United States Department of Agriculture

Marketing and Regulatory Programs

Animal and Plant Health Inspection Service

Final Human Health and Ecological Risk Assessment for Carbaryl Rangeland Grasshopper and Mormon Cricket Suppression Applications

November 2019

Final Human Health and Ecological Risk Assessment for Carbaryl Rangeland Grasshopper and Mormon Cricket Suppression Applications

November 2019

Agency Contact:

William Wesela
National Policy Manager
Plant Protection and Quarantine – Policy Management
Animal and Plant Health Inspection Service
U.S. Department of Agriculture
4700 River Road, Unit 134
Riverdale, MD 20737

Table of Contents

EXECUTIVE SUMMARY

The United States Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Plant Protection and Quarantine (PPQ) is proposing the use of the insecticide carbaryl in its cooperative rangeland grasshopper and Mormon cricket suppression program (Program). Carbaryl is a carbamate insecticide. The proposed carbaryl spray formulation, Sevin® XLR Plus, is a liquid containing 5% of the active ingredient that can be applied using ultra-low volume (ULV). The bait formulations (2% Sevin® bait and Sevin® 5 bait) are solids containing 2% and 5% of the active ingredient carbaryl. The Program applies both the spray and bait formulations by ground-based equipment or aerially at reduced rates compared to current conventional labelled rates for grasshopper control.

APHIS evaluated the potential human health and ecological risks from the proposed use of carbaryl ULV sprays and carbaryl bait applications and determined that the risks to human health and the environment are low. The lack of risk to human health and the environment is based on the low probability of human exposure and the favorable environmental fate and effects data. The proposed use of carbaryl as a ULV spray or a bait and adherence to label requirements substantially reduces the potential for exposure to humans and the environment. APHIS does not expect adverse health risks to workers because of the low potential for exposure to carbaryl when applied according to label directions and use of personal protective equipment (PPE) during applications. APHIS quantified the potential risks associated with accidental exposure of carbaryl for workers during mixing, loading, and application. The quantitative risk evaluation results indicate no concerns for adverse health risk for Program workers from carbaryl applications in accordance with program standard operating procedures for safety. APHIS treatments are conducted in rural rangeland areas where agriculture is a primary economic factor. Rural rangeland areas consist of widely scattered, single dwellings in ranching communities with low population density. Risk to the general public from carbaryl ground or aerial applications is also expected to be minimal due to the low-population areas proposed for treatment, adherence to label requirements, and additional Program measures designed to reduce exposure to the public.

Risk to non-target fish and wildlife is low for many taxa and is reduced with the implementation of program measures designed to reduce off-site deposition. Some wildlife within blocks where program treatments occur may be at risk from carbaryl applications but these risks are reduced with use of rates lower than specified on the label, the implementation of reduced agent area treatments and the use of carbaryl bait applications that are more selective than ULV treatments.

1.0 INTRODUCTION

This human health and ecological risk assessment (HHERA) is a qualitative and quantitative evaluation of the potential risks and hazards to human health, non-target fish, and wildlife from the exposure to the N-methyl carbamate insecticide carbaryl. The N-methyl carbamate insecticide group has a common mechanism of toxicity that affects the functioning of the nervous system (carbamylation of acetylcholinesterase (AChE)). The Program applies the insecticide as a bait or ultra-low-volume (ULV) spray using aerial or ground equipment to suppress populations of rangeland grasshopper species, such as the migratory grasshopper, valley grasshopper, bigheaded grasshopper, clearwinged grasshopper, and Mormon cricket.

The methods used to assess potential human health effects follow standard regulatory guidance and methodologies (NRC, 1983; USEPA, 2016), and generally conform to other Federal agencies such as the U.S. Environmental Protection Agency, Office of Pesticide Programs (USEPA/OPP). The methods used to assess potential ecological risk to nontarget fish and wildlife follow USEPA and other published methodologies regarding ecological risk assessment, where applicable.

The HHERA is divided into four sections beginning with problem formulation (identifying hazard), a toxicity effect analysis (the dose-response assessment), and an exposure assessment (identifying potentially exposed populations and determining potential exposure pathways for these populations). The fourth section (risk characterization) integrates the information from the exposure and the dose-response assessments to characterize the risk of carbaryl applications to human health and the environment.

2.0 PROBLEM FORMULATION

Grasshoppers and Mormon crickets are closely related insects that belong to the Order Orthoptera. Nearly 400 grasshopper species inhabit the 17 western States involved in APHIS' grasshopper Program, but only a small percentage are pest species. Anywhere from 15 to 45 species of grasshoppers can be found in a particular rangeland ecosystem, and economic damage can occur when grasshopper populations exceed population thresholds.

Mormon crickets (*Anabrus simplex*) are flightless, shield-backed katydids. Although they do not fly, Mormon crickets are highly mobile and capable of migrating great distances. They move by walking or jumping, and may devour much of the forage in their path.

These insects damage grasses and other vegetation by consuming plant stems and leaves. Their feeding causes direct damage to plants' growth and seed production, thus reducing valuable livestock forage. In addition, the damage they cause to plants may result in: soil erosion and degradation, disruption of nutrient cycles, interference with water filtration, and potentially irreversible changes in the flora and fauna of the rangeland ecosystem. In addition, some populations that develop on rangelands can invade adjacent cropland where the value of crop plants is much higher than rangeland grasses (USDA APHIS, 2015a).

Carbaryl is one of the most widely used broad-spectrum insecticides in agriculture, professional turf management, professional ornamental production, and residential lawns and gardens, as well as in wide-area pest control and public health programs (USEPA, 2017a). APHIS uses carbaryl spray and carbaryl bait formulations in its grasshopper and Mormon cricket suppression Program. The carbaryl ULV spray (Sevin® XLR Plus) formulation is effective against grasshoppers and crickets season-long, and can be used in wet and cool conditions. The carbaryl bait formulation is effective against Mormon crickets which consume the bait almost immediately, but it is not consumed by all species of grasshoppers. Therefore, carbaryl bait is used mostly for Mormon cricket control. The bait formulation can be used season-long and has little drift when applied (USDA APHIS, 2015b).

Carbaryl, an N-methyl carbamate insecticide, affects the inhibition of acetylcholinesterase (AChE) through carbamylation of the serine hydroxyl group (USEPA, 2010a, 2017a). AChE is an enzyme in the nervous system that is necessary for the degradation of the neurotransmitter acetylcholine (ACh). Inhibition of AChE causes an accumulation of ACh and ultimately leads to neurotoxicity in the central and/or peripheral nervous system (USEPA, 2017a).

The following sections discuss the Chemical Description and Product Use; Physical and Chemical Properties; Environmental Fate; and Hazard Identification for carbaryl.

2.1 Chemical Description and Product Use

Carbaryl (CAS No. 63-25-2, $C_{12}H_{11}NO_2$) is the common name of 1-naphthyl methylcarbamate in the carbamate chemical family. The chemical structure is illustrated in figure 2-1.

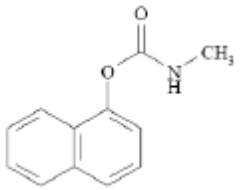

Figure 2-1 The chemical structure of carbaryl

First registered with USEPA in 1959, carbaryl is the active ingredient (a.i.) in the Sevin® formulation (USEPA, 2004). APHIS uses carbaryl baits (Sevin® 5 Bait, EPA Reg. No. 2935-366, and 2% Sevin® Bait, EPA Reg. No. 2935-556) and spray (Sevin® XLR Plus, EPA Reg. No. 61842-37) by aerial or ground application to suppress rangeland grasshoppers during outbreaks (USDA APHIS, 2008; 2016). Sevin® 5 Bait and 2% Sevin® Bait contain 5% or 2% of the active ingredient carbaryl and 95% or 98% inert ingredients, respectively. For both bait formulations, carbaryl is added into wheat bran, rolled oats, or pellets made from grape or apple pomace, or expired human food products (USDA APHIS, 2018). APHIS applies baits at 10 lbs 5% carbaryl bait per acre (ac) (0.5 lb a.i./ac) or 10 lbs 2% carbaryl bait per acre (0.2 lb a.i./ac). Sevin® XLR Plus contains 44.1% carbaryl and 55.9% other ingredients by weight (4 lbs carbaryl per gallon). The Sevin® XLR Plus carbaryl formulation is a microfine suspension in an aqueous medium. It readily disperses in water to form a spray and is applied at 16 or 32 fl. oz. of carbaryl spray per acre (0.5 and 0.25 lb a.i./ac). APHIS uses different formulations based on site-specific conditions. Baits are easier to direct toward the target area, are more specific to grasshoppers and Mormon crickets, and affect fewer nontarget organisms. In addition, baits can be applied when the air temperature is too high to permit effective application of sprays. Baits can also be applied at a safe altitude when the terrain is too rough to allow flying at the low altitude required for spray applications. On the other hand, spray applications typically produce a quicker, higher, and more predictable grasshopper mortality rate (USDA APHIS, 2002).

2.2 Physical and Chemical Properties

Carbaryl is a white to light tan solid with molecular weight of 201.22 g mol, a melting point of 142 ºC, and a vapor pressure of 1.36 $x10^{-7}$ torr at 25 ºC. The Henry's law constant of carbaryl is 1.28 x 10^{-8} atm m^3/mol, its density is 1.21 kg/L at 20 ºC, and its octanol/water partition coefficient (Kow) is 229. Carbaryl has a water solubility of 32 mg/L at 20 ºC (USEPA, 2010b). The Sevin® XLR Plus formulation is a white to beige liquid suspension with a weak phenolic odor (Tessenderlo Kerley, Inc., 2015). The bait formulations are in solid form and the 2% Sevin® Bait is in the form of tan pellets with a sweet odor (Wilbur-Ellis Company LLC, 2016a,b).

2.3 Environmental Fate

The environmental fate describes the processes by which carbaryl moves and is transformed in the environment. The environmental fate processes include: 1) persistence and degradation, 2) mobility and migration potential to groundwater and surface water, and 3) plant uptake.

Carbaryl can be transported in the atmosphere through volatilized spray drift or via particulate residues. Carbaryl's degradation in aerobic soil varies from rapid to slow with half-lives ranging from 4 to 253 days (USEPA, 2017a). Half-lives decrease with increasing pH from acidic to alkaline conditions. Under anaerobic soil conditions, carbaryl has a half-life of 72 days. Carbaryl degrades fairly rapidly in aerobic aquatic systems with a half-life of 4.9 days. However, carbaryl degrades relatively slowly under anaerobic aquatic conditions with a half-life of 68.9 days. The hydrolysis of carbaryl is pH dependent with half-lives of 3.2 hours at pH 9, 12 days at pH 7, and no evidence of degradation at pH 5. Carbaryl degrades rapidly to 1-naphthol through aqueous photolysis with half-life values ranging from 5 hours to 1.8 days. The primary degradate, 1-naphthol, degrades very rapidly with a half-life of less than 1 hour (USEPA, 2010b). Forestry field dissipation data show carbaryl dissipation half-lives of 21 days (foliar), 65 days (soil), and 75 days (leaf litter) (USEPA, 2010b). Terrestrial field dissipation data show carbaryl dissipation half-lives of 62 to 116 days in the upper 30 cm of the soil profile (USEPA, 2017a). 1-naphthol is the major degradate under both aerobic and anaerobic conditions in soil and water. Limited data indicate that 1-naphthol appears to be less mobile and more volatile than carbaryl. Sorption of 1-napthol to soil also increases with increasing organic carbon content. The degradate, 1-naphthol, is expected to be less persistent in the field than carbaryl, but may transport farther in air because of its greater volatility (USEPA, 2017a).

Carbaryl released in soil is expected to have moderate mobility based on its Freundlich Kf values ≤ 3.52. Sorption increases with the increasing soil organic matter content with a Koc of 196 L/kg. Carbaryl has moderate water solubility ranging from 10–1,000 mg/L (NPIC, 2016). Column leaching experiments indicated that carbaryl is slightly mobile in columns (30 cm long) of sandy loam, silty clay loam, silt loam, and loamy sand soils.

The half-life of carbaryl for foliar degradation is 3.71 days and the foliar washoff rate is 0.91 cm-1. The bioaccumulation potential for carbaryl is expected to be low based on its low Kow of 229 (USEPA, 2010b). Carbaryl is not expected to bioconcentrate significantly with bioconcentration factors in fish of 14x in edible tissue, 45x in whole fish, and 75x in visceral tissues (USEPA, 2007a).

2.4 Hazard Identification

Carbaryl is a hazard to human health mainly due to its neurotoxicity (USEPA, 2007b). Carbaryl can cause AChE inhibition (i.e., overstimulation of the nervous system) in humans resulting in nausea, headache, dizziness, anxiety, and mental confusion, as well as convulsions, coma, and respiratory depression at high levels of exposure (CDPR, 2014; NIH, 2009).

2.4.1 Toxicological Effects

Carbaryl targets the nervous system. The carbaryl mode of action (MOA) is carbamylation of AChE resulting in accumulation of the neurotransmitter ACh. The carbamylation of the serine

hydroxyl group located in the active site of the AChE enzyme is reversible with rapid spontaneous recovery of inhibited cholinesterase (USEPA, 2017a; NIH, 2009). However, the carbamylation binding process is reversible which allows for the rapid reactivation of the enzyme. Therefore, only acute exposures are a concern for neurotoxic effects, and repeated daily exposure does not result in an increased inhibition of AChE. AChE inhibition is the most sensitive non-cancer endpoint for carbaryl (USEPA, 2017a).

2.4.2 Metabolism

Carbaryl is rapidly absorbed primarily through the oral route with peak radiolabelled ^{14}C-carbaryl values in various tissues reached at 15 minutes. Dermal absorption of carbaryl is slower with peak values reached at 4 hours, and the highest absorption was 12.7% of a carbaryl formulation (43.9% a.i.). Carbaryl binding to cholinesterase has a short duration with a cholinesterase inhibition half-life of approximately 1.7 hours in rats. Excretion of carbaryl is through urine (approximately 85% of a single oral dose) and also feces (approximately 10% of the administered dose). The primary metabolite, 1-naphthol, is excreted free or conjugated with glucuronide or naphthyl. Excretion of carbaryl metabolites occurs through the bile and undergoes extensive enterohepatic recirculation (USEPA, 2007b, 2017a).

Kidney and blood have the highest concentrations (µg/g tissue) of residual radiolabelled ^{14}C-carbaryl based on metabolism studies in the rat. Rat studies identified carbaryl in the brain, fat, and liver; 1-naphthol in the brain, fat, liver, blood, plasma, and red blood cells (RBC); the sulfate conjugate of 1-naphthol in plasma; and N-hydroxymethyl carbaryl in the brain. There were negligible levels of ^{14}C-carbaryl radioactivity detected in rat tissues at 168 hours following dosing. The metabolic pathway of carbaryl degradation in rats is mainly through hydrolysis of the carbamate ester to yield 1-naphthol, and 1-naphthol is subsequently conjugated to generate a variety of polar metabolites, including 1-naphthol sulfate. Carbaryl was detected in the plasma of intravenously dosed animals and the detected level decreased to below the detection limit one hour after dosing. Both 1-naphthol and 1-naphthol sulfate were detected in the plasma of oral and intravenously dosed animals. The 1-naphthol level decreased to below the detection limit 2 hours post-dosing; however, the 1-naphthol sulfate level did not decrease at 8 hours post-dose (USEPA, 2017a).

2.4.3 Human Incidents

USEPA (2007b, 2010c, and 2017b) performed human poisoning incident reviews for incidents relevant to carbaryl as an active ingredient from the following sources:

1) the USEPA/OPP's incident data system (IDS), comprised of reports of adverse effects submitted by registrants, other federal and state health and environmental agencies, and the public through the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) 6(a)2 since 1992;
2) Center for Disease Control/National Institutes of Occupational Safety and Health (NIOSH) Sentinel Event Notification System for Occupational Risks (SENSOR), which has provided surveillance in 12 states since 1998;

3) California Department of Pesticide Regulation's pesticide poisoning surveillance program, comprised of reports from physicians of illness suspected to be related to pesticide exposure since 1982;
4) Poison Control Center (PCC) data covering the years 1993 through 2005 for all pesticides; and
5) National Pesticide Information Center (NPIC) from January 1, 2011 to August 19, 2016.

USEPA (2010c) identified 347 incident cases for carbaryl from IDS between the years 2002 and 2009. Two hundred and ten incidents occurred between 2007 and 2009 from exposure during and after application, and were mostly of low to moderate severity, though a few incidents were classified as major severity (but no fatalities). The health effects and symptoms from the 2007-2009 IDS reports included dermal (40%) (e.g., rash, hives, blisters, swellings and itchiness), neurological (21%) (e.g., dizziness, headache, tingling and numbness sensation, muscle spasms, tremors, poor coordination, and loss of consciousness), respiratory (17%) (e.g., coughing, respiratory irritation, sore throat, shortness of breath, asthma, difficulty in breathing, and chest pain), gastrointestinal (17%) (e.g., nausea, vomiting, abdominal cramps, and diarrhea), ocular (4%) (e.g., redness, pain and swelling of eyes, itchy watery eyes, pin-point pupils, and blurred vision), and others such as fever, joint pain, and changes in blood pressure and heart rhythm (USEPA, 2010c). An updated review of incident reports identified 356 cases involving carbaryl (322 incidents of carbaryl as a single active ingredient and 34 incidents of multiple active gradients) in the IDS from January 1, 2011 to April 26, 2016 (USEPA, 2017b). Of the 322 single carbaryl incidents, there were 21 classified as major severity, 300 of moderate severity, and one of minor severity. Further review of 101 carbaryl incidents reported to the IDS between 2014 and 2016 identified 60 cases from residential application exposures, 32 cases from residential contact with carbaryl products (post-application, drift, or accidental contact), 5 cases from consumed items treated with carbaryl, and 4 cases from child exposure (1 accidental ingestion, 1 post application exposure, and 2 children getting into the product). There were 15 additional incidents of residential indoor misuse. The symptoms most often reported to the IDS during this time frame were gastrointestinal (diarrhea, hemoptysis, melena, anorexia, abdominal pain, hematemesis, nausea and vomiting), dermal (rash, hives/welts, itchiness, blisters, pruritus, dermal irritation, redness, and swelling), neurological (sweating, headaches, dizziness, numbness, and muscle weakness), respiratory (swelling of throat, shortness of breath, wheezing, throat irritation, nasal irritation, coughing, congestion, bronchitis, and respiratory irritation), ocular (redness, ocular irritation, ocular swelling, burning eyes, lacrimation, and blurred vision), and cardiovascular (tachycardia, elevated blood pressure, heart palpitations, chest pain, and chest tightness).

