again to adults. Once females breach the root zone, they release sex pheromones that attract males for fertilization. After fertilization, embryos will develop in the egg until the second-stage juvenile emerges. PCN usually has a single generation during a host growing season (Turner and Evans, 1998).

Host plants are those in the family Solanaceae, which includes the potato, tomato, and eggplant, as well as other nonagricultural hosts (appendix A).

In cases where PCN populations exceed 5 to 10 eggs per gram of soil, the plants can exhibit reduced root systems and altered total mineral uptake. Plants may also have yield reductions due to water stress, altered mineral ratios, and early senescence (DEFRA, 1996; Phillips et al., 1998).

2. Spread of PCN in Idaho

It is not known from where or how PCN came to southeast Idaho. Analysis of the fields' infestation levels and inconclusive investigations of potential pest origins suggest that it was likely unintentionally established in the area decades ago. APHIS continues to find additional lightly-infested fields, but there is no evidence that additional PCN detections are the result of new movement from known infested fields because the PCN regulations designed to prevent PCN spread were implemented in 2006. The most recent detection of a newly infested field was in late December 2016. Detection of these incipient infestations has been made through ongoing cooperative monitoring of associated fields by APHIS and the ISDA. Associated fields are those that have grown a PCN host crop in the last ten years with a relationship with an infested field, or the field shares a border with an infested field; or the field has come into contact with a regulated article from an infested field in the last ten years, or within the last ten years the field shared ownership, tenancy, seed, drainage or runoff, farm machinery with an infested field that could allow spread of PCN. Infested fields are still being found because incipient infestations take numerous (2–3) crop cycles to build up to detectable levels, and PCN eggs can remain dormant in soil for up to 30 years. PCN reproduces primarily on crops and weeds in the Solanaceae plant family. Depending upon a field's crop rotation, a low level infestation may take several years to detect. Some low population level detections of PCN were made in 2011 which was followed by analysis by the Center for Plant Health Science and Technology (CPHST) and discussions with growers. After further consideration at the 2012 PCN Program Review, and as is allowed by the PCN regulations, the APHIS deregulation protocol was amended. This protocol was then adapted into the May 2014 PCN Guidelines (USDA APHIS, 2014).

3. Previous NEPA Documentation

Since 2006, APHIS has prepared numerous NEPA documents for the PCN program. Initially, APHIS prepared a categorical exclusion decision for an interim rule to establish a PCN quarantine in September, 2006. This interim rule was published in 2007 (USDA APHIS, 2007b). At that time, no treatments for infested fields were included in the proposed action, only activities restricting movement of infested and potentially infested materials.

Once a PCN eradication program in Bonneville and Bingham Counties was proposed for treatment of infested fields, APHIS prepared an EA in May 2007, and amended it in July 2007 (USDA APHIS, 2007a) because of a change in application rate of DCP used by the program. From 2007 to 2010, APHIS prepared several categorical exclusion decisions as acres were removed from regulation. In 2011, APHIS prepared an addendum to the finding of no significant impact (FONSI) because of a proposed program fumigant change. APHIS continued to prepare additional FONSI addenda in 2012, 2013, and 2014 for additions and removals of acres to the regulated area.

Whenever there is an expansion or reduction of the regulated area, APHIS will continue to determine whether further NEPA documentation is necessary. However, this supplemental EA is expected to eliminate or reduce the need for APHIS to prepare an additional NEPA document each time there is an expansion or reduction of the regulated area within the two counties.

II. Alternatives

This EA analyzes the potential environmental consequences of the proposed action to eradicate PCN from fields in Idaho where the nematode has been detected. Four alternatives are being considered: (1) maintain current eradication program (no action), which includes the quarantine and use of fumigants such as MeBr and DCP, (2) no eradication program to eliminate PCN but the quarantine would remain in place (3) no quarantine or eradication, and (4) the treatment alternative (preferred alternative), which includes the quarantine, application of chemical treatments using DCP, and trap cropping using litchi tomato. The no action and preferred alternatives have the same goal which is to eradicate PCN from infested fields, however, the methods to reach that goal differ.

A. No Action Alternative

Under the no action alternative APHIS would maintain the current PCN eradication program as codified in 7 CFR 301.86 to 301.86-9 and analyzed in the 2007 EA (USDA APHIS, 2007a). This alternative consists of maintaining a Federal quarantine, as well as treatment of currently infested fields, with a chemical treatment in the spring and fall. Chemical treatments would consist of either MeBr or DCP.

1. Federal Quarantine

APHIS maintains a federal quarantine regarding PCN that is designed to restrict the interstate movement of regulated articles. Regulated articles include, but are not limited to, PCN cysts, soil, PCN host crops, and any other article that could result in the movement of PCN (USDA, APHIS, 2014). The designation of a quarantine area is based on a field being identified as infested with PCN, fields that have been found to be associated with an infested field, and any area that the Administrator considers necessary to quarantine because of its inseparability for quarantine enforcement purposes from infested or associated fields. APHIS will publish the description of the quarantined area and a map on the APHIS Plant Protection and Quarantine (PPQ) Web site, http://www.aphis.usda.gov/planthealth/pcn. The description of the quarantined area will include the date the description was last updated and a description of the changes that have been made to the quarantined area. The description of the quarantined area may also be obtained by request from any local office of APHIS PPQ; local offices are listed in telephone directories. After a change is made to the quarantined area, APHIS will publish a notice in the Federal Register informing the public that the change has occurred and describing the change to the quarantined area (7 CFR §301.86-3). The phytosanitary measures and environmental monitoring related to quarantined fields is summarized under the treatment (preferred) alternative in this chapter.

2. Methyl Bromide/ Chloro-picrin

A standard application of MeBr is injected approximately 12 inches below the soil surface at a rate of 400 lbs of MeBr that also contains chloropicrin. MeBr is odorless and the chloropicrin serves as a warning agent. An impermeable tarp covers the treated field for approximately five days for safety and to enhance efficacy, reduce offsite transport, and promote degradation of the fumigant. There is a 14-day plant-back restriction after fumigation. MeBr use has not occurred in the PCN program since May 2014 due to concerns about its use in PCN-infested fields. No MeBr use occurred in 2015 or 2016.

3. 1,3-Dichloro-propene

Telone II®, which contains the active ingredient 1,3 dichloropropene (DCP), will be applied at a rate of 18–36 gallons per acre, or approximately 177–354 lbs ai/ac depending on site conditions. Applications occur as an injection at least 12 inches below the soil surface. The point of injection is sealed by compacting the soil to minimize volatilization. Telone II® can be applied once or twice a year; however, in most cases the applications would occur once per year in the late summer or fall.

APHIS conducts environmental monitoring during fumigations of infested fields when fumigants are applied. Factors monitored during the fumigation include date, time, wind speed and direction, air temperature,

acres fumigated on day of reading, and the atmospheric concentrations of the fumigants used.

B. No Eradication Alternative

Under the no eradication alternative, APHIS would not eradicate PCN from Bingham and Bonneville Counties, ID. A Federal domestic quarantine would remain in effect because APHIS is authorized under the Plant Protection Act to implement a quarantine for a regulated pest such as the PCN. In addition, regulated articles including potatoes, nursery stock, and soil may not be moved interstate from regulated fields except under specified conditions that these articles are sufficiently free of soil, or accompanying soil is appropriately contained during movement to prevent its entry into agricultural areas, and ultimately disposed of at an APHIS-approved site. Farm equipment moving interstate may not be moved from an infested field unless it has been pressure washed to ensure that all soil has been removed and it has been steam treated in accordance with schedule T406–d of the PPQ Treatment Manual (USDA APHIS, 2015).

Some control or management measures might be taken by other entities; within the State of Idaho; however, these actions would not be funded or controlled by APHIS. In addition, local business owners and area residents could attempt to control PCN. Due to the difficulty in controlling PCN and the several methods of dispersal from infested areas, the nematode would likely expand its range into other potato-growing areas, as well as infest areas in other states containing other solanaceous species. Other agricultural crops, such as tomato and eggplant, could be expected to be impacted, as well as nonagricultural solanaceous species, which could also serve as a source for re-infestation into previously treated fields.

C. No Quarantine or Eradication Alternative

Under the no quarantine or eradication alternative APHIS would not maintain a Federal quarantine or eradicate PCN from Bingham and Bonneville Counties, ID. Any state-implemented quarantine may remain in place because APHIS does not have authorization over those quarantines, and other states could establish their own regulations on articles at risk for spreading PCN. Some control measures could also take place but those would be taken by the state or private landowners and would not be funded or controlled by APHIS.

D. Treatment Alternative (Preferred Alternative)

The preferred alternative consists of maintaining a Federal quarantine, as well as treatment of PCN-infested fields, with a chemical treatment in the

spring and/or fall, a trap crop, and monitoring in Bingham and Bonneville counties. Trap crops are those plants that are planted to attract a particular pest by inducing hatch while not providing a usable food source. Trap crops are not included in the no action alternative. The preferred alternative also differs from the no action alternative in that MeBr and chloropicrin treatments used in the no action alternative would not be used under the preferred alternative. DCP would be the only fumigant proposed for use. The proposed DCP treatments would continue until PCN is eradicated. PCN population levels will be monitored on a regular basis to assess the progress of the eradication effort. The preferred timing for DCP treatments is late summer/early fall although a spring treatment could occur within the first part of May, depending on soil temperature. In addition, phytosanitary requirements are in place for application equipment to ensure that PCN is not artificially spread from treated fields. Specific details on protocols and requirements growers must follow to prevent spread are provided regularly in face-to-face meetings with growers and detailed in Compliance Agreements provided by the program headquarters in Idaho Falls. Additional information regarding surveillance and phytosanitary actions are available in recently revised guidelines between the United States and Canada (USDA APHIS, 2014). The guidelines were developed by both countries, with input from stakeholders, to outline the phytosanitary measures to be taken on the detection of PCN, provide guidance on long-term control and release of infested and associated regulated fields from quarantine, and to provide guidance on how seed potatoes and regulated articles could move between the two countries. The guideline document (USDA APHIS, 2014) is incorporated by reference in this EA, and portions of it are included below.

1. Federal Quarantine

The Federal Quarantine that would be in place would be the same as the one described under the no action alternative. If a change is made to the quarantined area, APHIS will publish a notice in the Federal Register informing the public that the change has occurred and describing the change to the quarantined area (7 CFR §301.86-3). Phytosanitary measures for infested fields are summarized below.

Phytosanitary measures in fields where a PCN infestation is confirmed include: an investigation of any historical movement of regulated articles that may have been associated with the infested field in order to identify potentially exposed fields; restriction of movement of regulated articles from infested fields and adjacent or exposed fields; and, identifying all adjacent, exposed, and infested fields as the initial regulated area that will be subject to all sampling and regulatory controls. If the PCN infested field was used for seed potato production, trace forward information must be collected for the seed lots produced on the infested field. The fields planted with seed potatoes originating from the last potato crop grown on the infested field must be part of the regulated area as exposed fields.

Fields used as seed sources for the infested field will be prioritized for surveys but are not necessarily included as part of the regulated area. Seed potato movement from these fields is restricted until the surveys of these individual fields have been completed. (From: USDA APHIS, 2014)

Any fields undergoing confirmation of PCN infestation are considered suspect fields and should be treated as follows: Restrict movement of regulated articles from the suspect field where the sample was collected; and, initiate investigations of any historical movement of the regulated articles that may have been associated with the suspect field in order to identify potentially exposed fields. If a suspect field cannot be confirmed as infested with PCN, after a series of negative soil surveys following harvest of host crops, all phytosanitary measures are removed. If the suspect field is confirmed as PCN positive, that field as well as adjacent and exposed fields will be regulated and will be subject to all sampling and regulatory controls. While the investigation is being conducted, the PCN phytosanitary certification requirements for seed potatoes traded between the United States and Canada will provide the necessary safeguards to permit the undisrupted trade of seed potatoes from fields outside of the regulated area. (From: USDA APHIS, 2014)

PCN-Infested Field Treatments

A chemical treatment option is available and is discussed below, as well as trap cropping.

2. 1,3-Dichloro-propene

Telone II®, which contains the active ingredient 1,3 dichloropropene (DCP), will be applied at a rate of 18–36 gallons per acre, or approximately 177–354 lbs ai/ac depending on site conditions. Applications occur as an injection at least 12 inches below the soil surface. The point of injection is sealed by compacting the soil to minimize volatilization. Telone II® can be applied once or twice a year, however, in most cases the applications would occur once per year in the late summer or fall. Two applications per year would only be used if an accelerated treatment schedule is implemented that includes both a spring and a late summer/fall treatment.

APHIS has conducted environmental monitoring during fumigations of infested fields and this would continue under the preferred alternative. DCP would be the only product monitored because MeBr is not being proposed for use under the preferred alternative.

3. Trap Cropping

Trap crops are those plants that are planted to attract a particular pest. In the case of PCN, the trap crop proposed for use is the litchi tomato (*Solanum sisymbriifolium* Lam.), which is an annual herb that is native to South America. It is used in Europe as a management tool for PCN and is

also grown around the world for ornamental and culinary uses. The litchi tomato plant can reach up to 3 feet in height. The stems and branches have thorn-like prickles that can be up to ½ inch in length, and the flowers are white to pale blue.

APHIS is proposing to use litchi tomato as a trap crop for PCN in fields where PCN has been detected. The roots of litchi tomato stimulate nematode eggs in the soil to hatch, but do not support nematode feeding or reproduction (Timmermans et al., 2007). Because hatched nematodes have limited food reserves, they die because they cannot successfully parasitize litchi tomato roots. An additional benefit is that the roots of the litchi tomato can reach to greater depths in the soil than program fumigants. Initially, litchi tomato has been planted on a limited basis in three PCN-infested fields to determine its efficacy against PCN.

Litchi tomato is not native to Idaho, and may become invasive in the environment if not carefully managed. The ISDA has restricted the use of litchi tomato and requires that growers and the University of Idaho researchers complete a detailed permitting process prior to planting. The current weed management plan has been developed at the University of Idaho to prevent litchi tomato from becoming a weed in years subsequent to planting. Planted litchi tomato will be monitored every two weeks for emergence and plant development thresholds. ISDA will be notified immediately at germination, flowering, and the first sign of berry development. Once the crop has reached the trigger for destruction the crop will be treated with an herbicide (see below for specific types) and flailed 5–10 days post treatment, and all plant residues incorporated into the soil within 10 days of flailing. Planted fields will be monitored for regrowth, and if any is observed, the process will be repeated. Following planting and prior to leaving the field, all equipment will be visually inspected by APHIS PCN program staff for litchi tomato seed. Monitoring outside the planted fields will also occur every two weeks throughout the growing season and until the onset of winter.

Additional proposed monitoring and treatment of escaped litchi tomato are listed below from the weed management plan:

- APHIS PCN Program staff will conduct a field perimeter survey to include fence lines, ditches, roadways, and neighboring fields approximately every two weeks throughout the growing season to locate any escaped litchi tomato plants.
- If litchi tomato is detected in rotation crops and/or non-crop areas, spot spraying with herbicide is required. Hand removal may not remove all growing plants.
- In the five years following the litchi tomato field plantings, rotational crops must be of appropriate height to allow effective

monitoring for escaped or volunteer litchi tomato plants. Examples of acceptable crops are small grains, canola, hay, and potatoes.

- Small grain or corn herbicides labeled for use in those crops, such as bromoxynil, 2,4-D, 2-methyl-4-chlorophenoxyacetic acid (MCPA), saflufenacil, and dicamba, can control litchi tomato. Glyphosate could be used in Roundup Ready® corn, alfalfa, and sugar beet. Non-crop herbicides, including glyphosate, could be used in areas outside of the crop to control litchi tomato. Other herbicides that may be used in potatoes and other crops to control litchi tomato include flumioxazin, metribuzin, pendimethalin, dimethenamid-p, EPTC, rimsulfuron, ethalfluralin, metribuzin, thifensulfuron, imazapic, aminopyralid, and fluroxypyr. Herbicide recommendations for controlling litchi tomato were provided by the University of Idaho and are currently registered for use in Idaho.
- Crops such as corn may be grown when:
    - At least one year separates the litchi tomato crop and corn, and:
    - No litchi tomato plants are identified as a result of monitoring in the field in the previous year.

Release of Infested Fields from Regulatory Control

Infested fields may be released from regulatory control via the following procedures outlined in the guideline document (USDA APHIS, 2014).

a. Regulated non-agricultural land

There are a number of regulated fields in both Canada and the United States that have been converted to non-agricultural uses. Examples of non-agricultural land are highways or other paved roads, paved parking lots, industrial parks, other commercial developments (such as shopping malls, apartment housing, and office complexes), residential developments, state or national parks, other recreational areas, racetracks, and golf courses. All regulated land in this category may be released if it meets the following criteria (USDA APHIS, 2014):

1. Records must be available to determine that the land has been out of agricultural production for the last 20 years and will not return to agriculture; or,

2. Construction for non-agricultural purposes has rendered the land non-tillable and is not likely to ever return to agricultural production.

b. Regulated agricultural land no longer in host crop production.

There are some fields in the United States that are regulated and where agriculture does still occur but where all host crop production was prohibited or has ceased for a minimum of 30 years. This could include formerly infested, adjacent, or exposed fields. During this time, the fields may have been used for various purposes, including but not limited to hobby farms, fallow fields, forage crops, grain fields, nurseries, pasture, riding academies, sod farms, etc. All regulated land in this category may be released if it meets all of the following criteria (except formerly infested fields, which may never be used for seed potato production) (USDA APHIS, 2014):

1. Records must be available to determine that land has been out of host crop production for the last 30 years.

2. The field is surveyed using protocols outlined in USDA APHIS (2014).

3. If PCN cysts are found, a viability test must be performed on these cysts.

4. If no PCN cysts are found, or no viable larvae or eggs are detected after a viability test, then the field can be released from regulatory control.

5. If host crops are grown after regulatory changes are made, continued surveillance is strongly suggested.

c. Adjacent and exposed fields used for host crop production

Adjacent and exposed fields are subject to regulatory measures due to their association with infested fields and the consequent risk they pose for soil-borne spread of PCN. Host crops may be grown in the field. Processing or fresh market potatoes may be grown on adjacent and exposed fields only for non-seed purposes under regulatory control (i.e., compliance agreements or equivalent). Potatoes may be grown for seed purposes under regulatory control (i.e., compliance agreements or equivalent); however, seed potatoes harvested from adjacent and exposed fields may be used only within that regulated area. Exposed fields are eligible for the lifting of all regulatory controls when conditions 1 and 3, listed below, are met. Adjacent fields, however, are eligible for lifting of all regulatory controls when all of the following conditions are met (USDA APHIS, 2014):

1. Negative surveys. In order to proceed to steps 2 and 3, negative test results must be obtained.

2. Removal of equipment-cleaning requirement. Provided surveys are negative, and on a case-by-case evaluation, equipment-cleaning requirements may be removed.

3. Additional surveillance. Following a susceptible host plant crop, conduct one additional survey. If this survey is negative then all regulatory controls may be lifted on an exposed field.

4. Adjacent fields. The lifting of all regulatory controls on adjacent fields may occur only following negative bioassay results from the corresponding infested field.

d. Infested fields used for host crop production

Infested fields to be used for host plant production are subject to the most stringent phytosanitary measures due to the high risk of soil-borne PCN spread. Potatoes may only be grown under the currently approved management plan, unless potatoes are being planted as part of a bioassay. The following measures are required for release of infested fields used for host crop production (USDA APHIS, 2014):

1. Negative viability assay. Fields must be surveyed using the Viability Assay Survey as described in USDA APHIS (2014) and viable PCN must not be detected as per the PCN viability assay protocol.

2. Negative bioassay. After a negative viability assay is completed a bioassay must be conducted, using methods described in USDA APHIS (2014).

3. Release from equipment cleaning requirement. If the bioassay is negative, and on a case-by-case evaluation, equipment-cleaning requirements may be removed and host crops may be grown in the field.

4. Continued monitoring or in-field bioassay. Conduct three additional full field surveys using the viability assay method. Each survey must be conducted after the harvest of a susceptible host crop.

5. Further release from regulatory control. If no viable cysts are detected, the field can be released from most regulatory controls except that the field remains restricted for seed potato production.

III. Affected Environment

The current area being considered for treatment consists of potato growing areas that are or could become infested with PCN in Bingham and Bonneville Counties (see figure 2).

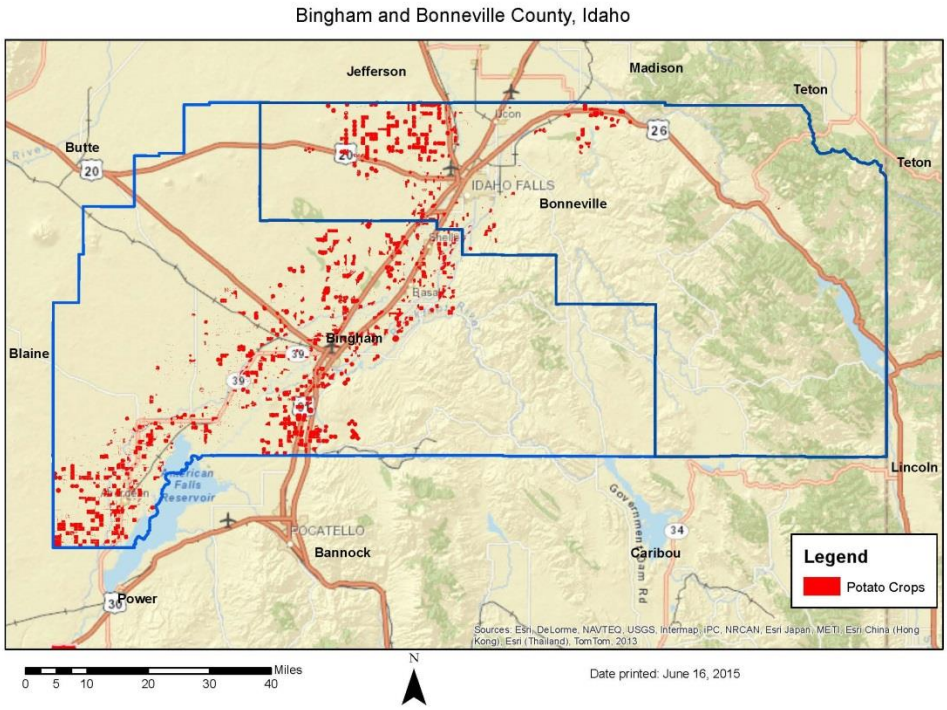

Figure 2. Location of potato fields (in red) in Bingham and Bonneville Counties, Idaho, 2012.

A. Land Characteristics and Agricultural Production

Bingham County

Bingham County is 2,184 square miles including the 359 square miles in the Fort Hall Indian Reservation. The county is fairly level, with the Blackfoot mountain range on the east, and lies entirely within the Snake River plain. The economy of Bingham County is heavily dependent on agriculture (Cravens, 2015). The county has 1,265 farms averaging 687 acres in size (U.S. Department of Agriculture, 2012a). Farmland use in the county is 43 percent cropland, 54 percent pastureland, and 3 percent other uses (U.S. Department of Agriculture, 2012a). The top crop item (acres) for Bingham County is wheat for grain (145,820 acres), while potatoes are fourth in acreage, grown on 77,204 acres in the county (U.S. Department of Agriculture, 2012a). The value of vegetables and potatoes for the county is $179,169,000, and this commodity group value is ranked highest

in the State. Bingham County has the highest acreage in all Idaho Counties for wheat for grain, all harvested vegetables, and potatoes (U.S. Department of Agriculture, 2012a). The market value of all agricultural products sold in Bingham County, including crops, nursery and greenhouse, livestock, poultry, and their products is reported as $453,267,000 (U.S. Department of Agriculture, 2012a). It is known as the potato capital of the world (Safety, 2005). Bingham County soil mostly falls into three classifications: Sagemoor, Declo, and Bannock (Safety, 2005).

Land ownership in Bingham County is as follows: 392,484 acres of Federal land, mainly owned by the Department of Interior, Bureau of Land Management; 156,198 acres of State land; 786,156 acres of privately owned land; 5,480 acres of County land; and 354 acres of Municipal land (Safety, 2005). Land use is divided as: 3,200 acres of urban land; 428,200 acres of agricultural land; 632,000 acres of rangeland; 51,900 acres of forest; 18,400 acres of water; 16,000 acres of wetland; and 201,800 acres of barren land (1997 Census of Agriculture-County Profile, as cited in (Safety, 2005).

Bonneville County

Bonneville County is approximately 1,900 square miles in size and is part of the Upper Snake River Valley. By population, Bonneville County is the fourth largest in Idaho, with a population of 108,623 as of 2014 (St.Jeor, 2015). The county's economy is less dependent on agriculture than Bingham County, with 893 farms with an average size of 458 acres (U.S. Department of Agriculture, 2012b). Farmland use in the county is 68 percent cropland, 26 percent pastureland, and 6 percent other uses (U.S. Department of Agriculture, 2012b). The top crop item (acres) for Bonneville County is barley for grain (72,280 acres), and is ranked highest in the State (U.S. Department of Agriculture, 2012b). Potatoes are not reported as a top crop item in the County (U.S. Department of Agriculture, 2012b). The market value of all agricultural products sold from Bonneville County, including crops, nursery and greenhouse, livestock, poultry, and their products is less than half of that for Bingham County, reported as $204,176,000 (U.S. Department of Agriculture, 2012b).

Although other land in the counties includes portions of the Caribou-Targhee National Forest and Grays Lake National Wildlife Refuge (NWR) these areas are not considered part of the affected environment because these are not areas where potatoes would be grown.

B. Air Quality

The Clean Air Act (CAA) (42 U.S.C. §§ 7401 et seq.) is the primary Federal legislation that addresses air quality. In any given region or area of the United States, air quality is measured by the concentration of pollutants in the atmosphere, and is influenced by surface topography and prevailing meteorological conditions. The USEPA established National Ambient Air Quality Standards (numerical concentration-based standards) for six criteria pollutants that impact human health and the environment (40 CFR § 50). These pollutants are common and accumulate in the atmosphere as a result of natural processes and normal levels of human activity. They include carbon monoxide, nitrogen dioxide, ozone, sulfur dioxide, small particulate matter, and lead.

There are no air quality non-attainment or maintenance areas in Bingham or Bonneville Counties. However, a non-attainment area for particulate matter with an aerodynamic diameter of less than or equal to 10 micrometers (PM-10) occurs in the Fort Hall Indian Reservation in Bannock County, near Bingham County. To improve air quality in the area, the USEPA published a final rule for a Federal Implementation Plan on August 23, 2000 (Agency, 2000) to impose emission limits and work practice requirements for an elemental phosphorus facility located on the reservation. Next to this non-attainment area is the Portneuf Valley Maintenance Area for PM-10, also in Bannock County. It includes federal land managed by the Bureau of Land Management and the Caribou National Forest, as well as privately owned land in the cities of Pocatello and Chubbuck. The USEPA issued a final rule in 2002 indicating that a finding of attainment for PM-10 was achieved for this area (formerly known as the Portneuf Valley Non-Attainment Area) as of December 31, 1996 (Agency, 2002).

C. Water Quality

Idaho has more than 95,000 miles of rivers and streams and 437 lakes and reservoirs, making water one of the state's most important resources. These rivers, lakes, streams, reservoirs, and wetlands provide natural beauty as well as water necessary for drinking, recreation, industry, agriculture, and aquatic life.

The Snake River flows northwest through Bonneville County, beginning at the Wyoming border as the Palisades Reservoir. The river exits the county about midway on its northern border, turns and re-enters approximately 20 miles west to flow southwest through Idaho Falls. The river flows southwest through the middle of Bingham County; at the county's southwest corner the river is the American Falls Reservoir.

Other waterbodies in Bingham and Bonneville Counties include: the Ririe Reservoir, located on Willow Creek, a popular fishery close to Idaho Falls; Palisades Reservoir which is part of the Greater Yellowstone ecosystem, is surrounded by forested mountains and is used for boating, fishing, camping, and wildlife viewing; the Blackfoot River that joins the Snake River in Bingham County, formed at the convergence of Lanes Creek and Diamond Creek, and flows into the Blackfoot Reservoir in Caribou County.

Mercury can be found in Idaho's environment from historic gold mining practices and much of it is still present in Idaho water bodies today (Quality, 2015). In a 2007 lake and reservoir survey in Idaho, 20 out of the 50 lakes sampled had at least one fish species in which the mercury criterion (0.3 milligrams/kilogram) was exceeded. As of February 2009, there were 13 lakes or reservoirs and two streams across the state of Idaho with fish consumption advisories for mercury (Essig, 2010), including the American Falls Reservoir for Utah suckers and South Fork Snake River for brown trout (Quality, 2013). In addition, a statewide consumption advisory for smallmouth and largemouth bass was issued in 2008 (Essig, 2010).

Nitrate is one of the contaminants responsible for groundwater degradation and is one of the most widespread ground water contaminants in Idaho. High levels of nitrate in drinking water are associated with adverse health effects in humans and livestock. High levels of nitrate also adversely affect fish and surface waters such as lakes and rivers. Nitrate priority area ranking is used to prioritize the development and implementation of strategies to help reduce nitrate loading from land-use activities. Two nitrate priority areas (NPAs) have been identified in Bingham County, the Fort Hall and Blackfoot NPAs. These NPAs are areas where elevated levels of nitrate have been found in ground water. The minimum criterion for a Priority 1 NPA is 25 percent of sampled wells that have nitrate levels at or above 5 milligrams per liter (mg/L) (Quality, 2008). The state and federal drinking water standard, as well as the Idaho Ground Water Quality Standard for nitrate is 10 mg/L. In water samples from the Fort Hall NPA, 88 percent of wells were found to have greater than 10 mg/L of nitrates (Quality, 2008). In the Blackfoot NPA, 20 percent of wells had greater than 10 mg/L of nitrates (Quality, 2008).

D. Vegetation and Wildlife

Vegetation

The Bingham County Comprehensive Plan (Safety, 2005) describes the vegetation in Bingham County as follows: In the desert and mountains, Wyoming big sagebrush, basin big sagebrush, rocky mountain juniper,

Utah juniper, mountain big sagebrush and three-tipped sagebrush are found. In the mountains, mountain penstemon, mountain eriogenum, aspen, Douglas fir, rocky mountain juniper, and Utah juniper can be found. Green rabbit brush, four wing salt bush, tall rabbit brush, balsam root, hawksbeard, and herbaceous sage can be found throughout Bingham County. Along the river bottoms, black cottonwood and several types of willows can be found that are also found in the mountains. Native grasses found in Bingham County consist of blue bunch wheat grass, stream band wheat grass, basin wild rye grass, Nevada bluegrass, and sandburg grass. Sedges, rushes and tufted hair grass are found along the river bottoms. In the mountains, blue bunch wheat grass, basin wild rye, stream bench wheat grass, western wheat grass, slender wheat grass, Idaho fescue and pine grass occur. Mountain shrubs consist of serviceberry, snowberry, chokecherry, and snowbush.

Wildlife

The Sterling Wildlife Management Area (WMA) in Bingham County is located along the northwest shore of American Falls Reservoir, and these areas likely support the greatest variety of shorebirds in Idaho. Bufflehead, Canada goose, gadwall, mallard, pintail, redhead, ring-necked duck, ruddy duck, scaup, shoveler, teal, and widgeon are common in the area at various times. Avocet, black-necked stilt, sandhill crane, and a variety of sandpipers use the area. Antelope, badger, beaver, cottontail rabbit, coyote, marmot, mink, mule deer, muskrat, pocket gopher, raccoon, red fox, striped skunk, and jackrabbits are some of the mammals which commonly occur in the area. The marshes provide good duck hunting. Food and cover plots provide opportunity for goose and pheasant hunting. (From: (Game, 2015)).

The Tex Creek WMA is located east of Idaho Falls in eastern Idaho's Bonneville County. Rocky Mountain elk and mule deer begin moving north toward Tex Creek in the late fall. More than 3,000 elk, 3,000 mule deer and 50 moose may winter on WMA lands each year. Sage and sharp-tailed grouse and gray partridge are found in the dry shrublands of Tex Creek WMA. Black-capped chickadees, brown creepers, wrens, goldfinches, shrikes, and chipping sparrows inhabit Tex Creek WMA's forest, riparian and upland communities. Bald and golden eagles, goshawks, and American kestrels also occur in the area. When water flows are sufficient, the lower reaches of Tex Creek WMA's streams support native cutthroat trout and introduced brook and German brown trout. Hunting is popular at Tex Creek WMA. Big game, upland bird and small game hunting are all allowed in season. (From: (Game, 2015)).

IV. Environmental Impacts

A. No Action Alternative

This alternative consists of maintaining a Federal quarantine, as well as treatment of currently infested fields, with a chemical treatment in the spring and fall. Chemical treatments would consist of either MeBr with chloropicrin, or DCP. The analysis of the potential environmental impact from the use of each fumigant is incorporated by reference from the previous amended EA, with an updated analysis for MeBr and chloropicrin in appendix C of this supplemental EA. An updated analysis for DCP is presented in the preferred alternative section of this supplemental EA.

B. No Eradication Alternative

The no eradication alternative in the PCN program would be the continuation of the domestic quarantine that is currently in place in Idaho. In addition to the ISDA regulation which restricts intrastate movement of regulated articles and prevents farmers on fields classified as infested from growing potatoes and other host crops, the Federal regulations restrict interstate movement of regulated articles including—

- Pale cyst nematodes.
- The following pale cyst nematode host crops:
    - Eggplant (*Solanum melongena* L.)
    - Pepper (*Capsicum* spp.)
    - Potato (*Solanum tuberosum* L.)
    - Tomatillo (*Physalis philadelphica* Lam.)
    - Tomato (*Lycopersicon esculentum* L.)
- Root crops.
- Garden and dry beans (*Phaseolus* spp.) and peas (*Pisum* spp.).
- All rooted nursery stock.
- Soil, compost, humus, muck, peat, and manure, and products on or in which soil is commonly found, including grass sod and plant litter.
- Hay, straw, and fodder.
- Any equipment or conveyance used in an infested or associated field that can carry soil if moved out of the field.
- Any other product, article, or means of conveyance not listed in paragraphs (a) through (h) of this section that an inspector determines presents a risk of spreading the PCN, after the inspector provides written notification to the person in possession of the

product, article, or means of conveyance that it is subject to the restrictions of this subpart.

The no eradication alternative would provide a means of slowing the spread of PCN with the use of the Federal and State quarantines, but due to the difficulty of inspecting all the regulated articles listed above, it would be difficult to contain the infested acreage to the small area where it currently occurs. Over time, PCN would be expected to expand its range beyond the currently infested fields and possibly infect other potato growing areas within the State of Idaho, as well as other potato-growing regions in the United States (figure 3).

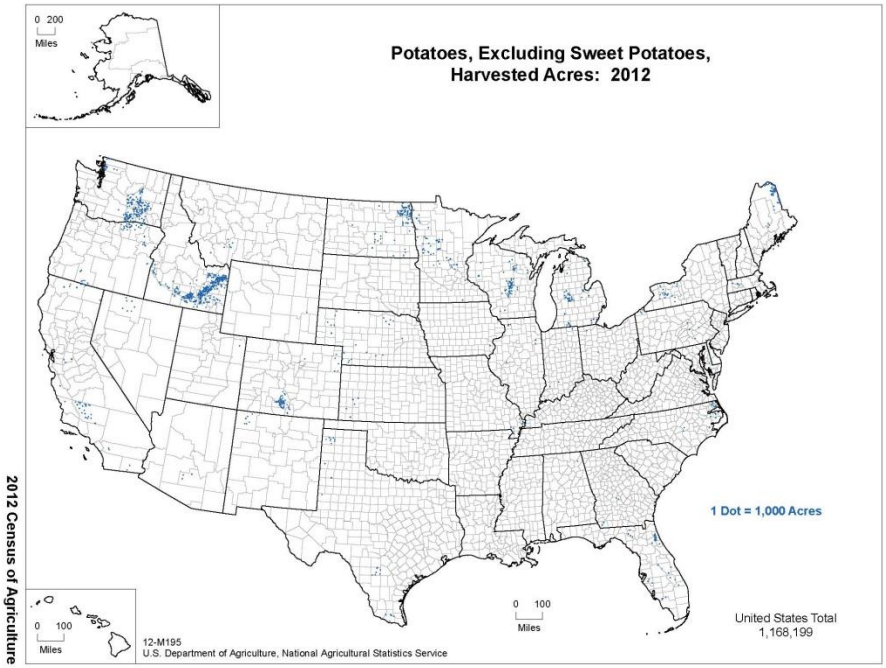

Figure 3. Harvested potato acreage in the United States – 2012 (blue areas).

While the current infestation is localized and affects only potatoes, PCN is known to have additional host plants within the plant family Solanaceae (appendix A). These include other agricultural crops, such as tomatoes and eggplant, but also a wide variety of nonagricultural species. Impacts of PCN to nonagricultural Solanaceae would be expected for those plants where nematode levels increase to damaging levels. In addition, these areas could serve as sources for PCN to be spread to other areas and be reintroduced into previously treated fields.

Movement of PCN to other potato-growing areas of the United States would eventually result in nematode levels reaching economic threshold levels that would justify additional pesticide applications. Controlling PCN in agricultural and nonagricultural areas would require increased pesticide use that would result in an increase in pesticide loading to the environment with fumigants, such as MeBr and DCP, and other nematicides. High-use rates are common with fumigants so any additional pesticide applications to control PCN could dramatically increase environmental loading while also increasing potential risk. Environmental concerns could result from the increased use of pesticides while also increasing production costs for any crops that would require additional pesticide applications.

C. No Quarantine or Eradication Alternative

The no quarantine and no eradication alternative would include removing the Federal quarantine for PCN in Idaho and no eradication of PCN from infested fields. ISDA regulations regarding intrastate movement of regulated material could still be in place but there would be no Federal regulations regarding interstate movement, allowing PCN-infested material to move to other states. Any funding that APHIS provides to ISDA to maintain their quarantine and other activities would not be available. APHIS would also not implement any control measures to eradicate PCN in Idaho. The environmental consequences from selecting this alternative are similar to some of those described in the no eradication alternative; however, the impacts would be expected to be more significant because PCN-infested material would be allowed to move interstate increasing the probability of introducing PCN into other states and leading to the likely regulation of PCN-regulated commodities in other states and countries. Potatoes are the most economically significant crop grown in Idaho with revenues of approximately $1 billion annually. Idaho fresh packers provide one-third of the nation's fresh potato shipments. In 2002, Idaho's potato production, packing, and processing sectors created approximately 15,500 jobs and $3.4 billion in sales of potatoes and potato products (Taylor et al, 2007). These types of economic impacts would be expected to increase if the quarantine and eradication efforts were discontinued in Idaho. Foreign markets would be expected to react in similar fashion to the initial detection in 2006, with many immediately closing markets to Idaho potatoes, and some possibly to U.S. potatoes again. This potentially exposes the entire U.S. potato industry valued at over $8 billion (National Potato Council, 2014).

There would likely be a spread of PCN to other areas of the United States, and in cases where nematodes levels reach economic thresholds, there would be additional need for pesticide applications. It is likely other states would establish regulations restricting entry of Idaho potatoes to mitigate

the pest risk, which would be a significant negative impact to Idaho's industry. This would also increase pesticide loading to the environment and also increase production costs for crops that would require treatment.

D. Treatment Alternative (Preferred Alternative)

The preferred alternative consists of maintaining the quarantine to slow any further movement of PCN, and to eradicate PCN from currently infested fields using fumigant applications. In addition, trap cropping using the litchi tomato may be used if proven effective. Litchi tomato is a non-native plant to Idaho that may become invasive if not properly managed. The potential for the litchi tomato to become established in the immediate area of its proposed use, and other parts of the United States, is high if not managed properly (USDA APHIS, 2013). However, the potential for environmental impacts from litchi tomato plantings in the proposed program will be reduced based on its restricted use by the ISDA, and the implementation of a weed management plan designed to prevent its unintended introduction into other areas. A summary of the potential impacts of the available herbicides that may be needed to treat litchi tomato is available in appendix D. Herbicides will only be used in cases where litchi tomato has been positively identified and only herbicides that are currently registered for use in Idaho will be used. Many of the post emergent treatments may be spot treatments reducing the potential for off-site transport due to reduced application rates.

The quarantine and associated monitoring for PCN has been categorically excluded under the APHIS NEPA Implementing Regulations; therefore, the discussion of potential environmental impacts from the preferred alternative will focus on pesticide use. The fumigant being considered for use in the PCN eradication program is DCP which is considered a MeBr replacement fumigant (UNEP, 2002). A summary of the risk profile for DCP is presented below.

a. Toxicity

DCP (1,3-Dichloropropene) has moderate acute oral and dermal toxicity while having comparably lower inhalation toxicity. Acute toxicity values for DCP in the rat range from an oral median lethal dose (LD_{50}) value of 224 (females) and 300 mg/kilogram (kg) (males). The dermal LD_{50} in rabbits is reported as 333 mg/kg while the inhalation median lethal concentration (LC_{50}) values in rats were 3.88 (males) to 4.69 mg/L (females) (USEPA, 1998). In 13-week subchronic feeding studies, the rat and mouse no observed adverse effect level (NOAEL) values were 5 and 15 mg/kg/day, and lowest observed adverse effect level (LOAEL) values were 15 and 50 mg/kg/day based on hyperkeratosis and/or basal cell hyperplasia in the stomachs of the rat, and decreased weight gain in the

mouse. Subchronic inhalation studies in mice and rats resulted in NOAEL values of 10 and 30, and LOAEL values of 30 parts per million (ppm), based on histopathological lesions in the nasal turbinates (USEPA, 2007). The oral or inhalation animal studies showed no evidence of developmental or reproductive effects (USEPA, 2008b). Human incident reports show that health effects from accidental exposure of DCP spills are skin injuries (blistering, burning sensation, or dermal irritation) and respiratory effects.

USEPA classifies DCP as likely to be carcinogenic to humans based on a 2-year chronic feeding study using rats. The chronic study reported the NOAEL of 2.5 mg/kg/day and the LOAEL of 12.5 based on a decrease in body weight gain and an increase in the number of cells in an epithelium resembling the basal cells of the nonglandular mucosa in the stomach. The study also revealed liver cell adenoma formation at the highest dose tested in the study, 25 mg/kg/day. DCP has also been shown to be genotoxic based on mutagenicity studies (USEPA, 2007). DCP is absorbed, conjugated with glutathione to form mercapturic acid, and excreted in the urine quickly (Schneider et al., 1998; USEPA, 2008b). DCP does not bioaccumulate in target tissues based on its chemical properties and rapid metabolism (USEPA, 2008b).

Acute effects to birds demonstrate that DCP is moderately toxic with a reported LD_{50} value of 152 mg/kg for the bobwhite quail. Dietary LC_{50} values for the bobwhite quail and mallard duck are greater than 10,000 ppm; however, these values should be interpreted with caution because the product is highly volatile and was most likely lost during the duration of the study. No chronic avian studies are available due to the short dissipation half-life and the typical one application per year scenario for DCP (USEPA, 1998).

The formulated material, Telone® II Soil Fumigant, (Dow AgroSciences, 2015) has moderate acute toxic to birds with an oral LD_{50} of 139.8 mg/kg for the bobwhite quail. It is practically non-toxic to birds via a dietary route with a dietary LC_{50} greater than 6,243 mg/kg diet for the mallard. The toxicity to soil-dwelling organisms reported the 14-day LC_{50} of 55.6 mg/kg for earthworms. DCP is moderately toxic to honey bees with a 48-hour (hr) LD_{50} of 6.6 micrograms/bee (µg/bee) based on a dusting technique (USEPA, 1998). Another study showed that the applications of 1,3-D do not adversely affect soil arthropods, but have a transient effect on earthworms and soil microflora with full recovery within six months and 4.5 months of DCP application, respectively (CalEPA, 2012; Small et al., 2008).

DCP is considered to be moderately toxic to fish and very highly toxic to aquatic invertebrates based on standard toxicity tests. Several fish LC_{50}

values exist for DCP with the most sensitive species being the walleye (LC_{50} = 1.08 ppm) and most tolerant being the bluegill sunfish (LC_{50} = 7.1 ppm). Toxicity to freshwater invertebrates appears to be limited to *Daphnia magna* with a reported 48-hour EC_{50} value of 0.09 mg/L. No chronic aquatic vertebrate or invertebrate data is available due to the short half-life of DCP in aquatic systems and the typical one application per season use pattern (USEPA, 1998).

The DCP formulation proposed for use in the PCN program is highly toxic to fish with the 96-hour LC_{50} for sheepshead minnow of 0.87 mg/LL. The 96 hours LC_{50}s for rainbow trout and bluegill sunfish are 2.78 mg/LL, and 3.7 mg/L, respectively. The acute toxicity to aquatic invertebrates' data show that the 48 hour median effective concentration (EC_{50}) for the water flea (*D. magna*) and eastern oyster are 3.58 mg/L and 0.64 mg/LL, respectively. The acute toxicity to algae/aquatic plants data show the 72-hr EC_{50} for green algae is 14.9 mg/LL (biomass), while the 120-hr EC_{50} for the diatom, *Navicula* sp., is 2.35 mg/Ll (biomass), with a 14-day EC_{50} of 14.56 mg/LL for that aquatic plant, *Lemna gibba*. Chronic exposures using the fathead minnow estimated a no observable effect concentration (NOEC) of 0.0318 mg/L for the fathead minnow based on survival impacts. The chronic toxicity to aquatic invertebrates using *D. magna* reported a 21-day NOEC of 0.0701 mg/L.

b. Exposure and Risk

The dissipation of DCP from soil after application occurs primarily through volatilization, leaching, abiotic hydrolysis, and aerobic soil metabolism (USEPA, 2008). Field volatility studies with DCP have shown that 45 to 53 percent of the material volatilizes from the field within 14 days (Kim et al., 2003), while field dissipation half-life values range from 1 to 7 days (USEPA, 1998). The dissipation half-lives were based on application rates approximating the highest application rate allowed on the 24(c) label for Telone II® in the PCN program (354 lbs ai/ac). The initial half-life reported in a sandy loam was 1 day with a secondary half-life of 7 days at an application rate of 345 lbs ai/ac. The dissipation half-life in another study using a sandy soil was reported as 7 days when applying DCP at 342 lbs ai/ac. Laboratory metabolism half-life values in soil range from 12 to 54 days under aerobic conditions, but is much shorter under anaerobic soil conditions with a half-life of 2.4 to 9.1 days. Increased microbial degradation of DCP occurs with increasing temperature in most cases (Dungan and Yates, 2003). In aquatic systems, DCP volatilizes from water or can be degraded through hydrolysis. Hydrolysis half-lives are temperature dependent with reported half-lives of approximately 100 days at 2^{o} C, 13 days at 15^{o} C, and 2 days at 29^{o} C. Hydrolysis half-lives do not appear to be pH dependent with a reported half-life of 13.5 days for pH values of 5, 7, and 9 at a constant temperature of 20^{o} C (USEPA, 1998).

Increased light intensity and nitrogen dioxide concentration can greatly increase photodegradation of DCP (CalEPA, 2012). The atmospheric half-life via photodegradation is 7 to 12 hours (Dow AgroSciences, 2015). Plants such as bush beans, carrots, and tomatoes can absorb DCP from the soil. DCP absorbed by the plants is metabolized and converted into 3-chloroallyl alcohol and then to normal plant products. The isomers of DCP and 3-chloroallyl alcohol were generally non-detectable 120 hours after administration (Berry, 1980; CalEPA, 2012). DCP plant residues are not a concern after fumigation because of the rapid degradation of DCP in plants, and that crops are typically planted after most of the fumigant has dissipated (WHO, 1993; EFSA, 2009; CalEPA, 2012). In addition, the 24(c) label for DCP applications provides label restrictions allowing dissipation of DCP prior to planting which will further reduce the possibility of DCP uptake in plants.

DCP is mobile in soil and has high water solubility (2,800 mg/L at 20°C); however, due to the low rainfall in the area, the distance of the treated fields from surface water (approximately 0.25 miles), and the method of application, no residues are expected to occur via drift or runoff to aquatic water bodies. Site-soil characteristics and the location of the water table (50 to 60 feet (ft)) reduce the potential for DCP, or its metabolites, to contaminate groundwater through leaching. Data collected by the U.S. Geological Survey (USGS) in soil types similar to those in the area where the eradication program is being proposed demonstrated that DCP residues were at, or below, detection limits at 3 ft below the surface in a majority of the soils tested. One sampling site did have concentrations above detection at 3 ft below the soil surface, but levels were low (<3.0 parts per billion (ppb)) (USGS, 2000). The label for DCP requires 100-ft buffers adjacent to water wells and occupied structures, further reducing human health risks.

The potential exposure routes for DCP as a pre-plant soil fumigant include inhalation, incidental ingestion, and dermal contact for workers, and inhalation exposure as a result of DCP fumigant off-gassing for the general public who live or work in the vicinity of a fumigation site. The actual exposure to DCP for workers is reduced because of: protection by using personal protective equipment (PPE); engineering controls requirements such as a mechanical transfer system, end-row spillage control, and transferring Telone II® through connecting hoses, pipes, and/or couplings sufficiently tight with all bulk and non-bulk containers; and other label required mitigation measures. Other mitigation measures include best management techniques in the field, such as use of impermeable tarps and soil injection reaching at least 12 inches below the soil surface designed to protect workers and the public. Telone II® is a restricted use pesticide due to its acute inhalation toxicity and carcinogenicity, and is only used by certified applicators or persons under their direct supervisions. The Telone

II® label (DowAgroScience, 2012) includes specific requirements such as PPE for handlers, entry restriction, posting fumigant warning signs at entrances to treated areas, and a buffer zone to mitigate potential exposures to workers and the general public. Examples of specific label required restrictions to prevent exposure to the general public include:

- Telone II® should not be applied within 100 ft of an occupied structure (i.e., a school, hospital, business, or residence),
- No person shall be present at this structure at any time during the seven consecutive day period following application,
- Telone® II shall not be applied to soils more frequently than twice each year, and
- Individuals without proper training and PPE are prohibited to enter the area from the start of application until five days after application.

Consequently, human health risks from direct contact are low due to reduced exposure. APHIS personnel measured the atmospheric concentrations of DCP during fumigation treatments from August 14 to 28, 2008. Field measurements detected trace concentrations (0.1 ppm) of DCP, below the established regulatory threshold limits (USDA APHIS, 2008b). Additional monitoring in 2010 and 2011 demonstrated similar results with most residues at or below detection. The highest concentration measured in 2010–2011 was 3.5 ppm in one sample collected in the treatment field in 2010.

DCP exposure to terrestrial nontarget organisms can occur through direct or indirect exposure. The likelihood of direct exposure (other than to soil invertebrates in the treated fields) is low because DCP will not drift due to the method of application which involves injecting the material into the soil at a minimum depth of 12 inches. In compliance with the label, the soil will then be sealed by compaction after injection of DCP which serves to reduce volatilization (Wang et al., 2001). Plant residues of DCP from a cover planting that could serve as forage for nontarget organisms are not expected due to the lack of residues that have been determined in multiple crop residue studies (USEPA, 1998). A lack of measurable DCP residues in plants is related to the rapid degradation of DCP, and its dissipation after fumigation (WHO, 1993; CalEPA, 2012). The higher application rates that are part of the program are not expected to result in an increase in risk for most nontarget organisms based on the toxicity profile for DCP and the dissipation half-lives that have been reported to be less than 7 days at the higher proposed application rate. Screening level risk assessments for birds and mammals using an application rate of 342 lbs ai/ac have also demonstrated low risk to both groups from direct ingestion of contaminated food items (USEPA, 1998). The increased application rates could result in impacts to any soil-borne invertebrates that are within the

treated areas and were not impacted at the lower application rates. These impacts are expected to be localized to treated fields that are intensively managed agriculture production areas.

Field dissipation and degradation of DCP could result in soil residues that could be ingested by mammals and birds that serve as prey for predators and scavengers. The residues would be low due to the short dissipation half-life and method of application of DCP. Additionally, DCP residues from increased application rates are not expected to occur at levels that could impact predators and scavengers based on metabolism studies with DCP. Dosing studies with rats and mice show rapid excretion of DCP through the urine, indicating predators and scavengers would not accumulate significant DCP residues (USEPA, 2000; USEPA, 2008b). Therefore, indirect exposure via contaminated prey is not expected to occur based on the metabolism and environmental fate of DCP at the proposed rates.

c. Summary

DCP poses minimal risk to human health based on the method of application, label requirements for engineering controls and exposure prevention, and the lack of expected residues from any crop or in drinking water. The application site will also be posted to ensure no incidental human exposure occurs by accessing treated fields. The USEPA has also updated protection measures for all fumigants that are designed to further reduce the risk to human health (USEPA, 2012a–f). The current labels incorporate USEPA mitigation measures to reduce potential risks to fumigation workers and the general public. The mitigation measures for soil fumigants (as a restricted use pesticide) include a clear description of handler activities, training and on-site supervision, respirator protection, air monitoring, tarp perforation and removal, entry-restricted period, establishing and posting of a buffer zone (unless a physical barrier exists to prevent access to a buffer zone), good agricultural practices, emergency preparedness and response plans, notice to state and tribal lead agencies, and site-specific fumigant management plans.

The use of DCP also poses minimal risk to most nontarget organisms. Aquatic organisms will not be impacted because rainfall in the area is low and the application sites are far enough from any water source to minimize residues from drift, runoff, or leaching. Risk to nontarget terrestrial organisms at the higher proposed rates (other than soil invertebrates which are expected to succumb) is also minimal due to the method of application and environmental fate of DCP. Risk to human health and the environment is further reduced by other management practices such as soil injection during application, sealing the injection site to reduce offsite transport, and a 100-ft buffer around water wells and occupied structures.

E. Cumulative Effects

The cumulative effects discussed in this section are focused primarily on the preferred alternative. The no action alternative is the current program that includes the use of fumigants, such as MeBr. APHIS has not used MeBr in the PCN program since 2014. If MeBr were proposed for use in the future, it would be analyzed in a new NEPA document. The no eradication and no quarantine and no eradication alternatives are also not addressed in this section because they are not considered viable options.

Cumulative effects from the preferred alternative relate to the management actions in the proposed treatment area. The proposed areas of treatment are agricultural fields and will continue to be planted in crops based on any label restrictions, or may be planted in some type of cover planting; therefore, no cumulative impacts related to soil erosion are expected. A cover planting may be used in the winter; however, it will be dependent on whether environmental conditions allow the planting to establish prior to the end of the growing season. Historically, winter cover plantings are not used in this area; therefore, any soil erosion related to the preferred alternative is not expected to be any greater than would occur under typical agricultural practices in the area. Cumulative impacts from the use of the litchi tomato are expected to be negligible because these plantings will be restricted and managed by the ISDA, and require a weed management plan to prevent spread to other areas where control measures would be required. The weed management plan provides for herbicide use for fields that are planted in litchi tomato and for spot treatments for areas where litchi tomato is found outside of a planted field. In cases where litchi tomato is planted, there would be additional use of herbicides. However, these products are currently registered for use in Idaho for other agricultural and non-agricultural uses.

The potential cumulative impacts of DCP to aquatic resources are expected to be incrementally negligible. The label for DCP does contain a groundwater advisory; however, the soil conditions and depth to the water table reduce the likelihood of DCP moving into groundwater even with the additional proposed application and higher rates. The Telone II® 24(c) label does allow for applications twice per year, however, it will only be used if an accelerated treatment schedule is implemented that includes both a spring and a late summer/fall treatment. Only cover crops would be grown on a field receiving two treatments per year. The increased application rate and increased frequency of use is not expected to result in cumulative impacts to human health or the environment due to the method of application and rapid dissipation/degradation that has been reported at application rates approaching the maximum single rates proposed for the PCN program. DCP would also not be expected to have cumulative

impacts related to elevated nitrate levels that have been reported in groundwater in the area. DCP is regularly used in the area proposed for eradication; however, groundwater monitoring for DCP and its metabolites have shown no historical detections (USGS, 2000).

Based on the chemical properties of DCP, it will volatilize into the atmosphere. Additional DCP use does occur in Idaho primarily on potatoes, sugar beets and onions (figure 6).

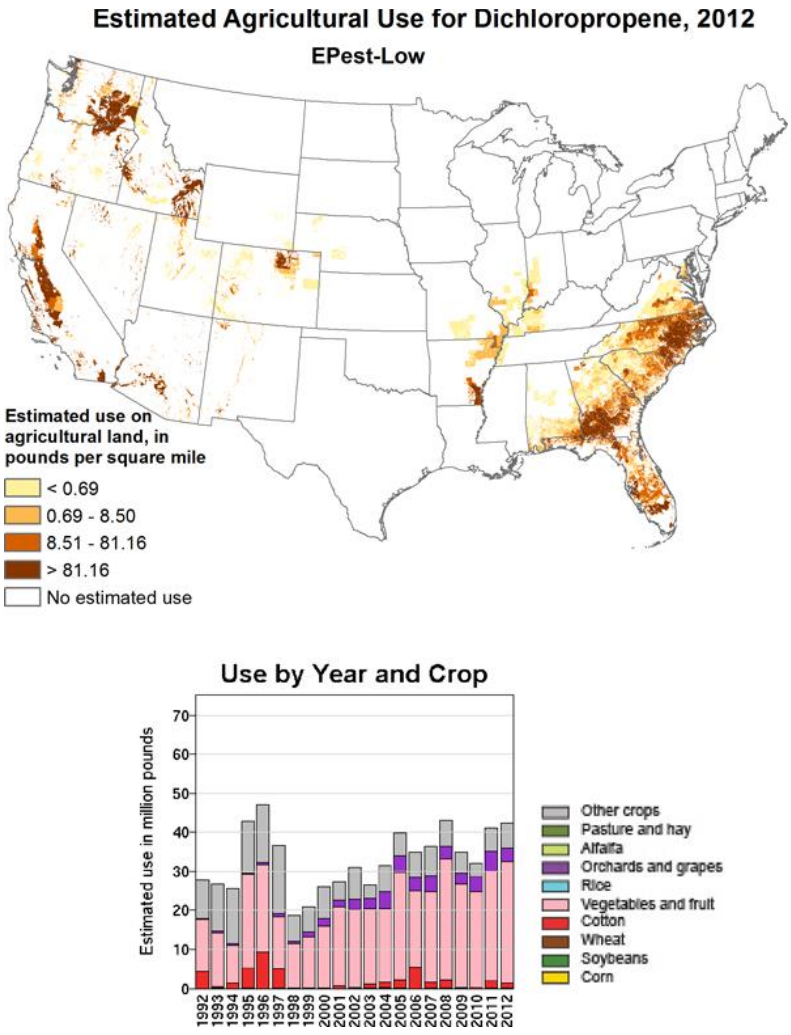

Figure 6. Estimated dichloropropene use in the United States. (Source: USGS, 2015. Method for Estimating Pesticide Use for County Areas in the Conterminous United States.)