USEPA's review of cases involving carbaryl as a single active ingredient in the NIOSH SENSOR database from 1998 to 2013 shows that 71% (205 of 287) of the cases occurred from residential exposure and 29% of the cases from occupational exposure (USEPA, 2017b). The most frequent cases occurred when applying or handling the product, and other cases included exposure to residential bystanders or exposed to residues post-application. There were 29 cases of children under the age of 16 exposed to carbaryl residues in the home or outside of the home. Among the 287 cases, most (233 cases, 81%) were low severity, 42 moderate severity, and 12

high severity. Five high severity cases were from ingestion of the product with other high severity cases occurring from exposures during application (one worker at an agricultural establishment and six homeowners). The most frequently reported symptoms include nausea, vomiting, headache, upper respiratory pain, shortness of breath, and dizziness.

USEPA's review (2017b) of the California Pesticide Illness Surveillance Program (PISP) identified 17 (2010–2013) cases involving carbaryl as the active ingredient. The health effects reported include neurological (e.g. shaking, confusion, and headache), gastrointestinal (e.g. vomiting, nausea, and diarrhea), respiratory (e.g., sinus and coughing), ocular (e.g., blurred vision, watering eyes, and burning eyes), dermal (e.g., rash, peeling skin, and blisters), and cardiovascular (e.g., bradycardia, tachycardia, palpitations, and high blood pressure) symptoms. The California Department of Pesticide Regulations (CDPR) human exposure assessment (CDPR, 2014) evaluated a total of 103 illness cases in 76 episodes reported in PISP during the years 1992 through 2009. Most of the illnesses occurred in fieldworkers and handlers. The health effects in these cases include nausea, dizziness, headache, confusion, and weakness; skin effects such as irritation, rashes, itching, and blisters; respiratory illnesses such as sore throat, congestion, coughing, wheezing, and shortness of breath; and eye effects such as irritation, pain, and blurry vision.

USEPA's review (2010c) of epidemiological studies in the National Institutes of Health/National Cancer Institute-led Agricultural Health Study (AHS) concluded that carbaryl exposure may be a risk factor in the development of cutaneous melanoma. Dennis et al. (2010) evaluated 42 AHS publications and found an association between carbaryl and melanoma as well as other adverse effects. USEPA's additional review (2017b) on epidemiological literature from the AHS found insufficient evidence of a clear associative or causal relationship between exposure to carbaryl and the health outcomes investigated in the AHS.

Between January 1, 2011 and August 19, 2016, the National Pesticide Information Center (NPIC) reported 94 human incidents involving carbaryl, with 42 classified as "consistent, possible or probable", meaning that the majority of reported symptoms were consistent with exposure to carbaryl based on the time course between exposure, onset, and duration of symptoms. Based on the symptoms and exposure scenarios of the 42 cases, 26 incidents were classified as minor severity, 15 incidents were classified as moderate severity, and one incident was classified as major severity. Most of the incidents occurred from contact during homeowner application of carbaryl. The symptoms most often reported to NPIC were gastrointestinal (diarrhea, nausea, stomach pain, vomiting, and stomach cramps), neurological (excessive sweating, tingling, dizziness, disorientation, and headaches), ocular (burning, eye irritation, and blurred vision), respiratory (throat irritation, bronchitis, nasal irritation, choking, shortness of breath, and difficulty breathing), dermal (itching, irritation, redness, and burning), and cardiovascular (chest tightness and elevated blood pressure).

The USEPA's incident review (2017b) indicates that the frequency and severity of carbaryl cases since 2010 in IDS, SENSOR, PISP, and NPIC have declined, or have remained consistent at a low frequency.

2.4.4 Acute Toxicity

Technical carbaryl (99% a.i.) has moderate oral acute toxicity in rats (Category II, a combined LD_{50} of 307.0 mg/kg (302.6 mg/kg for males/311.5 mg/kg for females)), low acute dermal toxicity in rabbits (Category III, a LD_{50} of > 2,000 mg/kg), and very low acute inhalation toxicity in rats (Category IV, a LC_{50} of > 3.4 mg/L). Carbaryl is not a primary eye or skin irritant in rabbits. It is not a dermal sensitizer in the guinea pig (USEPA, 2007b). However, human incidents as discussed in the previous section reported dermal irritation and other dermal symptoms caused by carbaryl. The Sevin® XLR Plus formulation safety data sheet (Tessenderlo Kerley, Inc., 2015) reports an acute oral LD_{50} of 699 mg/kg in rats (Category III), an acute dermal LD_{50} of >4,000 mg/kg in rabbits (Category III), and an acute inhalation LC_{50} of 3.84 mg/L in 4-hr exposures to rats (Category IV). The formulation is less toxic in the oral route compared to technical carbaryl, but is considered a mild irritant to the eye and skin.

2.4.5 Subchronic and Chronic Toxicity

A 4-week dermal toxicity study in rats reported a systemic no-observed adverse-effect level (NOAEL) of 20 mg/kg/day, and a LOAEL (lowest observed adverse effect level) of 50 mg/kg/day based on decreased red blood cell AChE activity in males and females and brain AChE activity in males. The study also established a dermal NOAEL of 100 mg/kg/day without a LOAEL (USEPA, 2017a).

Two chronic dermal toxicity studies in dogs reported a LOAEL of 3.1 mg/kg/day based on plasma and brain AChE inhibition and a LOAEL of 4.11 mg/kg/day based on plasma AChE inhibition (USEPA, 2007b, 2017a). In the first study, a NOAEL was not determined for females while in the second study a NOAEL of 1.43 mg/kg/day was reported for males (USEPA, 2007b, 2017a).

2.4.6 Nervous System Effects

An acute neurotoxicity screening battery in rats administering doses of 0, 10, 50, or 125 mg/kg/day reported a NOAEL <10 mg/kg, and a LOAEL of 10 mg/kg based on increased inhibition of AChE in red blood cell, plasma, blood, and brain (USEPA, 2017a).

A subchronic neurotoxicity screening battery in rats administering doses of 0, 1, 10, or 30 mg/kg/day reported a neurotoxicity NOAEL of 1 mg/kg/day, and a neurotoxicity LOAEL of 10 mg/kg/day based on increased functional observational battery (FOB) changes. FOB is a neurobehavioral screening tests for various nervous system effects. The study also reported an AChE inhibition NOAEL of 1 mg/kg/day, and a LOAEL of 10 mg/kg/day based on increased inhibition of AChE in plasma, blood, RBC, and brain (USEPA, 2017a).

A developmental neurotoxicity study in rats administering doses of 0, 0.1, 1.0 and 10 mg/kg/day reported a maternal NOAEL of 1.0 mg/kg/day, and a LOAEL of 10 mg/kg/day based on decreased body weight gain; FOB changes; RBC, plasma, whole blood, and brain AChE

inhibition. The study also reported an offspring NOAEL of 1.0 mg/kg/day, and a LOAEL of 10 mg/kg/day based on alterations in brain morphometrics, including decreased cerebellar length in female pups, increased cerebellar length in adult females, and thickened cerebral cortex thickness in adult males. Morphometric measurements were not evaluated at lower doses. Although effects for parents and offspring were observed at the same dose levels in the developmental neurotoxicity study, offspring effects were more severe than parental effects (USEPA, 2017a).

2.4.7 Reproductive or Developmental Effects

A two-generation reproduction and fertility effects study in rats reported a parental systemic NOAEL of 23.49 (male)/26.91 (female) mg/kg/day, and a LOAEL of 92.43 (male)/110.78 (female) mg/kg/day based on decreased body weight, decreased body weight gain, and feed consumption. The reproductive toxicity NOAEL was 92.43 (male)/110.78 (female) mg/kg/day (the highest doses tested) without a LOAEL. The offspring NOAEL was 4.67 (male)/5.56 (female) mg/kg/day, and the LOAEL was 23.49 (male)/26.91 (female) mg/kg/day based on increased numbers of F2 pups with no milk in the stomach and decreased pup survival. This study showed evidence of quantitative susceptibility because offspring effects of decreased pup survival occurred at a lower dose than parental toxicity (USEPA, 2017a).

A prenatal developmental toxicity study in rats (carbaryl administered at doses of 0, 1, 4 and 30 mg/kg/day by oral gavage) reported a maternal NOAEL of 4 mg/kg/day, and a LOAEL of 30 mg/kg/day based on clinical signs, decreased body weight gain and food consumption. The study also reported a developmental NOAEL of 4 mg/kg/day, and a LOAEL of 30 mg/kg/day based on decreased fetal body weight and incomplete ossification of multiple bones. A prenatal developmental toxicity study in rabbits (0, 5, 50, 150 mg/kg/day by oral gavage) reported a maternal NOAEL of 5 mg/kg/day, and a LOAEL of 50 mg/kg/day based on decreased body weight gain and plasma AChE inhibition. The study also reported a developmental NOAEL of 50 mg/kg/day, and a LOAEL of 150 mg/kg/day based on decreased fetal weight. These developmental toxicity studies in rats or rabbits did not show evidence of increased quantitative or qualitative susceptibility (USEPA, 2017a).

2.4.8 Carcinogenicity and Mutagenicity

The Cancer Assessment Review Committee classified carbaryl as “likely to be Carcinogenic to Humans” based on malignant vascular tumors (an increased incidence of hemangiosarcomas) in male mice (USEPA, 2017a). A carcinogenicity study using the mouse reported a NOAEL of 14.73 mg/kg/day, and a LOAEL of 145.99 mg/kg/day based on increased intracytoplasmic droplets in the bladder of males and females, chronic progressive nephropathy in males, and RBC AChE inhibition in males (USEPA, 2017a). A combined chronic toxicity/carcinogenicity in rats reported a NOAEL of 10 (males)/12.6 (females) mg/kg/day, and a LOAEL of 60.2 (males)/78.6 (females) mg/kg/day based on RBC AChE inhibition. There was an increase in liver adenomas in females, an increase in benign transitional cell papillomas and transitional cell carcinomas in males and females, transitional cell carcinoma in the kidney of one male, an increase in benign thyroid follicular cell adenomas in males, and follicular cell carcinoma in one

male observed at 349.5 and 484.6 mg/kg/day for males and females, respectively (USEPA, 2017a).

In vitro mutagenicity studies indicate that carbaryl is clastogenic based on carbaryl metabolites that react with DNA and cause chromosomal aberrations in cultured mammalian cells, and carbaryl's effects on karyokinesis and cytokinesis and stress genes associated with oxidative damage. However, the in vivo mutagenicity studies did not show effects (USEPA, 2017a).

2.4.9 Endocrine System Effects

USEPA included carbaryl in the endocrine disruptor screening program (EDSP) list 1 chemicals, and performed a weight-of-evidence (WoE) analysis of the potential interaction of carbaryl with the estrogen (E), androgen (A) or thyroid (T) signaling pathways. The WoE evaluation for each pathway (E, A and T) begins with the results of the Tier 1 *in vitro* assays followed by *in vivo* mammalian and wildlife results, then the results of scientifically relevant cited information for mammalian and wildlife studies. The WoE analysis concluded that there is no convincing evidence for interaction of carbaryl with the E or T pathways in mammals or wildlife. There is also no convincing evidence for interaction of carbaryl with the A pathway in mammals. Based on the WoE conclusions in mammals, USEPA did not recommend mammalian EDSP Tier 2 testing. USEPA recommended the EDSP Tier 2 Medaka Extended One-Generation Reproduction Test because of the potential interaction with the A pathway in the fish short-term reproduction assay (USEPA, 2015).

2.4.10 Immune System Effects

There were no significant effects on the immune system observed in most studies in rabbits, mice, and rats at doses permitting survival (USDA FS, 2008). The USEPA guideline immunotoxicity study in rats (administered doses of 0, 24.5, 73.2, or 215.3 mg/kg/day) reported a systemic NOAEL of 73.2 mg/kg/day, and a LOAEL of 215.3 mg/kg/day based on decreased absolute body weights and decreased absolute spleen weights. The study established an immunotoxicity NOAEL of 215.3 mg/kg/day (the highest tested dose), but an immunotoxicity LOAEL was not established (USEPA, 2017a).

2.4.11 Toxicity of Other Ingredients and Metabolites

Approximately 55.9% of the Sevin® XLR Plus formulation is inert ingredients, of which 5% is propylene glycol (1,2-propanediol) (Tessenderlo Kerley, Inc., 2015). The other ingredients are considered confidential business information and are not disclosed.

Propylene glycol absorbs water and is used to make polyester compounds and a base for deicing solutions (antifreeze). Propylene glycol is widely used in the chemical, food, and pharmaceutical industries. The Food and Drug Administration (FDA) has classified propylene glycol as a food additive that is "generally recognized as safe (GRAS)", and is a solvent for food colors and flavors (21 CFR §184.1666). Propylene glycol is a bactericide and fungicide registered with USEPA as an air sanitizer and a hard surface disinfectant, as well as a miticide/insecticide (such

as for fleas and mites) (USEPA, 2007c). Propylene glycol is also an inert ingredient formulated into end-use agricultural and antimicrobial pesticide products.

Propylene glycol has high volatility with a vapor pressure of 0.129 mm Hg at 25 ^{o}C. Propylene glycol in the atmosphere degrades rapidly through photochemical oxidation by reacting with hydroxyl radicals (estimated half-life of 32 hours) (USEPA, 2007d). Propylene glycol in soil rapidly degrades to carbon dioxide (CO_2) in 4 to 9 days under aerobic and anaerobic conditions. Propylene glycol has a low soil organic carbon-water partition coefficient (Koc = 8) and would be expected to be highly mobile in soil. Propylene glycol is highly miscible with water and can be transported to aqueous media (ATSDR, 2008; USEPA, 2007c). Propylene glycol is not likely to bioaccumulate in aquatic organisms due to its low octanol/water partition coefficient (log Kow of -0.92). The potential for propylene glycol to partition from surface water to air is low based on its low air/water partition coefficient Henry's Law Constant (1.31×10^{-10} atm-cu m/mole at 25 ^{o}C). Propylene glycol has a low potential for aquatic hydrolysis, oxidation, volatilization, bioconcentration, and absorptivity to soil (USEPA, 2007d).

Propylene glycol has low toxicity to humans (ATSDR, 2008; USEPA, 2007c). The acute oral LD_{50} values range from 8,000 mg/kg to 46,000 mg/kg in rats, 24,800 mg/kg in mice, and 18,350 to 19,600 mg/kg in rabbits and the guinea pig (USEPA, 2007c). Propylene glycol is not an acute irritant to eyes and skin, and is not a skin sensitizer (USEPA, 2007c). It does not normally irritate the skin although contact dermatitis may occur after a wide variety of topical preparations. Inhaling propylene glycol mist may result in irritation for some individuals (ATSDR, 2008). A subchronic (15-week) oral toxicity study in rats reported a NOAEL of 2,500 mg/kg/day (USEPA, 2007c). Another subchronic (140 days) toxicity study of propylene glycol administered to rats via drinking water reported clinical signs (such as central nervous system depression and minor liver abnormalities) at a dose of 13,200 mg/kg/day. There were clinical signs of toxicity (such as loss of balance, marked depression, and analgesia) reported in mice, guinea pigs, and rabbits at extremely high doses (ranging from 18,400–24,900 mg/kg/day following single oral dose exposures of propylene glycol). A subchronic (90-day) inhalation study in rats reported no changes in respiratory rates, minute volumes, or tidal volumes except for a significant increase in the number of goblet cells in the nasal passages at vapors of 1.0 or 2.2 mg/L. Propylene glycol is not a reproductive or developmental toxicant in mice, rats, hamsters, or rabbits, and there is negligible concern for reproductive or developmental toxicity in humans (NTP, 2004). There is no evidence of propylene glycol being carcinogenic or mutagenic to humans (USEPA, 2007c; ATSDR, 2008). During the USEPA reregistration review, no toxicological endpoints of concern for oral, dermal, or inhalation exposure to propylene glycol based on the available toxicity data were identified (USEPA, 2006). There is no evidence of dermal toxicity and no adverse effects in repeated dose inhalation toxicity studies up to and exceeding the limit dose of 1 mg/L (USEPA, 2007d). USEPA's human incident review did not identify any incidents reported from propylene glycol as an individual chemical exposure (USEPA, 2007c).

Propylene glycol has very low acute toxicity to terrestrial and aquatic animals (USEPA, 2007e). Propylene glycol is practically non-toxic to birds (LD_{50} > 2,000 mg/kg, NOAEL of 2,000 mg/kg), and mammals (LD_{50} > 5,000 mg/kg and NOAEC > 2,500 mg/kg/day). Propylene glycol

is practically non-toxic to freshwater fish (LC_{50} values ranging from 710 to 62,000 ppm), freshwater invertebrates (EC_{50} > 110 ppm, NOEC of 110 mg/kg, and LC_{50} values ranging from 1,020 mg/kg to 18,340 mg/kg), and estuarine and marine organisms (LC_{50} > 10,000 ppm). Propylene glycol used in BioLure traps has a synergistic effect resulting in increased captures of *Anastrepha* fruit flies (Leblanc et al., 2010).

Adverse health risks to humans from exposure to propylene glycol associated with Program use are not expected because of its low toxicity, low percentage of propylene glycol in the formulation, and handler adherence to label and Program safety requirements. Exposure and risk to nontarget fish and wildlife is unlikely based on the low quantity used in the formulation and lack of toxicity.

A primary environmental degradate of carbaryl is 1-naphthol. 1-naphthol is not a cholinesterase inhibitor. USEPA included 1-naphthol in its cancer risk assessment because there is no information to suggest that it does not contribute to the carcinogenicity of carbaryl. 1-naphthol appears to be less mobile and more volatile than carbaryl (USEPA, 2017a). 1-naphthol has limited persistence and is not expected to be found in significant concentrations resulting from carbaryl applications (USEPA, 2007b).

2.4.12 Fire Hazards

Wildfires can occur on rangeland. This section identifies fire hazards from pyrolysis products of carbaryl during a wildfire, and evaluates the associated potential exposure and risk for a firefighter. Fire hazards from other sources are also identified in this section.

Fire hazards from pyrolysis products of carbaryl

APHIS reviewed safety data sheets (SDS) for the carbaryl bait and spray formulations used by the program and conducted a literature search to identify carbaryl pyrolysis products. The SDS for the bait formulation (Wilbur-Ellis Company, 2017a, b) states that hazardous gases may be formed during fire without listing specific pyrolysis products of carbaryl. The SDS for the spray formulation (Tessenderlo Kerley, 2018) states that nitrogen oxides and carbon dioxide can be released from fire. Trace amounts of methyl isocyanate were listed under hazardous decomposition products along with carbon and nitrogen oxides. Toxic combustion products that may be released in a fire involving carbaryl include oxides of nitrogen, methylamine, and carbon monoxide (NIH, 2009). Specific studies of carbaryl pyrolysis products from a wildfire were not identified from the available literature.

Risks for rangeland firefighters from exposure to pyrolysis products of carbaryl was determined by comparing potential levels of carbaryl, and its pyrolysis products, in wildfire-associated smoke to human health hazard benchmarks. The U.S. Occupational Safety and Health Administration (OSHA) occupation safety standards (i.e. permissible exposure limits (PELs)) for carbaryl and its potential pyrolysis products were used as safe exposure levels for firefighters. The PELs are the highest levels of exposure that workers may be exposed to for 8 hours a day without incurring adverse health effects. The OSHA PELs for carbaryl, methyl isocyanate, methylamine, and carbon oxide are an eight-hour time-weighted average (TWA) of 5 mg/m^3,

0.05 mg/m^3, 12 mg/m^3, and 55 mg/m^3, respectively (CDC, 2018a, b, c, d). The OSHA PEL for nitrogen dioxide is a ceiling level of 9 mg/m^3 (CDC, 2018e). APHIS assumed that a wildfire event would occur immediately after application of a bait and that no degradation of carbaryl or removal by grasshoppers would occur after treatment. APHIS used a mixing height of 400 meters to calculate potential residues in the atmosphere from carbaryl and any known degradates that would occur as a result of a wildfire under the conventional rate (0.5 lb a.i./acre) and the reduced agent area treatment (RAAT) rate (0.2 lb a.i./acre). A mixing height of 400 meters in the air represents a conservative exposure scenario because it is the approximate smoke-plume height of the most intensive smoke particulates during initial stage of a prescribed fire using a ground-based scanning lidar (Kovalev, et al,. 2015). Under stagnant conditions, a smoke mixing height is 518 meters or less (Auburn University, No Date). Under the 400-meter mixing height scenario, 0.5 and 0.2 pounds of carbaryl produce 0.14 mg/m^3 and 0.06 mg/m^3 of total combustion products, respectively (appendix A). For trace amounts of methyl isocyanate (less than 1%) in the combustion products, the estimated exposure levels are 0.0014 mg/m^3 and 0.0006 mg/m^3, respectively. The comparison results show that the estimated potential exposure levels for carbaryl and its possible pyrolysis products under a 400-meter mixing height scenario for the conventional or RAAT application rates were all below the occupational health standards. APHIS further evaluated firefighter risks by comparing the exposure doses calculated from the estimated exposure levels to the USEPA's occupational inhalation point of departure (POD) of 1.0 mg/kg/day for carbaryl (USEPA, 2017a). The occupational inhalation POD is a human-equivalent dose of no adverse effect based on an acute inhalation toxicity study in rats. The potential exposure dose for a wildland fire fighter assumes a body weight of 80 kg (USEPA, 2017a), a breathing rate of 24 liter per minutes, and an average of 13.6 hours per daily shift (Navarro, et al., 2019). The estimated exposure dose levels (0.03 mg/kg/day and 0.01 mg/kg/day, appendix A) are below 1.0 mg/kg/day for both the conventional and RAAT application rates suggesting no adverse effects. The estimated margin of exposures (MOEs) under the mixing height of 400 meters for the conventional and RAAT application rates (33 and 100, respectively, appendix A) are higher than the USEPA's level of concern of 30 for the inhalation exposure, which indicates that there is no concern. A MOE is a numerical value that characterizes the amount of safety to a toxic chemical. The actual mixing heights could be greater than 400 meters during an actual wildfire and less bait would be available due to consumption by grasshoppers and Mormon crickets. Inhalation POD values are not available for pyrolysis products of carbaryl. A more detailed risk evaluation for rangeland firefighters from potential exposure to the pyrolysis products of carbaryl is included in appendix A.

In general, grasshopper mortality during the first 2 days after treatment may range from 30 - 80% depending on conditions (such as temperature), and may reach 90% mortality under especially good application conditions (Foster and Onsager 2001a, cited in Beauvais and Struttman, 2003). The best temperature for carbaryl to kill insects is in the 60^o-80^o F range with slower killing at lower temperatures (Foster and Onsager, 2001b, cited in Beauvais and Struttman, 2003). The bait formulations use apple pomace (the solid component of apple after pressing for juice) as a carrier for carbaryl. Apple pomace pellets without carbaryl are sold for livestock and dairy feed and would not contribute to the bait's toxicity (APHIS, 2018b). Although carbaryl in a carrier may be somewhat protected from degradation through microbial interactions, the baits are applied in areas of grasshopper and Mormon cricket activity where the bait would be consumed. The residue unconsumed baits would degrade. The specific half-life of the carbaryl bait formulations

are not available. Carbaryl in general is relatively short-lived in the environment with 14 to 21 days residual activity against grasshoppers (Beauvais and Struttman, 2003). Carbaryl's degradation in aerobic soil varies from rapid to slow with half-lives ranging from 4 days (sandy loam, pH 6.7) to 253 days (silty clay loam, pH 5.8) (USEPA, 2017b). Half-lives decrease with increasing pH from acidic to alkaline conditions. Under anaerobic soil conditions, carbaryl has a half-life of 72 days. Carbaryl generally degrades rapidly on foliage with a foliar degradation half-life of 3.71 days (USEPA, 2010).

Carbaryl burns at 379.4 ^{o}F (Volker, 2016). A shrub dominated fire would be expected to largely consume residual carbaryl bait, where as a grass dominated fire would consume less of the bait with more residual compounds remaining. Because grass dominated fires cool rapidly after burning they would typically require less mop-up activities than a shrub dominated fire (APHIS, 2018b).

Fire hazards from other sources

Various compounds are released in smoke during burning in wildfires including carbon monoxide (CO), CO_2, nitrous oxides (NO_x), sulfur dioxide (SO_2), hydrogen chloride, aerosols, polynuclear aromatic hydrocarbons contained within fine particulate matter (a byproduct of the combustion of organic matter such as wood), and aldehydes, most notably formaldehyde produced from the incomplete combustion of burning biomass (USDA FS, 2013; Reisen and Brown, 2009; Burling et al., 2010; U.S. Department of the Interior, 1992). Particulate matter, CO, benzene, acrolein, and formaldehyde have been identified as compounds of particular concern in wild land fire smoke (Reinhardt and Ottmar, 2004). Respirable particulates carrying absorbed and condensed toxicants can be inhaled into the deeper recesses of the lungs, the alveolar region, and cause inflammation of the lungs, and short-term effects such as cough, shortness of breath, and chest pain (Bytnerowicz, 2009). Symptoms of CO exposure from vegetative smoke include headaches, dizziness, nausea, loss of mental acuity, and fatigue (Occupational Safety and Health Administration, 2002). Symptoms of SO_2 exposure are severe irritation of eyes, skin, upper respiratory tract, and mucous membranes, and bronchoconstriction. SO_2 can damage the airways of humans, and long-term exposure to SO_2 reduces lung volume and its ability for gaseous diffusion (Bytnerowicz, 2009). Greater potential risk results from exposure to these compounds in smoke than to carbaryl pyrolysis products from a wildfire.

The SDS for carbaryl identifies the combustion products of carbaryl as well as recommendations for personal protective equipment (PPE), much of it similar to what is typically used in fighting wildfires. The SDSs for the 2% and 5% Sevin® bait formulations indicate that gases hazardous to health may be formed during fire, and provide instructions to use standard firefighting procedures and consider the hazards of other involved materials (Wilbur-Ellis Company LLC, 2016a,b). The SDS of Sevin® XLR Plus (Tessenderlo Kerley Inc., 2015) indicates that NO_x and CO_2 can be released during fire. Toxic combustion products that may be released in a fire involving carbaryl include oxides of nitrogen, methylamine, and CO (NIH, 2009). Gas or vapor of aliphatic amines such as methylamine is highly irritating, and can cause serious injury to eyes or skin and irritation of the respiratory tract. However, methylamine is not listed as a combustion product in the SDSs of the other formulations that may be used by the Program. Self-contained

breathing apparatus with a full face piece operated in positive pressure mode is specified in the SDS for the Sevin® XLR Plus formulation (Tessenderlo Kerley Inc., 2015), but is not specified in the other two formulations (Wilbur-Ellis Company LLC, 2016a,b). The self-contained breathing apparatus will prevent adverse health effects from smoke inhalation.

Many of the naturally occurring products associated with combustion from wildfires are also present on rangeland where carbaryl has been applied. These naturally occurring combustion byproducts will typically be at higher concentrations and may pose greater risk when compared to carbaryl residues. Removal of carbaryl bait by grasshoppers and Mormon crickets after treatment, and the low application rates and favorable environmental fate for carbaryl reduce the potential for exposure to wildfire firefighters. Carbaryl applied at low rates will degrade rapidly under field conditions that will further reduce exposure to combustion byproducts in the event a fire occurs after treatment. Considerations for treatment would also be made in the event that a grasshopper outbreak occurs in proximity to a wildfire because the effectiveness of the treatment would be less than the wildfire itself.

3.0 DOSE-RESPONSE ASSESSMENT

3.1 Human Health Dose-Response Assessment

A dose-response assessment evaluates the dose levels (toxicity criteria) for potential human health effects including acute and chronic toxicity.

As discussed in Section 2.4.1., the AChE inhibition of carbaryl is a reversible binding process that allows for rapid reactivation and recovery of the enzyme within minutes to hours. Therefore, only acute exposure durations are a concern for neurotoxic effects because the enzyme recovery is complete before the next acute exposure, and the repeated daily exposure does not result in increased inhibition of AChE (USEPA, 2017a).

USEPA has developed a benchmark dose (BMD) analysis that calculates a BMD_{10} and a benchmark dose level $(BMDL)_{10}$ for each exposure scenario for AChE-inhibiting compounds (USEPA, 2012). The BMD_{10} is the estimated dose where 10% inhibition of AChE occurs compared to background. The $BMDL_{10}$ is the lower confidence bound on the BMD_{10}. USEPA uses the BMDL as the point of departure (POD). A POD is the dose-response point that marks the starting point for a low-dose extrapolation.

USEPA (2017a) selected a $BMDL_{10}$ of 1.1 mg/kg (a BMD_{10} of 1.46 mg/kg based on inhibition of brain AChE activity in 11-day-old pups) from a comparative cholinesterase rat study as the incidental oral POD. The $BMDL_{10}$ of 1.1 mg/kg/day for the oral POD is based on AChE inhibition that is protective of effects in the developmental neurotoxicity study (Section 2.4.7.), and was approximately the same as the extrapolated NOAEL of 1.0 mg/kg/day from the developmental neurotoxicity study. The BMD_{10} of 1.46 mg/kg for brain AChE inhibition in 11-day-old pups is 16-fold less than doses resulting in offspring effects in the reproductive study discussed in Section 2.4.7.

USEPA (2017a) uses 1.0 mg/kg/day (a $BMDL_{10}$ of 0.0088 mg/L) as the POD for occupational inhalation exposure. In an acute inhalation dose-response study in rats, USEPA estimated a BMD_{10} of 0.013 mg/L based on brain AChE inhibition in female rats. A $BMDL_{10}$ of 30.56 mg/kg/day is used as the POD for occupational dermal exposure. In a rat adult dermal study, USEPA estimated a BMD_{10} of 49.35 mg/kg/day, based on brain AChE inhibition. The refined dermal POD for humans is 86 mg/kg/day because the comparative rat dermal penetration study showed that rat skin is 2.8 times more permeable than human skin at the low- and mid-dose. USEPA used a dermal absorption factor of 4.5% for risk assessment (USEPA, 2017a).

Carbaryl is classified as "Likely to be Carcinogenic in Humans" based on an increased incidence of hemangiosarcomas in male mice. The cancer potency factor Q1 of carbaryl is 8.75×10^{-4} $(mg/kg/day)^{-1}$ in human equivalent (USEPA, 2017a).

The USEPA has established tolerances for residues of carbaryl on various food commodities (40 CFR 180.169). The carbaryl tolerance level for grass (hay) is 15 ppm.

3.2 Ecological Dose-Response Assessment

3.2.1 Wild Mammal, Avian, Reptile, and Amphibian Toxicity

The acute oral LD_{50} of carbaryl to avian species ranges from 16 mg/kg to >2,000 mg/kg, with starlings (*Sturnis vulgaris*) and red-winged black birds (*Agelaius phoeniceus*) considered to be the most sensitive (Hudson et al., 1984; Schafer et al., 1983). Subacute LC_{50} dietary values for all tested species resulted in values greater than the highest test concentration (Table 3-1).

Table 3-1. Acute oral and dietary avian toxicity values for carbaryl

Test Organism	Endpoint	Toxicity Value	Reference
European starling *Sturnis vulgaris*	LD_{50}	16 mg/kg	Schafer et al., 1983
Red-winged blackbirds *Agelaius phoeniceus*	LD_{50}	56 mg/kg	Schafer et al., 1983
Ring-necked pheasant (male) *Phasianus colchicus*	LD_{50}	>2,000 mg/kg	Hudson et al., 1984
Ring-necked pheasant (female)	LD_{50}	707 mg/kg	Hudson et al., 1984
Canada goose *Branta canadensis*	LD_{50}	1,790 mg/kg	Hudson et al., 1984
Sharp-tailed grouse *Tympanuchus phasianellus*	LD_{50}	<1,000 mg/kg	Hudson et al., 1984
California quail *Lophortyx californicus*	LD_{50}	>2,000 mg/kg	Hudson et al., 1984
Mallard duck *Anas platyrynchos*	LD_{50}	>2,000 mg/kg	USEPA, 2003
Ring-necked pheasant (male) *Phasianus colchicus*	LC_{50}	>5,000 ppm	USEPA, 2003
Northern bobwhite quail *Colinus virginianus*	LC_{50}	>5,000 ppm	USEPA, 2003
Japanese quail *Coturnix japonica*	LC_{50}	>5,000 ppm	USEPA, 2003
Mallard duck *Anas platyrynchos*	LC_{50}	>5,000 ppm	USEPA, 2003

Several toxicity studies evaluating sublethal impacts to avian species have been conducted. Solomon and Robel (1980) dosed northern bobwhite quail (*Colinus virginianus*) twice over a 2-day period with 10, 50, or 90 mg/kg of carbaryl, and measured cholinesterase activity, gross

energy intake, metabolized and excretory energy, and body weight. No effects were seen on any of the assessed parameters. Brain cholinesterase inhibition was not observed because birds were not assessed until 48 hours after the last treatment, and any cholinesterase inhibition was reversed.

Bursian and Edens (1977) found no effects on the F_1 population after a 14-week exposure of carbaryl to mating pairs of Japanese quail (*Coturnix japonica*) at concentrations ranging from 0 to 1,200 ppm. In the adults, decreased body weight, and increased relative brain, liver, and kidney weights were noted in concentrations greater than 900 ppm, suggesting a NOEC of 600 ppm.

In standardized reproduction studies using the northern bobwhite quail (*Colinus virginianus*) and the mallard duck (*Anas platyrynchos*) a NOEC of greater than 3,000 ppm was determined for the northern bobwhite quail while the NOEC for the mallard duck was 300 ppm. The sensitive endpoint in the mallard study was based on the number of eggs produced.

The acute oral LD_{50} of carbaryl to bullfrogs (*Rana catesbeiana*) is greater than 4,000 mg/kg (Hudson et al., 1984). Acute toxicity studies testing other species have demonstrated lower LC_{50} values for tadpoles. Boone and Bridges (1999) demonstrated temperature-related differences in 96-hour LC_{50} values for the green frog (*Rana clamitans*). At 27 $^{\circ}$C, the 96-hour LC_{50} was calculated as 11.32 mg/L, and at 17 $^{\circ}$C, the LC_{50} value was 22.02 mg/L. Bridges et al. (2002) and Dwyer et al. (2005) using tadpoles of the southern leopard frog (*Rana sphenocephala*) and the boreal toad (*Bufo boreas*) determined 96-hour LC_{50} values of 8.4 and 12.31 mg/L, respectively. Zaga et al. (1998) conducted 96-hour acute toxicity tests using *Hyla versicolor* and *Xenopus laevis* tadpoles and reported LC_{50} values of 2.51 and 1.73 mg/L, respectively. Toxicity was enhanced seven fold in the presence of solar ultraviolet radiation. Marian et al. (1983) reported an LC_{50} of 6.2 mg/L for Indian bullfrog (*Hoplobatrachus tigerinus* (=*Rana tigrina*)) tadpoles.

Several sublethal studies have been published assess a variety of endpoints related to potential direct and indirect effects of carbaryl to amphibians. Bridges (1999a) evaluated the effects of carbaryl on swimming behavior and predator avoidance in the gray treefrog (*Hyla versicolor*) at carbaryl concentrations of 1.25 and 2.50 mg/L. Results from the study revealed that swimming activity was significantly reduced at the 2.50 mg/L concentration which could result in increased predation. Another predator-prey interaction study (Bridges 1999b) evaluated the effects of a 24-hour exposure of carbaryl on activity and predation of the southern leopard frog tadpole (*Rana sphenocephala*) by the adult red-spotted newt (*Notophthalmus viridescens*). Southern leopard frog tadpole activity was diminished at 2.5 mg/L while activity for red-spotted newt was not affected. Results from the study suggest that when newts and tadpoles were exposed simultaneously, predation rates did not differ from those under natural conditions, but exposure of either predator or prey at different times can impact the predator-prey relationship. In another predator-prey interaction study, Relyea and Mills (2001) assessed the impacts of carbaryl on the gray treefrog (*Hyla versicolor*). In the first experiment, a 10-day exposure at nominal concentrations of 0.045 and 0.090 mg/L resulted in significant mortality when solutions were changed out every three days of the experiment. A significant predator-treatment interaction was noted using the larval salamander (*Ambystoma maculatum*) resulting in significantly more

predation of treated tadpoles than untreated. In the second and third studies, the gray treefrog was exposed for 16 days to nominal carbaryl concentrations ranging from 1.0 to 8.3 mg/L or 0.07 to 0.54 mg/L. Solutions were changed every 4 days during the experiment. In the higher dosing study, effects on survival were seen by day 3 at the 8.3 mg/L concentration and day 6 at the 1.0 mg/L concentration. The presence of a predator did not affect treefrog survivorship. In the final experiment, at the lower dose range, survivorship was affected at all concentrations after day 10 to 12 depending on dose. In the absence of a predator there was not a dose-dependent effect on survivorship as should be expected. At the end of the 16-day study, there were no effects on survivorship at the highest test concentration (0.54 mg/L) but there was approximately 40% mortality at 0.14 mg/L and 25% at 0.27 mg/L. The lack of a dose response in the presence of a predator also adds to the uncertainty of these results and their use in a risk assessment. In addition, the pH range (8.2–8.5) would suggest rapid hydrolytic degradation ($T_{1/2}$ = 3.2 hours at pH=9), and that changing solutions every three days in the first experiment, and every fourth day in the second and third experiment, subjected the test organisms to multiple pulse doses of carbaryl.