Currently, neither sugar beets nor onions are grown near the PCN-infested fields. The cumulative impacts to air quality would be minimal due to temporal differences in the PCN-related DCP applications. For a majority

of uses, DCP is used as a pre-plant fumigant which would mean applications would occur just prior to the growing season in early spring or the previous fall. The projected applications for DCP in the PCN eradication program will typically occur late summer or early fall, at a time when any volatilized DCP from earlier applications would have dissipated and been dispersed by wind. Cumulative effects to air quality would be expected to be minimal due to efforts to minimize volatilization by soil injection and sealing the soil where injections occur.

F. Threatened and Endangered Species

Section 7 of the Endangered Species Act (ESA) and its implementing regulations require Federal agencies to ensure that their actions are not likely to jeopardize the continued existence of endangered or threatened species or result in the destruction or adverse modification of critical habitat.

Two federally threatened species occur in Bingham County, Idaho; the yellow-billed cuckoo (*Coccyzus americanus*) and its proposed critical habitat and the Ute ladies'-tresses (*Spiranthes diluvialis*). In Bonneville County, there are four threatened species, the yellow-billed cuckoo and Ute ladies'-tresses, as well as the Canada lynx (*Lynx canadensis*) and grizzly bear (*Arctos ursos horribilis*). The North American wolverine (*Gulo gulo luscus*) is proposed for listing as a threatened species and occurs in both counties.

Western yellow-billed cuckoo

Western yellow-billed cuckoo habitat is comprised of riparian trees including willow (*Salix* sp.), Fremont cottonwoods (*Populus fremontii*), alder (*Alnus* sp.), walnut (*Juglans* sp.), sycamore (*Platanus* sp.), boxelder (*Acer* sp.), ash (*Fraxinus* sp.), mesquite (*Prosopis* sp.), and tamarisk (*Tamarix* sp.) that provide cover, shelter, foraging, and dispersing habitat (U.S. Department of the Interior, 2014b). Critical habitat has been proposed in Bingham County, along the Snake River. This proposed critical habitat unit is 9,294 acres in extent and is a 22-mile long continuous segment of the Snake River from the upstream end of the American Falls Reservoir in Bannock County upstream to a point on the Snake River approximately two miles west of the Town of Blackfoot in Bingham County, Idaho (U.S. Department of the Interior, 2014a). This proposed critical habitat within Bingham County is approximately 25 miles from the current treatment area near Shelley, ID.

Ute ladies'-tresses

Ute ladies'-tresses is known from moist meadows associated with perennial stream terraces, floodplains, and oxbows at elevations between 4,300-6,850 feet, as well as seasonally flooded river terraces, sub-irrigated or spring-fed abandoned stream channels and valleys, and lakeshores. In addition, 26 populations have been discovered along irrigation canals, berms, levees, irrigated meadows, excavated gravel pits, roadside barrow pits, reservoirs, and other human-modified wetlands. (From: (U.S. Fish and Wildlife Service, Undated)). These plants would not be present within agricultural fields.

Canada lynx

The Canada lynx range extends south from the classic boreal forest zone into the subalpine forest of the western United States, and the boreal/hardwood forest ecotone in the eastern United States. In Bonneville County, the lynx would not likely be present in potato growing areas. The Targhee National Forest has known Canada lynx habitat, but program actions would not occur there.

Grizzly bear

Agricultural fields are not considered suitable habitat for grizzly bears because this land type does not contain adequate food resources to support grizzly bears (U.S. Department of the Interior, 2007).

North American Wolverine

In the contiguous United States, wolverine habitat is restricted to high-elevation areas in the West (FWS, 2011). Wolverines concentrate their year-round activities in areas that maintain deep snow into spring and cool temperatures throughout summer. Because wolverine habitat is generally inhospitable to human use and occupation and most of it is also federally managed, wolverines are somewhat insulated from impacts of human disturbances from agricultural activities (FWS, 2011).

Assessment

Activities under all four alternatives will have no effect on threatened species, species proposed for listing, or proposed or designated critical habitat in Bingham and Bonneville Counties. Restriction of movement of regulated articles would have no effect on threatened species or habitat. Fumigation activities occur in potato fields; however, potato fields do not provide habitat for the listed or proposed species in Bingham and Bonneville Counties. The fruits of litchi tomato are not toxic to animal

species, and listed animals would not be exposed to them because the trap crop would be destroyed prior to fruit ripening. Application of herbicides in fields planted with litchi tomato or spot treatments of litchi tomato outside of fields would not expose listed species to herbicides.

If additional species are federally listed, critical habitat is designated, or program activities change so that they could affect federally listed species, APHIS will initiate consultation with the U.S. Fish and Wildlife Service (FWS) as necessary. In particular, if treatments of fields will occur within one mile of yellow-billed cuckoo critical habitat, APHIS will initiate consultation with FWS.

G. Migratory Birds

The Migratory Bird Treaty Act of 1918 (16 United States Code (U.S.C.) 703–712) established a Federal prohibition, unless permitted by regulations, to pursue, hunt, take, capture, kill, attempt to take, capture or kill, possess, offer for sale, sell, offer to purchase, purchase, deliver for shipment, ship, cause to be shipped, deliver for transportation, transport, cause to be transported, carry, or cause to be carried by any means whatever, receive for shipment, transportation or carriage, or export, at any time, or in any manner, any migratory bird or any part, nest, or egg of any such bird.

Executive Order 13186, "Responsibilities of Federal Agencies to Protect Migratory Birds," directs Federal agencies taking actions with a measurable negative effect on migratory bird populations to develop and implement a memorandum of understanding (MOU) with the FWS which promotes the conservation of migratory bird populations. On August 2, 2012, an MOU between APHIS and FWS was signed to facilitate the implementation of this Executive order.

Bingham and Bonneville Counties occur within the Pacific flyway. This flyway extends from the Arctic tundra to South American wetlands. This flyway includes Alaska, Arizona, California, Idaho, Nevada, Oregon, Utah, and Washington; portions of Colorado, Montana, New Mexico, and Wyoming west of the Continental Divide; and the Canadian provinces of British Columbia and Alberta; and the Yukon and Northwest Territories. Migratory birds of conservation concern in the two counties are listed in Table 1.

Table 1. Migratory birds of conservation concern in Bingham and Bonneville Counties, Idaho (Service, 2015a; 2015b).

Common name	**Scientific Name**	**County**
Black rosy-finch	*Leucosticte atrata*	Bingham, Bonneville
Brewer's sparrow	*Spizella breweri*	Bingham, Bonneville
Calliope hummingbird	*Stellula calliope*	Bingham, Bonneville
Cassin's finch	*Carpodacus cassinii*	Bingham, Bonneville
Eared grebe	*Podiceps nigricollis*	Bingham, Bonneville
Ferruginous hawk	*Buteo regalis*	Bingham, Bonneville
Fox sparrow	*Passerella iliaca*	Bingham, Bonneville
Greater sage-grouse	*Centrocercus urophasianus*	Bingham, Bonneville
Green-tailed towhee	*Pipilo chlorurus*	Bingham, Bonneville
Lewis' woodpecker	*Melanerpes lewis*	Bingham, Bonneville
Loggerhead shrike	*Lanius ludovicianus*	Bingham, Bonneville
Long-billed curlew	*Numenius americanus*	Bingham, Bonneville
Olive-sided flycatcher	*Contopus cooperi*	Bingham, Bonneville
Peregrine falcon	*Falco peregrinus*	Bingham, Bonneville
Pinyon jay	*Gymnorhinus cyanocephalus*	Bingham
Sage thrasher	*Oreoscoptes montanus*	Bingham, Bonneville
Short-eared owl	*Asio flammeus*	Bingham, Bonneville
Swainson's hawk	*Buteo swainsoni*	Bingham, Bonneville
Williamson's sapsucker	*Sphyrapicus thyroideus*	Bonneville
Willow flycatcher	*Empidonax traillii*	Bonneville

Program activities will occur only within potato fields. Migratory birds would not be expected to be present within these fields during application. For the preferred and no action alternatives, DCP toxicity to birds is considered moderate. For the no action alternative, MeBr is considered moderately toxic to birds. The method of application, environmental fate of each fumigant, and areas of treatment suggest that migratory birds would be at low risk from exposure to PCN or MeBr fumigation treatments. As previously stated, MeBr is no longer part of the PCN program.

Litchi tomato fruits are not toxic to birds, although birds are known to disperse seeds of litchi tomato via ingestion of the ripe tomato-like fruit (USDA APHIS, 2013). However, birds will not be exposed to mature litchi tomato fruits because the trap crop will be destroyed prior to that growth stage of the plants.

H. Bald and Golden Eagle Protection Act

The Bald and Golden Eagle Protection Act (16 U.S.C. 668–668c) prohibits anyone, without a permit issued by the Secretary of the Interior, from "taking" bald eagles, including their parts, nests, or eggs. The act provides criminal penalties for persons who "take, possess, sell, purchase, barter, offer to sell, purchase or barter, transport, export or import, at any time or any manner, any bald eagle…[or any golden eagle], alive or dead, or any part, nest, or egg thereof." The Act defines "take" as "pursue, shoot, shoot at, poison, wound, kill, capture, trap, collect, molest or disturb."

In Idaho, large concentrations of wintering bald eagles are found along Lake Coeur d'Alene, Lake Pend Oreille, and sections of the Snake, Salmon and Boise Rivers. Although some nesting pairs remain in Idaho year-round, the winter population is supplemented by migrants from Canada. The bald eagle count in Idaho has ranged from 480 to 832 birds. In Bingham and Bonneville Counties, eagle nests are concentrated along the Snake River (Game, 2008).

Eagles are not likely to be disturbed by routine activities that pre-date the eagles' successful nesting activity in a given area, and ongoing existing uses can with the same intensity with little risk of disturbing bald eagles (Service, 2007a). Farming activities routinely occur in treated fields, and fumigations would be very similar to those activities in those fields. Therefore, eagles would not likely be disturbed by program activities under any of the four alternatives. The risk of exposure to fumigants would also be low based on the method of application and the expected lack of prey that would be present in treated fields.

V. Other Considerations

Executive Order (EO) 13175, "Consultation and Coordination with Indian Tribal Governments," was issued to ensure that there would be "meaningful consultation and collaboration with tribal officials in the development of Federal policies that have tribal implications…."

The Fort Hall Reservation occurs within Bingham County, a reservation for the Shoshone-Bannock Tribes. Currently, the eradication program does not occur on the Fort Hall Reservation; however, potato fields occur on the Reservation and could be affected should PCN spread.

APHIS prepared a letter and sent it to the Shoshone-Bannock Tribes, describing the program, and requesting input regarding potential effects on the Tribes, and an invitation for consultation. Federal and State agriculture

officials will continue to collaborate with Indian tribal officials to ensure that they are well-informed and represented in policy and program decisions that may impact their agricultural interests.

Executive Order (EO) 12898, "Federal Actions to Address Environmental Justice in Minority Populations and Low-income Populations," focuses Federal attention on the environmental and human health conditions of minority and low-income communities and promotes community access to public information and public participation in matters relating to human health or the environment. This EO requires Federal agencies to conduct their programs, policies, and activities that substantially affect human health or the environment in a manner so as not to exclude persons and populations from participation in or benefiting from such programs. It also enforces existing statutes to prevent minority and low-income communities from being subjected to disproportionately high or adverse human health or environmental effects.

Using U.S. Census Bureau estimates, in Bingham County, 16 percent of the population speaks a language other than English at home, but only 6.8 percent of the population report speaking English less than "very well" (Bureau, 2013). Approximately 14 percent of Bingham County residents are considered persons in poverty (Bureau, 2014). The population reporting their race as Black is 0.5 percent, Asian as 0.7 percent, Hispanic or Latino as 17.6 percent, and American Indian and Alaska Native as 7.4 percent (Bureau, 2014).

In Bonneville County, approximately 11 percent of its residents are considered persons in poverty (Bureau, 2015). The population reporting their race as Black is 0.7 percent, Asian as 1 percent, Hispanic or Latino as 12.4 percent, and American Indian and Alaska Native as 1.1 percent (Bureau, 2015). Only 3.8 percent of the population report speaking English less than "very well" (U.S. Census Bureau, 2013).

The demographic information does not suggest low-income and minority residents would require additional outreach to ensure adequate understanding of the program. Consequently, APHIS finds additional outreach to these segments of the population is not needed. Because the preferred alternative is to apply fumigants in privately-owned potato fields, these segments of the population are not likely to be disproportionately adversely affected by the treatment. APHIS has determined that the environmental and human health effects from the proposed changes in applications for eradication of PCN in Idaho are minimal and are not expected to have disproportionate adverse effects to any minority or low-income populations.

EO 13045, "Protection of Children from Environmental Health Risks and Safety Risks," acknowledges that children, as compared to adults, may suffer disproportionately from environmental health and safety risks because of developmental stage, greater metabolic activity levels, and behavior patterns. This EO (to the extent permitted by law and consistent with the agency's mission) requires each Federal agency to identify, assess, and address environmental health risks and safety risks that may disproportionately affect children. Applications will follow label requirements designed to reduce risk if infested fields are in proximity to schools, parks, or day care facilities where children may be present. In addition, the method of application and management of the fields will minimize residues from drift, volatilization, and dietary exposure. Based on the distance of the application area from surface and groundwater resources, no residues from any of the proposed fumigants would be expected in drinking water. The preferred alternative is not expected to have disproportionately high or adverse human health or environmental effects to children.

Consistent with the National Historic Preservation Act of 1966, APHIS has examined the proposed action in light of its impacts to national historic properties. Several historic sites exist within the current quarantine as well as the counties (table 2), but treatments will occur in potato fields and these will not impact historic properties. Treatments for PCN on historic properties are not anticipated at this time. In the event that future treatments could occur on historic properties they would be coordinated with the State Historic Preservation Officer and other appropriate contacts.

Table 2. Historic sites within Bingham and Bonneville Counties, Idaho (National Register of Historic Places, 2015 http://www.nps.gov/nr/research/index.htm)

Historic Site	County	Address	City
Art Troutner Houses Historic District	Bonneville	3950, 4012 and 4032 S. 5th W.	Idaho Falls
Beckman, Andrew and Johanna M., Farm	Bonneville	US 20 0.5 mi. W of jct. with New Sweden Rd.	Idaho
Beckman, Oscar and Christina, Farmstead	Bonneville	SW corner of jct. of New Sweden--Shelley Rd. and US 20	Idaho Falls
Bonneville County Courthouse	Bonneville	Capital Ave. and C St.	Idaho Falls
Bonneville Hotel	Bonneville	400 Blk W. C St.	Idaho Falls
Douglas-Farr Building	Bonneville	493 N. Capital Ave.	Idaho Falls
Eagle Rock Ferry	Bonneville	N of Idaho Falls on Snake River	Idaho Falls
Eleventh Street Historic District	Bonneville	Roughly bounded by S. Boulevard, 13th, 10th, and 9th Sts., S.	Idaho Falls

		Emerson and S. Lee Aves.	
Farmers and Merchants Bank Building	Bonneville	383 W. A St.	Idaho Falls
First Presbyterian Church	Bonneville	325 Elm St.	Idaho Falls
Hasbrouck Building	Bonneville	362 Park Ave.	Idaho Falls
Holy Rosary Church	Bonneville	288 E. Ninth St.	Idaho Falls
Hotel Idaho	Bonneville	482 W. C St.	Idaho Falls
I.O.O.F. Building	Bonneville	393 N. Park Ave.	Idaho Falls
Idaho Falls Airport Historic District	Bonneville	2381 Foote Dr.	Idaho Falls
Idaho Falls City Building	Bonneville	303 W. C St.	Idaho Falls
Idaho Falls Public Library	Bonneville	Elm and Eastern Sts.	Idaho Falls
Iona Meetinghouse	Bonneville	In Iona	Iona
Kress Building	Bonneville	451 N. Park Ave.	Idaho Falls
Montgomery Ward Building	Bonneville	504 Shoup Ave.	Idaho Falls
New Sweden School	Bonneville	SW corner of jct. of New Sweden School Rd. and Mill Rd.	Idaho Falls
Ridge Avenue Historic District	Bonneville	Roughly bounded by N. Eastern Ave., Birch St., S. Blvd., Ash St., W. Placer Ave. and Pine St.	Idaho Falls
Rocky Mountain Bell Telephone Company Building	Bonneville	246 W. Broadway Ave.	Idaho Falls
Sealander, Carl S. and Lizzie, Farmstead	Bonneville	W end St. John Rd.	Idaho Falls
Shane Building	Bonneville	381 N. Shoup Ave.	Idaho Falls
Shelton L.D.S. Ward Chapel	Bonneville	SW of Ririe on Shelton Rd	Ririe
Trinity Methodist Church	Bonneville	237 N. Water Ave.	Idaho Falls
U.S. Post Office	Bonneville	581 Park Ave.	Idaho Falls
Underwood Hotel	Bonneville	343-349 W. C Street	Idaho Falls
Wasden Site (Owl Cave)	Bonneville	Address Restricted	Idaho Falls
Blackfoot I.O.O.F. Hall	Bingham	57 Bridge St.	Blackfoot
Blackfoot LDS Tabernacle	Bingham	120 S. Shilling St.	Blackfoot
Blackfoot Railway Depot	Bingham	Main St., NW	Blackfoot
Eastern Idaho District Fair Historic District	Bingham	97 Park Dr.	Blackfoot
Fort Hall Site	Bingham	16 mi. N of Fort Hall	Fort Hall
Idaho Republican Building	Bingham	167 W. Bridge St.	Blackfoot
Jones, J. W., Building	Bingham	104 Main St., NE	Blackfoot

Lincoln Creek Day School	Bingham	Rich Ln., eight mi. SE of St. Hwy. 91	Fort Hall
North Shilling Historic District	Bingham	N. Shilling Ave.	Blackfoot
Nuart Theater	Bingham	195 N. Broadway	Blackfoot
Ross Fork Episcopal Church	Bingham	Mission Rd.	Fort Hall
Ross Fork Oregon Short Lines Railroad Depot	Bingham	Agency Rd.	Fort Hall
Shilling Avenue Historic District	Bingham	Shilling Ave. between E. Idaho and Bingham Sts. and Bridge and Judicial Sts. to Stout Ave.	Blackfoot
St. Paul's Episcopal Church	Bingham	72 N. Shilling Ave.	Blackfoot
Standrod Bank	Bingham	59 and 75 Main St., NW	Blackfoot
US Post Office--Blackfoot Main	Bingham	165 W. Pacific	Blackfoot

VI. Listing of Agencies and Persons Consulted

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Plant Protection and Quarantine
Plant Health Programs
4700 River Road, Unit 134
Riverdale, MD 20737

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
Policy and Program Development
Environmental and Risk Analysis Services
4700 River Road, Unit 149
Riverdale, MD 20737

U.S. Department of Agriculture
Animal and Plant Health Inspection Service
State Plant Health Director
9118 W. Blackeagle Drive
Boise, ID 83709

Idaho PCN Program Director
U.S. Department of Agriculture
Animal and Plant Health Inspection Service
2281 West Heyrend Way
Idaho Falls, ID 83042

Bureau Chief
Plant Industries Division
Idaho State Department of Agriculture
P.O. Box 790
Boise, ID 83701

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Appendix A: Potential Host Plants for G. pallida

Bold= confirmed in the literature
Non Bold = listed in either CABI Compendium or GPDD

Note: Most papers were prepared before *Globodera pallida* was distinguished from *G. rostochiensis*. Many older papers refer to the potato cysts nematodes as a strain of *Heterodera schachtii*.

Primary Hosts:
Lycopersicon esculentum (tomato)
Solanum melongena (eggplant, aubergine)
Solanum tuberosum (potato)
Capsicum spp.

Minor Hosts:
Datura stramonium (Devil's trumpet, Jamestown-weed)
Lycopersicon pimpinellifolium (currant tomato) (syn. **Lycopersicon racemigerum)**
Oxalis tuberosa (oca)
Solanum aviculare (kangaroo apple)
Solanum gilo (syn. Solanum integrifolium) (scarlet or tomato eggplant)
Solanum indicum (Indian nightshade)
Solanum marginatum (white-edged (margined) nightshade)
Solanum mauritianum (tree tobacco, earleaf nightshade)
Solanum nigrum (black nightshade) (Winslow (1954) found as a non-host, appears there are multiple varieties that vary in susceptibility/resistance (Scholte (2000)).
Solanum quitoense (Naranjillo)
Solanum sarrachoides (hairy nightshade)

Other hosts:
Atropa belladonna? (deadly nightshade) - Reported as a host by Franklin (1940), Found to be negative by Winslow (1954)
Datura tatula (jimsonweed)
Hyoscyamus niger (black henbane)
Lycopersicon esculentum aureum
Lycopersicon glandulosum (Peruvian nightshade)
Lycopersicon hirsutum (hairy tomato)
Lycopersicon mexicanum
Lycopersicon peruvianum (wild tomato)
Lycopersicon pyriforme (garden tomato)
Physalis philadephica (Mexican groundcherry)
Physochlainia orientalis (purple trumpet flowers)
Salpiglossis spp. (painted tongue)
Saracha jaltomata

Other *Solanum spp*.
Solanum acaule (Wild Andean potato)
Solanum aethiopicum (Ethiopian nightshade, African eggplant)
Solanum ajanhuiri (Ajanhuiri)
Solanum alandiae
Solanum alatum (red fruited nightshade)
Solanum americanum (American black nightshade)
Solanum anomalocalyx
Solanum antipoviczii *(now* ***S. stoloniferum*****)**
Solanum armatum (forest nightshade)
Solanum ascasabii
Solanum asperum
Solanum berthaultii (wild potato)
Solanum blodgettii (mullein nightshade)
Solanum boergeri
Solanum brevimucronatum
Solanum brevidens (wild potato-diploid)
Solanum bulbocastanum – (ornamental nightshade) - also listed as *S. bulbocastana*
Solanum calcense
Solanum calcense x Solanum cardenasii
Solanum caldasii
Solanum canasense
Solanum capsibaccatum
Solanum capsicoides (cockroach berry)
Solanum cardiophyllum (heartleaf horsenettle)
Solanum carolinense (Carolina horsenettle)
Solanum chacoense – (Chaco potato) also reported as *S. chacoense* v. *subtilis*
Solanum chaucha
Solanum chenopodioides
Solanum chloropetalum
Solanum citrullifolium (watermelon nightshade) – also listed as *S. citrillifolium*
Solanum coeruleifolium (chaucha)
Solanum commersonii (Commerson's nightshade)
Solanum curtilobum (rucki)
Solanum curtipes
Solanum demissum (nightshade)
Solanum demissum x Solanum tuberosum
Solanum dulcamara (bittersweet)
Solanum durum
Solanum elaeagnifolium (silverleaf nightshade)
Solanum famatinae
Solanum fraxinifolium
Solanum fructo-tecto
Solanum garciae

Solanum gibberulosum
Solanum giganteum (African holly)
Solanum gigantophyllum
Solanum glaucophyllum (waxyleaf nightshade)
Solanum goniocalyx (yellow potato)
Solanum gracile (whitetip nightshade)
Solanum heterodoxum (melonleaf nightshade)
Solanum heterophyllum (unarmed nightshade)
Solanum hirtum (huevo de gato)
Solanum hispidum (devil's fig)
Solanum intrusum (garden huckleberry)
Solanum jamesii (wild potato)
Solanum jujuyense
Solanum juzepczukii (ckaisalla)
Solanum kesselbrenneri (phureja)
Solanum kurtzianum
Solanum lanciforme (heartleaf nightshade)
Solanum lapazense
Solanum lechnoviczii
Solanum leptostygma (potato)
Solanum longipedicellatum *(now S. stoloniferum)*
Solanum luteum (red-fruited nightshade)
Solanum macolae
Solanum macrocarpon (African eggplant)
Solanum maglia
Solanum mamilliferum (chauca)
Solanum miniatum (red-fruited nightshade)
Solanum multidissectum
Solanum muricatum (pepino melon)
Solanum nitidibaccatum (Argentinian nightshade)
*Solanum ochroleucum (*syn. *S. nigrum)*
Solanum ottonis (divine nightshade)
Solanum pampasense
Solanum parodii
Solanum penelli
Solanum phureja (chauca)
Solanum photeinocarpum (terimini inuhoozuki)
Solanum pinnatisectum (tansyleaf nightshade)
Solanum platypterum
Solanum platense
Solanum polyacanthos
Solanum polyadenium (potato)
Solanum prinophyllum (forest nightshade)
Solanum radicans (cusmayllo)
Solanum raphanifolium (wild potato)
Solanum rostratum (buffalobur nightshade)

Solanum rybinii (phureja)
Solanum salamanii
Solanum saltense
Solanum sambucinum
Solanum sanctae-rosae
Solanum scabrum
Solanum schenkii
Solanum schickii
Solanum semidemissum
Solanum simplicifolium
Solanum sinaicum (nightshade)
Solanum sodomaeum (apple of Sodom)
Solanum soukupii
Solanum sparsipilum
Solanum stenotomum (pitiquina)
Solanum stoloniferum
Solanum suaveolens
Solanum subandigenum (Andigena)
Solanum sucrense
Solanum tarijense
Solanum tenuifilamentum (chauca)
Solanum tomentosum
Solanum toralopanum (apharuma)
Solanum triflorum (cutleaf nightshade)
Solanum tuberosum ssp. andigena (potato)
Solanum tuberosum ssp. tuberosum (Irish potato)
Solanum tuberosum 'Aquila',
Solanum tuberosum ' Xenia N'
Solanum utile- South American genus-strongly attacked
Solanum vallis-mexicae
Solanum vernei (purple potato)
Solanum verrucosum
Solanum villosum (red-fruited nightshade)
Solanum violaceimarmoratum
Solanum wittmackii
Solanum wittonense
Solanum xanti (chaparral nightshade)
Solanum yabari (pitiquina)
Solanum zuccagnianum (gilo)

Web Resources:

CABI Crop Compendium. www.cabicompendium.org
Extensive list of hosts. List *Salpiglossis spp.* that are actually *Solanum spp.*

Global pest and disease database. https://www.gpdd.info.
Extensive list of hosts.

HYPP Zoology. *Globodera rostochiensis* (Wollenweber, (U.S. Department of Agriculture)
http://www.inra.fr/Internet/Produits/HYPPZ/RAVAGEUR/6gloros.htm

This species exclusively parasitizes the Solanaceae, especially potato, tomato, egg plant and a few volunteer plants such as bittersweet (*Solanum dulcamara*) and henbane (*Hyoscyamus niger*).

Society of Nematologists. *Globodera pallida*. http://nematode.unl.edu/pest5.htm

Potato (*Solanum tuberosum*) is the major host. Other hosts include many *Solanum* species, oca (*Oxalis tuberosa*), Jamestown-weed (*Datura stramonium*), tomato (*Lycopersicon* spp.), and *Salpiglossis* spp.

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Appendix B. Response to Comments for the Supplemental Environmental Assessment

Comment: There should be mandatory intercropping with sticky nightshade (*Solanum sisymbriifolium*, litchi tomato) until eradication is verified, and lost yields should be subsidized.

Response: Infested field treatments using litchi tomato are not required by the PCN program, and treatments are allowed to proceed by the Idaho State Department of Agriculture (ISDA) only after written and notarized consent for the treatment is provided by a field's owner. In cases where a landowner has consented to planting litchi tomato, the APHIS PCN program will enter into an agreement to reimburse landowners for certain services attendant to the planting, maintenance, and destruction of the litchi tomato crop. Yields that are lost due to the planting of litchi tomato are not part of the reimbursement plan.

Comment: A commenter stated that methyl bromide (MeBr) should never be used again in Idaho with the totally impenetrable film practices that were used in the past 10 years. Not enough time was spent studying the effects that MeBr would have on soil and on future crops after the MeBr applications. The consequences of the use of MeBr in Bingham and Bonneville Counties may not fully be known for many years. Links may not be made with MeBr in respect to many cancers; however, studies suggest that there is a risk associated with MeBr for stomach cancer. There needs to be continued monitoring of soil, water, and foliage to protect people as well as animals in this area.

Response: APHIS is not proposing that methyl bromide be used in the PCN program at this time. As stated in the draft supplemental environmental assessment (SEA), any proposed use of methyl bromide in the future by the PCN program would require additional analysis and NEPA documentation with input from the public.