In another swimming performance and activity level study Bridges (1997) used the plains leopard frog (*Rana blairi*) to test carbaryl effects at 3.5, 5.0, and 7.2 mg/L daily over a 96-hour period. There was a 90% reduction in activity at 3.5 mg/L with no activity reported at 7.2 mg/L. There was a slight recovery in activity at 24 hours post-exposure with no recovery of swimming performance. In another study to assess variation of carbaryl tolerance in different species of tadpoles, Bridges and Semlitsch (2000) tested the exposure of nine *Rana* spp. to 30 mg/L and monitored time of death over a 60-hour period while activity was monitored after 24 hours of exposure to 2.5 mg/L carbaryl. Statistically significant differences in time to death were noted with *R. sylvatica* being the most sensitive species and *R. aurora* being the least sensitive. There was no statistically significant interaction of treatment and species in assessing activity, suggesting that all species were equally sensitive. Bridges (2000) assessed the long-term effects of carbaryl exposure at 0.16, 0.40, and 1.0 mg/L to different life stages of the southern leopard frog (*R. sphenocephala*) during a 180-day study where solutions were changed every 3 days. No effects on hatching success and embryo survival were observed at any concentration for the egg and embryo life stages. Significant differences in survival were noted in the tadpole stage at the 0.40 mg/L concentration. Mean age at metamorphosis effects were noted at 0.40 and 1.0 mg/L with a chemical life stage interaction for mass and metamorphosis noted at all concentrations.

Lethal and sublethal impacts have also been assessed in the salamander *Ambystoma barbouri* at concentrations ranging from 0.5 to 50 µg/L (Rohr et al., 2003) during a 37-day exposure. Solutions were changed out every other day for the duration of the study. Carbaryl did not significantly affect hatching day or swimming activity at any of the test concentrations. There was a significant effect on larval survival at 50 µg/L but not at 5 µg/L. The difference in survival from the solvent control was not apparent until approximately 20 days or more after exposure.

3.2.2 Terrestrial Invertebrate Toxicity

Carbaryl is highly toxic to many insects. Carbaryl is highly toxic to honey bees (*Apis mellifera*), with an acute contact LD_{50} of 0.0011 mg/bee; however, acute contact toxicity testing using a soluble concentrate formulation, Carbaryl SC, indicates bees are slightly less sensitive to the formulated product with an LD_{50} of 0.0040 mg/bee (USEPA, 2003). The same trend is true regarding reduced toxicity of the formulation compared to the technical material when assessing oral toxicity studies with the honey bee. The acute oral LD_{50} for the technical material (LC_{50} = 0.0001 mg/bee) is 10 times more toxic than the Carbaryl SC LC_{50} value of 0.0016 mg/bee (USEPA, 2003). Carbaryl resides have been measured in colonies with average levels of 111 µg/kg measured in migratory colonies (Mullins et al, 2010). The 24- and 72-hour acute oral LD_{50} values for the bumble bee (*Bombus terrestris*) ranged from 3.92 to 3.84 µg/bee respectively, suggesting a similar sensitivity range as the honey bee (Marletto et al., 2003). Contact sprays can be very toxic to small, native bees because of direct contact with the insecticide or insecticide residue.

Based on toxicity data for several groups of terrestrial arthropod predators, carbaryl effects can range from moderately to highly toxic (USEPA, 2003). Spiders are not severely affected in carbaryl-treated fields, and recovery occurs within 3 weeks after spraying (Barrett, 1968; Shepard and Sterling, 1972). Carbaryl is severely toxic to predatory mites, but less toxic to phytophagous mites (Bartlett, 1968).

3.2.3 Terrestrial Plant Toxicity

Toxicity to terrestrial plants has also been evaluated for several agronomic crops using the formulation of Sevin® XLR Plus. Typically, USEPA/OPP requires terrestrial phytotoxicity testing using the formulated material. The plants tested that showed no effects at a rate of 0.803 lb a.i./ac were cabbage, cucumber, onion, ryegrass, soybean, and tomato (USEPA, 2003). The carbaryl application rate used in these studies is above the rates (0.50 lb a.i./ac full coverage rate or 0.25 lb a.i./ac Reduced Agent Area Treatment rate (RAAT)). APHIS proposes for use in this Program. Several terrestrial plant incident reports have been filed with USEPA under FIFRA Section 6(a)2; however, for a majority of the cases, the doses used were well above those used in the Program and involved potential misuse in home lawn applications.

3.2.4 Aquatic Vertebrate Toxicity

Acute carbaryl toxicity to fish ranges from slightly to highly toxic. The 96-hour median lethal concentration of carbaryl ranges from 0.25 milligrams/liter (mg/L) for the Atlantic salmon, (*Salmo salar*) to 20 mg/L for black bullhead (*Ameiurus melas*) (Mayer and Ellersieck, 1986). Species of catfish and minnow are generally 10 times more tolerant than salmonids (figure 3–1; appendix B-1).

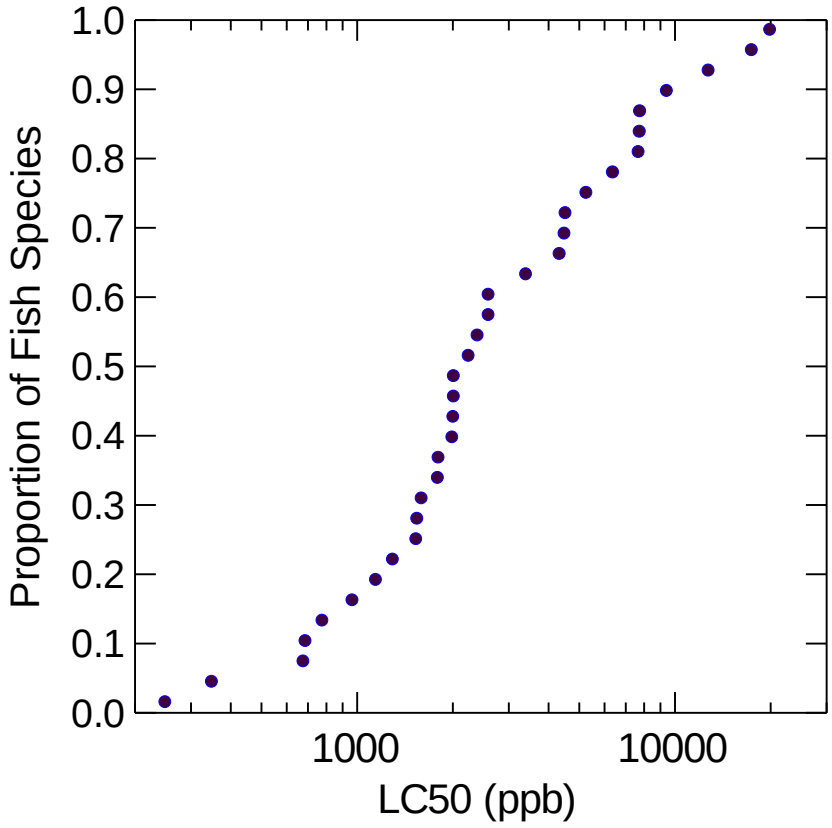

Figure 3–1. Cumulative distribution of acute fish toxicity values for carbaryl

Acute sublethal effect levels related to carbaryl can vary depending on the endpoint and test species. Little et al. (1990) noted several carbaryl-related behavioral effects after a 96-hour exposure period using the rainbow trout (*Oncorhynchus mykiss*). Effects on swimming capacity, swimming activity, and the number of *Daphnia* consumed were statistically significant at a concentration of 1.0 mg/L carbaryl. No effects on the above endpoints were noted at the next concentration, 0.1 mg/L, which represents the NOEC. In a 6-hour exposure of cutthroat trout (*Oncorhynchus clarki*) to carbaryl the concentrations where no effects were seen on predator avoidance or swimming performance was 200 and 500 µg/L, respectively (Labenia et al., 2007). In a 7-day exposure using the fathead minnow (*Pimephales promelas*) at different age classes, the NOEC value ranged from <250 µg/L to 500 µg/L, based on growth. The NOEC value that was less than 250 µg/L was repeated, and the second test demonstrated a NOEC value of 1.0 mg/L based on growth (Pickering et al., 1996).

In addition to behavioral responses, the inhibition of butyrylcholinesterase (BChe) and the regulation of the muscarinic cholinergic receptors (MChR) after carbaryl exposure have been evaluated for several species in short-term exposures (Ferrari et al., 2004a; Ferrari et al., 2004b; Beauvais et al., 2001; Jones et al., 1998; Beyers and Sikoski, 1994; Zinkl et al., 1987).

Ferrari et al. (2004a,b) determined the BChe inhibition concentration (IC_{50}) for larval rainbow trout and the goldfish (*Carassius auratus*) to be 19 µg/L to 2.62 mg/L, respectively. The IC_{50} value for trout (19 µg/L) was calculated using non-linear regression with 95% confidence intervals of 15 and 23 µg/L. The lowest concentration that appears to have been tested, (~ 6 µg/L) resulted in approximately 35% inhibition. Beauvais et al. (2001) documented a statistically significant effect on brain cholinesterase at carbaryl concentrations of 188 µg/L. No other concentrations were tested and, thus, a NOEC could not be established. Beyers and Sikoski (1994) determined the 24-hour NOEC for cholinesterase inhibition to be 30 µg/L for the Colorado pikeminnow (*Ptychocheilus lucius*). Jones et al. (1998) measured MChR in several cold and warmwater fish species. MChR was affected in rainbow trout at 2.2 mg/L and higher but not at doses below 1.3 mg/L. No effects on MChR were observed for the Lahontan cutthroat trout (*Oncorhynchus clarkii henshawi*) or Apache trout (*Oncorhynchus apache*) at the highest concentration tested, 2.2 and 1.3 mg/L, respectively. For the four warmwater species tested in the study (fathead minnow (*Pimephales promelas*), razorback sucker (*Xyrauchen texanus*), bonytail chub (*Gila elegans*), and Colorado pikeminnow), there was a species dependent effect on MChR; however, no impacts were observed for any species at or below a concentration of 1.3 mg/L.

In longer-term studies, chronic NOEC concentrations have been established for the fathead minnow, bonytail chub, and Colorado pikeminnow. In studies ranging from 32- to 35-day exposures, a NOEC value of 210, 445, and 650µg/L was calculated for the fathead minnow, Colorado pikeminnow, and bonytail chub, respectively. Both bonytail chub and Colorado pikeminnow are currently listed species (Beyers et al., 1994). Carlson (1972) reports a NOEC of 210 µg/L for the fathead minnow in a fish full-life cycle study.

3.2.5 Aquatic Invertebrate Toxicity

Carbaryl is very highly toxic to all aquatic insects, and highly to very highly toxic to most aquatic crustaceans. The toxicity from 96-hour static tests ranged from 1.5 µg/L in the shrimp (*Paneaus aztecus*) to 22.7 mg/L in the mussel (*Mytilus edulis*) (USEPA, 2003; Mayer, 1987) (figure 3–2; appendix B-2). Peterson et al. (1994) evaluated EC/LC_{50} values for crustaceans ranging from 5 to 9 µg/L (cladoceran, mysid), 8 to 25 µg/L (scud), and 500 to 2,500 µg/L (crayfish). Aquatic insects have a similar range of sensitivity.

Chronic toxicity of carbaryl to aquatic invertebrates is variable depending on the test species and endpoint measured. Reproductive and growth-related NOECs ranging from 1.0 to 15 µg/L have been reported for cladocerans while a NOEC of 500 µg/L was reported for the chironomid midge based on impacts on emergence (USEPA, 2003; Hanazato, 1991; US FS, 2008) (appendix B-3).

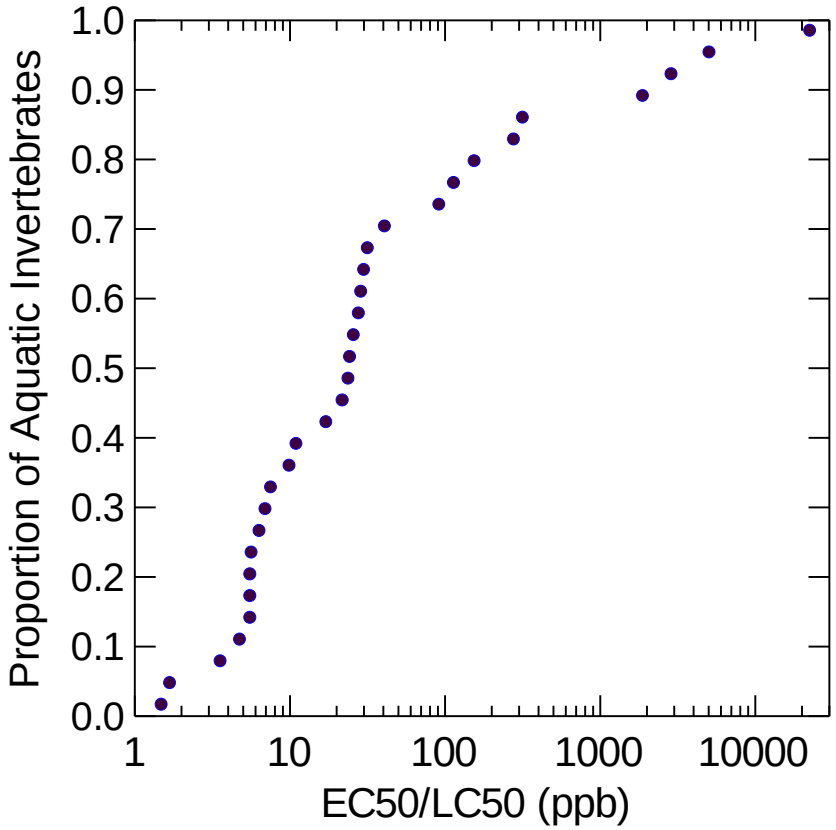

Figure 3–2. Distribution of acute aquatic invertebrate toxicity values for carbaryl

3.2.6 Aquatic Plant Toxicity

Aquatic plant toxicity testing is not typically required for insecticides under USEPA/OPP regulatory requirements. However, studies have been submitted testing the effects to the freshwater green algae (*Pseudokirchneriella subcapitata*), with a reported effective concentration (EC_{50}) and NOEC of 1.27 and 0.29 mg/L, respectively, for the technical active ingredient (US FS, 2008). In another study, the effects of carbaryl on four algal species, seven cyanobacteria species, and the aquatic macrophyte, duckweed (*Lemna minor*) found statistically significant effects at the one dose used in the study (3.7 mg/L) (Peterson et al., 1994). Boonyawanich et al. (2001) reported 96-hour EC_{50} values of 0.996, 0.785, and 0.334 g/L for the three aquatic plants, *Ipomoea aquatica*, *Pistia stratiotes*, and *Hydrocharis dubia*.

3.2.7 Aquatic Toxicity of Formulations and Metabolites

Based on the available toxicity data for various formulations of carbaryl, the toxicity appears to be comparable to the range of sensitivities shown for technical carbaryl and its effects on fish and aquatic invertebrates (table 3–2). Data for the proposed formulation used in this Program is

limited to two acute fish and one algal study. The formulation proposed for use in this Program, Sevin® XLR Plus, contains approximately 44 percent carbaryl and an unknown quantity of 1,2-propanediol according to the available SDS. Other inerts that have been noted in the Sevin® XLR Plus formulation are an unknown sticker material and fine particulates (US FS, 2008).

Table 3–2. Formulation Aquatic Toxicity Data for Carbaryl

Test Organism	Endpoint/Length	% AI	Toxicity Value	Reference
Onchorynchus mykiss	96-hour LC_{50}	44	1.4 mg/L	USEPA, 2003
Onchorynchus mykiss	96-hour LC_{50}	81.5	3.3 mg/L	USEPA, 2003
Onchorynchus mykiss	96-hour LC_{50}	95	1.35 mg/L	Katz, 1961
Onchorynchus kisutch	96-hour LC_{50}	95	0.99 mg/L	Katz, 1961
Onchorynchus mykiss	96-hour LC_{50}	50	3.45 mg/L	USEPA, 2003
Onchorynchus clarki	96-hour LC_{50}	49	6.7 mg/L	Woodward and Mauck, 1980
Cyprinus carpio	96-hour LC_{50}	50	3.30 mg/L	Kaur and Dhawan, 1993
Gambusia affinis	96-hour LC_{50}	5	204 mg/L	Naqvi and Hawkins 1988
Lepomis macrochirus	96-hour LC_{50}	44	9.8 mg/L	USEPA, 2003
Lepomis macrochirus	96-hour LC_{50}	30	49.0 mg/L	USEPA, 2003
Lepomis macrochirus	96-hour LC_{50}	50	22.0 mg/L	USEPA, 2003
Daphnia magna	48-hour EC_{50}	47.3	6.66 µg/L	USEPA, 2003
Daphnia magna	48-hour EC_{50}	81.5	7.2 µg/L	USEPA, 2003
Pseudokirchneriella subcapitata	96-hour EC_{50} 96-hour NOEC	XLR Plus	3.2 mg/L 1.8 mg/L	USEPA, 2003; US FS, 2008

Available toxicity data for the primary metabolite of carbaryl and 1-naphthol was compiled and compared to toxicity data for the parent compound (table 3–3). Available acute and chronic fish data for 1-naphthol is within the range of known EC_{50} /LC_{50} and NOEC values for carbaryl and fish. The same also holds true when comparing available aquatic invertebrate data for carbaryl and 1-naphthol. However, in studies where comparisons were made between technical carbaryl

and 1-naphthol, the metabolite appears to be more toxic. Rao et al. (1984) reported that the 96-hr LC_{50} for technical grade carbaryl was 5.9 mg/L while the comparative value for 1-naphthol was 1.46 when using the fish Cirrhinus mrigala. Tilak et al. (1981) demonstrated that the acute fish toxicity of formulated carbaryl was less toxic than the metabolite 1-naphthol. Calculated 96-hr LC_{50} of carbaryl for *Catla catla, Anabas testudinens, Mystus casius*, and *M. vittatus* were 6.4, 6.6, 4.6, and 2.4 mg/L, respectively, compared to 1-naphthol toxicity values which were 4.3, 3.0, 0.33 and 1.0 mg/L, respectively. Shea and Berry (1983) also reported higher toxicity of 1-napthol compared to technical grade carbaryl; however, no toxicity values were reported.

Table 3–3. 1-Naphthol Laboratory Acute and Chronic Aquatic Toxicity Values

Test Organism	Endpoint/Length	Toxicity Value	Reference
Lepomis macrochirus	96-hour LC_{50}	0.75 mg/L	USEPA, 2003
Cyprinodon variegatus	96-hour LC_{50}	1.2 mg/L	USEPA, 2003
Oncorhynchus mykiss	96-hour LC_{50}	1.4 mg/L	USEPA, 2003
Daphnia magna	48-hour EC_{50}	0.73 mg/L	USEPA, 2003
Mysidopsis bahia	96-hour LC_{50}	0.21 mg/L	USEPA, 2003
Crassostrea virginica	48-hour LC_{50}	2.1 mg/L	USEPA, 2003
Pimepheles pomalis	32-days NOEC	0.10 mg/L	USEPA, 2003

4.0 EXPOSURE ASSESSMENT

4.1 Human Health Exposure Assessment

The exposure assessment estimates the potential exposure of humans to carbaryl. Beginning with the use and application method for carbaryl, a complete exposure pathway then includes (1) release from a carbaryl source, (2) an exposure point where contact can occur, and (3) an exposure route such as ingestion, inhalation, or dermal. In this way, the potentially exposed human populations and complete exposure pathways were identified, and then exposure for the identified human populations was qualitatively or quantitatively evaluated.

4.1.1 Identification of Potentially Exposed Human Populations and Complete Exposure Pathways

The carbaryl bait used for grasshopper suppression is prepared by mixing the appropriate amount of 2% Sevin® bait and Sevin® 5 bait with a cereal grain substrate, as recommended on the current Section 3 label with an application rate of 0.5 lb a.i./ac (conventional) and 0.2 lb a.i./ac (RAATs). The ULV spray, Sevin® XLR Plus carbaryl insecticide is diluted and mixed as recommended on the current Section 3 label with an application rate of 0.5 lb a.i./ac (conventional) and 0.25 lb a.i./ac (RAATs).

Grasshopper suppression would be conducted on rangelands. These rural areas would have low population density, but some rangeland areas may have suburban developments nearby. Recreationists may use rangelands for hiking, camping, bird watching, hunting, falconry or other uses. Ranchers and sheepherders may work on the rangelands on a daily basis. Individuals with allergic or hypersensitive reactions to insecticides may live near or may utilize rangelands in the proposed suppression area. Some rural schools may be located in areas near the rangeland and might be included in treatment blocks. Children may visit areas near treatment blocks or may even enter treatment blocks before or after treatments (USDA APHIS, 2018).

Workers are the most likely human population segment to be exposed to Program carbaryl treatments. Occupational exposure to carbaryl may occur through inhalation and dermal contact during ground and aerial applications. Direct contact exposure from the applications of a carbaryl ULV spray or bait will be minimal when following label requirements regarding PPE, general safety hygiene practices, and restricted entry intervals (REI) into treated areas after application (Wilbur-Ellis Company LLC, 2012, 2014, Tessenderlo Kerley Inc., 2012). The label-required PPE for mixers, loaders, applicators, and handlers include a long sleeved shirt and long pants, shoes plus socks, and chemical-resistant gloves (a chemical-resistant apron is also required for the Sevin® XLR Plus formulation). A National Institute for Occupational Safety and Health (NIOSH)-approved dust/mist filtering respirator with NIOSH/Mine Safety and Health Administration approval number prefix TC-21C or A NIOSH-approved respirator with any N, R, P or HE filter is also required for mixers and loaders in aerial applications or handlers loading bait formulations into airplanes. Engineering controls, such as pilots using an enclosed cockpit in a manner that is consistent with the Worker Protection Standards for Agricultural Pesticides [40

CFR 170.240(d)(6)], are also included on the labels. Human flaggers directing ULV aerial applications from the ground are required to use an enclosed cab that meets the requirements of 40 CFR 170.240(d)(5) for dermal protection as well as respiratory protection. The SDSs also recommended tightly sealed goggles or safety glasses with side shields for eye protection and face protection (Wilbur-Ellis Company LLC, 2016a,b; Tessenderlo Kerley Inc., 2015). The occupational exposure limits (8-hour time weighted average) for carbaryl are 5 mg/m^3 (the Occupational Safety and Health Administration permissible exposure limit) and 0.5 mg/m^3 (inhalable fraction and vapor) (the American Conference of Governmental Industrial Hygienists threshold limit value). Accidental exposure to carbaryl may occur for a worker during application. This accidental exposure scenario is further described and quantified in the next section (4.1.2).

Carbaryl exposure to the general public is minimal from Program use based on label requirements and Program standard operating procedures (USDA APHIS, 2016) that prevent potential exposure to general public. Only protected handlers may be in the area during application and entry of the general public into the treated area is not allowed during the REI period. APHIS treatments are conducted on rural rangelands, where agriculture is a primary economic factor with widely scattered single rural dwellings in ranching communities with low population density. The Program requires avoiding flights over congested areas, water bodies, and other sensitive areas. The required buffer zones for water bodies are 500 feet for aerial liquid insecticides and 200 feet for ground applications. Aerial applications are not allowed while school buses are operating in the treatment area; within 500 feet of schools or recreational facilities. Aerial applications are made only when wind velocity does not exceeds 10 miles per hour (mph) (unless a lower wind speed is required under State law); air turbulence could seriously affect the normal spray pattern; and/or temperature inversions could lead to off-site movement of spray. Program personnel also notify residents within treatment areas, or their designated representatives, prior to proposed operations to reduce the potential for incidental exposure (USDA APHIS, 2016). Off-site drift of carbaryl ULV spray applications may occur, but will be reduced by following label guidance regarding management practices designed to minimize drift (Wilbur-Ellis Company LLC, 2012, 2014, Tessenderlo Kerley Inc., 2012). Potential exposures to the general public from Program application rates will be infrequent and of low magnitude. Inhalation exposures are expected to be negligible for post-application.

The primary use areas for carbaryl include rangeland that could be grazed by livestock. Farmers in areas near proposed suppression areas may grow crops such as alfalfa and corn that are used as feed for livestock (dairies and feedlots). They also grow potatoes, sugar beets, wheat, barley, sweet corn, beans, and a variety of other crops (USDA APHIS, 2018). The labels restrict carbaryl applications within 48 days of harvest of grain and fodder, or within 14 days of harvest or grazing of forage or silage (Tessenderlo Kerley Inc., 2012). Dietary exposure to the general public from carbaryl is minimal since reduced application rates compared to those on the label are being used and in accordance with other label restrictions designed to reduce exposure.

The potential exposure of the general public to carbaryl from drinking water sources is not expected based on Program adherence to the label requirements, the proposed use rates, and APHIS Program treatment guidelines (USDA APHIS, 2017; 2016). The Program restricts insecticide applications directly to water bodies, as stated on the label, and also requires a no treatment buffer from water bodies (500 foot buffer for aerial and 200 foot buffer for ground applications) to minimize drift from ULV applications. Bait applications are also not expected to result in detectable carbaryl residues in water due to the implementation of a 50-foot buffer. In addition, only one application is made per season to a treatment block and at rates below those on the label.

4.1.2 Exposure Evaluation

This section quantitatively evaluates worker exposure from accidental direct contact pathways while mixing, loading, and applying carbaryl based on Program application rates. Exposures are acute or short-term. Long-term exposure for workers to carbaryl used in the Program is not typically expected because only one application is proposed per season. The application rates of the carbaryl ULV spray for grasshopper treatment are 0.5 lb a.i./ac (conventional) and 0.25 lb a.i./ac (RAATs) with an approximate total applied volume of 32 fl. oz per acre (carbaryl and water in 1:1 ratio) (conventional) and 16 fl. oz per acre (RAATs). The application rates of carbaryl bait are 0.5 lb a.i./ac (conventional) and 0.2 lb a.i./ac (RAATs) with approximate total applied volumes of 10 lb/ac of bran flakes and apple pomace (conventional and RAATs).

To quantify the potential exposure from dermal and inhalation pathways during mixing, loading, and applications for workers, APHIS estimated dermal and inhalation doses using the following equation:

Dermal Dose = Application Rate (lb a.i./ac) × Area Treated (ac/day) x Dermal Unit Exposure (µg/lb a.i.) × Conversion Factor (0.001 mg/µg)) ÷ BW (kg)

Inhalation Dose = (Application Rate (lb a.i./acre) × Area Treated (ac/day) x Inhalation Unit Exposure (µg/lb a.i.) × Conversion Factor (0.001 mg/µg)) ÷ BW (kg)

The mixing/loading liquids exposure scenario in the Occupational Pesticide Handler Unit Exposure Surrogate Reference Table (USEPA, 2018) is the closest to the Program loading and application exposure scenario.

The dermal unit exposure of 37.6 µg/lb a.i. (single layer, gloves PPE level), and inhalation unit exposure of 0.219 µg/lb a.i. (no respirator PPE level) of the mixing/loading liquids exposure scenario were used for the exposure estimates. The dermal and inhalation doses were quantified for maximum and average exposure scenarios based on APHIS conventional and RAATs application rates of 0.5 and 0.25 lb a.i./ac (0.2 lb a.i. for baits). The area treated was assumed to be 10,000 acres per day for the mixing and loading using aerial application, and 500 acres per day for ground application. The conservative estimation of 10,000 acres per day is based on

recent data from 2016 where 16,963 acres were treated over 2 days. A dermal absorption factor of 4.5% applied to the cancer calculations when estimating a dermal dose. The standard body weight of 80 kg was used for the worker exposure estimation. The exposure dose estimates for dermal and inhalation routes are included in appendix C.

4.2 Ecological Exposure Assessment

Offsite insecticide deposition from Program applications can occur through various transport processes including volatility, drift, and runoff. Volatility is not considered a significant transport pathway based on the reported low vapor pressure values measured for carbaryl. Drift and runoff were also considered and are discussed in more detail below.

4.2.1 Terrestrial Exposure Assessment

Exposure levels on vegetation and other forage items for terrestrial non-target vertebrate organisms were calculated using the Terrestrial Residue Exposure Model (T-REX) (USEPA, 2005). T-REX provides an updated version of the Fletcher residue model that was originally based on the Kenaga nomogram used by USEPA/OPP in their risk assessment process for pesticide registration. T-REX allows the user to input variables such as use, application rate/type, percent active ingredient, soil or foliar dissipation half-life, application interval, and number of applications to calculate exposure concentrations on a variety of food items (table 2-4). For foliar sprays the estimates of exposure are based on the original Kenaga nomogram using field collected residue data for several pesticide classes to calculate residue levels for a wide variety of food items. Minimum and maximum residue levels were calculated for each food item (Hoerger and Kenaga, 1972). The model was updated by Fletcher to account for any potential differences in new chemistry classes that had been developed after Kenaga (Fletcher et al., 1994). Based on over 200 residue studies the model was shown to provide an accurate representation of residues for certain food items, but in some cases such as long grass, it overestimated residues. The current T-REX model provides daily residue values as a mean and upper bound estimate. All exposure values in this risk assessment are based on the upper bound residue estimates. In addition to the calculated residue data, the T-REX model allows the user to input toxicity endpoints that can be compared to exposure values to determine if exposure levels exceed benchmark effect levels.

Exposure concentrations for birds and mammals can be based on mg/kg diet or mg/kg body weight. Acute exposure concentrations were based on the upper bound estimate of mg/kg diet and represent residue levels that would be expected from a direct application to various food items. The exposure concentrations were used to determine residues for carbaryl for different mammals and birds based on their body size and relative food consumption on a daily basis. These values can then be compared to effects data with endpoints represented as mg/kg diet (i.e., LC_{50} and NOEC). The comparison of the specific mammals and bird exposure values to the lowest available effects data is discussed in the risk characterization section of this risk assessment.

4.2.2 Aquatic Exposure Assessment

The method of calculating aquatic exposure concentrations and effective buffer zones for the Program is through the use of two aerial drift deposition models. The models (AgDrift and AgDisp) allow for specific application information to be used as input into the model, and then determine the amount of drift that would occur at a user-defined distance from the spray block. The difference between deposition at the edge of a field and a selected buffer zone can be used as a means to reduce the total amount of insecticide that would be expected at a certain distance from the spray block.

AgDrift and AgDisp are pesticide drift deposition models that provide the user with the ability to provide site- and application-specific information as input to determine application efficiency and off-site drift residues. AgDisp is a model which was developed by the USDA Forest Service and served as the platform for the development of the AgDrift model which has become a regulatory tool for the USEPA/OPP in the registration of pesticides (Hewitt et al., 2002; Teske and Curbishley, 2003). Both models have a tiered approach that allows the user to choose default values or provide more specific data, based on the available information. Both models have been validated under various application scenarios in the literature (Duan et al., 1992a; Duan et al., 1992b; Teske et al., 2000; Teske and Thistle, 2004). In general, aerial application predictions slightly underestimate drift within the first 80 m, but over predict at increasing distances by a factor of two to four at distances up to approximately 300 m (Bird et al., 2002; Duan et al., 1992a.b; Teske and Thistle, 2003; Thistle et al, 2008).

For this risk assessment, the AgDrift model was used to simulate all ground applications, while AgDisp was used to simulate all aerial ULV applications. The AgDisp model was used in the aerial applications because the application heights chosen are beyond those that have been validated using AgDrift (Teske and Thistle, 2004). Input data for the AgDrift and AgDisp models were based on pesticide labels for each product and specific application information available in the APHIS workplan for the Program (USDA APHIS, 2016). While several types of aircraft are available for application in the Program, the quantitative differences in drift are minimal at the buffer zones being assessed. Therefore, the focus of the modeling work was to emphasize those parameters that have the greatest influence on drift. Multiple factors can influence pesticide drift; however, release height, wind speed and direction, and nozzle atomization/orientation are the primary factors influencing drift (Bird et al., 1996; Teske et al., 2000).

Unless otherwise specified, release height for aerial applications was set at 75 feet with a maximum allowed sustained wind speed of 10 mph at -90^{o} wind direction towards the sensitive habitat for the entire length of all swaths with no reduced area of application occurring over the spray block. The spray nozzles were set to the American Society of Agricultural and Biological Engineers (ASABE) droplet size distribution of very fine-to-fine (median diameter = 137.5 µm). ASABE has developed standardized parameters for different droplet size spectra that can be selected in both drift models. The very fine-to-fine droplet size spectrum selected for all of the

air and ground ULV simulations is consistent with an application recommended for use in the Program. The drift models do not allow for the selection of bait applications so those were not assessed in this exercise. Application rates selected for modeling were based on the maximum RAAT rates assuming 100% coverage during application. Lower RAAT rates may be used in cases where reduced application and coverage can be implemented to effectively suppress grasshopper and Mormon cricket populations. The RAATs rates were selected because they are the most common application method.

The intent of the Program is to make applications as close to the ground as possible. However, in some cases where rapid elevation changes are likely to occur, applications must be made at a height that will ensure pilot safety and the appropriate swath width. All applications were simulated on an area where the buffer was on a zero grade and there was no upslope or downslope between the spray block and off-site habitat. In addition, the maximum height of vegetation between the spray block and habitat was no greater than 0.1 meters high. This provides a conservative estimate regarding the ability of plants and terrain to intercept drift between the spray block and sensitive areas.

Other parameters that influence drift are meteorological conditions. In addition to wind speed, both drift models allow the user to input temperature and humidity. Temperature and humidity values for this exercise were selected from all geographically representative areas where the program could potentially make applications. Meteorological data was obtained from the AgDisp model which allows the user to view a 30-year compendium of meteorological data from 239 sites in the United States (1961–1990 National Solar Radiation Data Base, Version 1.0, Solar and Meterological Surface Observational Network (SAMSON)) (Teske and Curbishley, 2003).

The 25th percentile humidity value and the 75th percentile highest temperature were selected based on weather data from Lubbock, Texas, which reported a temperature value of (90 ^{o}F) with a humidity value of 36%. Bismarck, North Dakota, and Pocatello, Idaho, were also evaluated, and based on a combination of maximum temperature and minimum humidity values for those areas, all three had similar application efficiencies and drift fractions based on their respective worst-case temperature and humidity values. Therefore, the temperature and humidity value from Lubbock, Texas, was used because it would maximize the potential for insecticide drift.

AgDisp and AgDrift provide estimates of off-site residues related to drift in terrestrial and aquatic environments. However, they do not provide an estimate of the amount of runoff that could occur into aquatic habitats. Several aquatic fate models exist to estimate environmental loading into aquatic habitats. USEPA/OPP has developed a tiered approach for the use of aquatic fate models that allow the user to estimate aquatic concentrations based on default “reasonable worst-case conditions,” or to calculate estimated aquatic concentrations based on crop-specific soil and weather conditions (USEPA, 2004). None of the available models allow the user to calculate the effects of application buffers in reducing pesticide runoff.

The runoff contribution from applications in the Program is considered minimal due to the application buffers that are applied adjacent to aquatic environments. The effectiveness in the use of application buffers to reduce runoff can vary based on site conditions, the type of vegetation

present in the buffer, and the fate of the insecticide. However, the products used in the Program and the large buffers ensure that runoff will not be a significant contribution of off-site pesticide movement when products are applied according to label specifications and APHIS policy.

Aquatic residue estimates were made using the Program 200-foot ground buffer and the 500-foot aerial no treatment buffer. Water body sizes were one acre in area and 6.56 feet deep to simulate a pond scenario, and one acre in area and 0.49 feet deep to simulate a wetland scenario. All residues were average acute values assuming no degradation of the insecticide over time in a static system. Acute 96-hour residues from ground applications ranged from 1.4 to 19.15 parts per billion (ppb) while acute 96-hour aerial application residues ranged from 16.02 to 214.43 ppb. These are considered conservative estimates based on assumptions in the model and when compared to monitoring data that has been collected to validate field applications (USDA APHIS, 2015b). Drift card data collected as part of the monitoring program show that aerial drift modeling estimates are approximately 20 times greater than observed values. Potential residues in water from bait applications are expected to be less than those estimated for ULV applications due to the large coarse pellet that would be applied from ground and aerial equipment and the implementation of a 50 foot no treatment buffer from aquatic habitats.

5.0 RISK CHARACTERIZATION

Risks associated with potential adverse human health effects are characterized qualitatively and quantitatively in this section. Results from the risk characterization suggests that the use of carbaryl ULV spray or baits for the Program will pose minimal risks to human health. Grasshopper outbreaks usually occur every year with some exceptions such as 2012 and 2013 without any outbreaks.

5.1 Human Health

The risk to workers exposed to carbaryl via oral, inhalation, and dermal routes during applications is minimized by the use of PPE and adherence to other label requirements such as REIs. Carbaryl is a hazard to humans because of its ability to inhibit ChE through oral, inhalation, and dermal exposure. The low potential for significant exposure from the Program use of a carbaryl ULV spray or bait suggests there are minimal risks to workers.

Accidental exposure during mixing and loading, and applications for ULV spray or bait formulations may occur. APHIS quantified the risks of dermal and inhalation exposure for workers and calculated a hazard quotient (HQ) using the following equation for non-carcinogens:

HQ = Exposure Dose / Reference Dose

Cancer risk was also evaluated because USEPA has classified carbaryl as "likely to be carcinogenic to humans". Cancer risk is estimated over the anticipated lifetime using the following equation:

Cancer risk = Lifetime Average Daily Dose (mg/kg/day) × Q1*, where Q1* = 8.75E-04 $(mg/kg/day)^{-1}$.