Comment: Litchi tomato is a noxious weed. Although at the beginning of the research many believed that this would be a silver bullet to solve the PCN problem, after several years of research, the benefits do not outweigh the risks of bringing in a noxious weed that can spread throughout the area. The area experiences severe winds at times and the chances of spreading the seeds of litchi tomato are great. The commenters believe that the loss of a crop for an entire year is a huge stumbling block for the success of using litchi tomato. Economic costs are part of the consideration when making an environmental assessment. Litchi tomato has to have 14 weeks of growth and be at 25 pounds of biomass per square foot to achieve the needed mass to get PCN to hatch. Even with that much biomass, they were only able to get 50% of all PCN to hatch. Although University of Idaho researchers believe that the litchi tomato won't spread seeds when the fruit is green, it would have red fruits in order for it to reach the biomass needed to cause a hatch of PCN. Because of the extraordinary risk posed by introduction of invasive

species to control environmental problems the Final SEA should, for instance, analyze whether litchi tomato could out-compete farmed crops resulting in an economic and ecological catastrophe.

Response: Litchi tomato is not categorized as a noxious weed by either ISDA or APHIS but is listed as an invasive plant species by ISDA because it is not native to the state. Litchi tomato has been shown to be a potentially effective eradication tool in the treatment of PCN. Litchi tomato almost entirely eliminated reproduction of PCN on a succeeding potato crop in studies under greenhouse conditions conducted at the University of Idaho. PCN cyst numbers decreased between 70–95 percent (compared to original cyst numbers) when potato was planted following litchi tomato, whereas PCN increased by 340 percent in a potato following potato sequence. In field-scale trials planted on PCN-infested fields, litchi tomato reduced PCN viability to below detection levels after one planting.

While it is true that a field is taken out of commercial production during the crop year in which litchi tomato is grown, this occurs at the request and full consent of the field owner. Landowners are reimbursed for certain services attendant to the planting, maintenance, and destruction of the litchi tomato crop. While this is not the same as profiting from a commercial crop, there is additional value in receiving a treatment that will reduce the viable PCN population in a field.

USDA APHIS, in cooperation with the ISDA and University of Idaho, has developed a weed management plan to protect areas from the establishment of litchi tomato. This plan takes into account potential inadvertent movement by wind, animals and/or equipment. Planted litchi tomato will be monitored every two weeks for emergence and plant development thresholds. ISDA will be notified immediately at germination, flowering, and the first sign of berry development. The field operator will be required to use a chemical application or flail the crop when at least 30 percent of the crop is producing green (immature) berries, or when one or more ripening berries (turning from green to orange-red) have been identified at any time regardless of the percentage of green berries present. Once the crop has reached the trigger for destruction, the crop will be treated with an herbicide, flailed five to ten days post treatment, and plant residues incorporated into soil within 10 days following flailing. Fields will be monitored for regrowth and if any is observed the process will be repeated.

Following planting and prior to leaving the field, all equipment will be visually inspected by APHIS PCN program staff for litchi tomato seed. Monitoring outside the planted fields will also occur every two weeks throughout the growing season. This information and additional information about the weed management plan has been added to the final supplemental EA.

Monitoring of fields planted to litchi tomato and those surrounding areas are also critical parts of the weed management plan. The plan includes requirements for the year planted as well as an additional 5 years thereafter. During the planting year, required monitoring every two weeks includes field perimeter fence lines, access roads, ditches, canals, and approximately 50 feet into neighboring fields. Similar monitoring of fields planted to litchi tomato occurs for the following five years. In addition, crop selections for previously planted fields are required to be of appropriate height to effectively monitor for volunteer plants. Acceptable crops include small grains, hay, canola and potatoes.

Comment: The preferred alternative of the draft SEA states that litchi tomato has already been planted in PCN-infested fields (page 8). The Final SEA should tier to or incorporate by reference the NEPA analysis that preceded the decision to use an invasive species as a PCN control mechanism.

Response: The University of Idaho and USDA Agricultural Research Service, in cooperation with APHIS, planted litchi tomato in three small research plots to determine agronomic requirements in southeast Idaho, optimal timing of planting and crop destruction, rooting depth, above- and below-ground biomass production, herbicide susceptibility, and efficacy against PCN prior to recommending its use for the PCN eradication program. NEPA analysis regarding these research plots was not prepared; however, the small areas where planting occurred with most plots less than one acre, and three fields at 22, 34 and 75 acres respectively, meet the criteria for categorical exclusion based on the APHIS NEPA implementing regulations (7 CFR, part 372).

Comment: References are made to a University of Idaho weed management plan to prevent litchi tomato infestation but no details of the environmental impacts of this plan are provided nor is there any support for the statement that herbicides labeled for use in potatoes and other crops in southeast Idaho are sufficient to control litchi tomato. The Final SEA should explain and analyze what herbicides would be available to kill litchi tomato that escapes from the controlled planting areas.

Response: The supplemental EA has been updated to clarify how litchi tomato will be destroyed and what herbicides will be used based on recommendations from the University of Idaho and the USDA Agriculture Research Service.

Comment: One solution that was never discussed in the SEA dated April 2016 was that of growing a cover crop on the ground such as pasture grass and removing the ground from production. This option provides one of the best and safest ways to control PCN. If grass was grown, soil wouldn't be tilled and the chances for PCN to move would be severely limited. This option would be the most cost effective and environmentally friendly. Further consideration be given to this option.

Response: The use of a cover crop such as pasture grass can be an effective long-term means of PCN eradication under certain conditions. APHIS agrees that use of a cover crop such as pasture grass along with no-till practices would significantly reduce the probability of spreading PCN to other fields and over time would eradicate PCN from a given field. It is permissible for a landowner to exercise this option under the current program; however, there is currently no mechanism for APHIS or ISDA to reimburse the landowner for their lost revenue or weed control costs while a field remains out of crop production. For a successful eradication, the use of a cover crop would require the landowner to control any potato volunteers and/or solanaceous weeds that may host PCN, for a minimum of 30 years. Also, any animals that may graze these areas would be at risk for carrying soil out of the field and would be considered a regulated article that would require inspection and/or remediation prior to being moved from a field where

a cover crop is being used. Information regarding the use of a cover crop has been added to the SEA.

Comment: There are many possible biological remediation options available to combat PCN that could include a reasonable alternative. Using fungi to control PCN has been shown to be effective through the efforts of Instituto de Ecología (INECOL, in Mexico), who have identified a fungus that feeds on the PCN and eliminates the need for chemicals and improves crop yields, reducing cysts below the Economic Injury Level, from 6000 cysts per kg to 40 cysts per kg. While INECOL's methods are not available while they pursue a patent, timely investigation could find a solution appropriate for local use. It may be in the best interest of the USDA to investigate and implement possible bioremediation solutions.

Response: No fungal agents are currently registered for use to control PCN. APHIS continues to evaluate new eradication technologies and implement those technologies as they become available. If a new technology is identified it will be analyzed in future NEPA documents.

Comment: A commenter stated that methyl bromide does not pose a minimal risk. In a research article by H. Haldin-Davis, M.D. OXF, F.R.C.S. Eng., the author discusses "Bromide Eruptions" in humans. Halden-Davis indicates that "the salient characteristic of all cutaneous bromodermia is the production of puss," and that "Bromodermia in infants nursed by epileptic mothers who are taking the drug is well known". The commenter stated that they have observed and treated Bromodermia in cattle after being fed hay with inorganic bromide residue. The only way to allow healing is to lance the lesion and drain the puss creating an open wound. These lesions are caused by ingestion of inorganic bromide and in our world they are not a "minimal risk". The commenter stated that bromide levels pass through the cow in utero to her unborn calf, leaving the calf with the same symptoms as quoted in Knight and Costner, 1977 (lethargy, weakness and ataxia) which leads to death in newborn cattle. The commenter has also observed that bromide levels pass through the cow through her milk causing the same symptoms in her nursing calf.

Response: APHIS is not currently proposing to use MeBr in the PCN program. APHIS summarized the risk of MeBr in the appendix of the SEA with updated information about the various effects of MeBr and the bromide ion to various mammal species and other nontarget organisms. The assessment recognizes the potential adverse effects to livestock from feeding forage with elevated levels of bromide.

Comment: Within the Purpose and Need statement, APHIS states that it undertook action in response to discovery of PCN in two fields in June and July of 2006 and that an EA was then prepared in May 2007 to address "potential" action. From the Purpose and Need statement, it is clear that the agency had taken action prior to May 2007 such that the May 2007 EA followed, and did not precede, some of APHIS's actions in response to the 2006 discovery of PCN. This incongruity should be acknowledged and explained in the Final SEA. NEPA requires consideration of environmental effects of agency actions prior to those actions.

Response: APHIS proposed an eradication program in 2007. That program was analyzed in the May 2007 Environmental Assessment. APHIS activities prior to the proposed eradication program included establishing a quarantine which was categorically excluded under APHIS' NEPA implementing regulations (7 CFR, part 372). This has been clarified in the SEA.

Comment: The Purpose and Need statement contains the odd sentence that "APHIS prepared an amended EA in July 2007 to discuss how the proposed changes in use may have affected the quality of the human environment." If the amended EA preceded proposed changes that had not been implemented, then it is grammatically incorrect to say that the changes "may have affected," i.e., in the past tense, the environment. If the July 2007 amended EA followed the implementation of the changes, then it is incorrect to label those changes as "proposed" changes that would occur in the future. APHIS should clarify whether the July 2007 amended EA preceded or followed changes in the treatment program.

Response: The SEA was updated and the language "may have affected" was changed to "would affect" in the Purpose and Need section.

Comment: In the Purpose and Need statement, APHIS describes background information including previous NEPA documentation and changes in APHIS's protocols for the PCN program. Specially, under the heading "Previous NEPA Documentation," page 5, APHIS describes NEPA documents including FONSIs that are not available on the APHIS PCN website. The Final SEA must provide citations and brief descriptions if this SEA is incorporating other NEPA documents by reference pursuant to 40 C.F.R. § 1502.21 or tiering to other NEPA documents pursuant to 40 C.F.R. § 1502.20. If the incorporated material is not reasonably available to during this comment period, it may not be incorporated by reference. § 1502.21. Any tiered material must be summarized in the SEA and state where the material is available. § 1502.20. It is unclear whether the citations to previous NEPA documentation are intended to adopt those NEPA documents as part of the SEA.

Response: The reference to other NEPA documents in the SEA are not considered tiered material but was made to summarize previous NEPA work related to the PCN program and explain the need for a SEA.

Comment: The final SEA should state that it will not eliminate the need for a NEPA document each time there is an expansion or reduction of the regulated area in the two counties if (1) that expansion or reduction has the potential to create environmental impacts not previously analyzed in the Final SEA, (2) those changes would cause significant impacts to the human environment, or (3) the requirements for supplementation found at 40 C.F.R. § 1502.9(c) are otherwise met.

Response: The SEA was updated to state that if the program expands or is reduced that APHIS will re-evaluate whether there are any potential impacts that would require further NEPA analysis.

Comment: The purpose statement is overly broad. No rationale is provided for expanding the PCN program "to all potato growing areas in Bingham and Bonneville Counties should PCN be found in new areas." If PCN is found in new areas, the program could be expanded to those areas but not to "all" areas. Nor is "growing areas" defined. Is it current or potential areas in the counties where potatoes are grown or could theoretically be grown? An overly broad purpose statement unnecessarily expands and confuses the scope of the analysis.

Response: The purpose statement was updated to state that growing areas include any potato fields that are or could become infested with PCN. This does not mean that all potato growing areas are being brought under APHIS regulation but that in the event that an additional field is found to be infested, APHIS may not have to prepare another SEA after evaluation of the potential impacts and whether they differ from those described in this SEA.

Comment: The introduction to the "Alternatives" section states that the proposed action is "to eradicate PCN from fields in Idaho where the nematode has been detected." APHIS's proposed action does not appear to differ from APHIS's existing actions taken since the discovery of PCN in 2006. If the proposed action is not different from APHIS's previous actions over the last ten years, then (1) the proposed action must be redefined as an action that is different from that which has already been undertaken or (2) the Final SEA is meaningless as it analyzes the status quo.

Response: The proposed action to eradicate PCN differs between the original EA and the supplemental EA in the methods used to achieve eradication. The SEA provides an update to the original EA regarding new methods, such as using litchi tomato as a trap crop, a no quarantine and no treatment option and discontinuing the use of MeBr fumigation as a treatment option.

Comment: The agency's labels for its action alternatives are confusing. The no action alternative would maintain APHIS's current action through continuation of the PCN eradication program. The Federal District Court in Idaho has explained that no action means no action such that the current agency program would not be continued. Consequently, it appears that APHIS's Alterative B, no eradication, is closer to a no action alternative and the no action Alternative A is a continuation of the status quo. Even under Alternative B, APHIS would continue to take action by maintaining the federal domestic quarantine and regulating interstate movement of regulated articles. Consequently, there does not appear to be a true no action alternative.

Response: NEPA guidance and policy allows flexibility in how the alternatives may be described in an environmental assessment or environmental impact statement. In the case of this SEA, the no action alternative was defined as the “status quo” which is the current program that was described in the 2007 amended EA. A no eradication or quarantine alternative was added to the SEA as a means to discuss the impacts if no Federal program was in place.

Comment: Alternative C, the Preferred Alternative, discusses phytosanitary requirements that are in place to prevent PCN from artificially spreading from treated fields as well as protocols

and requirements that growers must follow to prevent the spread of PCN. Because the sole purpose of these requirements and protocols is to affect the environment where PCN might exist, they should be fully disclosed and analyzed as part of the Preferred Alternative and the environmental consequences that flow from that alternative. Reference is also made at pages 7 and 8 to surveillance and phytosanitary actions in the 2014 United States and Canada guidelines that outline mandatory phytosanitary measures. There is no tiering or incorporation by reference to previous NEPA documents that analyzed the impact of those actions on the environment. If there is no previous NEPA documentation, then the effect of those federal actions incorporating the United States and Canada guidelines must be disclosed and analyzed in the Final SEA. If there is existing NEPA analysis of those actions and measures, then that analysis should have been disclosed in the Draft SEA.

Response: The SEA has been updated to summarize aspects of the quarantine and information from the United States and Canadian guidelines. A summary of these measures is in the Alternatives chapter under the preferred alternative description in this SEA. The implementation of a quarantine has been categorically excluded under APHIS' NEPA Implementing Regulations (7 CFR, part 372). The SEA has been updated to state that quarantine measures are categorically excluded from further NEPA analysis.

Comment: The Preferred Alternative discusses use of DCP and covering the injection sites with a totally impenetrable film (page 8). The Final SEA should discuss whether DCP application has previously been covered with film and tier to the NEPA analysis for that program.

Response: APHIS is not proposing to cover DCP treated fields with a tarp. The supplemental EA has been updated to reflect that a tarp will not be used.

Comment: The Preferred Alternative discusses monitoring during fumigation of infected fields and lists various factors that are monitored in that process but noticeably omits monitoring of groundwater or foliage tests to determine whether fumigation levels are becoming toxic. No matter what program is adopted and used, the commenters request that soil, water, and plant samples be taken and monitored to prevent a problem in the future. DCP typically hasn't been applied 2 times per year at the highest labeled rate in our area, nor has it been applied year, after year, after year. The commenters stated that they want to make sure that any future program doesn't have the devastating impact that methyl bromide has had to growers and land owners.

Response: APHIS at this time is not proposing to monitor groundwater or foliage from treated fields. The depth to groundwater in the areas where the program may operate is approximately 50-60 feet suggesting that groundwater contamination would not occur. In addition, the Telone II® label requires a 100-foot buffer from groundwater wells to prevent contamination. Available environmental fate data for DCP that was summarized in the SEA suggests that the product will dissipate rapidly and that any DCP soil residues that are available for plant uptake will be metabolized quickly and that no residues would be anticipated at the proposed higher application rates. In addition, applications are planned for the fall and any crops planted the following spring would not be expected to have DCP residues. The Telone II® 24(c) label does allow for a

second application of DCP that could occur in the spring but this would not be a typical use pattern. Label restrictions state that any areas that receive a spring treatment would require a 30-day plant back restriction which would allow for DCP dissipation and degradation.

Comment: It is unclear from the Draft SEA how the temporal and spatial scope of the analysis area was established for each aspect of the affected environment and the cumulative effects analysis. For example, under the subheading of "Vegetation and Wildlife," and the sub-subheading "Wildlife ," a discussion of 300 potential species of nesting birds, 85 species of mammals, and 17 amphibians and reptiles occur on the Caribou-Targhee National Forest that is located in parts of four states. Clearly, a significant portion of the Caribou-Targhee National Forest, if not the entire forest, is outside the spatial range of the potentially affected area under the alternatives. If, in fact, there is a potential impact, the Draft SEA does not explain how that determination was made.

Response: The Affected Environment chapter provided a summary description of various environmental resources that occur within Bonneville and Bingham counties. The supplemental EA was updated to clarify those resources that would be expected to occur on potato fields, or in close proximity.

Comment: The no eradication alternative states, "While the impacts of PCN to nonagricultural Solanaceae are unknown, it could be expected to impact those species in cases where nematode levels increased to damaging levels" (page 16). This sentence is contradictory.

Response: The intent of the statement was to state that other Solanaceae may be impacted by PCN; however, the extent of damage is unknown. The statement was clarified in the SEA.

Comment: A curious statement is made that a quarantine of real property will have no environmental impacts. Limiting the uses to which farm property may otherwise be placed will have an environmental impact. Those impacts may be positive or they may be negative but they will not be nonexistent. The impacts from the quarantine should be disclosed and analyzed.

Response: The statements made in the supplemental EA were not to imply that there would be no environmental impacts related to the quarantine. Analysis of the impacts of the quarantine was conducted in a previous NEPA document and was categorically excluded from further analysis based on the APHIS NEPA Implementing Regulations (7 CFR, part 372). The APHIS NEPA implementing regulations list the following under actions that may be categorically excluded from further NEPA analysis: "Routine measures, such as identifications, inspections, surveys, sampling that does not cause physical alteration of the environment, testing, seizures, quarantines, removals, sanitizing, inoculations, control, and monitoring employed by agency programs to pursue their missions and functions." § 372.5(c)(1)(i).

Comment: The Preferred Alternative notes that the Telone II® label proscribes its application more frequently than twice each year but the Preferred Alternative does not specifically adopt this requirement and does not address the cumulative effects of either annual or bi-annual application of Telone II® into the foreseeable future. The Draft SEA does not discuss the cumulative effects of this 400% increase in application over each two year period.

Response: The SEA states that one to two applications per year of Telone II® may be made according the USEPA label. The preferred alternative section was updated to clarify that the program may make two applications per year and the potential for cumulative impacts from the increased use is also discussed in the cumulative effects section of the SEA

Comment: It is unclear if the Preferred Alternative adopts the twice-per-year DCP application under the special local use need label described on pages 2 and 3.

Response: The preferred alternative was updated to confirm that applications may occur twice per year for DCP. The Telone II® 24(c) label does allow for applications twice per year; however, it will only be used if an accelerated treatment schedule is implemented that includes both a spring and a late summer/fall treatment.

Comment: This would be four times the application rate prescribed on the Telone II® label. These effects from repeated application should also be analyzed as to other aspects of the affected environment such as groundwater and wildlife.

Response: The analysis in the SEA regarding DCP use was updated to clarify how increased rates may impact groundwater and wildlife.

Comment: The layout of the environmental impact section is confusing. Sections A, B, and second B (C) present the three alternatives followed by cumulative effects analysis. These are then followed at an equal heading level discussion of specific resources such as threatened and endangered species, migratory birds, and eagles. The Final SEA would be much clearer if these environmental impacts were discussed under each of the alternatives and under the cumulative impacts analysis rather than as standalone sections at an equal heading level.

Response: The effects of each of the alternatives on federally threatened species, migratory birds, and bald and golden eagles are clarified in the final SEA, but they remain as standalone sections.

Comment: Under section D, Threatened and Endangered Species, the second paragraph notes a federally threatened species by its Latin name but not by its common name in an apparently unintended omission.

Response: The section has been updated in the SEA to include the common name.

Appendix C. Methyl Bromide and Chloropicrin Summary Risk Analysis

The Environmental Protection Agency (EPA) approved pesticide label (Tri-Con 80/20, EPA Reg. No. 58266-1) for the proposed application of methyl bromide for the PCN eradication program contains two active ingredients. Methyl bromide is the primary active ingredient comprising 80 percent of the formulated product while chloropicrin makes up 19.9 percent of the product with 0.1 percent other ingredients. The purpose of adding chloropicrin to the formulation is to act as a warning agent because methyl bromide is odorless, while chloropicrin has a strong odor. Chloropicrin is also an active ingredient in the Tri-Con 80/20 formulation. Summary risk profiles for both chemicals are discussed in the following sections.

Methyl Bromide

a. Toxicity

Methyl bromide is an odorless gas. Human toxic effects from incidents of agricultural applications of methyl bromide exposure include symptoms such as headache, malaise, weakness, difficulty breathing (dyspnea), convulsions, severe skin burns, vomiting, and diarrhea. Animal studies show that methyl bromide has low to moderate toxicity via oral or inhalation exposure. Methyl bromide does have high toxicity through dermal and ocular routes of exposure (EPA, 2006; 2007a). The oral LD_{50} in the rat is 86 mg/kg, while the inhalation LC_{50} in rats is 3.03 mg/L (EPA, 2007a). Neurotoxicity is the major hazard concern in acute and chronic toxicity exposure studies. Decreased activity, ataxia, tremors, and paralysis are common signs of exposure in inhalation studies using methyl bromide. In developmental inhalation studies using the rabbit, the maternal no observed adverse effects level (NOAEL) was 40 ppm, while the developmental toxicity NOAEL was also 40 ppm. In subchronic studies (5 to 7 weeks) using the dog (the most sensitive species to the neurotoxic effects of methyl bromide), a systemic NOAEL of 26 ppm was established based on daily doses of methyl bromide. Chronic studies using the rat, over a 127-week period, resulted in a lowest observed adverse effects level (LOAEL) of 3 ppm, based on respiratory irritation and a systemic toxicity NOAEL of 30 ppm.

USEPA currently classifies methyl bromide as not likely to be a human carcinogen because there is not enough evidence to support a different classification at this time (EPA, 2007a). Despite epidemiologic studies suggesting methyl bromide exposure may be associated with prostate, stomach, and testicular cancers (Alvanja et al., 2003; Mills and Yang, 2003; Cockburn et al. 2011; Mills and Yang, 2007; Wong et al., 1984), a more recent study evaluated the associations of methyl bromide with the cancer cases of pesticide applicators in the Agricultural Health Study (AHS) (http://aghealth.nih.gov/) with follow-up from 1993 through 2007 (Barry et al., 2012). This study also evaluated interactions with a family history for four common cancers (prostate, lung, colon, and lympho-hematopoietic). The results indicated little evidence of methyl bromide association with cancer risks (including prostate cancer) except for stomach cancer risk. An association with prostate cancer with shorter follow-up (through 1999) previously was observed. The association, however, did not persist with longer follow-up. Therefore, the researchers

suggested in the report to re-evaluate the exposure-dependent increase in stomach cancer risk with longer follow-up in the AHS along with other epidemiologic studies.

Methyl bromide is genotoxic based on available human and animal studies. A study of methyl bromide fumigation workers reported lymphocyte-related genotoxic effects associated with methyl bromide exposure (Calvert et al., 1998). In animal studies methyl bromide exposure induced micronuclei formation in the bone-marrow and peripheral blood cells of rats and mice (USEPA, 2001; IARC, 1999). A rat testicular DNA alkaline elution assay showed genotoxic potential in testicular DNA from repeated short-term inhalation exposure of methyl bromide (USEPA, 2001). A DNA-binding study of methyl bromide exposure in rats detected DNA adducts in the liver, lung, stomach, and fore stomach (Gansewendt et al., 1991).

Metabolism studies using ^{14}C-MeBr in rats indicated that inhaled methyl bromide is absorbed and distributed in all tissues with the lungs, liver, and kidneys being the major organs, and is then metabolized, and excreted mainly as Br^- and carbon dioxide (NRC, 2012; Honma et al., 1985; Bond et al., 1985; Medinsky et al., 1985). Approximately 27–50 percent of methyl bromide vapor inhaled was absorbed after a six hour exposure (USEPA, 2006; Medinsky et al., 1985). For metabolism, methyl bromide may react with water and break down to methanol and bromide ion. Methyl bromide may also react with organic thiols to form S-methyl derivatives. Methanol and S-methyl derivatives further break down to form carbon dioxide (approximately 40–50 percent of the administered dose) and other nonvolatile metabolites (approximately 20–25 percent) (ATSDR, 1992). The excreted ^{14}C methyl bromide metabolites orally administrated were primarily found in urine (43%), and expired carbon dioxide (32 percent) with less amounts in carcass (14 percent), and feces (less than 3 percent) over a 3-day period (Medinsky et al., 1984). Bromine concentrations in tissues peaked 4–8 hours after inhalation exposure, and the half-life of elimination was about 5 days in rats (Honma et al., 1985). Bromide and chloride present in body fluids in animals in steady state are excreted readily. Increased chloride intake has been shown to increase bromide excretion (WHO, 2009).

In mammals, bromine converts to the bromide ion (USEPA, 2005). Acute oral and dermal studies using sodium bromide show low toxicity (oral LD_{50} of 4,200 mg/kg and dermal LD_{50} >2,000 mg/kg) with mild eye and skin irritation (USEPA, 1993a). Chronic diet studies in mice show NOAELs ranging between 400 and 1,200 mg/kg (NRC, 2005; Hansen and Hubner, 1983). Dietary studies in rats observed disturbances in thyroid and renal function at dietary levels between 1,200 and 19,200 mg/kg (NRC, 2005; Loeber et al., 1983). A decrease in fertility also occurred at 1,200 mg/kg (NRC, 2005; van Leeuwen et al., 1983). A 1-year sodium bromide exposure study in dogs with doses of 100 mg/kg/day of bromide as sodium bromide or doses up to 150 mg/kg/day of bromide as food fumigated with methyl bromide reported a NOEL of 100 mg/kg/day (USEPA, 1993b; Rosenblum, et al., 1960). Effects on weight gain and lethargy were observed at 150 mg/kg/day. A 2-year study in rats using feed fumigated with methyl bromide reported effects on body weight at a residual bromide level of 500 mg/kg, but no effect on body weight at a residual bromide level of 200 mg/kg (NRC, 2005; Mitsumori et al., 1990).

An evaluation of human studies on bromide indicated a daily NOAEL of 4 mg/kg of body weight (van Leeuwen and Sangster, 1987). Neurotoxicity appeared to be the most sensitive effect at higher levels in humans. The Food and Agriculture Organization/World Health Organization

(1967) set the acceptable daily intake of bromide at 1 mg/kg estimated from all food sources (NRC, 2005).

In nontarget organisms, such as birds, the clinical signs of toxicity are comparable to mammals. Decreased activity, ataxia, and tremors were observed in the bobwhite quail with a reported LD_{50} value of 73 mg/kg and a no observable effect concentration (NOEC) of 33 mg/kg. Methyl bromide is moderately toxic with the acute (4 hour) inhalation LC_{50} of 561 ppm in bobwhite quail, and 780 ppm in mouse. The chronic (11 week) reproductive study in Norway rat reported a NOAEL of 30 ppm (24 mg/kg/day) for parental/systemic toxicity and a LOAEL of 90 ppm (73 mg/kg/day) based on reduced body weight during gestation. The study also reported a juvenile survival no observed adverse effects concentration (NOAEC) of 3 ppm and LOAEC of 30 ppm based on pup weight (USEPA, 2011).

Methyl bromide is moderately to highly toxic to aquatic organisms. The range of acute LC_{50} values in five different fish species ranges from 0.7 to 17 ppm. Chronic fish toxicity is lower with a reported no observable effect concentration (NOEC) of 0.1 ppm. Toxicity to the freshwater aquatic invertebrate, *Daphnia magna*, appears to be similar to fish with a reported 48-hour LC_{50} value of 2.6 ppm and a NOEC of 1.2 ppm. The breakdown product of methyl bromide, the bromide ion, has also been evaluated for aquatic toxicity and found to be much less toxic to aquatic fauna. For acute exposures to fish and invertebrates, the bromide ion was approximately four to five orders of magnitude less toxic for invertebrates and fish, respectively. Chronic fish toxicity values for the bromide ion were also less toxic than methyl bromide with a NOEC value that is an order of magnitude less than the parent.

b. Exposure and Risk

The primary mechanism of methyl bromide dissipation is through volatilization into the atmosphere. Twenty four percent to seventy four percent of methyl bromide applied as a soil fumigant dissipates into the atmosphere (Yagi et al., 1993; 1995; Majewski et al., 1995; Yates et al., 1996bc; Williams et al., 1999). Volatilized methyl bromide degrades in the upper troposphere through its reaction with the hydroxyl radical (half-life 210 days), and stratosphere via photoionization by ultraviolet (UV) light (lifetime 35 years). The estimated total global lifetime of methyl bromide in the atmosphere is 0.7 years (USEPA, 2011). Field dissipation studies show half-lives ranging between 4 and 11 days. Methyl bromide that does not volatilize is susceptible to hydrolysis (half-life 11 to 15 days), as well as microbial activity, with reported aerobic and anaerobic soil half-lives ranging from 6 to 59 days, depending on soil type (USEPA, 2011). Methyl bromide breaks down to bromine (inorganic bromide). The PCN program environmental monitoring reported residual bromide soil concentrations ranging between 0.724 mg/kg and 10.6 mg/kg mostly detected in subsurface soil of the fumigated fields at depths between two and three feet (APHIS, 2015). Degradation of methyl bromide is dependent on soil organic matter with increased rates of degradation in soils with increasing levels of organic matter. Methyl bromide degradation in water is somewhat pH-dependent with hydrolysis half-life values ranging from 29 days at a pH of 3, to 9 days at a pH of 8 (USEPA, 2011). The high pH of the soil in the areas to be treated will contribute to the rapid breakdown of methyl bromide.

Bromine in soil is a negatively charged ion, and can be taken up by plants. Crops (fruits, grains, and vegetables) grown in soils after methyl bromide fumigation may have higher levels of bromide (NRC, 2005; Brown et al., 1979; Roughan and Roughan, 1984) with potential for increased bromide accumulation (Ellis et al., 1995; Kempton and Maw, 1972). High bromine concentrations (up to 8,400 mg/kg) were reported in plants such as barley, bur clover, filaree, wild oat, ryegrass, spinach, lettuce, and oat hay with no phytotoxic symptoms (Brown et al., 1979; Kempton and Maw, 1972; and Knight and Costner, 1977). Bromide residues are especially high in plants planted closely after soil fumigation (Roughan and Roughan, 1984) and during the first year of the fumigation (Brown et al., 1979). APHIS (2015) reported an average level of 9,545 mg/kg in fodder samples of baled and grain stage peas, oats, and barley harvested from a field in the same year of soil fumigation in 2013. Average concentrations of 6,265 mg/kg and 4,827 mg/kg were reported in baled hay samples collected from the first and second cutting of 2014. An average concentration of 1,443 mg/kg was reported in baled hay samples collected from the first cutting in the same fields in 2015 (fumigation was not performed in 2015). Elevated levels of bromine in plants used for animal feeds have shown adverse health effects such as lethargy, weakness, and ataxia in horses, goats, and cattle (Knight and Costner, 1977) and motor incoordination in cattle (Knight and Reinea-Guerra, 1977). Reported bromide intoxication of livestock in California was caused by ingestion of volunteer oat hay cut from a field treated with methyl bromide the previous year (Knight and Costner, 1977). The bromide levels in the hay ranged from 6,800 to 8,400 ppm. Bromide levels in plants grown in methyl bromide treated fields may result in exposure to nontarget vertebrates, such as wildlife and domestic animals that consume plant material. Residues of bromine in soil and plants will be dependent upon site conditions that affect methyl bromide degradation.

The maximum tolerable level (MTL) of bromine is the dietary level consumed for a duration of time that will not impair animal health or performance. The National Research Council (NRC) established a MTL of 300 mg/kg in rodents. NRC uses an estimated MTL of 200 ppm in animal feed for swine and cattle (NRC, 2005). The level was estimated based on no observed effects seen in a pig diet study (pigs exposed to bromide salts at level of 200 mg/kg/day) (Barber et al., 1971) and cattle diet studies (cattle exposed to inorganic bromide at levels of 19 mg/kg/day and 43 mg/kg/day) (Lynn et al., 1963). Limited information is found in the open literature on residue levels in meat and milk of animals at various dietary bromide levels. A dietary study in dairy cows (Vreman et al., 1985) reported muscle and milk bromide levels of 3 mg/kg and 6 mg/kg, respectively (dietary bromide level of 22 mg/kg), and 20.8 mg/kg and 31 mg/kg, respectively (dietary bromide level of 115 mg/kg). In the study, dairy cows were fed diets contained 22, 69, or 115 mg/kg inorganic bromide residues from the decomposition of methyl bromide fumigate for 5 weeks.

Human exposure to methyl bromide gas can occur during and after application because of its volatility and ability to move off site for an extended period of time after application. Fumigant applications result in exposures up to several thousand feet from a treated field depending on the size of the fumigated field, the amount of fumigant applied, and the rate at which the fumigant escapes from the treated field. The rate of a fumigant off-gassing from a treated field after application is dependent on factors such as the application method, soil moisture, soil temperature, organic matter levels, water treatments, the use of tarps, biological activity in soil, soil texture, weather conditions, and soil compaction (USEPA, 2008a). The potentially exposed

human populations include workers (applicators and handlers) with inhalation, incidental ingestion, and dermal contact as the exposure routes, and the general public who live or work in the vicinity of a fumigation site with inhalation as the primary exposure route.

Concerns regarding potential human exposure to fumigants resulted in EPA implementing additional safety requirements in 2012 to increase protection for agricultural workers and bystanders who live, work, or spend time near fumigated fields (USEPA, 2012a).

The safety measures to be incorporated into a soil fumigant product label include: (1) agriculture worker protection, (2) handler training information, (3) good agricultural practices, (4) application method, practice and rate restrictions, (5) restricted use pesticide classification, (6) buffer zone and posting requirements, (7) site-specific fumigant management plans, (8) emergency preparedness and response requirements, (9) applicator training programs, (10) information for handlers, communities, and first responders, and (11) compliance assistance and assurance measures. As specified in the site-specific Fumigant Management Plan (FMP) and post-application summary factsheet (USEPA, 2012b), a site-specific FMP must contain information such as: (1) certified applicator information, (2) buffer zone determination, (3) provisions for state and/or tribal lead agency advance notification, and (4) applicable mandatory good agricultural practices. A FMP also contains plans for air monitoring, emergency response, and communication among key parties. The post-application summary also is delineated in the factsheet (USEPA, 2012b). The summary must describe any deviations from the FMP requirements for measurements taken to comply with good agricultural practices, and any complaints and whether any reportable incidents occurred.

For worker protection, mitigation measures include a clear description of handler activities on labels, on-site supervision and training, respiratory protection requirements, tarp perforation and removal requirements, and entry-restricted period requirements (USEPA, 2012c). For the general public such as bystanders, a buffer zone will reduce the potential exposure to air concentrations that may cause acute adverse health effects. The buffer zone distance is based on application rate, field size, application equipment and methods; and credits (USEPA, 2012d). Posting requirements for buffer zones (USEPA, 2012e) will inform bystanders the location of the buffer to ensure they do not enter areas designated as part of the buffer zone. The applicators must perform on-site monitoring of the buffer zone perimeter in areas where residences and other occupied structures are within a specific distance. As an alternative, the applicators can provide emergency response information directly to neighbors when the buffer zones are greater than 25 feet, and there are residences and businesses within 50, 100, 200, or 300 feet from the outer edge of the buffer zones of >25 feet and <100 feet, >100 feet and <200 feet, >200 feet and <300 feet, and >300 feet, respectively (USEPA, 2012f).

Fumigation site monitoring will reduce exposures during or after the fumigation to people who may be near a buffer zone. Emergency response information for neighbors is provided through mail, telephone, door hangers, or other methods. The information includes the location of the application block, information on the fumigant product, time period (must not range more than 4 weeks), early signs and symptoms of exposure to the fumigant(s), what to do, and emergency responder phone number, and additional information about fumigants.

The Tri-Con 80/20 formulation is a restricted use pesticide with use only by certified applicators, or persons under their direct supervision. The label (2014) incorporated EPA required safety measures and includes specific requirements to mitigate exposure to workers and the general public. For example, fumigation workers must have certified applicator training. The label requires personal protective equipment and specifies a National Institute for Occupational Safety and Health (NIOSH)-certified full-face piece air-purifying respirator with cartridges certified by the manufacturer for protection from exposure to methyl bromide at concentrations up to 5 ppm when an air-purified respirator is required. Air monitoring is required at least every 2 hours in the breathing zones of a handler performing a representative handling task when full-face piece air-purifying respirators are worn. Stop work is triggered when a methyl bromide air sample is greater than 5 ppm. A direct read detection device with sensitivity of at least 1 ppm (methyl bromide) and 0.15 ppm (chloropicrin) must be used for air monitoring. No respirator is required when air concentrations are less than 1 ppm and no sensory irritation is experienced. Only correctly trained personnel with required personal protective equipment (PPE) can enter the application block. The entry restriction periods are 5 days for untarped applications and 14 days after the completion of tarp applications. The maximum application rate for nematode control is 400 lbs methyl bromide/acre (cannot exceed 500 lbs Tri-Con 80/20 per acre). The maximum application block sizes allowed are 100 acres except for untarped deep applications in orchard replant applications. A buffer zone is required that extends outward from the edge of the application block perimeter equally in all directions. The buffer zone distance (a minimal distance of 25 feet) is calculated using the application rate and size of the application block to reduce the potential exposure for the general public. The planting or transplanting interval is at least 14 days after the completion of application and can vary based on what crops may be planted and soil conditions. Per label requirements, APHIS also develops a site-specific fumigation management plan that reflects current site conditions and contains information about EPA required safety measures for each application block.

Management techniques in the field also have a large influence on methyl bromide volatilization and degradation. The use of a tarp after methyl bromide application has been shown to be an effective means of reducing volatilization and increasing degradation of methyl bromide (USEPA, 2011). The Tri-Con 80/20 label requires that tarps must not be perforated until a minimum of 5 days (120 hours). Soil injection has also been shown as an effective means of limiting methyl bromide volatilization (Yagi et al., 1995). Both management actions are to be implemented in the PCN eradication program as a means to limit off-site movement of methyl bromide. Language on the label regarding placards for the site, as well as the use of the warning agent chloropicrin, will further reduce potential human-related exposure. Consequently, human health risks from direct contact are minimal due to reduced exposure. The lack of exposure is supported by environmental air monitoring data that was collected between 2008 and 2014 in fields after application. Approximately 119 samples have been collected over that time period with approximately 81 percent of the samples having methyl bromide residues below analytical detection. Of the collected samples most were at trace levels (0.2 ppm and 0.5 ppm) of methyl bromide which is below established regulatory threshold limits.

Exposure is expected to be minimal in both terrestrial and aquatic environments due to the location of the application sites in relation to sensitive areas and the safety language present on the label. While methyl bromide is highly soluble (15.2 g/L) and mobile in soil, the distance of

the application area from surface and groundwater precludes any exposure that could impact human health or nontarget aquatic organisms. The closest surface water is approximately 0.25 miles from the application area, while soil type and water table depth mitigate groundwater exposure. Surface to groundwater distance ranges from 35 to 50 feet based on data collected in proximity to the proposed application area (USGS, 2000). The low rainfall in the area, coupled with the ability to manage irrigation water, provide additional confidence that movement of methyl bromide into ground and surface water is unlikely.

Soil invertebrates, as well as any other nontarget animals present during the fumigation and unable to escape, are expected to succumb to the fumigation. The fumigated areas, however, are small and likely to be recolonized within a short time.

There is the potential for small nontarget terrestrial organisms to be exposed through inhalation or ingestion of contaminated soil. The proposed treatment areas are agricultural fields which are highly disturbed areas. The likelihood of small terrestrial organisms being exposed is expected to be minimal. The use of a tarp and the warning agent, chloropicrin, will act as a deterrent for small mammals that may try to forage in or near treated fields. Any exposure to nontarget terrestrial organisms related to the ingestion of treated soil or inhalation should not be at levels sufficient to cause adverse effects. Small terrestrial nontarget organisms that could serve as prey would not be expected to accumulate sufficient residues to impact predators. Methyl bromide has been shown to be rapidly excreted primarily through urine or exhaled as carbon dioxide (EPA, 2006a). The environmental fate and limited exposure pathway, as well as the rapid metabolism of methyl bromide, would suggest that methyl bromide does not accumulate in the tissue of exposed animals.

Methyl bromide has been identified by USEPA and the United Nations as a product that can cause ozone layer depletion. The human health effects from thinning of the ozone layer include skin cancer, cataracts, and immunosuppression due to increased UV radiation reaching the earth's surface (USEPA, 2008a). However, manmade sources of methyl bromide contribute a minor amount of ozone-depleting compounds to the atmosphere when compared to other chlorine and bromine gas sources (figure 4). Total chlorine gas sources are more than 100-fold above bromine sources.

Atmospheric methyl bromide levels peaked in the mid- to late-1990's and have been decreasing at a rate of 4 to 6 percent per year in the northern hemisphere since 1996 (UNEP, 2007; Yokouchi et al., 2002). Methyl bromide contributions from human sources have decreased by 61.8 percent between 1998 and 2012 as a result of Montreal Protocol (Hegglin et al., 2015). While many of the ozone-depleting substances have long half-lives in the atmosphere, the half-life for methyl bromide is comparatively shorter (0.7 years) and, therefore, any decline in methyl bromide use is reflected more quickly in atmospheric levels.

Methyl bromide uses related to the PCN eradication program represent a small percentage of total use in the United States. Recent data regarding methyl bromide use in the United States for critical use exemptions (CUE) and quarantine pre-shipment (QPS) treatments shows that 3,670 metric tons, or approximately 8.09 million pounds were used in 2011. Methyl bromide use for the PCN program in Idaho has ranged from 144,640 to 438,609 lbs between 2007 and 2014. The

range of methyl bromide quantities used in the PCN program represents approximately 1.8 to 5.4 percent of the total used for CUE and QPS in the Unites States. When compared to global use of methyl bromide the percent contribution would be much less. The contribution of methyl bromide from PCN use would be considered negligible when compared to all ozone-depleting substances since the contribution relative to other bromine and chlorine source gases is minor (Figure 4).

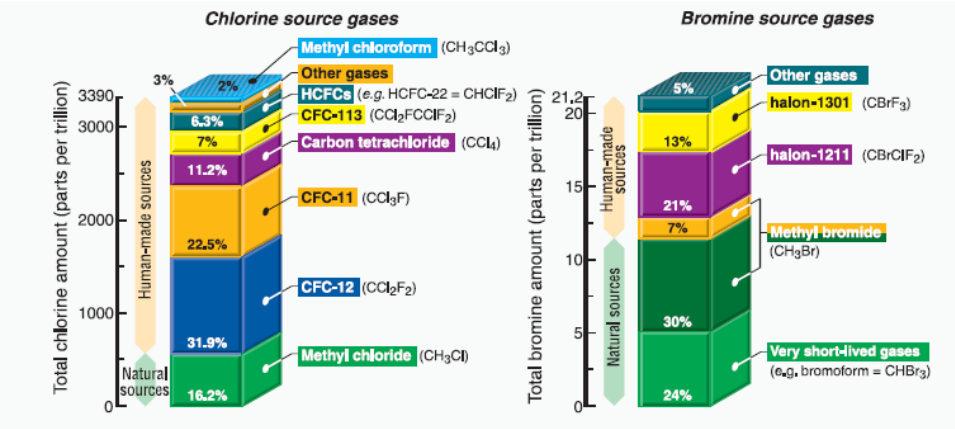

Figure 4. Primary source of chlorine and bromine gases for the stratosphere in 2004. (Source: UNEP, 2006. Twenty Questions and Answers about the Ozone Layer: 2006 Update.)

c. Summary

Based on the method of application, label restrictions and other mitigation measures required for most fumigants the risk to human health is expected to be minimal. The proposed use of methyl bromide also poses minimal risk to nontarget organisms. Aquatic organisms will not be impacted because the application sites are far enough from any of the treated fields to minimize residues from drift or runoff. In addition, high soil pH will speed degradation and low rainfall will greatly limit any potential for runoff or leaching into ground and surface waters. Risk to terrestrial organisms (other than the soil invertebrates in the treated fields that would be impacted) is also minimal due to the method of application and the environmental fate of methyl bromide. Risk to human health and the environment is further reduced by other management practices such as soil injection of methyl bromide in the soil, posting warning signs at the application site, and the use of a tarp to reduce volatilization and enhance degradation. Air quality impacts related to ozone depletion is also low because methyl bromide is not a large source of manmade ozone depleting gases, and its use in this program relative to global methyl bromide use is negligible. Risks to bromine in the environment as a result of methyl bromide applications and degradation are expected to pose low risk to human health and most nontarget organisms. The potential for exposure and risk to nontarget organisms is greatest for terrestrial vertebrates that feed on plants or crops that grow on fields after they are treated. This risk diminishes as soil bromine levels decrease over time with less available for uptake by plants.

Chloropicrin

a. Toxicity

Chloropicrin is the other active ingredient that is present in the methyl bromide formulation proposed for use in the PCN eradication program. Chloropicrin is a fumigant (19.9 percent of the formulation) as well as a warning agent to prevent accidental exposure. It has chemical properties similar to other fumigants, such as high volatility (vapor pressure of 23.8 mm @ 25 ^{o}C and Henry's Law Constant (2.05 x 10^{-3} atm M^3/mole) and a low affinity for binding to soil (Koc 36.05 ml/g).

Mammalian toxicity data for chloropicrin demonstrates high acute toxicity based on median lethal oral (LD_{50} = 37.5 mg/kg), inhalation (LC_{50} = 17 ppm), and dermal (LD_{50} = 100 mg/kg) studies. Chloropicrin is corrosive to skin and causes irritation to the eye, nose, throat, and upper respiratory with the most sensitive effect being eye irritation in humans (USEPA, 2009a). The human sensory irritation study shows that study participants felt mild eye irritation within 30 minutes at 0.1 ppm and 20 minutes at 0.15 ppm. Effects ceased 1 hour after the exposure ended with no irritation effects the following day. Based on the sensory irritation the studies, USEPA determined a bench mark concentration level ($BMCL_{10}$) of 0.073 parts per million (ppm) (no eye or nose irritation, or upper respiratory changes) (USEPA, 2009b). Sub-chronic inhalation studies report a NOAEL of 0.3 ppm in both the mouse and rat. The inhalation developmental studies report a maternal NOAEL of 0.4 ppm in the rat and rabbit. Chronic feeding studies using the rat and dog resulted in a NOAEL of 0.1 mg/kg/day for both test species and a LOAEL of 1 mg/kg/day based on liver and immune system effects in the rat, and gastrointestinal irritation and blood chemistry alterations in the dog. EPA does not consider chloropicrin to be carcinogenic based on oral or inhalation routes of exposure (USEPA, 2009b).

Limited studies show that chloropicrin is metabolized and excreted rapidly in the body (USEPA, 2009b). A 48-hour study administering ^{14}C-chloropicrin to male mice showed that urine was the major route of excretion (43-47% excreted in the first 24 hours, and another 8-8.5% between 24 and 48 hours) (Sparks *et al.*, 1997). The other routes of excretion were expired air (6.5-15% of the applied dose excreted as CO2 in 48 hours), and feces (only 2.5-9% in 48 hours). Tissue radiological measurements show that the liver had the highest level of radioactivity, followed by the kidney, lung, blood, fat and skin at 1 hour and 48 hours.

As a pre-plant soil fumigant, EPA considers the use of chloropicrin to be a non-food use and tolerances are not needed (USEPA, 2009b). This is because chloropicrin is degraded in both aerobic and anaerobic soil to carbon dioxide (CO_2), and used by the plants to be incorporated into starch, proteins, pectin, lignin, hemicellulose, and cellulose (USEPA, 2009b).

Chloropicrin is considered highly toxic to wild mammals through oral, inhalation and dermal exposures. No acute or chronic data appear to be available that describe effects to avian species. Chloropicrin is considered very highly toxic to aquatic organisms, with fish LC_{50} values ranging from 16.5 ppb for the rainbow trout to 105 ppb for the bluegill sunfish. Toxicity to aquatic invertebrates is similar to fish with a 48-hour median effective concentration (EC_{50}) value of 63

ppb for *Daphnia pulex*. No chronic aquatic toxicity values appear to be available for chloropicrin; this may be due to its extremely short half-life in water (EPA, 2006d).

b. Exposure and Risk

Based on the chemical properties of chloropicrin, the primary route of dissipation is through volatilization. Airborne chloropicrin is sensitive to light with half-lives less than 8 hours in direct sunlight. Chloropicrin left in soil degrades quickly with half-lives ranging from 3.7 to 4.5 days (USEPA, 2009). Chloropicrin is highly soluble in water and has low adsorption potential in soil suggesting it may be mobile. Chemical and physical properties for chloropicrin, such as high solubility and lack of partitioning to tissue, suggest that it will not bioconcentrate or bioaccumulate in animals.

Similar to methyl bromide, the potential exposure routes for chloropicrin include inhalation, incidental ingestion, and dermal contact for workers, and acute inhalation exposure for the general public who live or work in the vicinity of a treatment. The actual inhalation exposure to chloropicrin for fumigation workers and the general public are minimal due to the use of PPE, the label required mitigation measures, and best management techniques in the field (e.g., impermeable tarp and soil injection approximately 12 inches below the soil surface). As discussed in the methyl bromide section, the Tri-Con 80/20 formulation label (2014) includes specific requirements such as certified applicator training, PPE, air monitoring, entry restriction, the maximum application rate and maximum application block size, and establishment and posting of a buffer zone to mitigate potential exposures to fumigation workers and the general public. Consequently, human health risks from direct contact are minimal due to reduced exposure. Available air monitoring data for chloropicrin collected in treated fields between 2008 and 2014 supports a lack of exposure potential since all samples were below detection with the exception of two samples that had trace levels of chloropicrin (0.1 ppm).

Chloropicrin is highly soluble and mobile; however, due to the low rainfall in the area, the location of the treatment fields relative to aquatic resources and the application method, chloropicrin migration from runoff into surface water or leaching into groundwater is unlikely. Residues in water and aquatic organisms are not expected.

Direct and indirect exposure to nontarget terrestrial organisms (other than soil invertebrates in the treated fields which are expected to succumb), is highly unlikely due to the method of application and the use of an impermeable tarp during treatment. There is a slight possibility that terrestrial prey could be contaminated if they ingest soil from the treated area after tarp removal. However, prey would have to occupy the treated fields immediately after tarp removal to be exposed. Because its use for this application is as a warning agent, any terrestrial prey would most likely not forage in treated areas due to the eye and nasal irritability of chloropicrin. In the event of chloropicrin exposure, residues would not accumulate in tissue based on its chemical properties that suggest it would not partition to tissue, and its rapid metabolism in mammals.

c. Summary

Based on the method of application, mitigation measures required by the Tri-Con 80/20 label, and the lack of residues from any crop or drinking water, the use of chloropicrin poses minimal risk to human health. The use of chloropicrin also poses minimal risk to nontarget organisms (other than to soil invertebrates in the treated sites which are expected to succumb). Aquatic organisms will not be impacted because of low rainfall in the area and the application sites are far enough from any aquatic habitats to minimize residues from leaching, drift, or runoff. Risk to terrestrial organisms is also minimal due to the method of application and the environmental fate of chloropicrin. Risk to human health and the environment is further reduced by its use as a warning agent and other management practices such as soil injection during application, posting warning signs at the application site, and the use of a tarp to reduce volatilization and enhance degradation. Based on the lack of exposure and available toxicity data, the use of chloropicrin and methyl bromide as a formulated mixture will not significantly increase environmental risk compared to their associated risks when used individually.

Appendix D: Summary Risk Assessment Information for Herbicide Use Related to Litchi Tomato Control

Glyphosate

Glyphosate is a non-selective phosphonomethyl amino acid herbicide widely used to control weeds on various agricultural crops (such as fruits, and vegetables) and non-agricultural (such as greenhouses, and residential) areas (USEPA, 2009a). Glyphosate is formulated as a water dispersible granule, emulsifiable concentrate, water-dispersible liquid, ready to use, and soluble concentrate/solid that can be applied pre- or post-emergence by aerial and ground equipment. Glyphosate is a potent and specific inhibitor of 5-enolpyruvylshikimate 3-phosphate synthase enzyme. The enzyme is essential for the biosynthesis of aromatic amino acids such as tyrosine, tryptophan, and phenylalanine and other aromatic compounds in algae, higher plants, bacteria, and fungi. Inhibition of this enzyme causes plant cell death (USEPA, 2009a). The program proposes the use of glyphosate for pre-emergent treatments before litchi tomato has emerged, or spot post-emergent treatment.

Glyphosate has low acute oral, dermal, and inhalation toxicity to mammals (USEPA, 2009b). It is a mild eye irritant, and slight skin irritant, but is not a dermal sensitizer. A chronic feeding study in rats showed no systemic effects on body weight, food consumption, clinical signs, mortality, clinical pathology, organ weights, and histopathology. A second chronic feeding study in rats tested at higher dietary levels reported toxicity effects including decreased body weight gains in females, and increased incidence of cataracts and lens abnormalities, decreased urinary pH, increased absolute liver weight, and increased relative liver weight/brain weight in males. There were no developmental toxic effects observed in glyphosate toxicity studies in rats and rabbits. The 3-generation reproductive study in rats reported a focal tubular dilation of the kidneys. However, the 2-generation reproductive study in rats reported no adverse reproductive effects. There is no evidence of increased susceptibility of offspring observed in the glyphosate developmental and reproductive studies (USEPA, 2009b). The toxicology studies conducted indicate that glyphosate is not neurotoxic or immunotoxic (USEPA, 2009b; 2012). USEPA Endocrine Disruptor Screening Program weight of evidence evaluation on the glyphosate Tier 1 screening assay concluded that there was no convincing evidence of potential glyphosate interaction with the estrogen, androgen or thyroid pathways in mammals or wildlife (USEPA, 2015). Glyphosate is classified as "not likely to be carcinogenic to humans" based on lack of convincing carcinogenic evidences in rats or mice (USEPA, 2009b) and a USEPA recent evaluation on carcinogenic potential of glyphosate (USEPA, 2016). There is no concern of mutagenicity for glyphosate based on a lack of evidence that glyphosate induces mutations in vivo through the oral route and overall weight of evidence from available mutagenicity studies (USEPA, 2016).

Exposure and risk to all human population groups from the program use of glyphosate as pre- or post-emergence treatments is expected to be negligible. The potential for exposure is greatest for workers during mixing, loading, and applying, as well as during post-application activities. Following label directions including restricted entry interval, and properly using personal protective equipment and general hygiene practices results in minimal exposure and risk to this

subgroup of the population. Available human health risk assessment results from USEPA (2009b; 2012) suggest no risks of concern to the public, including children, from glyphosate use based on risk estimates for potential exposure scenarios including residential post-application exposure, and dietary exposure (food and drinking water). These exposure scenarios would be considered conservative when compared to the proposed program use of glyphosate since no residential applications or direct applications to food items would occur in the PCN program. For potential dietary exposure, there were no chronic dietary risks of concern for all U.S. population subgroups including infants and children. There were no short-term (food, water and residential incidental oral), intermediate-term (food, water, residential incidental oral), and chronic (food and water) aggregate risks of concern for various population groups, including children (USEPA, 2012). USEPA has not performed a quantitative post-application inhalation exposure assessment for a residential bystander for glyphosate primarily because there is no inhalation point of departure (USEPA, 2012).

Glyphosate in soil degrades through aerobic soil metabolism with half-lives ranging from 1.8 to 5.4 days under laboratory conditions. In water, glyphosate degrades more quickly under aerobic conditions with a half-life of 14.1 days compared to the anaerobic aquatic metabolism half-life of 208 days. Glyphosate is stable to abiotic hydrolysis, direct soil photolysis, and direct aqueous photolysis. Terrestrial field dissipation studies indicate that glyphosate dissipated with half-lives ranging from 1.7 to 142 days. The half-life in an aquatic field dissipation study was 7.5 days. Glyphosate has low potential to volatilize from soils (low vapor pressure) or from water (low Henry's Law constant) to air. Glyphosate adsorbs strongly to soil. It has slight to low mobility in soil with low potential to reach surface water as dissolved runoff and groundwater from leaching. There is potential for glyphosate to reach surface water from spray drift or transport of residues absorbed to soil particles suspended in runoff. Glyphosate is unlikely to bioaccumulate in fish (USEPA, 2009a).

Glyphosate is slightly toxic to avian species in acute oral and acute dietary studies. Chronic toxicity to birds is also low with a lack of effects reported in reproductive studies. Glyphosate has low toxicity to wild mammals based on the above discussion regarding mammalian data and potential human health effects. Glyphosate is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. Glyphosate is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots. Dicots are more sensitive than monocots based on vegetative vigor studies (USEPA, 2009a). Spray drift presents potential risks to nontarget plants in close proximity to treated fields. Exposure and risk to vertebrate nontarget terrestrial wildlife is expected to be low based on the low toxicity of glyphosate and the proposed applications of glyphosate. Risks to nontarget plants is the greatest but will be minimized by following labeled directions designed to reduce the amount of drift from applications.

For aquatic organisms, glyphosate is slightly acutely toxic to freshwater fish and freshwater invertebrates. A chronic toxicity study using the fathead minnow reported no observed adverse effects at the highest glyphosate concentration tested. The chronic toxicity to freshwater invertebrates indicates that glyphosate can reduce reproductive capacity at higher concentrations. Glyphosate is practically non-toxic acutely to marine/estuarine fish and slightly acute toxic to marine/estuarine invertebrates. Glyphosate is toxic to non-vascular and vascular aquatic plants

(USEPA, 2009a). The exposure and risk to aquatic organisms from the program use as pre- or post-emergence treatments will be reduced by adherence to the label requirements regarding applications in proximity to aquatic sites.