Tables 5-1, 5-2, and 5-3 summarize the results for accidental direct contact exposures for three exposure scenarios: mixing and loading, ground application with a mechanical spreader, and aerial application. For each exposure scenario, the maximum exposure represents the conventional application rate and the average exposure represents the RAATs application rate. The acute dermal reference dose of 0.086 mg/kg/day is an estimated human POD of 86 mg/kg divided by 100 (10x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF) for dermal exposure. The inhalation reference dose of 0.033 mg/kg/day is the POD of 1.0 mg/kg divided by 30 (3x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF) for inhalation exposure.

For the mixing and loading exposure scenario (table 5-1), 10,000 acres per day was used for the treated area based on the highest recent actual acreage of 16,953 acres applied over 2 days in 2016. The unit exposures (single layer with gloves for the dermal route, no respirator for the inhalation route, and the engineering control with a closed loading system for the both routes)

represent various exposure protections applied for calculating the estimated risk associated with the mixing/loading liquids exposure scenario (USEPA, 2018). The engineering control with a closed loading system exposure scenario provides more protection to workers. Table 5-1 shows that the risk estimates for workers under the mixing and loading exposure scenario, including the dermal and inhalation combined maximum, average, and a closed loading system HQ values of 3, 2, and 0.8, respectively. The HQ of 0.8 from a closed loading system does not exceed the USEPA's level of concern (HQ of 1) indicating no concerns for adverse health risk. However, the dermal and inhalation combined maximum/average HQs of 3/2 under the single layer with gloves exposure scenario without a closed loading system exceed the USEPA's level of concern (HQ of 1), indicating potential concerns for adverse health risk. Because of the potential risk associated with dermal exposure (maximum and average) with the single layer with gloves protection for workers under the mixing and loading exposure scenario, an engineering control with a closed loading system protection should be used during mixing and loading if the treated area is 10,000 acre per day.

APHIS estimated MOEs and compared a calculated MOE to USEPA's levels of concern for carbaryl. A MOE is a ratio of a toxicological endpoint (usually a NOAEL) to exposure that characterizes the amount of safety to a toxic chemical. A MOE is calculated using a dermal or an inhalation POD divided by a dermal or an inhalation exposure dose. The estimated MOEs (160 for dermal route in a closed loading system and 73/146/193 for inhalation route) for the mixing and loading exposure scenario (table 5-1) are higher than the USEPA's levels of concern of 100 (dermal) and 30 (inhalation) indicating that there is no concern of adverse health effects. The estimated MOEs of 37 and 73 for dermal route without a closed loading system are lower than the level of concern of 100, indicating that there is concern of adverse health effects. A total aggregated risk index (ARI) was calculated because the LOCs for dermal exposure (100) and inhalation exposure (30) are different. The calculated ARIs for the maximum, average, and a closed loading system exposures are 0.3, 0.6, and 1.3, respectively. The USEPA's target ARI is 1. The calculated ARI of 1.3 for the exposure with a closed loading system is higher than 1 indicating that there is no concern of adverse health effects. The calculated ARIs of 0.3 and 0.6 for the exposures without a closed loading system are less than 1 indicating risk estimates of concern. The MOE evaluation results are consistent with the HQ evaluation. The combined maximum, average, and a closed loading system cancer risks of 4×10^{-6}, 2×10^{-6}, and 9.5×10^{-7} are within the cancer risk range of 1×10^{-4} to 1×10^{-6} (USEPA, 2000) for the mixing and loading exposure scenario indicating no concerns for adverse health risk for workers.

Table 5-1. Cancer risks and hazard quotients estimated for dermal and inhalation exposures of workers for the mixing and loading exposure scenario

	Dermal Exposure	Inhalation Exposure
	Maximum/Average/Closed system	Maximum/Average/Closed system
Exposure dose (mg/kg-day)	2.4/1.2/5.4E-01 (non-cancer) 1.1E-01/5.3E-02/2.4E-02 (cancer)	1.4E-02/6.8E-03/5.2E-03
Reference Dose (mg/kg-day)	0.86	0.033
Q1*(mg/kg-day)$^{-1}$	8.75E-04	8.75E-04
HQ	2.7/1.4/0.6	0.4/0.2/0.2

Lifetime Average Daily Dose	3.9E-03/2E-03/8.9E-04	5E-04/2.5E-04/1.9E-04
	Combined dermal and inhalation HQ = 3/2/0.8 Cancer risk from dermal and inhalation exposure = 4E-06/2E-06/9.5E-07	
POD (mg/kg-day)	86	1
MOE	37/73/160	73/146/193
	ARI = 0.3/0.6/1.3	

For the ground application exposure scenario (table 5-2), 500 acres per day was used for the treated area. The unit exposures (single layer without gloves for the dermal route and no respirator for the inhalation route for an applicator with open cab solid broadcast spreader) represent various exposure protections applied for calculating the estimated risk associated with the ground application exposure scenario (USEPA, 2018). Table 5-2 shows that the risk estimates including dermal and inhalation combined maximum/average HQ values of 0.1/0.06, as well as the combined cancer risks of $2 \times 10^{-7}/7 \times 10^{-8}$ do not exceed the USEPA's levels of concern (HQ of 1 and cancer risk of less than 1×10^{-6}) for indicating no concerns for adverse health risk. Based on the risk calculation results, the exposure protections for workers with single layer without gloves and no respirator for an applicator with open cab solid broadcast spreader are sufficient without the engineering control of a closed loading system.

Under the ground application exposure scenario, the estimated MOEs for the maximum and average exposures are 2780 and 6949 (a dermal route) and 267 and 667 (an inhalation route) (table 5-2). The calculated ARIs for the maximum and average exposures are 6.7 and 16.8, respectively. The ARIs are higher than 1 indicating that there is no concern of adverse health effects.

Table 5-2. Cancer risks and hazard quotients estimated for dermal and inhalation exposures of workers for the ground application exposure scenario

	Dermal Exposure	Inhalation Exposure
	Maximum/Average	Maximum/Average
Exposure dose (mg/kg-day)	3.1E-02/1.2E-02 (non-cancer) 1.4E-03/5.6E-04 (cancer)	3.8E-03/1.5E-03
Reference Dose (mg/kg-day)	0.86	0.033
$Q1^*(mg/kg\text{-}day)^{-1}$	8.75E-04	8.75E-04
HQ	0.04/0.01	0.1/0.05
Lifetime Average Daily Dose	5E-05/2E-05	1E-04/6E-05
	Combined dermal and inhalation HQ = 0.1/0.06 Cancer risk = 2E-07/7E-08	
POD (mg/kg-day)	86	1
MOE	2780/6949	267/667
	ARI = 6.7/16.8	

For the aerial application exposure scenario (table 5-3), the 10,000 acres per day was used for the treated area based on the highest recent actual acreage of 16,953 acres applied over a 2 day period. The unit exposure for an aerial applicator with fixed-wing, liquid, and enclosed cockpit represents the exposure protection for workers applied for calculating the estimated risk (USEPA, 2018). Table 5-3 shows that the risk estimates including the dermal and inhalation combined maximum/average HQ values of 0.2/0.08, as well as the combined cancer risks of 2 x 10^{-7}/1 x 10^{-7} do not exceed the USEPA's levels of concern (HQ of 1 and Cancer risk of less than 1 x 10^{-6}) for indicating no concerns for adverse health risk. Consistent with the HQ evaluation results, the calculated ARIs for the maximum and average exposures of 6.2 and 12.5 are higher than 1 indicating that there is no concern of adverse health effects. Based on the risk calculation results, the exposure protection for the aerial applicator with fixed-wing, liquid, and enclosed cockpit protection is sufficient under the aerial application exposure scenario. The risk calculations are included in appendix C.

Table 5-3. Cancer risks and hazard quotients estimated for dermal and inhalation exposures of workers for the aerial application exposure scenario

	Dermal Exposure	Inhalation Exposure
	Maximum/Average	Maximum/Average
Exposure dose (mg/kg-day)	1.3E-01/6.5E-02 (non-cancer) 5.9E-03/2.9E-03 (cancer)	3.1E-04/1.5E-04
Reference Dose (mg/kg-day)	0.86	0.033
Q1*(mg/kg-day)$^{-1}$	8.75E-04	8.75E-04
HQ	0.15/0.076	0.009/0.005
Lifetime Average Daily Dose	2E-04/1E-04	1E-05/6E-06
	Combined dermal and inhalation HQ = 0.2/0.08 Cancer risk = 2E-07/1E-07	
POD (mg/kg-day)	86	1
MOE	662/1323	3265/6531
	ARI = 6.2/12.5	

Risks to the general public in treatment areas from ground or aerial applications are not expected because APHIS treatments are conducted in rural rangeland areas, where agriculture is a primary economic factor. These areas consist of widely scattered, rural dwellings in ranching communities with low population density. Historically, a majority of the applications occur on Federal lands. Program personnel notify residents and implement mitigation measures beyond label requirements to ensure that no treatments occur within the required buffer zones from structures, such as homes and schools where there is potential exposure for residents including children (USDA APHIS, 2018). There are no adverse health risks associated with eating treated

food because Program treatments occur in rangeland and there is no primary food consumption pathway from direct intake of crops. Adverse health risks from indirect consumption of cattle grazed on carbaryl-treated rangeland are not expected because of the low application rate of the ULV spray or bait treatments and natural degradation of the carbaryl. Dietary exposure and risk from carbaryl exposure in drinking water is also not expected due to the environmental fate of carbaryl, low application rates, and use of treatment buffers from surface drinking water sources.

5.2 Terrestrial and Aquatic Risk Characterization

5.2.1 Terrestrial Risk Characterization

5.2.1.1. Direct and Indirect Risk to Mammals

To assess the acute and chronic risk to mammals the most sensitive acute and chronic endpoints were used. Instead of using the LD_{50} value for acute effects, the acute NOEL based on AChE inhibition was used (10 mg/kg). The lowest chronic value that was used was the offspring NOEL of 4.67 mg/kg/day from the rat study. This value was selected rather than the parental NOEL (23.49 mg/kg/day), or the reproductive NOEL (92.43 mg/kg/day) in order to provide a very conservative endpoint for comparison to residue values. These values were used to calculate adjusted acute and chronic NOEL values for each mammal class and size (table 5-4).

Table 5-4. Different mammal class parameters used to calculate adjusted LD_{50} and NOEL carbaryl values

Mammalian Class	Body Weight (g)	Ingestion (dry) (g bw/day)	Ingestion (wet) (g/day)	% body weight consumed	(kg-diet/day)	Adjusted Acute NOEL	Adjusted Chronic NOEL
Herbivores/	15	3	14	95	1.43E-02	21.98	10.26
Insectivores	35	5	23	66	2.31E-02	17.78	8.30
	1000	31	153	15	1.53E-01	7.69	3.59
	15	3	3	21	3.18E-03	21.98	10.26
Granivores	35	5	5	15	5.13E-03	17.78	8.30
	1000	31	34	3	3.40E-02	7.69	3.59

Based on the comparison of the acute and chronic NOEL values to residues that would be expected with no application buffer zone, risk quotient (RQ) values exceeded 1 for all three mammal sizes that feed exclusively on short and tall grass as well as broadleaf plants and small insects (table 5-5).

Table 5-5. Calculated mammalian risk quotient values for carbaryl assuming no application buffer zone

Dose-based RQs (Dose-based EEC/NOEL)	15 g mammal		35 g mammal		1000 g mammal	
	Acute	Chronic	Acute	Chronic	Acute	Chronic
Short Grass	2.60	1.11	2.22	0.95	1.19	0.51
Tall Grass	1.19	0.51	1.02	0.43	0.55	0.23
Broadleaf plants/small insects	1.46	0.62	1.25	0.53	0.67	0.29
Fruits/pods/large insects	0.16	0.07	0.14	0.06	0.07	0.03
Seeds (granivore)	0.04	0.02	0.03	0.01	0.02	0.01

Carbaryl has a reported half-life on vegetation of 3 to 10 days, suggesting exposure will be short term. The exposure also assumes exclusive consumption of contaminated short grass throughout the life cycle of each mammal, which is unlikely.

Using the 2-generation reproduction rat study NOEC of 75 ppm, and comparing that value to the maximum residue for carbaryl on short grass (120 ppm) the resulting chronic RQ value based on diet is 1.6 with no application buffer zone. This is a very conservative risk estimate because only one carbaryl application will occur and the effects endpoint was based on a 2-generation effect study.

Direct risk of carbaryl bait to wild mammals was calculated using the LD_{50}s per square foot method. The LD_{50} per square foot method provides a measure of the amount of pesticide in a square foot that can result in mortality to 50% of the animals. The method has limited ecological relevance due to the selection of an arbitrary area for exposure but assumes as the value increases there is an increase in risk.

The LD_{50} per square foot method also assumes that mammals will consume all of the bait present in a square foot. This would not occur in field applications since applications are timed to coincide with maximum removal by grasshoppers and Mormon crickets. Based on the highly conservative exposure assumptions, and the low risk values that have been calculated using sublethal endpoints, the direct risk to mammals from carbaryl bait applications is expected to be low for most species.

The lowest acute NOEL value (10 mg/kg) was used in this estimate rather than the lowest LD_{50} value, thus providing a conservative estimate of effects for mammals of different weight classes. Values were above 1.0 for each size class with the exception of the 1,000 g mammal class for applications within a spray bloc (table 5-6)k. Risk quotient values exceeding one demonstrate a potential for risk to smaller mammals, however based on the conservative assumptions in the exposure and effects analysis the actual risk is reduced for mammals.

Table 5-6. Estimated risk quotient values for mammals from carbaryl bait applications

Mammalian Size Class (g)	Risk Quotient
15	15.79
35	8.37
1000	0.68

Indirect risk to mammals can be defined as impacts on habitat or prey base. Loss of habitat can occur through carbaryl-related effects to terrestrial plants. Based on the available terrestrial phytotoxicity data, no effects at rates as high as 0.803 lb a.i./ac have been observed in several agronomic crops. There have been reported cases of terrestrial phytotoxicity, mostly in urban applications, but these rates are much higher than full application rates of 0.50 lb a.i./ac that are used in the Program. Based on the lack of known effects at the highest Program rates for ULV and bait applications of carbaryl, there is minimal indirect risk to mammalian habitat.

Another area of possible indirect risk to mammals is the loss of food items attributed to ULV and bait carbaryl applications. Based on the known toxicity data for terrestrial plants there would be minimal risk of indirect effects to mammals that rely on plant material for food. The other food items that could impact small mammal populations are terrestrial invertebrates. Weiland et al. (2002) assessed the impacts of Sevin® XLR Plus applications at 750 g a.i./ha to several invertebrate groups over a 21-day period. This rate equates to 0.67 lb a.i./ac which is 1.34 times higher than the highest rate allowed in the Program. Results from the study demonstrated no negative effects on abundance in the following insect groups: Homoptera, Hymenoptera, Coleoptera, Hemiptera, Lepidoptera, or Neuroptera.

5.2.1.2 Direct and Indirect Risk to Birds

Based on the lowest reported toxicity value for birds (LD_{50} = 16 mg/kg) the adjusted LD_{50} values for different size birds ranged from 12.88 to 23.16 mg/kg (table 5-7).

Table 5-7. Adjusted avian carbaryl toxicity values for different sized avian classes

Avian Class	Body Weight (g)	Ingestion (dry) (g bw/day)	Ingestion (wet) (g/day)	% body weight consumed	(kg-diet/day)	Adjusted LD_{50} (mg/kg-bw)
Small	20	5	23	114	2.28E-02	12.88
Mid	100	13	65	65	6.49E-02	16.39
Large	1000	58	291	29	2.91E-01	23.16

Based on the adjusted LD_{50} values for each avian class, and the calculated dose each class would receive, RQ values exceeded 1 for 20 and 100 g bird sizes feeding on short grass, for 20 g birds that rely solely on tall grass and broadleaf plants and small insects (table 5-8). These values

represent risk based on the conservative assumption that birds feed exclusively within treated areas.

Table 5-8. Calculated acute avian risk quotient values for carbaryl assuming no application buffer zone

Dose-based RQs (Dose-based EEC/adjusted LD_{50})	Avian Acute RQs		
	20 g	100 g	1000 g
Short Grass	5.31	2.38	0.75
Tall Grass	2.43	1.09	0.35
Broadleaf plants/small insects	2.98	1.34	0.42
Fruits/pods/seeds/large insects	0.33	0.15	0.05

The risk quotient values in table 5-8 assume 100% of the diet for a bird is obtained from one type of food, and that the bird consumes only contaminated food items. These values also reflect upper bound estimates of residues that were calculated using the T-REX model.

To calculate the acute RQ value based on dietary carbaryl concentrations. the lowest reported avian LC_{50} value (>5,000 ppm) was used to compare to the highest estimated food concentration (60 ppm) in short grass calculated using maximum carbaryl RAAT rates. The calculated risk quotient value was <0.012 since all reported avian LC_{50} values are greater than the highest test concentration.

Based on the lowest NOEC chronic reproduction value for avian species (300 ppm) and the highest estimated environmental food concentration (60 ppm) in short grass, the resulting RQ value is 0.4, suggesting minimal chronic avian risk from direct applications. Using the lowest acute and chronic dietary effect concentrations, carbaryl poses minimal acute and chronic risk to birds.

Direct risk to avian species from carbaryl bait was calculated using the LD_{50} per square foot method that was described and used for mammals in the previous section. The lowest acute avian LD_{50} value (16 mg/kg for the European starling) was used in this estimation. Values were greater than 1.0 for each size class with the exception of the 1,000 g avian class when no application buffer is applied to potential avian habitat (table 5-9). The estimate of risk in table 5-9 is highly conservative due to multiple conservative assumptions that are discussed in the previous section regarding the risk of carbaryl bait to mammals.

Table 5-9. Estimated risk for various sized avian classes from carbaryl bait applications using LD_{50}/sq. ft.

Avian Size Class Body Weight (g)	(no buffer)
20	20.21
100	3.18
1000	0.22

A number of studies have reported no effects on bird populations in areas treated with carbaryl (Buckner et al., 1973; McEwen et al., 1996; Richmond et al., 1979). Some applications of formulated carbaryl were found to cause depressed AChE levels (Gramlich, 1979; Zinkl et al., 1977) however, the doses were twice those proposed for the full coverage application in the Program.

AChE inhibition at 40 to 60% affects coordination, behavior, and foraging ability in vertebrates. This could lead to death from weather, predators, or other stresses of survival in the wild. Studies over several years for multiple grasshopper treatment areas have shown AChE inhibition at levels of no more than 40% with most at less than 20% (McEwen et al., 1996).

The use of Sevin® 4-Oil, at the formulation rate of 1.25 lbs a.i./acre has demonstrated no toxicity-caused mortality of upland birds, mammals, or reptiles, and none has been observed as part of the grasshopper integrated pest management (IPM) monitoring effort (McEwen et al., 1996).