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Rimsulfuron

Rimsulfuron is a pre- and post-emergent sulfonylurea herbicide registered for use on several crops using ground or aerial equipment. Rimsulfuron inhibits the plant enzymes acetolactate synthase (ALS)/acetohydroxy acid synthase (AHAS) resulting in the disruption of cell division. The ALS enzyme is unique to plants resulting in lower toxicity to other organisms. Rimsulfuron is considered practically non-toxic to mammals in acute oral, dermal, and inhalation exposures. Rimsulfuron is not a skin irritant but is considered a moderate eye irritant. Chronic toxicity of rimsulfuron to mammals is also low based on the available toxicity data. Rimsulfuron is not considered to be mutagenic, carcinogenic or a developmental toxicant (USEPA, 2015a). Conservative aggregate exposure scenarios for the general public and workers who apply rimsulfuron show very low risk to human health from the use of rimsulfuron (USEPA, 2015a).

Rimsulfuron does not persist in the environment with soil and water half-lives of approximately 47 and 3 days, respectively (USDA, 2015b). Rimsulfuron is soluble in water and does not bind tightly to soil suggesting it may be mobile and could move off-site in runoff. Rimsulfuron is not

expected to volatilize into the atmosphere or bioconcentrate in the environment based on available chemical fate data (USDA, 2015b).

Rimsulfuron has low toxicity to wild mammals, reptiles and birds based on available toxicity data. Median lethality values typically exceed the highest test concentration suggesting that rimsulfuron is practically non-toxic to this group of organisms. Rimsulfuron is also expected to have low toxicity to terrestrial invertebrates based on its mode of action and lack of toxicity to honey bees in contact toxicity studies. Rimsulfuron is also considered practically non-toxic to aquatic invertebrates and vertebrates with available data showing median lethality values exceeding 100 ppm (EPA, 2015b). Rimsulfuron is considered highly toxic to vascular and non-vascular aquatic plants with toxicity values in the low ppb range. Rimsulfuron is also toxic some terrestrial plants with the most sensitive test plant species being sorghum in seedling emergence studies and oilseed rape in vegetative vigor studies. The risk to nontarget sensitive aquatic and terrestrial plants is reduced by adherence to label directions and avoiding conditions that favor drift or runoff during and after application.

References

USEPA. 2015a. Memorandum – Rimsulfuron. Human Health Draft Risk Assessment for Registration Review, 36 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0178-0018, last accessed Oct. 27, 2016.

USEPA. 2015b. Preliminary Ecological Risk Assessment for Registration Review of 22 Sulfonylurea Herbicides, 318 pp, available at https://www.regulations.gov EPA-HQ-OPP-2010-0626-0028, last accessed Oct. 27, 2016.

Ethalfluralin

Ethalfluralin is a pre-emergent herbicide that has a variety of registered agricultural uses, including potatoes, and can be applied as a liquid or granule using ground or aerial equipment. It is in the dinitroaniline class of herbicides and acts by inhibiting the formation of cell walls in plants and causing dessication of xylem and phloem in sensitive plant species. Ethalfluralin has low acute oral, dermal and inhalation toxicity to mammals. It is considered moderately irritating to the eye and can produce skin irritation (USEPA, 2016a). Ethalfluralin has not been shown to be neurotoxic or mutagenic. Ethalfuralin is classified as a possible human carcinogen based on the increased incidence of the formation of mammary gland fibroadenomas at higher concentrations. Available risk assessment data from EPA suggests low risk to the public from ethalfluralin use based on aggregate exposures from food and drinking water and the available toxicity data (EPA, 2016a).

Ethalfluralin has low to moderate persistence in the environment. In the presence of light ethalfluralin will degrade quickly in water with a half-life of 6.3 hours. Ethalfluralin is also susceptible to microbial degradation with aerobic soil half-lives ranging from 17 to 46 days. Field dissipation half-lives range from 23 to 51 days. Ethalfluralin has low water solubility and binds tightly to soil and would be expected to have a low runoff risk to water in a dissolved state. Ethalfluralin is volatile and may partition into the atmosphere but would degrade quickly based

on its sensitivity to light (EPA, 2016b). Ethalfluralin transport off-site would primarily occur as drift or as soil bound material in runoff.

Ethalfluralin has low toxicity to wild mammals, reptiles and birds based on available ecotoxicity data. Median lethality values for mammals and birds are greater than the highest test concentration suggesting ethalfluralin is practically non-toxic to this group of vertebrates. Toxicity is also low to pollinators such as the honey bee based on acute contact toxicity studies. Ethalfuralin is considered highly toxic to most aquatic vertebrates and invertebrates with median lethality values ranging in the low to mid parts per billion range (EPA, 2016b). Toxicity is also high to aquatic plants and several terrestrial plant test species. Vegetative vigor and seedling emergence toxicity studies show monocot species to be more sensitive to ethalfuralin than dicots. The risk to aquatic organisms and nontarget terrestrial plants can be reduced by adherence to pesticide label requirements.

References

USEPA. 2016a. Ethalfluralin: Human Health Draft Risk Assessment for Registration Review. 44 pp, available at https://www.regulations.gov EPA-HQ-OPP-2011-0094-0016, last accessed Oct. 27, 2016.

USEPA. 2016b. Memorandum - Preliminary Environmental Fate and Ecological Risk Assessment for the Registration Review of Ethalfluralin, 133 pp, available at https://www.regulations.gov EPA-HQ-OPP-2011-0094-0019, last accessed Oct. 27, 2016.

Bromoxynil

Bromoxynil is a selective contact foliage applied herbicide used to control broadleaf weeds. Bromoxynil negatively affects plant respiration by inhibiting photosynthetic electron transport and oxidative phosphorylation in mitochondria, which stops energy production in plants (USEPA, 2012). The USEPA registered bromoxynil products include bromoxynil (or bromoxynil phenol), and its esters bromoxynil octanoate and bromoxynil heptanoate as active ingredients for agricultural and non-agricultural uses in grass crops and some tolerant broadleaf crops. Bromoxynil esters formulated in the end products quickly break down to bromoxynil following application in the environment (USEPA, 2013a).

Bromoxynil has moderate acute oral and inhalation toxicity, and low acute dermal toxicity to mammals. It is a moderate eye irritant and is not a dermal irritant or skin sensitizer. Bromoxynil has liver and developmental effects from repeated exposure at elevated levels. Effects include increased panting, increased absolute liver weights and liver/body weight ratios, elevated rectal temperature, hyperthermia, and death. Developmental effects include increased incidence of supernumerary ribs, and malformations in the brain, eye, fused ribs, spine, and thoracic centrum at higher dose levels. Bromoxynil is not a reproductive toxin and is not neurotoxic or immunotoxic based on available information (USEPA, 2012). The endocrine disruptor screening program estrogen receptor (ER) bioactivity screening results indicates bromoxynil has ER bioactivity. However, the screening results alone do not provide a scientific basis for whether the chemical has the potential for endocrine disruption without further evaluation (USEPA, 2015). Bromoxynil is classified as a "possible human carcinogen" based on the presence of

hepatocellular tumors in male and female mice. Bromoxynil is not considered to be mutagenic (USEPA, 2012).

Exposure and risk to all human population groups from the use of the herbicide as spot treatments is expected to be negligible. The potential for exposure is greatest for workers during mixing, handling, and applying. Following label directions including personal protective equipment along with general hygiene practices results in minimal exposure and risk to this subgroup of the population. There is no registered residential use of bromoxynil (USEPA, 2012). Available human health risk assessment data from USEPA suggests low risk to the public from bromoxynil use based on aggregate exposures from food and drinking water and available toxicity data (USEPA, 2011).

Bromoxynil is not persistent and rapidly degrades in aerobic terrestrial and anaerobic aquatic environments. The aerobic soil metabolism half-lives are 1.3 days (loam) and 2.1 days (sandy loam). The anaerobic aquatic metabolism half-life is 4.5 days. There is no observed substantial degradation via photolysis in soil. Bromoxynil is stable via hydrolysis at a range of pH values. Bromoxynil has low vapor pressure and volatilization is not a major source of dissipation. Bromoxynil is moderately mobile in soil and has high water solubility with the potential for runoff and leaching (USEPA, 2013b).

Bromoxynil heptanoate, and octanoate are classified as moderately toxic to practically non-toxic to birds in acute oral exposures. They are considered slightly toxic in subacute dietary exposures to birds. Bromoxynil, heptanoate and octanoate have moderately acute oral toxicity to small mammals. Bromoxynil octanoate is practically nontoxic to pollinators such as the honey bee based on acute contact toxicity studies (USEPA, 2013b). Bromoxynil is toxic to some nontarget plants. The most sensitive effect concentration (EC_{25}) values for nontarget terrestrial plants are 0.014 lbs ai/ac for seedling emergence and 0.011 lbs ai/ac for vegetative vigor based on bromoxynil heptanoate effects on shoot weight (USEPA, 2013b). The exposure and risk to most terrestrial organisms from the proposed applications of bromoxynil as spot treatments will be low based on the lack of significant exposure since bromoxynil rapidly degrades in aerobic terrestrial environments and the proposed use pattern for controlling litchi tomato. The risk to sensitive plants will be reduced by following label directions designed to minimize off-site drift and runoff.

Bromoxynil is moderately toxic and bromoxynil octanoate is highly toxic to most nontarget aquatic organisms. The available acute data indicate bromoxynil is moderately toxic to freshwater fish and is slightly toxic to freshwater aquatic invertebrates. Bromoxynil octanoate is highly toxic to estuarine/marine fish and highly toxic to oysters and very highly toxic to estuarine/marine shrimp. USEPA's ecological risk assessment for registration review of bromoxynil and its esters concluded a chronic risk concern from fish exposed to bromoxynil are not anticipated (USEPA, 2013b). The chronic aquatic invertebrate studies indicate that aquatic invertebrate reproductive impairment may occur at bromoxynil octanoate levels greater than 2.5 ppb. Available aquatic plant toxicity data suggests bromoxynil octanoate and heptanoate are highly to very highly toxic to algae and diatoms. Bromoxynil heptanoate is highly toxic to the vascular aquatic plant *Lemna gibba*. The most sensitive effect concentration (EC_{25}) values for nontarget terrestrial plants are 0.014 lbs ai/ac for seedling emergence and 0.011 lbs ai/ac for

vegetative vigor based on bromoxynil heptanoate effects on shoot weight (USEPA, 2013b). The risk to aquatic organisms and nontarget terrestrial plants can be reduced by adherence to label requirements.

References

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USEPA. 2013a. Bromoxynil Final Work Plan, Registration Review Case Number 2070, Docket Number EPA-HQ-OPP-2012-0896. 7 pp.

USEPA. 2013b. Memorandum – EFED Registration Review Problem Formulation for Bromoxynil and Bromoxynil Esters, 62 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0896-0002, last accessed Aug. 18, 2016.

USEPA. 2012. Memorandum – Bromoxynil. Human Health Assessment Scoping Document in Support of Registration Review, 37 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0896-0003, last accessed Aug. 18, 2016.

USEPA. 2011. Memorandum – Bromoxynil: Human Health Risk Assessment for Amended Uses on Grass Grown for Seed, Conservation Reserve Program Areas, and Grain Sorghum, 72 pp, available at https://www.regulations.gov EPA-HQ-OPP-2010-0268-0004, last accessed Aug. 18, 2016.

2,4-D

2,4-D is a synthetic auxin herbicide in the phenoxy or phenoxyacetic acid family. It is most commonly used postemergence for selective control of broadleaf weeds in agricultural crops, as well as non-agricultural and industrial sites including residential turf. The registered uses include 2,4-D acid, salts, amines, and esters. 2,4-D is a plant growth regulator. It disrupts multiple growth processes in susceptible plants by affecting proteins in the plasma membrane, interfering with RNA production, and changing the properties and integrity of the plasma membrane. Excessive cell division and growth destroy the plant's vascular transport system (USEPA, 2013).

2,4-D has low acute dermal, inhalation and oral toxicities to mammals. It is not a skin irritant or a dermal sensitizer. However, it is a severe eye irritant. The principal organs impacted following repeated 2,4-D exposure include kidney, thyroid, liver, adrenal, eye, and ovaries/testes in test mammals. Developmental effects observed from developmental toxicity studies include skeletal variations, and abortions at higher doses. The observed reproductive toxic effect at elevated doses includes decreased female body weight/body weight gain and male renal tubule alteration, increased gestation length, and pup death. The observed neurotoxic effects include an increased incidence of incoordination and slight gait abnormalities and decreased total motor activity (acute neurotoxicity), and increased relative forelimb grip strength, and increased incidence of bilateral retinal degeneration (subchronic neurotoxicity). USEPA concluded there

was no convincing evidence of potential interaction of 2,4-D with the estrogen, androgen or thyroid pathways based on the Endocrine Disruptor Screening Program (EDSP) weight of evidence evaluation on the Tier 1 screening assay (USEPA, 2015). 2,4-D is classified as "not classifiable as to human carcinogenicity" based on no treatment-related increase in the incidence of any tumor type in the carcinogenic studies in rats and mice. 2,4-D is not considered mutagenic based on the overall evaluation of mutagenic testing results (USEPA, 2012).

Exposure and risk to all human population groups from the use of the herbicide as spot treatments is expected to be negligible. The potential for exposure is greatest for workers during mixing, handling, and applying. Following label directions including restricted entry intervals and proper use of personal protective equipment will minimize exposure and risk to this subgroup of the population. Available human health risk assessment results from USEPA suggests low risk to the public from 2,4-D use based on aggregate risk estimates of concern from combined food, drinking water, and residential exposure for adults or children. USEPA's screening level assessment for residential receptors indicates that residential handler and post-application exposures are not likely to be of concern (USEPA, 2012).

2,4-D is not persistent in the terrestrial environment under aerobic soil conditions. It rapidly degrades through oxidative microbially-mediated mineralization with half-lives ranging from 1.4 days to 12.4 days and a median half-life of 2.9 days. 2,4-D is stable to photodegradation in soil. Terrestrial field dissipation half-lives range from 1.1 to 42.5 days with a median half-life of 6.1 days. 2,4-D was not persistent in aerobic aquatic environments (half-life of 15.0 days); but was moderately persistent to persistent (half-lives of 28.5 to 333 days) in anaerobic aquatic laboratory studies. 2,4-D in water degrades through photodegradation (half-life of 12.9 days in a pH 5.0 buffer solution). Aquatic field dissipation half-lives range from 2.7 to 20.7 days after two applications. 2,4-D is stable to abiotic hydrolysis in buffered aqueous solutions at a range of pH levels. 2,4-D has low volatility to air. 2,4-D is mobile to moderately mobile in soil (intermediately mobile to very mobile in mineral soil), and is soluble in water with a potential to move off site in runoff and leachate (USEPA, 2013).

2,4-D is moderately toxic from acute oral exposure and is slightly toxic from subacute dietary exposure to avian species. 2,4-D has moderately acute oral toxicity and low acute inhalation toxicity to small mammals. 2,4-D is practically nontoxic to pollinators such as the honey bee based on acute contact and oral toxicity studies. 2,4-D is toxic to terrestrial plants and some plant species such as lettuce are more sensitive to 2,4-D based on seedling emergence and vegetative vigor toxicity studies (USEPA, 2013). The exposure and risk to terrestrial organisms from the proposed applications of 2,4-D as spot treatments will be low. Drift from spot treatments are expected to be minimal, and adherence to label directions on any runoff mitigation measures will reduce the potential for exposure to nontarget organisms.

2.4-D acid, amines, and salts are slightly toxic to freshwater and estuarine/marine fish and invertebrates, and practically non-toxic to amphibians. 2,4-D ester is highly toxic to freshwater and estuarine/marine fish, moderately toxic to freshwater invertebrates, and very highly toxic to estuarine/marine invertebrates. Chronic studies with freshwater and estuarine/marine fish reported survival effects at elevated concentrations. 2,4-D affects both nonvascular and vascular plants based on available toxicity studies (USEPA, 2013). There is no proposed direct

application to aquatic system by the program. The risk to aquatic organisms will be reduced based on the program use as a spot treatment and adherence to the label requirements designed to reduce the potential for exposure to aquatic habitats.

References

USEPA. 2013. Memorandum - EFED Registration Review Problem Formulation for 2,4-D – Revised, 86 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0330-0025, last accessed Aug. 19, 2016.

USEPA. 2012. Memorandum – 2,4-D. Human Health Assessment Scoping Document in Support of Registration Review, 36 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0330-0004, last accessed Aug. 19, 2016.

Dicamba

Dicamba is a benzoic acid herbicide widely used in agricultural, industrial and residential settings. Dicamba controls annual, biennial and perennial broadleaf weeds in crops and grasslands, as well as brush and bracken in pastures. Dicamba is primarily formulated in an aqueous solution as a salt. The registered uses include different forms of dicamba (acid and salts). Dicamba has similar structure and mode of action to phenoxy herbicides. Dicamba mimics a plant hormone (auxins) and causes abnormal cell growth by affecting cell division. After absorption by leaves and roots, dicamba moves throughout the plant (USEPA, 2011). Dicamba has low acute oral and dermal toxicity, and very low acute inhalation toxicity to mammals. It is not a dermal sensitizer. However, it is an eye and dermal irritant. Dicamba administered orally is rapidly absorbed and excreted in urine and feces without significant metabolism. The repeated oral and inhalation studies report liver and lung effects in test mammals. Dicamba acid or the salts are not a potential dermal hazard, because there was no evidence of dermal or systemic toxicity from the repeated dermal application of dicamba acid or the salts at a limited dose of 1000 mg/kg/day in rats and rabbits. A developmental toxicity study in rabbits reported an increased incidence of abortion and maternal toxicity at elevated doses. A two-generation reproductive toxicity study with dicamba acid reported offspring toxicity (decreases in pup weight) and parental toxicity. There is no qualitative or quantitative evidence for increased susceptibility following in utero or postnatal exposure of dicamba acid, or its salts, in reproduction and developmental studies using rats and rabbits. Reported neurotoxic effects include ataxia, decreased motor activity, and impaired righting reflex and gait. There is no concern for immunotoxicity (USEPA, 2016). USEPA Endocrine Disruptor Screening Program showed dicamba has no estrogen receptor bioactivity (USEPA, 2015). Dicamba is classified as "not likely to be carcinogenic to humans" based on studies in rats and mice. Dicamba is also not considered to be mutagenic (USEPA, 2016).

Exposure and risk to all human population groups from the use of the dicamba as spot treatments in a crop field is expected to be negligible. The potential for exposure is greatest for workers during mixing, handling, and applying. Label directions including restricted entry intervals and personal protective equipment results in minimal exposure and risk to this population segment. Human health risk assessment results suggest no risks of concern to the public from dicamba

uses based on risk estimations for various potential exposure scenarios (USEPA, 2016). There were no risk estimates of concern for non-occupational exposure from spray drift for aerial and ground boom application or to vapor phase at the edge of treated fields. Additionally, there were no acute and chronic dietary risks of concern for the U.S. population or any population sub-group. With respect to potential post-application exposure, there were no risk estimates of concern for children (1 to <2 years old) from incidental oral routes of exposure. Finally, there were no aggregate risk estimates of concern from short-term combined food, drinking water, and residential exposure for children (USEPA, 2016).

Dicamba is not persistent in soil under aerobic conditions. Aerobic soil metabolism is the main degradative process for dicamba acid in soil. Under aerobic soil conditions, dicamba degrades to the intermediate non-persistent degradate 3,6-dichlorosalicylic acid (DCSA) with a half-life of 6 days, and further degrades to carbon dioxide and microbial biomass at approximately the same degradation rate. Dicamba is more persistent in anaerobic soil or anaerobic aquatic conditions with anaerobic half-life of 141 days. Under anaerobic conditions, DCSA was the major degradate, which was persistent (comprising > 60 percent of the applied after 365 days of anaerobic incubation). Dicamba degrades slowly in soil and water in the presence of light. Field dissipation studies with the dimethylamine salt of dicamba indicate that dicamba dissipated with calculated half-lives ranging from 4.4 to 19.8 days. DCSA was the major degradate. Dicamba is stable to abiotic hydrolysis. It degrades more rapidly in aquatic systems when sediment is present with a half-life of 24 days in a sediment water system. Dicamba is considered volatile and may move off-site in the atmosphere. Dicamba is very mobile in laboratory soil studies and very soluble in water and may reach aquatic resources through runoff or drift. The proposed use pattern for dicamba in the program and label restrictions will reduce the potential for dicamba exposure in surface and groundwater. Dicamba is not expected to bioaccumulate in aquatic organisms (USEPA, 2011, 2005).

Dicamba has moderate to slight acute oral toxicity to birds. Dicamba acid and dicamba salts are practically non-toxic (diglycoamine salt of dicamba is slightly toxic) to avian species based on subacute dietary studies. Dicamba acid is practically non-toxic to small mammals from acute oral exposure. Dicamba is practically non-toxic to pollinators such as the honey bee based on an acute contact toxicity study. Dicamba is toxic to terrestrial plants. It negatively impacts seed germination, seedling emergence, and vegetative vigor in both monocots and dicots. Non-lethal effects include brown leaf tips, necrosis, decrease in size, leaf curling, chlorosis, and stem tumors. Spray drift, runoff, or leaching to roots present potential risks to nontarget plants in close proximity to treated fields because dicamba acid and salts in formulated typical end-use products are readily absorbed through the foliage and roots of plants (USEPA, 2005). The exposure and risk to most terrestrial organisms from the proposed applications of dicamba as spot treatments will be low based on the toxicity and use pattern. The risk to sensitive plants will be reduced by following label directions designed to minimize off-site drift and runoff.

Dicamba acid is slightly toxic and dicamba salts are practically non-toxic to freshwater fish based on acute toxicity studies. The sodium salt of dicamba was slightly toxic and dicamba acid and the other salts were practically non-toxic to freshwater invertebrates in acute exposures. Dicamba acid is practically non-toxic to estuarine/marine fish and invertebrates in acute exposures. Dicamba affects non-vascular and vascular aquatic plants, and is more toxic to non-

vascular plants based on available studies (USEPA, 2005). The exposure and risk to aquatic organisms from the program use as spot treatment is expected to be low based on the proposed use pattern and adherence to label requirements intended to protect aquatic resources.

References

USEPA. 2016. Memorandum – Dicamba and Dicamba BAPMA Salt: Human-Health Risk Assessment for Proposed Section 3 New Uses on Dicamba-tolerant Cotton and Soybean, 105 pp, available at https://www.regulations.gov EPA-HQ-OPP-2016-0187-0009, last accessed Aug. 26, 2016.

USEPA. 2011. Memorandum – Ecological Risk Assessment for Dicamba and its Degradate, 3,6-dichlorosalicylic acid (DCSA), for the Proposed New Use on Dicamba-Tolerant Soybean (MON 87708), 34 pp, available at https://www.regulations.gov EPA-HQ-OPP-2016-0187-0008, last accessed Aug. 26, 2016.

USEPA. 2005. Memorandum – EFED Reregistration Chapter for Dicamba/Dicamba Salts, 220 pp, available at https://www.regulations.gov EPA-HQ-OPP-2009-0081-0073, last accessed Aug. 26, 2016.

Metribuzin

Metribuzin is a systemic triazinone herbicide that selectively controls certain grasses and broadleaf weeds in vegetable and field crops, and non-crop areas including residential turfgrass. The registered metribuzin uses on food and feed crops including alfalfa, asparagus, barley, carrot, corn (field, sweet, and pop), grass (grown for seed only), lentil, pea (including chick pea/garbanzo bean), potato, sainfoin, soybean, sugarcane, tomato, and wheat (USEPA, 2012a), with the largest average amount applied annually on potatoes (USEPA, 2012b). Metribuzin inhibits electron transport in photosynthesis (USEPA, 2012c).

Metribuzin has low to moderate acute oral and low acute inhalation and dermal toxicity to mammals. It is not a dermal or an ocular irritant, nor a dermal sensitizer. The principal toxicities of concern following repeated metribuzin exposure observed in testing mammals are liver and thyroid effects. The reported liver effects include decreased body weight, changes in clinical chemistry associated with liver damage and anemia, changes in liver enzyme activity, increased liver enzymes, increased liver weights, and alterations in hematological parameters. The reported thyroid effects include increased thyroid weights. The liver and thyroid as target organs for metribuzin toxicity is also based on liver hypertrophy in adult rats (a reproductive study), and increased thyroid weights and increased thyroxine hormone levels in rat pups (a developmental study). There were no developmental or reproductive toxicity observed with metribuzin in studies using rats and rabbits. The available data does not show evidence of neurotoxicity or immunotoxicity (USEPA, 2012a). USEPA EDSP Tier 1 screening concluded there was no convincing evidence for potential interaction of metribuzin with the estrogen or androgen pathways. Metribuzin is "not classifiable as to human carcinogenicity" based on conflicting evidence for carcinogenicity in rats and no evidence of carcinogenicity in mice from

the chronic carcinogenicity studies. The mutagenicity testing results show metribuzin is not mutagenic (USEPA, 2012a).

Exposure and risk to all human population groups from the use of the herbicide as spot treatments is expected to be negligible. The potential for exposure is greatest for workers during mixing, handling, and applying. Following label directions and properly using personal protective equipment along with general hygiene practices results in minimal exposure and risk to workers who apply metribuzin. Available human health risk assessment results from USEPA suggest low risk to the public from metribuzin use including infants and children from chronic dietary (food and drinking water) exposure (USEPA, 2012a).

Metribuzin is relatively stable to both aerobic and anaerobic soil metabolism (half-lives of 106 and 112 days, respectively). Metribuzin degrades rapidly by direct photolysis in water and on soil (half-lives of 4.3 hours, and 2.5 days, respectively). Metribuzin is persistence in soil with field half-lives of 40-128 days because only the soil surface (approximately the top 1mm of soil) is actually exposed to sunlight. Metribuzin is stable to abiotic hydrolysis. Metribuzin is expected to persist in groundwater due to its stability to hydrolysis and the lack of light penetration. Metribuzin is not likely to persist in clear, well-mixed, shallow surface water with good light penetration because metribuzin degrades rapidly by aqueous photolysis (half-life of 4.3 hours). However, metribuzin is expected to persist in surface water receiving runoff containing significant sediments because it is stable to hydrolysis and light penetration would be limited. Metribuzin has low vapor pressure and is not considered to be volatile. Metribuzin is mobile in soil and soluble in water suggesting it may move to surface water or ground water (USEPA, 2012c).

Metribuzin has moderately acute oral toxicity to birds. Subacute and chronic dietary exposures to birds suggest low toxicity. Metribuzin has slight acute oral toxicity to mammals. Metribuzin is practically nontoxic to pollinators such as the honey bee based on acute contact toxicity studies. Metribuzin has adverse effects on certain sensitive terrestrial plants. Some plant species such as turnip are more sensitive to metribuzin than others based on seedling emergence and vegetative vigor toxicity studies (USEPA, 2012c). The exposure and risk to most terrestrial organisms from the proposed applications of dicamba as spot treatments will be low based on the toxicity and use pattern. The risk to sensitive plants will be reduced by following label directions designed to minimize off-site drift and runoff.

Metribuzin is slightly toxic to practically nontoxic to freshwater fish and moderately to slightly toxic to freshwater invertebrates in acute exposures. Metribuzin has slightly acute toxicity to estuarine/marine fish and invertebrates. Chronic studies using freshwater fish and invertebrates observed reproductive and growth effects at elevated concentrations. Metribuzin affects nonvascular and vascular plants based on aquatic plants studies (USEPA, 2012c). There is no proposed direct application to aquatic system by the program. The risk to aquatic organisms will be reduced based on the proposed program use of metribuzin and adherence to label requirements designed to protect aquatic resources.

References:

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USEPA. 2012b. Memorandum - BEAD Chemical Profile for Registration Review: Metribuzin (101101), 6 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0487-0005, last accessed Aug. 22, 2016.

USEPA. 2012c. Memorandum - Registration Review: Preliminary Problem Formulation Environmental Fate and Ecological Risk, Endangered Species, and Drinking Water Assessments for Metribuzin, 61 pp, available at https://www.regulations.gov EPA-HQ-OPP-2012-0487-0002, last accessed Aug. 22, 2016.

Pendimethalin

Pendimethalin is a dinitroaniline herbicide that selectively controls broadleaf and grassy weeds in agricultural crops and non-crop areas. Pendimethalin is registered for uses on a variety of agricultural crops (including soybean, cotton, corn, citrus, nuts, pome and stone fruits, vegetables, and berries), turf, and ornamentals (USEPA, 2012a). Pendimethalin acts by inhibiting cell division and cell elongation in plants, and disrupts the process of mitosis in the growth of shoots and roots. After absorption into shoots and roots, pendimethalin generally stays at the intake site with very little translocation (USEPA, 2012a).

Pendimethalin has low mammalian acute toxicity via oral, inhalation, and dermal routes. It is not a dermal or an ocular irritant, nor a dermal sensitizer. The principal toxicity of concern following repeated pendimethalin exposure observed in test mammals is thyroid effects. The reported thyroid effects include alternations in thyroid hormones, increased thyroid weight, and microscopic thyroid lesions (increased thyroid follicular cell height, follicular cell hyperplasia, and follicular cell adenomas). There was no developmental, reproductive, or offspring toxic effects from pendimethalin exposure observed in rabbit or rat developmental studies and the 2-generation reproduction rat study. A developmental thyroid study indicated no toxicity concern of pendimethalin to thyroid glands in the fetus and offspring. There is no evidence of neurotoxicity or immunotoxicity from pendimethalin exposure (USEPA, 2012b). The endocrine disruptor screening program estrogen receptor (ER) bioactivity screening results indicates pendimethalin has ER bioactivity. However, the screening results alone do not provide a scientific basis for whether the chemical has the potential for endocrine disruption without further evaluation (USEPA, 2015). Pendimethalin is classified as “possible human carcinogen” based on thyroid follicular cell adenomas in male and female rats. Pendimethalin was non-mutagenic in mammalian somatic cells and germ cells (USEPA, 2012b).

Exposure and risk to population groups from the use of the pendimethalin as a spot treatment is expected to be negligible. The potential for exposure is greatest for workers during mixing, handling, and applying. Label directions including restricted entry interval and information about personal protective equipment along with general hygiene practices results in minimal exposure and risk to this subgroup of the population. Available human health risk assessment results suggest no risks of concern to the public from pendimethalin uses based on risk estimations for various potential exposure scenarios USEPA (2012b). First, there were no risk estimates of concern for residential handler and post-application exposures. There were also no acute and chronic dietary (food and drinking water) risks of concern for the general U.S. population and all population subgroups. Additionally, there were no short-term aggregate risk estimates of concern from combined background food and water, and residential exposure. An inhalation exposure assessment was performed for flaggers during flagging for aerial spray applications, and there were no risk estimates of concern for this exposure scenario. This flagger exposure scenario would not be used in the program but is considered protective of most outdoor agricultural and commercial post-application inhalation exposure scenarios because it is representative of a worse case inhalation exposure from drift (USEPA, 2012b, c).

Pendimethalin dissipates in the terrestrial environment via sorption to soil, metabolism by microbes, and volatilization into air. The extent of sorption of pendimethalin is related to soil organic content. Microbes can degrade pendimethalin to many non-significant (< 10 percent of applied radio activity) degradates. Pendimethalin has relatively high vapor pressure and may volatilize into the atmosphere. Pendimethalin degrades slowly under aerobic soil conditions with a half-life of 1,322 days. It is stable to soil photolysis and anaerobic soil metabolism. Persistence of pendimethalin in soil decreases with increasing temperature, moisture, or decreasing soil organic carbon. Terrestrial field dissipation studies for pendimethalin reported half-lives ranging from 4 to 147 days at different locations due to soil and climatic differences. Pendimethalin is not mobile in soil with very limited water solubility. There was no leaching observed in the field dissipation studies. In the aquatic environment, pendimethalin degrades by aqueous photolysis with a calculated half-life of 17 days, by aerobic aquatic metabolism with a half-life of 27 days, and by anaerobic aquatic metabolism with a half-life of 68 days. Pendimethalin is stable to sterile hydrolysis. The aquatic field dissipation study reported a half-life of 15 days. Pendimethalin accumulated readily in the bluegill sunfish study with bioconcentration factors of 1400x in edible portions, 5800x in non-edible portions, and 5100x in whole fish. However, depuration of pendimethalin residues occurred 14 days post exposure, which reduces the potential for bioaccumulation (USEPA, 2012a).

Pendimethalin has slight acute oral toxicity to avian species. Pendimethalin is also slightly toxic to avian species based on sub-acute dietary studies. Pendimethalin has low acute oral toxicity to wild mammals. Pendimethalin effects to small mammals from chronic exposure include decreased body weight gain and food consumption, and decreases in the number of pups born and pup weight. Pendimethalin is practically non-toxic to pollinators such as the honey bee based on an acute oral bee toxicity study. Pendimethalin has adverse effects on terrestrial plants. It negatively impacts seedling emergence, and vegetative vigor in both monocots and dicots. Some plant species are more sensitive to pendimethalin than others. Ryegrass and lettuce are the most sensitive monocot and dicot plants, respectively. Radish and cucumber showed no effect in the vegetative vigor test at the highest treatment level (USEPA, 2012a). The exposure and risk

to most terrestrial organisms from the proposed applications of dicamba as spot treatments will be low based on the toxicity and use pattern. The risk to sensitive terrestrial plants will be reduced by following label directions designed to minimize off-site drift and runoff.

Pendimethalin has high acute toxicity to freshwater and estuarine fish and invertebrates. Chronic studies of freshwater fish and invertebrates observed reduced egg production and reduced production of young, respectively. Pendimethalin affects both non-vascular (e.g. growth inhibition) and vascular aquatic plants (e.g. reduced front number), and is more toxic to non-vascular plants based on aquatic plants studies (USEPA, 2012a). The exposure and risk to aquatic organisms will be reduced based on the program use of pendimethalin as a spot treatment and adherence to label restrictions designed to protect aquatic areas.

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Saflufenacil

Saflufenacil is a selective herbicide that controls broadleaf weeds in agricultural crops, fallow croplands, and non-agricultural areas such as pine plantations, rights-of-way, and bare ground. The registered agricultural crops include pre-plant and pre-emergence uses to cereal small grains, corn, chickpeas, cotton, edible beans, edible peas, lentils, lupine, sorghum, soybeans, sunflowers, and olives, and post-emergence uses to fruit tree orchards, nut tree orchards, and vineyards. There are no registered residential uses for saflufenacil (USEPA, 2009). Saflufenacil acts as a cell membrane disruptor and causes cell membrane damage and subsequent plant death by inhibiting protoporphyrinogen oxidase (PPO) (USEPA, 2014a).

Saflufenacil has low acute toxicities via oral and dermal routes and very low acute toxicity via inhalation route to mammals. It is slightly irritating to the eye. It is not a dermal irritant or sensitizer. The primary target organ of saflufenacil following repeated exposure observed in test mammals is the hematopoietic system. PPO inhibition in mammals may disrupt heme synthesis and cause anemia. The observed decreased hematological parameters included red blood cells, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, and mean corpuscular hemoglobin concentration. Other observed toxicity effects included increased weight,

centrilobular fatty change, and lymphoid infiltrate in the liver in mice; increased spleen weight and extramedullary hematopoiesis in the spleen in rats; and increased iron storage in the liver and extramedullary hematopoiesis in the spleen in dogs. There was evidence of increased susceptibility in the developmental and reproduction studies. The developmental studies reported decreased fetal body weights and increased skeletal variations in rats, and increased liver porphyrins in fetuses in rabbits. The 2-generation reproduction study in rats reported an increased number of stillborn pups, decreased pup viability and lactation indices, decreased pre-weaning body weight and/or body-weight gain, and changes in hematological parameters. There was no evidence of neurotoxicity (acute and subchronic neurotoxicity studies), dermal or systemic effects (a 28-day dermal toxicity study) or immunotoxicity from saflufenacil exposure. Saflufenacil is classified as "Not Likely Carcinogenic to Humans" based on no evidence of increased incidence of tumors in rats and mice. Saflufenacil was not considered as mutagenic because it was not mutagenic in bacterial cells, nor clastogenic in rodents *in vivo* (USEPA, 2009).

Exposure and risk to various population groups from the broadcast use of the saflufenacil is low. The potential for exposure is greatest for workers during mixing, handling, and applying. Following label directions including restricted entry intervals, and properly using personal protective equipment along with general hygiene practices results in minimal exposure and risk to this subgroup of the population. Available human health risk assessment results from suggest no risks of concern to the public from saflufenacil uses based on risk estimations for the potential exposure scenarios (USEPA, 2009, 2014a, b). There are no acute or chronic dietary (food and drinking water) risks of concern for the general U.S. population and all population subgroups.

Saflufenacil dissipates in the terrestrial environment via abiotic and biotic degradation, and leaching. It is not persistent in aerobic soil with half-lives of 1 to 5 weeks. The terrestrial field dissipation study for saflufenacil reported half-lives ranging from 1 to 36 days. Saflufenacil is not considered to be volatile based on its low vapor pressure. The water solubility of saflufenacil is pH-dependent. Saflufenacil is hydrophilic and mobile to highly mobile in soil with leaching observed in the field dissipation study. In the aquatic environment, saflufenacil is not persistent in alkaline water bodies with a half-life of less than 1 week, and moderately persistent in acidic to neutral water bodies with half-lives of 4 to 10 weeks (USEPA, 2009).

Saflufenacil is practically non-toxic in acute oral and sub-acute dietary toxicities in birds. An avian reproduction study of saflufenacil using the bobwhite quail reported a reduction in hatchling body weight at high doses. Saflufenacil is practically non-toxic to wild mammals based on information used to evaluate human health. Saflufenacil is practically non-toxic to nontarget terrestrial insects. Saflufenacil has adverse effects on terrestrial plants with dicots being more sensitive than monocots (USEPA, 2009). The exposure and risk to most terrestrial organisms will be negligible from the proposed applications of saflufenacil as spot treatments following label requirements. The risk to sensitive terrestrial plants will be reduced by following label directions designed to minimize off-site drift and runoff and the proposed use pattern of saflufenacil which will be spot treatments.

Saflufenacil is practically non-toxic to fish and freshwater invertebrates, and moderately toxic to estuarine/marine invertebrates. There were no sublethal effects observed in any of the acute aquatic animal studies for saflufenacil. Chronic studies observed 5 percent reduction in embryo survival in fish, and 30% reduction in parental survival and 5 percent reduction in growth in invertebrates at higher doses. Saflufenacil stays in water instead of partition to sediment based on sediment toxicity testing. Exposure of saflufenacil to benthic invertebrates reported a 17 percent reduction in emergence rate (USEPA, 2009). The exposure and risk to aquatic organisms from the program use as spot treatments will be negligible by adherence to label requirements designed to reduce drift and runoff to aquatic areas.

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Thifensulfuron

Thifensulfuron or thifensulfuron methyl is a sulfonylurea herbicide for agricultural (various crops, agricultural fallow, idle, and conservation reserve land), and non-agricultural (cotton, flax) use sites (USEPA, 2011a). Thifensulfuron is registered for postemergence application to barley, canola, chicory, cotton, flax, field corn, oats, safflower, soybeans, and wheat for selective control of broadleaf weeds, as well as for use as a preemergence burn-down broadcast application for barley, field corn, oats, soybeans, and wheat, and as a preplant burn-down application for canola, cotton, rice, grain sorghum, and sugar beets (USEPA, 2015a). Thifensulfuron is formulated as a dry flowable or soluble granules, and applied as a foliar application to weeds (USEPA, 2015a; 2016). After absorption by leaves and roots, thifensulfuron translocates extensively in the xylem and phloem, and accumulates in the meristematic tissue (USEPA, 2016). Thifensulfuron adversely affects plant growth and reproduction through inhibition of the plant enzyme acetolacetate synthase. The enzyme inhibits amino acid synthesis (USEPA, 2016). The program proposes to use thifensulfuron for weed control in litchi tomato after litchi tomato and weeds have emerged.

Thifensulfuron has very low acute oral toxicity (category IV), and low acute inhalation and dermal toxicities (category III) to mammals. It is not a dermal sensitizer. However, it is mildly irritating to the eye (category III) and a slight dermal irritant (category IV). Thifensulfuron methyl administered orally is excreted in the urine (primary route of excretion) and feces within 72 hours with most as parent and almost no tissue or carcass accumulation. The reported toxicity effects for thifensulfuron methyl include decreases in body weights, body weight gains, or organ weights (adrenal, spleen, and thyroid) with no specific target organ toxicity. A developmental toxicity study in rats reported an increased quantitative susceptibility with decreased fetal body

weights, and an increased incidence of small renal papillae observed in fetuses without maternal toxicity. The rabbit developmental or rat reproduction toxicity studies did not show increased susceptibility. Thifensulfuron is not neurotoxic or immunotoxic based on rat neurotoxicity and immunotoxicity studies (USEPA, 2015a). USEPA Endocrine Disruptor Screening Program Estrogen Receptor (ER) Bioactivity showed thifensulfuron has no ER bioactivity (USEPA, 2015b). Thifensulfuron is classified as "not likely to be carcinogenic to humans" based on carcinogenic studies in rats or mice. There was no indication of mutagenicity for thifensulfuron based on available mutagenicity studies (USEPA, 2015a).

Exposure and risk to all human population groups from program use of the thifensulfuron is negligible based on human health risk assessments conducted for broadcast applications. The potential for exposure is greatest for workers during mixing, loading, and applying, as well as during post-application activities. Label directions and proper use of personal protective equipment along with general hygiene practices results in minimal exposure and risk to workers. Human health risk assessment results suggest no risks of concern to the public from thifensulfuron uses based on risk estimations for potential exposure scenarios including dietary exposure (food and drinking water) and indirect exposure (incidental oral and dermal) to thifensulfuron methyl related to spray drift (USEPA, 2015a). For potential dietary exposure, there were no acute dietary risks of concern for females of child-bearing age and no chronic dietary risks of concern for all population subgroups. With respect to potential indirect exposure, there were no risks of concern for adults and children from spray drift using aerial, groundboom or airblast equipment at the edge of treated fields (USEPA, 2015a).

Thifensulfuron in soil degrades through microbial metabolism with an aerobic soil metabolism half-lives of 27.3 to 36.1 days and photolysis with half-lives of 41 to 55 days. The major degradate of the aerobic soil metabolism is IN-L9226, which has similar structure as the parent compound. Thifensulfuron dissipates in water primarily through photodegradation with half-lives of 5.3 to 5.7 days and aerobic aquatic metabolism with half-lives of 21 to 27 days. Thifensulfuron has an anaerobic aquatic metabolism half-life of 19, 21, and 29 days, and hydrolysis half-life of 5.5 days (pH 5), 184 days (pH 7), and 145 days (pH 9). Thifensulfuron is not expected to adsorb to suspended solids and sediment based on its soil adsorption coefficient values (Koc). Terrestrial field dissipation studies indicated that thifensulfuron dissipated with linear half-lives ranging from 0.8 to 2.8 days. Thifensulfuron has low vapor and is unlikely to volatilize into air. Thifensulfuron is highly soluble (water solubility of 2.24 g/L at pH 7), and very mobile in soil suggesting it could contaminate surface and ground water (USEPA, 2011b).

Thifensulfuron is practically non-toxic acute oral and subacute toxicities using upland game bird and waterfowl test species. The chronic toxicity studies for the tested avian species did not show significant chronic effects, but observed a slight and non-significant reduction in the production of eggs and hatchings. Thifensulfuron is practically non-toxic to wild mammals from acute oral exposures. Thifensulfuron is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. Thifensulfuron is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots. Onion and sugarbeet were the most sensitive monocot and dicot species tested in the seedling emergence study. Onion and rape were the most sensitive monocot and dicot species tested in the vegetative vigor study. Spray drift, runoff, or leaching to roots present potential risks to nontarget plants in close

proximity to treated fields because thifensulfuron is soluble and may move into soil and wash-off from foliage and surface soil (USEPA, 2011b). The risks to most terrestrial nontarget organisms is low based on the proposed use pattern and low toxicity however sensitive terrestrial plants in proximity to treated areas could be impacted. The use of spot treatments and adherence to label language designed to minimized off-site movement of thifensulfuron will reduce the risk to sensitive terrestrial plants.

Thifensulfuron is practically non-toxic to freshwater fish and invertebrates based on acute toxicity studies. Thifensulfuron is toxic to both non-vascular and vascular aquatic plants, and is more toxic to vascular plants based on aquatic plants studies (USEPA, 2011b). The exposure and risk to aquatic organisms such as fish and invertebrates is low based on the proposed use pattern and low toxicity of thifensulfuron however there is the potential for impacts to aquatic plants. These risks will be reduced based on the proposed use pattern and label restrictions that reduce risk to aquatic habitats.

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S-ethyl dipropylthiocarbamate (EPTC)

EPTC is a thiocarbamate herbicide used to control the growth of germinating annual weeds (not for established plants) for agricultural and non-agricultural (such as ornamental trees and herbaceous plants) uses. Similar to other thiocarbamate herbicides, EPTC inhibits germination and seedling development through inhibition of cuticle formation at the early stages of seedling growth (USEPA, 2012, 2013a). EPTC is formulated as a granular or emulsifiable concentrate and applied with ground application, soil band treatment, soil broadcast, direct spray, chemigation, flood treatment, and aerial application as a pre-emergence and early post-emergence herbicide (USEPA, 2013a). The program proposes to use EPTC for control of litchi tomato before litchi tomato has emerged.

EPTC has low acute mammalian toxicity via oral and dermal exposure routes, and has moderately high toxicity via inhalation exposure. EPTC is a moderate eye irritant, and a slight skin irritant. It is not a skin sensitizer. ETPC has toxic effects on the central and peripheral nervous systems, produces cardiomyopathy and neuronal cell necrosis, and cholinesterase inhibition in various toxicology studies. EPTC is not a developmental or reproductive toxicant based on studies using the rabbit or rat. Only the developmental neurotoxicity rat study observed evidence of increased qualitative and quantitative susceptibility in the offspring (USEPA, 2013b). The endocrine disruptor screening program weight of evidence indicates there was no convincing evidence of potential interaction with the estrogen, androgen or thyroid pathways (USEPA, 2015). EPTC is classified as "not likely to be carcinogenic to humans". EPTC is not mutagenic based on negative results in mutagenicity studies (USEPA, 2013b).

Exposure and risk to all human population groups from the program use of the herbicide for pre-emergence treatments is expected to be negligible. The potential for exposure and risk is greatest for workers however the low toxicity of EPTC and adherence to label requirements regarding protective personnel equipment results in minimal risk to this subgroup of the population. Available human health risk assessment results suggest no risks of concern to the public from EPTC uses based on risk estimations for various potential exposure scenarios (USEPA, 2013b). For potential dietary exposure, there were no acute and chronic dietary (food and drinking water) risks of concern for all population subgroups. For potential inhalation exposure from volatilization, there were no acute and/or short- and intermediate-term risks of concern for residential bystanders using air monitoring data.

EPTC has low to moderate persistence in the environment. The major dissipation and degradation processes of EPTC are volatilization and microbial-mediated metabolism. In air during daylight hours, EPTC has a half-life of less than one day. The reported aerobic soil metabolism half-life was 153.3 days. The degradation half-lives of EPTC are stable for soil photolysis and anaerobic soil metabolism. Terrestrial field dissipation studies for EPTC reported half-lives ranging from 2 to 56.8 days. The reported aerobic aquatic metabolism half-life was 306.6 days. The degradation half-lives of EPTC are stable for aquatic photolysis and hydrolysis. EPTC is moderately mobile in soil with high water solubility. EPTC reaches surface water via runoff and groundwater via leaching. EPTC has fish bioconcentration factors and depuration rate constants of 30x and 0.14 (edible fish), 57x and 0.15 (whole fish), and 80x and 0.21 (non-edible fish), respectively suggesting a low potential to bioconcentrate (USEPA, 2013a).

EPTC is practically non-toxic to slightly acutely toxic to various bird species. EPTC effects to bird species from chronic dietary exposures include reduction in proportion of viable embryos of eggs. EPTC has low acute oral toxicity to mammals. EPTC effects to small mammals from chronic exposure include decreased body weight, degenerative cardiomyopathy, and renal tubule degeneration. EPTC is practically non-toxic to pollinators such as the honey bee based on an acute oral bee toxicity study. EPTC can have adverse impacts to sensitive emerging terrestrial plants. It negatively impacts seedling emergence (emergence and shoot dry weight and phytotoxicity), and vegetative vigor (shoot dry weight and phytotoxicity) in both monocots and dicots (USEPA, 2013a). The risks to most terrestrial nontarget organisms is low based on the proposed use pattern and low toxicity however sensitive terrestrial plants in proximity to treated areas could be impacted. The adherence to label language designed to minimize off-site movement of EPTC from drift or runoff will reduce the risk to sensitive terrestrial plants.

EPTC is slightly toxic to freshwater and saltwater fish, and moderately to highly toxic to freshwater and saltwater invertebrates in acute exposures. The available aquatic animal chronic study using freshwater invertebrates showed reproductive, survival and growth effects at elevated doses. EPTC affects both growth and biomass of aquatic vascular and non-vascular plants (USEPA, 2013a). The exposure and risk to aquatic organisms will be reduced based on the proposed use pattern and label restrictions that reduce risk to aquatic habitats.

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Imazapic

Imazapic is an imidazolinone herbicide used to control certain grasses and broadleaf weeds in agricultural (such as peanuts, and pastureland/rangeland) and non-agricultural (such as utility rights-of-way, and turf) areas (USEPA, 2014a, b). Imazapic is formulated as a granule, water dispersible granule (dry flowable), soluble concentrate, and a ready-to-use solution that can be applied pre- or post-emergence by aerial and ground equipment (USEPA, 2014a, c). Similar to

other imidazolinone herbicides, imazapic is an inhibitor of acetolactate synthase, which is a key plant enzyme for the biosynthesis of essential branched chain amino acids (i.e., valine, leucine, and isoleucine). These amino acids are important for plant growth. Imidazolinone herbicides generally have very little toxicity to mammals, birds, fish, or insects because animals do not synthesize the amino acids via this pathway (USEPA, 2014c). The program proposes to use imazapic for post-emergence treatments after litchi tomato has emerged.

Imazapic has very low acute oral, dermal and inhalation toxicities to mammals (USEPA, 2014d). It is minimally irritating to the eye, and non-irritating to the skin. It is not a dermal sensitizer. A 90-day oral study in rats and a 21-day dermal study in rabbits observed no toxicity. A one-year dog feeding study reported minimal degeneration, and/or necrosis of the skeletal muscle of the thigh and/or abdomen in both sexes at the lowest doses tested. This study also found toxic effects in liver (increased absolute weights and changes in clinical chemical parameters), kidney (decreased urinary pH in females), and erythropoietic system (changes in hematological parameters and microscopic changes in the bone marrow and spleen) at intermediate doses. In addition, the study found inflammation of esophagus and skeletal muscle, and discoloration of the lung in both sexes at the highest doses. There were no effects observed in imazapic developmental toxicity studies in rats and rabbits. The two-generation imazapic reproductive study in rats reported no parental, offspring or reproductive toxicity. There was no evidence for imazapic to be neurotoxic or immunotoxic (USEPA, 2014d). USEPA Endocrine Disruptor Screening Program Estrogen Receptor (ER) Bioactivity showed imazapic has no ER bioactivity (USEPA, 2015). Imazapic is classified as "not likely to be carcinogenic to humans" based on the absence of carcinogenicity in acceptable studies in rats or mice. Imazapic is not mutagenic based on available studies (USEPA, 2014d).

Exposure and risk various population groups from the use of imazapic as post-emergence treatments in a potato field is expected to be negligible. The potential for exposure and risk is greatest for workers however the low toxicity of EPTC and adherence to label requirements regarding protective personnel equipment results in minimal risk to this subgroup of the population. Available human health risk assessment results suggest no risks of concern to the public from imazapic uses based on various risk estimations for exposure (USEPA, 2014d). There were no chronic dietary risks of concern for all U.S. population subgroups including infants and children. There were also no chronic (food and water) aggregate risks of concern for general U.S. population subgroups. Imazapic can be applied as a residential spot-treatment for weed control on turf. USEPA did not perform quantitative risk estimations for residential post-application exposure because residues on turf from a residential spot-treatment are expected to be negligible (USEPA, 2014d).

Imazapic is persistent based on various laboratory studies (USEPA, 2014c). The major route of dissipation for imazapic is aqueous photolysis (half-life of less than 8 hours) and transport with water. Imazapic is stable to hydrolysis at pH 5, 7, and 9, and has a half-life of 106 days to soil photolysis, an extrapolated half-life of 2010 days to aerobic soil metabolism, and an extrapolated half-life of 2400 days to anaerobic aquatic metabolism. Imazapic is expected to be in an ionized form in the natural environment. Terrestrial field dissipation studies indicated that imazapic dissipated with half-lives ranging from 31 to 223 days. Imazapic has low vapor pressure and is not volatile under normal field conditions. Imazapic is mobile to very mobile in soil and highly

soluble with a potential to reach surface water (via runoff) and groundwater (via leaching). Imazapic is not expected to bioaccumulate in fish (USEPA, 2014c).

Imazapic is considered practically non-toxic in acute oral and subacute dietary toxicities using bird species (USEPA, 2014c). A chronic dietary study in bobwhite quail observed effects on 14-d hatchling weight, live embryos/viable embryos, hatchlings/live embryos. A chronic dietary study in mallard ducks observed effects on 14-d hatchling weight. Effect levels in both studies were at concentrations that would not occur based on the proposed use in the program. Imazapic is practically non-toxic to small mammals from an acute oral exposure. Imazapic is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. Imazapic is toxic to terrestrial plants. It negatively impacts seedling emergence in both monocots and dicots (USEPA, 2014c). The low toxicity of imazapic to most terrestrial nontarget organisms and the proposed use pattern suggest minimal risk to terrestrial vertebrates and pollinators. Spray drift and runoff may pose a risk to sensitive nontarget plants in close proximity to treated fields. Label language designed to minimize off-site transport of imazapic drift or runoff will reduce the risk to sensitive terrestrial plants.

For aquatic organisms, imazapic is practically non-toxic to freshwater fish and invertebrates based on acute toxicity studies. The chronic data indicate that imazapic has no mortality or sublethal effects to freshwater fish and invertebrates. Imazapic is practically non-toxic to estuarine/marine fish and invertebrates. Imazapic is toxic to both non-vascular and vascular aquatic plants (USEPA, 2014c). The exposure and risk to aquatic organisms such as fish and invertebrates is low based on the proposed use pattern and low toxicity of imazapic, however, there is the potential for impacts to aquatic plants. These risks will be reduced based on the proposed use pattern and label restrictions that reduce risk to aquatic habitats.

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Flumioxazin

Flumioxazin is an N-phenylphthalimide herbicide for uses on fruits, vegetables, grains, and field crops; forestry, ornamental plants, lawns and trees; and non-agricultural uses such as right-of-way, commercial and industrial sites (USEPA, 2011a). Flumioxazin generally is formulated as water dispersible granules that can be applied pre- or post-emergence by aerial, ground, or chemigation (USEPA, 2011a, b). Flumioxazin is a light-dependent peroxidizing herbicide. It is an inhibitor of protoporphyrinogen oxidase by blocking heme and chlorophyll biosynthesis resulting in an endogenous accumulation of phototoxic prophyrins (USEPA, 2011b). The program proposes to use flumioxazin to control litchi tomato before it has emerged. Flumioxazin has very low acute oral and inhalation toxicities (category IV), and low acute dermal toxicities (category III) to mammals. It is not an eye or skin irritant, or a dermal sensitizer. Flumioxazin's inhibition of the protoporphyrinogen oxidase enzyme can interfere with the porphyrin component of heme in mammals. The reported toxic effects for flumioxazin include anemia, effects on the liver and the cardiovascular system, and increased renal toxicity in male rats. Hematologic effects of anemia consisted of alterations in hemoglobin parameters. Developmental toxicity studies in rats reported fetal cardiovascular anomalies. The 2-generation reproduction toxicity study reported systemic effects in adult animals, and effects such as decreased pup body weights, a decrease in the number of live born, decreased mating index and testicular atrophy in offspring animals (USEPA, 2011c). Flumioxazin is not neurotoxic or immunotoxic (USEPA, 2012a). USEPA Endocrine Disruptor Screening Program Estrogen Receptor (ER) Bioactivity showed flumioxazin has no ER bioactivity (USEPA, 2015). Flumioxazin is classified as "not likely to be carcinogenic to humans" based on the lack of carcinogenic evidences in rats or mice. There was no concern of mutagenicity for flumioxazin based on available studies (USEPA, 2011c).

Exposure and risk to all population groups from the program use of the flumioxazin as a pre-emergence treatment is expected to be negligible. The potential for exposure is greatest for workers during mixing, loading, and application. Label directions and proper use of personal protective equipment along with general hygiene practices results in minimal exposure and risk to workers. Human health risk assessment results suggest no risks of concern to the public from flumioxazin uses based on risk estimations for potential exposure scenarios including dietary exposure (food and drinking water) and potential post-application inhalation exposure for a residential bystander due to spray drift from neighboring treated fields (USEPA, 2012a,b). There were no acute and chronic dietary risks of concern for all population groups including infants and children. With respect to potential post-application inhalation exposure from spray drift at the edge of treated fields, there were no risks of concern for a residential bystander based on no risk estimates of concern for a flagger. The risk estimation for a flagger is considered to be protective of most outdoor post-application inhalation exposure scenarios because the flagger exposure scenario is representative of a worse case inhalation (drift) exposure. There were no

short-term aggregate risks of concern for females of child-bearing age and for children (1<2 years old) (USEPA, 2012a).