Field studies in North Dakota were conducted to determine the effects of Sevin® 4-Oil treatment on killdeer populations. At treatment rates of 0.5 and 0.4 lb a.i./acre, no toxic signs and no mortality were observed in the killdeer population. Effects on foraging and diet of the killdeer were examined by both direct observation and analysis of stomach contents (Fair et al., 1995a). The insect capture rate by foraging killdeer increased during the 2-day period after treatment when affected insects were easily obtainable (Fair et al., 1995b). There were no other differences or changes in food habits observed.

As part of the grasshopper IPM monitoring studies, a study was conducted in North Dakota on the effect of carbaryl bait on the nestling growth and survival of vesper sparrow (Adams et al., 1994). This study was designed to simulate the treatment of a small grasshopper infestation with carbaryl bait. There was no difference reported in any of the productivity parameters between nests on treated and untreated sites (Adams et al., 1994). Adult sparrows on treated sites had to forage farther from the nests to obtain food but did so successfully (McEwen et al., 1996).

5.2.1.3. Direct and Indirect Risk to Amphibians and Reptiles

Direct risk to amphibians from ULV and bait carbaryl applications was assessed by taking the highest instantaneous aquatic carbaryl concentration that was calculated for all application techniques (214.43 µg/L) and comparing that value to acute and chronic values that have been published for a variety of amphibians in aquatic systems. The focus on the aquatic habitat for amphibians was done based on the limited toxicity data that show very high LD_{50} values for adult amphibians. Based on the range of reported tadpole LC_{50} values (1.73–22.02 mg/L), the range of acute toxicity values was above the highest calculated carbaryl residue, suggesting minimal acute risk from carbaryl applications. Direct sublethal and chronic effects were evaluated using the known laboratory toxicity data. Following a review of the acute sublethal and chronic effects data, the lowest endpoint for the most sensitive endpoint was selected from each study where a NOEC and LOEC could be determined. Parameters evaluated included

several reproductive related endpoints, swimming activity and predator avoidance. Parameters where effects were not seen at the lower end of the dosing in each study were not considered further in the risk characterization. Based on the above assumptions, a NOEC for swimming behavior of 1.25 mg/L was assumed while a tadpole NOEC for survival, and mean age at metamorphosis was also used as another endpoint (NOEC 0.16 mg/L). The most sensitive NOEC value was 5.0 µg/L based on the reported effects from Rohr et al. (2003) who conducted a 37-day exposure using *Ambystoma barbouri*. Using the range of sublethal NOEC values from the studies discussed above and the highest aquatic residue value for carbaryl (214.43 µg/L), NOEC values were below the highest aquatic residue value, suggesting sublethal risk to amphibians. The reported NOEC value of 5 µg/L using larval survival as an endpoint, is considerably lower than other sublethal effect endpoints, which may be due to the exposure period. Concentrations in solution were based on nominal levels, and water was renewed every other day during the length of the exposure. The pH was not presented so it is unclear whether the carbaryl levels remained constant due to rapid degradation that can occur for carbaryl at high pH values. Regardless, the observed effect at 50 µg/L was not observed until after day 20 in the study. One application of carbaryl would be made in the Program and with the favorable environmental fate profile of carbaryl in water levels would decrease quickly. In addition the estimated residues from drift modeling are conservative instantaneous estimates of exposure based on multiple conservative input parameters and do not allow for degradation which would occur in shallow static waterbodies.

The range of laboratory concentrations where effects have been observed are also supported by a field study that was conducted using Woodhouse's toad (*Bufo woodhousii*) and gray treefrog (*Hyla versicolor*) in exposures to 3.5 and 7.0 mg/L of carbaryl as a Sevin® formulation (Boone and Semlitsch, 2001). Mass at metamorphosis, days to metamorphosis, and survival to metamorphosis were measured in outdoor exposures with effects on survival in both species seen at the highest test concentration (7 mg/L). No effect on mass at metamorphosis was observed at any dose in the Woodhouse's toad exposures, but an effect was seen at the highest dose in the gray treefrog exposure. Both species had statistically significant effects on days to metamorphosis when compared to controls at the highest test concentration. Green frog (*Rana clamitans*) metamorphs and tadpoles were also assessed and no statistically significant effects or interactions were observed at either test concentration.

Indirect effects to amphibians can include loss of habitat and food items. From a habitat perspective, this can include carbaryl effects to terrestrial and aquatic plants. More detailed assessments of potential carbaryl risk to aquatic and terrestrial plants are below; however, in summary, carbaryl at all Program rates poses minimal risk to aquatic and terrestrial plants. The other area of indirect risk that should be addressed is the loss of food items which can include aquatic plants and invertebrates. Both of these risks are discussed in more detail below and demonstrate minimal indirect risk to food items that amphibians would use in aquatic systems.

Due to the lack of data, assessing risk to reptiles is not possible. Currently USEPA/OPP assumes that the range of sensitivities for avian species represents reptiles; however, there is uncertainty in making that type of extrapolation. In the absence of data however, making that assumption

provides some insight regarding potential direct and indirect risk to reptiles from carbaryl applications. Based on the risk characterization for avian species using residues from the most conservative application method, carbaryl applications are expected to have a low risk to reptiles outside of the treatment blocks.

5.2.1.4. Risk to Terrestrial Invertebrates

Smith et al. (2006) assessed changes in non-target arthropod populations following applications of diflubenzuron, carbaryl, or malathion using RAATs. In the 2-year study, post application surveys of the major insect fauna revealed that only ants were negatively affected by grasshopper applications within treatment areas. As stated previously, Weiland et al. (2002) assessed the impacts of Sevin® XLR Plus applications at 750 g a.i./ha to several invertebrate groups over a 21-day period. This rate equates to 0.67 lb a.i./ac which is 1.34 times higher than the highest rate allowed in the Program. Results from the study demonstrated no negative effects on abundance in the following insect groups: Homoptera, Hymenoptera, Coleoptera, Hemiptera, Lepidoptera, and Neuroptera.

5.2.1.5. Direct and Indirect Risk to Terrestrial Plants

Based on the available toxicity data for terrestrial plants and the proposed application rates of carbaryl, the direct risk to terrestrial plants from phytotoxic effects is expected to be negligible. However, there is concern regarding indirect effects to terrestrial plants due to impacts of carbaryl applications on pollinators. Laboratory studies have indicated that bees are sensitive to carbaryl applications but at rates above those proposed in the Program. This may be attributed to the lower contact toxicity of carbaryl to bees compared to oral toxicity. In addition, formulated carbaryl is up to 10 times less toxic than the technical which would result in lower risk to pollinators. The reduced rates of carbaryl used in the Program and the implementation of application buffers will significantly reduce exposure of carbaryl applications to pollinators. In areas of direct application where impacts may occur, alternating swaths and/or reduced rates (i.e., RAATs) will reduce risk. Little field data appears to be available that discusses carbaryl effects to honey bees. Based on a field study using Carbaryl SC at a rate of 0.80 lb a.i./ac in a fruit orchard, there were no effects on bee mortality or behavior 7 days post-application (USEPA, 2003). Potential negative effects of Program insecticides on bee populations may also be mitigated by the use of carbaryl bran baits. Studies with carbaryl bran bait have found no sublethal effects on adults or larvae (Peach et al., 1994; 1995).

5.2.2 Aquatic Risk Characterization

Comparison of the distribution of acute, sublethal and chronic effects data for fish to the residues estimated using ground and aerial ULV and bait applications show that the range of residues do not overlap with acute toxicity values, suggesting there is no acute risk to fish species. There is some overlap with chronic and sublethal effect values and estimated residues (figure 5-1). However, carbaryl half-lives in water are typically short and with the proposed one time

application chronic exposure and risk to fish is not anticipated. Effects from consumption of contaminated prey are also not expected to be a significant pathway of exposure, based on the low residues and low BCF values reported for carbaryl.

Indirect risk to fish species can occur through the loss of habitat or reduction in prey base. To determine potential habitat loss from carbaryl applications, the most sensitive aquatic plant endpoint was used as a benchmark to compare to estimated aquatic residues that would be expected from aerial and ground ULV applications. Several aquatic plant toxicity values are available for carbaryl; however, the most sensitive species was the green algae *Pseudokirchneriella subcapitata* that had a reported NOEC value of 0.37 mg/L. Comparing the NOEC to the aquatic residue range estimated for all application methods of carbaryl (1.44-214.43 µg/L) resulted in residues that are below the threshold NOEC value. This suggests that carbaryl risk to aquatic plants that may serve as habitat or food for fish and aquatic invertebrates is low.

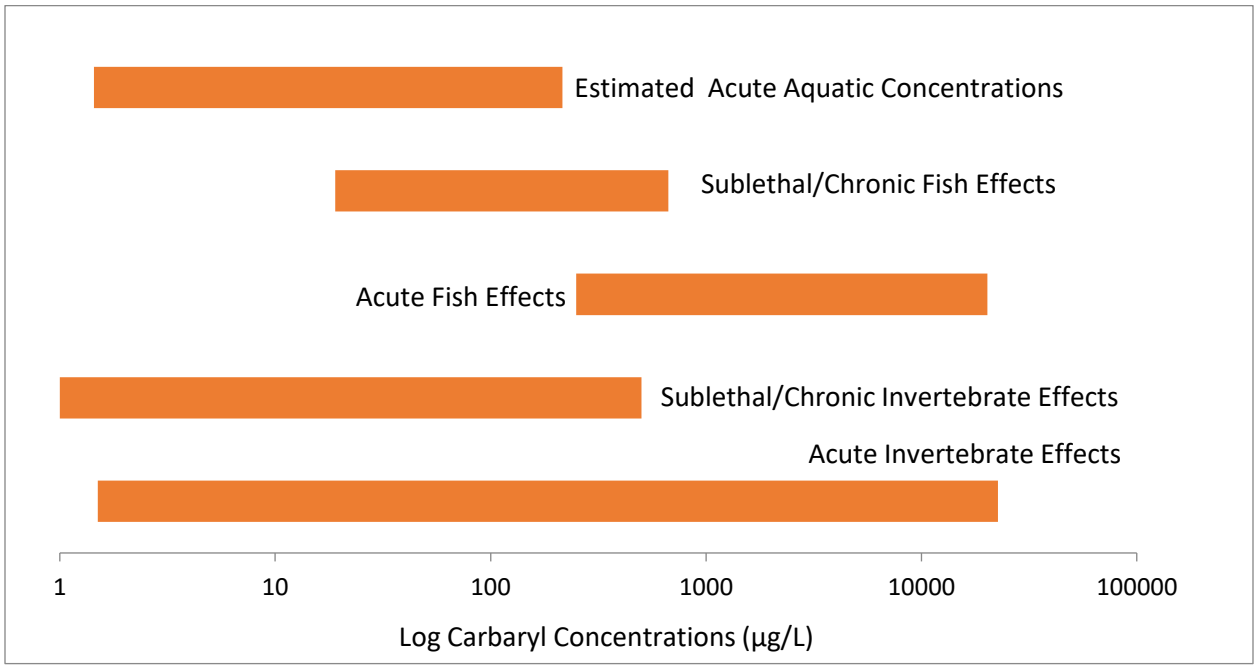

Figure 5-1. Carbaryl risk characterization for fish and aquatic invertebrates using liquid ground and aerial applications.

There was overlap of acute aquatic invertebrate effect values and estimated aquatic residues suggesting risk to some sensitive species. Based on a similar distribution of sensitivities, the Department of Commerce's National Marine Fisheries Service (NMFS) calculated a probability distribution plot for carbaryl and aquatic invertebrates and determined EC_{50} concentrations at the 50^{th}, 10^{th}, and 5^{th} percentiles (NMFS, 2009). The values from this exercise, when incorporating all studies, was 45.23, 2.29, and 0.98 µg/L for the 50^{th}, 10^{th}, and 5^{th}, percentiles, respectively. Values were slightly higher when using geometric means with 50^{th}, 10^{th}, and 5^{th} percentile values

of 69.53, 4.33 and 1.97 µg/L. Residues from aerial ULV applications in static wetland habitats exceeded the geometric mean 50th percentile value for aquatic invertebrates. Residues from ground applications in a static waterbody such as a pond were below the range of carbaryl aquatic invertebrate effect values, including the estimated 5th percentile geometric mean values estimated by NMFS. Residues exceeded the 10th percentile effect distribution when assessing residues in static wetland habitats. Residue estimates from this exercise are considered conservative when compared to observed residues that have been measured in the field. Multiple conservative assumptions were used to make estimates regarding aquatic residue values. Average residue values from drift cards collected at 500 feet from actual applications were greater than 20 times lower than values determined using both models (USDA APHIS, 2015b). Chronic risk is also a conservative estimate because chronic toxicity data is based on long-term exposures that what would not be expected to occur from a single application, based on the environmental fate of carbaryl in aquatic environments.

5.2.2.1. Aquatic Field Studies Regarding Fish and Aquatic Invertebrates

Several aquatic field studies have been published and summarized using carbaryl to determine impacts to aquatic invertebrates and fish (NMFS, 2009; Relyea and Diecks, 2008; US FS, 2008). The value of these studies in providing insight into aquatic community impacts from carbaryl applications is limited because all studies had dosing levels and/or frequencies much higher than what would occur from activities in this Program. Select studies and their results are summarized below.

In a field study related to the Program, applications of carbaryl were made in proximity to the Little Missouri River over a two-year period and impacts to fish and aquatic invertebrates were assessed (Beyers et al., 1995). Measured carbaryl concentrations were 85.1 ppb in a drought year and 12.0 ppb in a non-drought year 1 hour after application. Brain cholinesterase was measured in the fathead chub (*Platygobio gracilis*) in a drought and non-drought year after applications of Sevin-4-Oil® for the control of rangeland grasshoppers. No effects were seen on brain cholinesterase activity for either season when compared to chubs from the reference site. Invertebrate sampling resulted in an increase in the coefficient of variation in invertebrate drift 3 hours after treatment at a measured concentration of 12.3 µg/L 4 hours post-treatment. The increase in variability was not observed after that sampling event, and concentrations of carbaryl decreased to 0.100 µg/L 96 hours post-treatment. No impacts in invertebrate drift were noted in the second year of application where carbaryl concentrations of 12.6 µg/L were measured 2 hours post-treatment. Drift in this case is defined as stream invertebrates that leave their substrate and move downstream. It should be noted that the residues measured in this study are not based on current methods of carbaryl liquid applications and do not incorporate current rates and Program application restrictions.

Courtemanch and Gibbs (1980) reported similar impacts on invertebrate drift in field studies after direct application of Sevin-4-Oil® to streams. Residues were not measured; however, correlations to other studies in the manuscript suggest aquatic residues of 26 to 42 µg/L caused the increase in invertebrate drift, which is well above residues predicted from Program

applications. In another field study that assessed brain cholinesterase levels after carbaryl treatment, Haines (1981) noted a depression in brook trout (*Salvelinus fontinalis*) cholinesterase activity when Sevin-4-Oil® was applied at 1 lb a.i./ac in a forestry application in Maine. Similar results have been seen in other field studies, with brook trout AChE depression following 1 lb/ac treatments. Due to the rapid reversibility associated with carbaryl, AChE levels returned to normal within 48 hours (Hurlbert, 1978). In another field study a split application of Sevin-2-Oil®, at 280 g/hectare (ha) for each application, was used to evaluate impacts to brook trout and slimy sculpin (*Cottus cognatus*) as well as aquatic invertebrates (Holmes et al., 1981). Maximum measured residues were 313.7 and 122.6 µg/L after each application and declined to less than 1 µg/L after 10 days. Invertebrate drift was impacted; however, overall impacts to aquatic invertebrates was reported as negligible and stomach contents from both fish species demonstrated that there was no reduction in food availability.

The effects measured in the above studies are difficult to extrapolate and apply to conditions in the current Program. While sublethal effects have been noted in fish with depressed AChE, as well as some impacts to invertebrates in the field due to carbaryl, the application rates and measured aquatic residues where it was observed in these studies is well above values that would be expected from current Program operations.

6.0 UNCERTAINTIES AND CUMULATIVE IMPACTS

The uncertainties associated with this risk evaluation arise primarily from lack of information about the effects of carbaryl, its formulations, metabolites, and potential mixtures to non-target organisms that can occur in the environment. These uncertainties are not unique to this assessment but are consistent with uncertainties in human health and ecological risk assessments with any environmental stressor. APHIS may conduct a treatment to suppress economically damaging grasshopper and/or Mormon cricket populations on rangeland in 17 Western States (Arizona, California, Colorado, Idaho, Kansas, Montana, Nebraska, Nevada, New Mexico, North Dakota, Oklahoma, Oregon, South Dakota, Texas, Utah, Washington, and Wyoming). However, rangeland does not occur over the entire area of most of those States. There is uncertainty in where and how often an infestation may occur in a specific state, and the extent of carbaryl use because use is based on site-specific factors. The size of the treatment area for grasshoppers and Mormon crickets varies each year. Between 2006 and 2017, the actual acreage of treatment ranged from 1,264,676 acres (2010) to 3,693 acres (2013). The application rates used in the Program also vary based on the formulation. Of the total acres treated with Program insecticides between 2006 and 2017, approximately 6% of the total acres received carbaryl, a majority total acres (93%) received diflubenzuron, and only 1% total acres received malathion. Carbaryl ground liquid and bait applications are the most common carbaryl treatments that have historically been used by the Program.

Another area of uncertainty is the potential for cumulative impacts to human health and the environment including: 1) repeated worker and environmental exposures to carbaryl from Program activities in conjunction with other crop use sources, 2) co-exposure to other chemicals with a similar mode of action, and 3) exposures to other chemicals in mixtures and how that may affect the toxicity of carbaryl.

Carbaryl is one of the most commonly used conventional pesticide active ingredients in the home and garden market sector with an annual use of approximately 2 to 4 million pounds of active ingredient in the United States in 2012 (USEPA, 2017a). The Grasshopper Program use of carbaryl in rangelands is much less. The Program mostly uses carbaryl bait in RAAT applications compared to liquid formulations and conventional applications. Between 2006 and 2017, approximately 88% of the total number of acres treated with carbaryl used RAATs and 12% of the total number used conventional applications (only in 2008). Since 2009, the Program applied all carbaryl applications using RAATs. .

Cumulative impacts from the potential for co-exposure of carbaryl use and other insecticides used in the Program that have a similar mode of action resulting in synergism, potentiation, additive, or antagonistic effects are not expected. The other insecticides used in the Program include malathion, diflubenzuron, and chlorantraniliprole. Diflubenzuron and chlorantraniliprole do not have the same mode of action as carbaryl. Diflubenzuron causes methemoglobinemia and/or sulfhemoglobinemia. Chlorantraniliprole acts on the ryanodine receptor. Carbaryl targets the nervous system (carbamylation of AChE resulting in accumulation of the neurotransmitter, acetylcholine) as well as malathion (inhibition of the enzyme AChE function in the central and

or peripheral nervous system). Although carbaryl and malathion have the same mode of action, the Program would use only one insecticide in a treatment area and no more than one treatment per year would normally be applied at any location. Other pesticide use may occur on rangelands besides treatments for grasshoppers and Mormon crickets. Insecticide use for other pests of rangeland are not expected; however, herbicide use may occur on these lands to manage invasive weeds. There is uncertainty for the potential cumulative mixture effects with other pesticide use due to the temporal and spatial variability when other pesticide rangeland applications would occur relative to Program grasshopper treatments.