Flumioxazin degrades rapidly in soil and aquatic environments in the presence of microbes. In soil, flumioxazin degrades through anaerobic soil metabolism with a half-life of 0.2 days, soil photolysis with half-lives of 3.2 to 8.4 days, and aerobic soil metabolism with half-lives of 11.9 and 17.5 days. In water, flumioxazin degrades through hydrolysis with half-lives of 4.2, 1 and 0.01 days at pH 5, 7, and 9, respectively, aqueous photolysis with a half-life of 1 day, aerobic aquatic metabolism with half-lives of 3 to 5 days, and anaerobic aquatic metabolism with half-lives of 40.1 to 45.9 days. Flumioxazin dissipates in the environment mainly through rapid hydrolysis, photolysis, and metabolism of the parent compound. Terrestrial field dissipation studies indicate that flumioxazin dissipates with half-lives ranging from 4.8 to 42 days. Flumioxazin volatilizes slowly into air. Flumioxazin has medium soil mobility potential with low potential to reach surface water through runoff and groundwater via leaching. Flumioxazin is not expected to bioaccumulate in fish (USEPA, 2011b).

Flumioxazin is practically non-toxic in acute oral and subacute dietary toxicity studies using avian species. An avian reproduction study using mallard ducks observed significant reductions in the number of viable embryos and live 3-week embryos at the highest concentration of 500 ppm. Flumioxazin is practically non-toxic to small mammals from an acute oral exposure. A chronic reproduction study observed reproductive effects such as decreased number of live-born pups and decreased pup weights. Flumioxazin is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. Flumioxazin is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots (USEPA, 2011b). The low toxicity of flumioxazin to most terrestrial nontarget organisms and the proposed use pattern suggest minimal risk to terrestrial vertebrates and pollinators. Spray drift and runoff may pose a risk to sensitive nontarget plants in close proximity to treated fields. Label language designed to minimize off-site transport of flumioxazin drift or runoff will reduce the risk to sensitive terrestrial plants.

Flumioxazin is moderately to slightly toxic to freshwater fish and moderately toxic to freshwater invertebrates based on acute toxicity studies. An early life-stage toxicity test indicated that flumioxazin significantly affected larval growth (length and weight) of freshwater fish. The chronic data indicate that flumioxazin significantly reduced reproduction, survival and growth (length and weight) of freshwater invertebrates. Flumioxazin is moderately acutely toxic to estuarine/marine fish and moderate to highly acute toxic to estuarine/marine invertebrates. The chronic data indicate that flumioxazin significantly reduced reproduction, growth (length and weight), and survival in estuarine/marine invertebrates. Flumioxazin is toxic to both non-vascular and vascular aquatic plants (USEPA, 2011b). The exposure and risk to aquatic organisms will be reduced based on the low toxicity to some organisms, as well as the proposed use pattern and label restrictions that reduce exposure to aquatic habitats.

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Dimethenamid-p

Dimethenamid-p is a chloroacetamide family herbicide used to control broadleaf and grass weed species on agricultural crops and non-agricultural (such as ornamental plants and non-crop) areas. Herbicides in the chloroacetamide family inhibit long-chain amino acid synthesis. Dimethenamid-p generally is formulated as emulsifiable concentrate, soluble concentrate, or granular that can be applied pre-plant incorporated or surface, pre- or early post-emergence, or layby treatment by aerial broadcast, ground (broadcast and banded), or chemigation (USEPA, 2016a). The program proposes to use dimethenamid-p to control litchi tomato before it has emerged.

Dimethenamid-p has moderate acute toxicity (Category II) via the oral route and low acute toxicity (Category III) from dermal and inhalation routes. Dimethenamid-p is minimally irritating to both the eye (Category III) and the skin (Category IV), and is a mild skin sensitizer. The primary target organ for dimethenamid-p is the liver. Effects from subchronic exposure in test animals included decreased body weights, increased cholesterol and changes in liver weights along with histopathology showing microscopic effects in the liver. Effects from chronic exposure in tested animals include decreases in body weight and food efficiency. Other

observed effects include liver pathology, stomach hyperplasia, and kidney effects at higher dose levels. There were no developmental or reproductive toxicities from utero exposure observed in rats or rabbit developmental studies and the rat reproduction studies from pre- and post-natal exposure. Dimethenamid-p has no neurotoxicity or immunotoxicity effects (USEPA, 2016b). USEPA Endocrine Disruptor Screening Program Estrogen Receptor (ER) Bioactivity showed dimethenamid-p has ER bioactivity (USEPA, 2015a). Dimethenamid-p is classified as a "possible human carcinogen". Dimethenamid-p is not mutagenic based on negative results of mutagenicity studies (USEPA, 2016b).

Exposure and risk to all population groups from the program use of the dimethenamid-p for pre-emergence treatments is expected to be negligible. The potential for exposure and risk is greatest for workers however the low toxicity of dimethenamid-p and adherence to label requirements regarding protective personnel equipment results in minimal risk to this subgroup of the population. Available human health risk assessment results suggest no risks of concern to the public from dimethenamid-p uses based on risk estimations for various potential exposure scenarios (USEPA (2016b). There were no acute and chronic dietary (food and drinking water) risks of concern for all population groups. For potential residential exposure from uses on ornamental plants and post-application exposure, there were no short-term dermal and inhalation risks of concern for residential handlers, and no risks of concern for post-application exposure via dermal and incidental exposure for children 1 to <2 years old. There were no short-term aggregate risks of concern for adults and children (USEPA, 2016b).

Dimethenamid-p persists in soil based on laboratory studies with half-lives ranging from approximately two weeks to more than one month. It degrades through soil aerobic metabolism with a half-life of 13.5 days, and soil photolysis with a half-life of 90 days. Dimethenamid-p has low vapor pressure and volatilization is not a major dissipation process. Terrestrial field dissipation studies indicate dimethenamid-p dissipates with calculated half-lives of 8 to 41 days. Dimethenamid-p in water degrades through water anaerobic aquatic metabolism degradation with a half-life of 35 days and aqueous photolysis with a half-life of 51.4 days. Dimethenamid-p is stable to hydrolysis. Dimethenamid-p is mobile in soil and may move to surface water and groundwater via runoff and leaching. Dimethenamid-p is not expected to bioconcentrate (USEPA, 2015b).

Dimethenamid-p has slight acute oral toxicity and is practically non-toxic via subacute dietary exposures to avian species. Its effects to avian species from chronic exposure include reduced eggshell thickness and body weight at elevated doses. Dimethenamid-p has slightly to moderate acute oral toxicity to wild mammals. Dimethenamid-p is practically non-toxic to pollinators such as the honey bee based on an acute oral bee toxicity study. Dimethenamid is toxic to terrestrial plants. It impacts seedling emergence and vegetative vigor in both monocots and dicots (USEPA, 2015b). The low toxicity of dimethenamid-p to most terrestrial nontarget organisms and the proposed use pattern suggest minimal risk to terrestrial vertebrates and pollinators. Spray drift and runoff may pose a risk to sensitive nontarget plants in close proximity to treated fields. Label language designed to minimize off-site transport of dimethenamid-p drift or runoff will reduce the risk to sensitive terrestrial plants.

Dimethenamid-p has moderate acute toxic to freshwater fish and estuarine/marine invertebrates. It has slight acute toxic to estuary/marine fish and freshwater invertebrates. The aquatic animal chronic studies show reduced larval growth in freshwater fish and reduced survival and growth in freshwater invertebrates. Dimethenamid-p is highly toxic to aquatic vascular plants and adversely affects cell density in aquatic non-vascular plants (USEPA, 2015b). The exposure and risk to aquatic organisms will be low based on the low toxicity to some organisms, as well as the proposed use pattern and label restrictions that reduce exposure to aquatic habitats.

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Aminopyralid

Aminopyralid is a systemic herbicide used to control broadleaf weeds on agricultural crops (such as wheat and pasture crops) and non-agricultural (such as wildlife habitat and industrial vegetation management) areas (USEPA, 2014a, b). Aminopyralid is formulated as an emulsifiable concentrate that can be applied pre- or post-emergence by aerial, ground, and handheld equipment. Aminopyralid is a synthetic auxin analogue in the pyridine carboxylic acid class of herbicides. Absorbed by the plants through foliage and roots, aminopyralid translocates throughout the plant. Aminopyralid acts as a plant growth inhibitor by mimicking natural plant growth hormones (auxins) and causes uncontrolled cell divisions resulting in death of susceptible plant species (USEPA, 2014a). The program proposes to use aminopyralid for post-emergent spot treatment after litchi tomato has emerged.

Aminopyralid has very low acute oral, dermal, and inhalation toxicities to mammals (USEPA, 2014b). Aminopyralid is not a skin irritant or a dermal sensitizer. Aminopyralid (acid form) is severely irritating to the eye (Toxicity Category I) while aminopyralid triisopropanolammonium salt is not irritating to the eye (Toxicity Category IV). The main target organs for aminopyralid are the stomach, ileum, and cecum. The subchronic and chronic studies in rat and dog reported toxic effects such as hyperplasia of the mucosal epithelium of the ileum and cecum, hypertrophy

and hyperplasia of the mucosal epithelium, cecal enlargement, thickening of the stomach mucosa, slight lymphoid hyperplasia of the gastric mucosa, and chronic mucosal inflammation. A developmental study in rabbits observed ulcers and erosions in the glandular mucosa of the stomach in maternal animals as well as decreased body weights and uncoordinated gait. However, no developmental effects were seen in fetuses. There were no developmental or reproductive toxic effects observed in studies in rats, and no evidence of increased pre-and/or post-natal quantitative and qualitative susceptibility in rats or rabbits. Aminopyralid is not neurotoxic or immunotoxic based on available studies. Aminopyralid is not one of the chemicals that has been screened for bioactivity in several endocrine pathways (USEPA, 2016). Aminopyralid is classified as "not likely to be carcinogenic to humans" based on absence of significant tumor increases in rats and mice studies. Aminopyralid is not mutagenic based on negative results of all mutagenicity studies except for an in vitro chromosome aberration assay in rats. However, the clastogenic response was induced secondary to toxicity because aminopyralid induced chromosome aberrations only at cytotoxic concentrations (USEPA, 2014b).

Exposure and risk to all population groups from the program use of the aminopyralid as a post-emergence treatment is expected to be negligible. The potential for exposure and risk is greatest for workers however the low toxicity of aminopyralid and adherence to label requirements regarding protective personnel equipment results in minimal risk to this subgroup of the population. Available human health risk assessment results suggest no risks of concern to the public from aminopyralid uses based on risk estimations for potential exposure scenarios such as potential dietary (food and drinking water) exposure and potential residential post-application exposure (USEPA, 2014b). For potential dietary exposure, there were no chronic dietary risks of concern for the general U.S. population and all population groups. There were no risk estimates of concern for potential residential post-application incidental oral exposure from hand-to-mouth transfer, incidental ingestion of pesticide-treated turf grass and soil scenarios assessed. There were no short-term aggregate risk estimates of concern for children less than 12 years old (i.e., all infants < 1 year old, children 1-2, children 3-5, and children 6-12) (USEPA, 2014b).

Aminopyralid is in an ionic form under most environmentally relevant conditions. Aminopyralid may range from non-persistent to very persistent in the environment based on fate studies. Aminopyralid in soil degrades through aerobic soil metabolism with half-lives ranging from 15 to 148 days under laboratory conditions. Aerobic soil metabolism half-lives for aminopyralid plus unextracted residues ranged from 31 to 193 days. The soil photolysis half-life was 72.2 days (pH 7.7). In water, aminopyralid degrades through aqueous photolysis with a half-life of 0.6 days (pH 5). The aerobic aquatic metabolism half-life ranged from 462 days to 990 days. Aminopyralid is stable to hydrolysis (pH 5, 7, and 9), and to anaerobic aquatic metabolism. Terrestrial field dissipation studies indicate that aminopyralid dissipates with half-lives ranging from 24 to 36 days (half-lives in un-reviewed terrestrial field dissipation studies ranging from 9 to 54 days). Time to 90% dissipation ranged from 40 to 430 days. The half-lives of aquatic field dissipation studies were 10.8 and 14.6 days. Aminopyralid has a low potential to volatilize from soils (low vapor pressure) or from water (low Henry's Law constant) to air. Aminopyralid is relatively soluble and is mobile to highly mobile with the potential to reach surface water (via runoff) and groundwater (via leaching). Aminopyralid may also transport to adjacent area via spray drift. Aminopyralid is unlikely to bioconcentrate in organisms or to accumulate in terrestrial organisms (USEPA, 2014c).

Aminopyralid is practically nontoxic to birds in acute oral and dietary toxicity studies. Chronic effects to birds were not observed with no observed adverse effects at the highest test concentration. Aminopyralid is practically non-toxic to wild mammals from acute oral and inhalation exposures. A chronic toxicity study using small mammals reported no observed adverse effects at the highest aminopyralid concentration tested. Aminopyralid is practically non-toxic to pollinators such as the honeybee based on acute contact and oral toxicity studies. Aminopyralid is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots. Dicots are more sensitive than monocots on both seedling emergence and vegetative vigor (USEPA, 2014c). The low toxicity of aminopyralid to most terrestrial nontarget organisms and the proposed use pattern suggest minimal risk to terrestrial vertebrates and pollinators. Spray drift and runoff may pose a risk to sensitive nontarget plants in close proximity to treated fields. Label language designed to minimize off-site transport of aminopyralid from drift or runoff will reduce the risk to sensitive terrestrial plants.

Aminopyralid is practically non-toxic to freshwater fish and slightly toxic to freshwater invertebrates in acute exposures. A chronic toxicity study using the fathead minnow reported early life stage toxicity with decreased post-hatch survival and sublethal effects. Aminopyralid is practically non-toxic to estuarine/marine fish and invertebrates. There were no observed adverse effects for aminopyralid found in non-vascular plants at concentrations ranged from 6 mg/L to 23 mg/L and in vascular aquatic plants at concentration of 44 mg/L (USEPA, 2014c). The exposure and risk to aquatic organisms from the proposed use of aminopyralid is low for most aquatic organisms based on the favorable toxicity profile and lack of significant exposure.

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Fluroxypyr

Fluroxypyr is a pyridine herbicide used to control broadleaf weeds on various grains (such as wheat, barley, oats, millet, corn, and sorghum), fruits (such as pome fruits), and other areas such as fallow cropland, and range and pasture grasses (USEPA, 2014a). Fluroxypyr is generally formulated as an emulsifiable concentrate and soluble liquid concentrate formulation of fluroxypyr-methylheptyl ester (fluroxypyr-MHE) that can be applied post-emergence by broadcast spray, chemigation, and spot treatment (USEPA, 2014a; b; c). After foliar uptake, fluroxypyr-MHE hydrolyzes to fluroxypyr acid, the herbicidally active form of fluroxypyr (USEPA, 2014c). Fluroxypyr is an auxin analogue that induces auxin-like responses in the plant causing cellular effects such as alterations in cell wall elasticity and gene expression. Fluroxypyr also induces non-productive tissue growth resulting in epinasty and phloem disruption, preventing the movement of photosynthates, and causing death in days to weeks (USEPA, 2014d). The program proposes the use of fluroxypyr for post-emergence treatments after litchi tomato has emerged.

Fluroxypyr has low acute oral and dermal toxicity and moderate acute inhalation toxicity to mammals (USEPA, 2012). Fluroxypyr-MHE is less acutely oral or inhalation toxic than the acid. Both chemicals are non-irritating to the skin. Fluroxypyr-MHE is a mild eye irritant, but is not a dermal sensitizer. The target organ of fluroxypyr is the kidney based on oral exposure studies in rats, mice, and dogs. A 90-day feeding study in rats observed increased kidney weight, nephrotoxicity, and death in both sexes at elevated doses. A chronic study in rats observed increased kidney weight and chronic progressive glomerulonephropathy in both sexes. The developmental toxicity study using fluroxypyr observed increased kidney weight in maternal rats. The 2-generation reproduction study in rats observed kidney effects (e.g. renal failure, increased kidney weight, and microscopic kidney lesions). A 28-day feeding study in dogs found early signs of acute tubular nephrosis, however, the chronic feeding study in dogs did not indicate kidney effects or other treatment related toxicity at the same dose used in the 28-day study. Following long-term exposure of fluroxypyr, kidney lesions (increased incidences of renal papillary necrosis and regenerative nephrosis in females) were found in mice. There were no developmental toxicity in rats or rabbits. There were no reproductive toxicity observed in rats, but abortions were observed in rabbits following exposure to fluroxypyr-MHE at the limit dose. No increased susceptibility was observed following *in utero* exposure to the acid and the ester in rats and rabbits, or following pre and/or postnatal exposure to the acid in rats. Flyoxypyr is not a developmental, immunotoxic, neurotoxic or endocrine disrupting compound based on available data (USEPA, 2012; USEPA, 2015). Fluroxypyr is classified as "not likely to be carcinogenic to humans" based on no evidence of carcinogenicity in acceptable studies in rats or mice. There is no concern of mutagenicity for fluroxypyr based on negative results in mutagenicity studies (USEPA, 2012).

Exposure and risk to all population groups from the program use of fluroxypyr as a post-emergence treatment is expected to be negligible. The potential for exposure is greatest for workers during mixing, loading, and applying, as well as during post-application activities. Following label directions including restricted entry interval and properly using personal protective equipment and general hygiene practices will minimize exposure and risk to this

workers. Available human health risk assessment results suggest no risks of concern to the public, including children, from fluroxypyr use based on risk estimates for potential exposure scenarios including residential post-application exposure (residential turf), and dietary exposure (food and drinking water) (USEPA 2012; 2014c). These exposure scenarios would be considered conservative when compared to the proposed program use of fluroxypyr since no residential applications or direct applications to food items would occur in the PCN program. For potential dietary exposure, there were no chronic dietary risks of concern for all U.S. population subgroups including infants and children. There were no short-term (food, water and residential incidental oral), intermediate-term (food, water, residential incidental oral), and chronic (food and water) aggregate risks of concern for various population groups, including children (USEPA, 2012).

Fluroxypyr-MHE is not considered persistent with an aerobic soil half-life of less than one day and an aerobic aquatic half-life of approximately one day under basic conditions (pH 8.2) (USEPA, 2014d). Fluroxypyr acid is expected to be more persistent with aerobic soil metabolism half-lives from 8.2 to 30 days, and an aerobic aquatic half-life of approximately 3 days. Hydrolysis of fluroxypyr-MHE is pH dependent with a rapid half-life of approximately 3 days under basic conditions (pH 9) and a half-life of 454 days under acidic conditions. Fluroxypyr-MHE is subject to both abiotic (aqueous photolysis and alkaline hydrolysis) and biotic (metabolism) degradation. Fluroxypyr acid is expected to be present as the anion in the environment. Fluroxypyr-MHE undergoes rapid deesterification to fluroxypyr acid following application based on laboratory data. Terrestrial field dissipation studies indicate that combined residues of fluroxypyr-MHE and fluroxypyr acid degrade with half-lives ranging from 5 to 55 days. These studies also showed that fluroxypyr-MHE residues (i.e., fluroxypyr acid) may leach through the soil profile. Fluroxypyr-MHE is not expected to be volatile to air based on its low vapor pressure. Fluroxypyr-MHE is not expected to be mobile in soil and has limited solubility. Fluroxypyr is expected to be mobile and soluble with regard to its potential to reach to surface water (via runoff) and groundwater (via leaching). Fluroxypyr-MHE is not expected to bioaccumulate in fish (USEPA, 2014d).

Fluroxypyr-MHE is practically nontoxic to avian species in acute oral and sub-acute dietary studies. Chronic toxicity of fluroxypyr-MHE to birds is low based on available avian reproduction data. Fluroxypyr-MHE also has low toxicity to wild mammals based on the above discussion regarding mammalian data and potential human health effects. Fluroxypyr-MHE is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. Fluroxypyr-MHE is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots. Dicots are more sensitive than monocots based on seedling emergence and vegetative vigor studies (USEPA, 2014d). Spray drift presents potential risks to nontarget plants in close proximity to treated fields. Exposure and risk to vertebrate nontarget terrestrial wildlife is expected to be low based on the low toxicity of fluroxypyr and the proposed applications of fluroxypyr. Risks to nontarget plants is the greatest but will be minimized by following labeled directions designed to reduce the amount of drift from applications.

Fluroxypyr-MHE has low acute toxicity to all aquatic organisms with the exception estuarine/marine mollusks where it is considered highly toxic. Fluroxypyr acid is much less

toxic to aquatic animals than fluroxypyr-MHE. Fluroxypyr acid is slightly toxic to freshwater fish and saltwater fish based on acute toxicity studies. Fluroxypyr acid is practically non-toxic to freshwater invertebrates and slightly toxic to saltwater invertebrates. The available chronic toxicity data for fluroxypyr acid and freshwater fish (fathead minnow) show effects on in length and wet weight at concentrations and durations of exposure not anticipated under the proposed use pattern. The aquatic toxicity study in freshwater algae reported a NOAEC of 100 mg ai/L (EC50 > 100 mg ai/L) indicating algae are relatively insensitive to fluroxypyr acid (USEPA, 2014d). The exposure and risk to aquatic organisms from the program use as post-emergence treatments will be reduced by adherence to the label requirements regarding applications in proximity to aquatic sites.

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Dimethylamine Salt of 2-Methyl-4-Chlorophenoxyacetic Acid (MCPA Amine)

2-methyl-4-Chlorophenoxyacetic acid (MCPA) is a phenoxy or phenoxyacetic acid herbicide used to control broadleaf weeds on various agricultural (such as barley, flax, oats, peas, rye, wheat, range and pasture grasses, and grasses grown for seed), and non-agricultural (uncultivated fields, ornamental plants, residential lawns, and turf) areas (USEPA, 2004a; 2015a). MCPA formulations include a soluble concentrate/solid, water dispersible granule (dry flowable), and wettable powder that can be applied post-emergence by aerial or ground equipment. The four

active ingredients associated with MCPA are MCPA acid, MCPA sodium salt, MCPA dimethylamine salt (DMAS), and MCPA 2-ethylhexyl ester. Plants uptake MCPA primarily through foliage but may also take up the herbicide via the roots. MCPA is translocated throughout the plant in the xylem and phloem (USEPA, 2004a). MCPA is a synthetic auxin that causes disruption of plant hormone responses and disruption of plant growth processes (USEPA, 2004b). The program proposes the use of MCPA amine for post-emergence treatments.

MCPA amine has low acute oral, dermal, and inhalation toxicity to mammals (USEPA, 2004a). MCPA amine is a severe eye irritant causing corneal opacity (Toxicity Category I), and a slight skin irritant (Toxicity Category III). It is not a dermal sensitizer. The major target organs of MCPA are the kidney and liver. A 90-day oral study in rats reported increased absolute and relative kidney weights, increased clotting time, increased creatinine levels, and presence of crystaluria (oxalate, calcium phosphate, and urate). A 90-day oral study in dogs reported impaired renal function without histopathological change. A 21-day dermal study in rabbits reported kidney effects (increase in incidence of mineralization in renal tubule) and a decrease in body weight gain. A 28-day subchronic inhalation study in rats found epithelial alteration in the larynx and clinical signs at portal of entry following repeat inhalation exposure. There were no developmental toxicity effects found in rats at dose levels producing maternal toxicity, and no developmental toxicity observed in rabbits. There was also no reproductive toxicity observed in rats. MCPA is neurotoxic based on acute oral and subchronic oral neurotoxicity studies in rats and a rat developmental toxicity study using MCPA DMAS. However, the developmental neurotoxicity study did not identify developmental neurotoxicity. The neurotoxic effects include gait impairment in male rats, reduced values of forelimb grip strength and reduced values in the foot splay test in males and reduced values of hindlimb grip strength in females. There is no concern of MCPA for immunotoxicity, and USEPA waived the data requirement for immunotoxicity testing (USEPA, 2014a). USEPA Endocrine Disruptor Screening Program Estrogen Receptor (ER) Bioactivity show that MCPA has no ER bioactivity (USEPA, 2015b). MCPA is classified as “not likely to be carcinogenic to humans” based on long-term carcinogenicity studies in rats and mice. There is no concern of mutagenicity for MCPA based on negative results of inducing mutations in mutagenicity studies (USEPA, 2004a, 2014a).

Exposure and risk to all population groups from the program use of MCPA amine as a post-emergence treatment is expected to be low. The potential for exposure is greatest for workers during mixing, loading, and applying, as well as during post-application activities. Following label directions including restricted entry interval and properly using personal protective equipment and general hygiene practices results in minimal exposure and risk to this group of the population. Available human health risk assessment results suggest no risks of concern to the public, including children, from MCPA amine use based on risk estimates for potential exposure scenarios including residential post-application exposure (residential turf), and dietary exposure (food and drinking water) (USEPA 2004a, 2014a). For potential dietary exposure, there were no acute and chronic dietary risks of concern for all U.S. population subgroups including infants and children. There were no acute and chronic (food and water) aggregate risks of concern for various population groups, including children (USEPA, 2014a).

MCPA DMAS is rapidly converted to MCPA acid in the environment. A dissociation study indicated MCPA DMAS completely dissociated to form MCPA acid in aqueous environments.

Therefore, studies conducted with MCPA acid provide "surrogate data" for the MCPA DMAS. MCPA acid is moderately stable in the environment. It degrades in soil through aerobic soil metabolism with a half-life of 24 days, and photodegradation with a half-life of 67 days. MCPA acid degrades in water through aerobic aquatic metabolism with half-lives of 16 days and 17 days, and photodegradation with a half-life of approximately 25 days in sterile buffer at pH 5. Under aerobic aquatic conditions, MCPA acid degrades in water-sediment systems with half-lives ranged from 13 to greater than 100 days. MCPA acid did not degrade through anaerobic soil or anaerobic aquatic metabolism. MCPA acid does not hydrolyze in sterile buffered solutions at pH 5 to 7. Field studies conducted with MCPA DMAS indicate MCPA dissipated with calculated half-lives of 4.2 days and 8.5 days for grass, and 3.5 days and 10 days for thatch. Field studies conducted with MCPA acid indicate dissipation half-lives of 3.8 and 5.6 days on bare ground, and 3.2 and 6.6 days in wheat plots, and observed half-lives ranging between 7 and 14 days post-treatment. MCPA has low vapor pressure and does not volatilize to air. MCPA acid is mobile in soil and slightly soluble in water with regard to its potential to reach to surface water (via runoff) and groundwater (via leaching). MCPA acid is not expected to bioaccumulate in fish based on low bioconcentration values in fish (USEPA, 2004b, 2014b).
MCPA acid did not degrade anaerobically through anaerobic soil metabolism or anaerobic aquatic metabolism. MCPA acid does not hydrolyze in sterile buffered solutions at pH 5 to 7. Field studies conducted with MCPA DMAS indicated MCPA dissipated with calculated half-lives of 4.2 days and 8.5 days for grass, and 3.5 days and 10 days for thatch. Field studies conducted with MCPA acid indicated MCPA dissipated with calculated half-lives of 3.8 days and 5.6 days on the bare ground, and 3.2 days and 6.6 days on the wheat plot, and the observed half-lives ranged between 7 and 14 days post-treatment. MCPA has low vapor pressure and does not volatilize to air. MCPA acid is mobile in soil and slightly soluble in water with regard to its potential to reach to surface water (via runoff) and groundwater (via leaching). MCPA acid is not expected to bioaccumulate in fish based on no bioconcentration in fish study (USEPA, 2004b; 2014b).

MCPA DMAS is moderately to practically non-toxic to avian species in acute oral and sub-acute dietary studies. The avian reproduction toxicity study using MCPA acid reported no adverse effects at the highest dose. MCPA DMAS is slight toxic to mammals and has low toxicity to wild mammals based on the above discussion regarding mammalian data and potential human health effects. MCPA DMAS is practically non-toxic to pollinators such as the honeybee based on an acute contact toxicity study. MCPA DMAS is toxic to terrestrial plants. It negatively impacts seedling emergence and vegetative vigor in both monocots and dicots. Dicots are more sensitive than monocots based on seedling emergence and vegetative vigor studies (USEPA, 2004b, 2014b). Spray drift presents potential risks to nontarget plants in close proximity to treated fields. Exposure and risk to vertebrate nontarget terrestrial wildlife is expected to be low based on the low toxicity of MCPA DMAS and the proposed applications. Risks to nontarget plants is the greatest but will be minimized by following labeled directions designed to reduce the amount of drift from applications.

MCPA DMAS is slightly too practically non-toxic to freshwater fish and freshwater invertebrate in acute exposures. MCPA DMAS is practically non-toxic to estuarine/marine fish, and moderately to practically non-toxic to estuarine/marine invertebrates. A fish early life-stage toxicity study using MCPA DMAS and fathead minnows indicated effects on length, wet and dry

weights, with a reported no observable adverse effect concentration of 12 ppm. An invertebrate life–cycle toxicity study using MCPA DMAS and freshwater cladocerans reported no observable adverse effect concentration of 11 ppm based on impacts to reproduction. The aquatic plant toxicity studies suggest that MCPA DMAS is highly toxic to algae and aquatic vascular plants with effect concentrations occurring below one ppm. The low toxicity of MCPA DMAS to fish and invertebrates and the proposed use pattern suggest risk to this group of organisms is low. Risk is greater for algae and plants due to the higher toxicity however the proposed use pattern and adherence to label requirements will reduce the exposure to aquatic habitats.

References

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