7.0 REFERENCES

Adams, J.S., R.L. Knight, L.C. McEwen, and T.L. George. 1994. Survival and growth of nestling vesper sparrow exposed to experimental food reductions. The Condor 96:739–748.

ATSDR – see Agency for Toxic Substances and Disease Registry

Agency for Toxic Substances and Disease Registry. 2008. Addendum to the toxicological profile for propylene glycol. Division of Toxicology and Environmental Medicine Atlanta, GA, December 2008, 17 pp.

Arunachalam, S., Jeyalakshmi, K., and S. Aboobucker. 1980. Toxic and sublethal effects of carbaryl on a freshwater catfish, Mystus vittatus (Bloch). Arch. Environ. Contam. Toxicol. 9:307–316.

Barrett, G.W. 1968. The effects of an acute insecticide stress on a semi-enclosed grassland ecosystem. Ecology 49(6):1019–1035.

Bartlett, B.R. 1968. Outbreaks of two-spotted spider mites and cotton aphids following pesticide treatments. I. Pest stimulation vs. natural enemy destruction as the cause of outbreaks. Journal of Economic Entomology 61(1):297–303.

Beauvais, S.L., Jones, S.B., Parris, J.T., Brewer, S.K., and E.E. Little. 2001. Cholinergic and behavioral neurotoxicity of carbaryl and cadmium to larval rainbow trout (*Oncorhynchus mykiss*). Ecotox. Env. Safety 49:84–90.

Beyers, D.W., Keefe, T.J., and C.A. Carlson. 1994. Toxicity of carbaryl and malathion to two federally endangered fishes, as estimated by regression and ANOVA. Env. Toxicol. and Chem. 13:101–107.

Beyers, D.W., Farmer, M.S., and P.J. Sikowski. 1995. Effects of rangeland aerial application of Sevin-4-Oil® on fish and aquatic invertebrate drift in the Little Missouri River, North Dakota. Archives of Environmental Contamination and Toxicology 28:27–34.

Beyers, D.W. and P.J. Sikoski. 1994. Acetylcholinesterase inhibition in federally endangered Colorado squawfish exposed to carbaryl and malathion. Environ. Toxicol. and Chem. 13(6): 935–939.

Bird, S.L., Perry, S.G., Ray, S.L., and M.E. Teske. 2002. Evaluation of the AgDisp aerial spray algorithms in the AgDrift model. Env. Toxicol. Chem. 21(3): 672-681.

Bird, S.L., Esterly, D.M., and S.G. Perry. 1996. Off-target deposition of pesticides from agricultural aerial spray applications. J. Environ. Qual. 25(5): 1095–1104.

Boonyawanich, S., Kruatrachue, M., Upatham, E.S., Soontornchainaksaeng, P., Pokethitiyook, P., and S. Singhakaew. 2001. The effect of carbamate insecticide on the growth of three aquatic plant species: *Ipomoea aquatica*, *Pistia stratiotes* and *Hydrocharis dubia*. Sci. Asia. 27:99–104.

Boone, M.D. and C.M. Bridges. 1999. The effect of temperature on the potency of carbaryl of tadpoles of the green frog (*Rana clamitans*). Env. Toxicol. Chem. 18(7):1482-1484.

Boone, M.D. and R.D. Semlitsch. 2001. Interaction of an insecticide with larval density and predation in experimental amphibian communities. Conservation Biology 15(1):228-238.

Bridges, C.M. 1997. Tadpole swimming performance and activity affected by acute exposure to sublethal levels of carbaryl. Env. Toxicol. and Chem. 16:1935–1939.

Bridges, C.M. 1999a. Effects of a pesticide on tadpole activity and predator avoidance behavior. J. of Herpetology. 33(2): 303-306.

Bridges, C.M. 1999b. Predator-prey interactions between two amphibian species: effects of insecticide exposure. Aquatic Ecology 33: 205–211.

Bridges, C.M. 2000. Long-term effects of pesticide exposure at various life stages of the southern leopard frog (*Rana sphenocephala*). Arch. Environ. Contam. Toxicol. 39:91–96.

Bridges, C.M. and R.D. Semlitsch. 2000. Variation in pesticide tolerance of tadpoles among and within species of Ranidae and patterns of amphibian decline. Conservation Biology 14(5):1490-1499.

Bridges, C.M., Dwyer, F.J., Hardesty, D.K. and D.W. Whites. 2002. Comparative contaminant toxicity: are amphibian larvae more sensitive than fish? Bull. Environ. Contam. Toxicol. 69:562–569

Buckner, C.H., P.D. Kingsbury, B.B. McLeod, K.L. Mortensen, and D.G.H Ray. 1973. The effects of pesticides on small forest vertebrates of the Spruce Woods Provincial Forest, Manitoba. The Manitoba Entomologist 7:37–45.

Burling, I., Yokelson, R., Griffith, D., Johnson, T., Veres, P., Roberts, J., Warneke, C., Urbanski, S., Reardon, J., Weise, D., Hao, W., and J. de Gouw. 2010. Laboratory measurements of trace gas emissions from biomass burning of fuel types from the southeastern and southwestern United States. Atmospheric Chemistry and Physics 10: 11115-111130.

Bursian, S.J. and F.W. Edens. 1977. The prolonged exposure of Japanese quail to carbaryl and its effects on growth and reproductive parameters. Bull. Environ. Contam. Toxicol. 17(3): 360-368.

Bytnerowicz, A., Arbaugh, M., Riebau, A., and C. Andersen. (Eds.). 2009. Wildland fires and air pollution. Developments in Environmental Science 8. Oxford, UK: Elsevier.

CDPR - California Environmental Protection Agency Department of Pesticide Regulation.

California Environmental Protection Agency Department of Pesticide Regulation. 2014. Human Exposure Assessment Document for Carbaryl, by Beauvais, S. HS-1788, November 5, 2014, 136 pp.

California Department of Fish and Game (CA DFG). 1998. Hazard assessment of the insecticide carbaryl to aquatic life in the Sacramento-San Joaquin river system. Admin. Rep. No: 98–1. 62 pp.

Carlson, A.R. 1972. Effects of long-term exposure to carbaryl (Sevin®) on survival, growth, and reproduction of the fathead minnow (*Pimephales promelas*). J. Fish. Res. Board Can. 29: 583–587.

Courtemanch, D.L. and K.E. Gibbs. 1980. Short and long term effects of forest spraying of carbaryl (Sevin-4-Oil®) on stream invertebrates. Can. Ent. 112:271-276.

Duan, B., Yendol, W.G., Mierzejewski, K., and R. Reardon. 1992a. Validation of the AGDISP aerial spray deposition prediction model. Pestic. Sci. 36:19–26.

Duan, B., Yendol, W.G., and K. Mierzejewski. 1992b. Statistical comparisons of the AGDISP model with deposit data. Atmospheric Environment 26A(9): 1635–1642.

Dwyer, F.J., Mayer, F.L., Sappington, L.C., Buckler, D.R., Bridges, C.M., Greer, I.E., Hardesty, D.K., Henke, C.E., Ingersoll, C.G., Kunz, J.L., Whites, D.W., Augspurger, T., Mount, D.R., Hattala, K., and G.N. Neuderfer. 2005. Assessing contaminant sensitivity of endangered and threatened aquatic species: Part I Acute toxicity of five chemicals. Arch. Environ. Contam. Toxicol. 48:143-154.

Fair, J.M., P.L. Kennedy, and L.C. McEwen. 1995a. Effects of carbaryl grasshopper control on nesting killdeer in North Dakota. Environ. Toxicol. Chem. 14:881–890.

Fair, J.M., P.L. Kennedy, and L.C. McEwen. 1995b. Diet of nesting killdeer in North Dakota. Wilson Bulletin 107:174–178.

Ferrari, A., Anguiano, O.L., Soleno, J., Venturino, A., and A.M. Pechen de D'Angelo. 2004a. Different susceptibility of two aquatic vertebrates (*Oncorhynchus mykiss* and *Bufo arenarum*) to azinphos-methyl and carbaryl. Comp. Biochem. Physiol. Part C. 139:239–243.

Ferrari, A., Venturino, A., and A.M. Pechen de D'Angelo. 2004b. Time course of brain cholinesterase inhibition and recovery following acute and subacute azinphosmethyl, parathion and carbaryl exposure in the goldfish (*Carassius auratus*). Ecotox. Environ. Safety 57:420–425.

Fletcher, J.S., J.E. Nellesson and T.G. Pfleeger. 1994. Literature review and evaluation of the EPA food chain (Kenaga) nomogram, an instrument for estimating pesticide residues on plants. Environ. Tox. and Chem. 13(9):1383-1391.

Gramlich, F.J. 1979. Effects of Sevin on songbird cholinesterase. In Maine Department of Conservation, Bureau of Forestry, Augusta, ME. Environmental monitoring of Cooperative Spruce Budworm Control Projects.

Haines, T.A. 1981. Effect of an aerial application of carbaryl on brook trout (*Salvelinus fontinalis*). Bul. Environ. Contam. Toxicol. 27:534–542.

Hanazato, T. 1991. Effects of long- and short-term exposure to carbaryl on survival, growth, and reproduction of *Daphnia ambigua*. Environ. Pollution 74:139–148.

Hardersen, S. and S.D. Wratten. 2000. Sensitivity of aquatic life stages of *Xanthocnemis zealandica* (Odonata: Zygoptera) to azinphos-methyl and carbaryl. New Zeal. J. Mar. Fresh. Res. 34:117-123.

Hernandez, D.A., Lombardo, R.J., and M.C. Tororelli. 1990. Toxicity of ethyl-parathion and carbaryl on early development of sea urchin. Bul. Env. Contam. Toxicol. 45:734–741.

Hewitt, A.J., Johnson, D.R., Fish, J.D., Hermansky, C.G., and D.L. Valcore. 2002. Development of the spray drift task force database for aerial applications. Env. Toxicol. Chem. 21(3): 648-658.

Hoerger, F. and E.E. Kenaga. 1972. Pesticide residues on plants: correlation of representative data as a basis for estimation of their magnitude in the environment. In: F. Coulston and F. Corte, eds., Environmental Quality and Safety: Chemistry, Toxicology and Technology. Vol 1. George Theime Publishers, Stuttgart, Germany. pp. 9-28.

Holmes, S.B., Millikin, R.L. and P.D. Kingsbury. 1981. Environmental effects of a split application of Sevin-2-Oil®. Forest Pest Mgt. Institute. Report FPM-X-46. 58 pp.

Hudson, R.H., Tucker, R.K., and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. Resource Publication 153. U.S. Department of the Interior, Fish and Wildlife Service, Washington, DC.

Hurlbert, P.J. 1978. Effects of Sevin®, a spruce budworm insecticide on fish and invertebrates in the Mattawamkeas River in 1976. In: Maine Department of Conservation, Bureau of Forestry,

Augusta, ME. Environmental monitoring of cooperative spruce budworm control projects, Maine 1976 and 1977.

Jadhav, S., Sontakke, Y.B., and V.S., Lomte. 1996. Carbaryl toxicity to freshwater bivalve *Corbicula striatella*. Environ. Ecol. 14(4): 863–865.

Jones, S.B., King, L.B., Sappington, L.C., Dwyer, F.J., Ellersieck, M., and D.R. Buckler. 1998. Effects of carbaryl, permethrin, 4-nonylphenol, and copper on mucarinic cholinergic receptors in brain of surrogate and listed fish species. Comp. Biochem. Physiol. Part C. 120:405–414.

Karnak, R.E. and W.J. Collins. 1974. The susceptibility to selected insecticides and acetylcholinesterase activity in a laboratory colony of midge larvae, *Chironomus tentans* (Diptera: Chironomidae). Bull. Environ. Contam. Toxicol. 12(1): 62-9

Katz, M. 1961. Acute toxicity of some organic insecticides to three species of salmonids and to the threespine stickleback. Trans. Amer. Fish. Soc. 90(3): 264–268.

Kaur, K., and A. Dhawan. 1993. Variable sensitivity of *Cyprinus carpio*, eggs, larvae, and fry to pesticides. Bul. Environ. Contam. Toxicol. 50:593–599.

Labenia, J.S., Baldwin, D.H., French, B.L., Davis, J.W., and N.L. Scholz. 2007. Behavioral impairment and increased predation mortality in cutthroat trout exposed to carbaryl. Mar. Ecol. Proj. Serv. 329:1–11.

LeBlanc, L., Vargas, R.I and D. Rubinoff. 2010. Attraction of *Ceratitis capitata* (Diptera: Tephritidae) and endemic and introduced nontarget insects to biolure bait and its individual components in Hawaii. Environ. Entomol. 39(3):989-998.

Little, E.E., Archeski, R.D., Flerov, B.A., and V.I. Kozlovskaya. 1990. Behavioral indicators of sublethal toxicity in rainbow trout. Arch. Environ. Contam. Toxicol. 19:380–385.

Marian, M.P., Arul, V. and T.J. Pandian. 1983. Acute and chronic effects of carbaryl on survival, growth, and metamorphosis in the bullfrog (*Rana tigrina*). Arch. Environ. Cont. Toxicol. 12:271-275.

Marletto, F., Patetta, A. and A. Manino. 2003. Laboratory assessment of pesticide toxicity to bumblebees. Bull Insect. 56(1): 155-158.

Mayer, F.L. 1987. Acute toxicity handbook of chemicals to estuarine organisms. U.S. Environmental Protection Agency. Environ. Res. Lab., Gulf Breeze, Florida.

Mayer, F.L., Jr., and M.C. Ellersieck. 1986. Manual of acute toxicity: interpretation and data base for 410 chemicals and 66 species of freshwater animals. Resour. Publ. 160. Department of the Interior, Fish and Wildlife Service, Washington, DC. As cited ECOTOX.

McEwen, L.C., C.M. Althouse, and B.E. Peterson. 1996. Direct and indirect effects of grasshopper integrated pest management (GHIPM) chemicals and biologicals on nontarget animal life. In Grasshopper Integrated Pest Management User Handbook, Tech. Bul. 1809. Sec. III.2. U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Washington, DC.

Mullins, C.A., Frazier, M., Frazier, J.L., Ashcraft, S. and R. Simonds. 2010. High levels of miticides and agrochemicals in North American apiaries: implications for honey bee health. PLoS One 5(3):e9574. doi:10.1371/journal.pone.0009754.

National Institutes of Health (NIH). 2009. HSDB: Carbaryl, CASRN: 63-25-2, U.S. National Library of Medicine, Toxnet, available at: https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~xEEdG9:3, last revision date: 2009-06-26, last field update on 2012-03-26, last accessed on Feb. 21, 2018.

NMFS—See National Marine Fisheries Service

National Marine Fisheries Service. 2009. National Marine Fisheries Service Endangered Species Act Section 7 Consultation. Final biological opinion for pesticides containing carbaryl, carbofuran and methomyl. Environmental Protection Agency Registration of Pesticides Containing Carbaryl, Carbofuran and Methomyl.

National Pesticide Information Center (NPIC). 2016. Water Solubility, available at: http://npic.orst.edu/envir/watersol.html, last updated Feb. 5, 2016, last accessed June 29, 2018.

National Research Council (NRC). 1983. Risk assessment in the Federal government: managing the process. National Academy Press, Washington, DC.

NTP – see National Toxicology Program.

NTP-CERHR – National Toxicology Program Center for the Evaluation of Risks to Human Reproduction.

National Toxic Program. 2004. NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Propylene Glycol, NIH Publication No. 04-4482, 117 pp.

Naqvi, S.M., and R. Hawkins. 1988. Toxicity of selected insecticides (Thiodan, Security, Spartan, and Sevin) to mosquitofish, *Gambusia affinis*. Bul. Environ. Contam. Toxicol. 40:779-784.

Occupational Safety and Health Administration – U.S. Department of Labor, Occupational Safety and Health Administration

Peach, M.P., Alston, D.G., and V.J. Tepedino. 1994. Bees and bran bait: is carbaryl bran bait lethal to alfalfa leafcutting bee (Hymenoptera: Megachilidae) adults or larvae? J. Econ. Entomol. 87:311–317.

Peach, M.l., Alston, D.G., and V. J. Tepedino. 1995. Sublethal effects of carbaryl bran bait on nesting performance, parental investment, and offspring size and sex ratio of the alfalfa leafcutting bee (Hymenoptera: Megachilidae). Environ. Entomol. 24(1): 34-39.

Peterson, H.G., Boutin, C., Martin, P.A., Freemark, K.E., Ruecker, N.J., and M.J. Moody. 1994. Aquatic phyto-toxicity of 23 pesticides applied at expected environmental concentrations. Aquatic Tox. 28:275–292.

Peterson, J., Jepson, P., and J.J. Jenkins. 2001a. Effect of varying pesticide exposure duration and concentration on the toxicity of carbaryl to two field-collected stream invertebrates, *Calineuria californica* (Plecoptera: Perlidae) and *Cinygma* sp. (Ephemeroptera: Heptageniidae). Environ Toxicol Chem. 20(10): 2215-23.

Peterson, J.L., Jepson, P.C., and J.J. Jenkins. 2001b. A test system to evaluate the susceptibility of Oregon, USA, native stream invertebrates to triclopyr and carbaryl. Env. Tox. Chem. 20(10):2205-2214.

Pickering, Q.H., Lazochak, J.M., and K.L. Winks. 1996. Subchronic sensitivity of one-, four-, and seven-day old fathead minnow (*Pimephales promelas*) larvae to five toxicants. Env. Toxicol. Chem. 15(5): 353–359.

Rao, D.M., Murty, A.S., and P.A. Swarup. 1984. Relative toxicity of technical grade and formulated carbaryl and 1-naphthol to, and carbaryl-induced biochemical changes in the fish *Cirrhinus mrigula*. Env. Poll. (Series A) 34:47-54.

Reddy, M.S. and K.V.R. Rao. 1992. Toxicity of selected insecticides to the penaeid prawn, *Metapenaeus monoceros* (Fabricius). Bull. Environ. Contam. Toxicol. 48(4): 622-629.

Reinhardt, T. and R. Ottmar. 2004. Baseline measurements of smoke exposure among wildland firefighters. Journal of Occupational and Environmental Hygiene 1: 593-606.

Reisen, F. and S. Brown. 2009. Australian firefighters' exposure to air toxics during bushfire burns of autumn 2005 and 2006. Environment International 35: 342-352.

Relyea, R.A. and N. Mills. 2001. Predatory-induced stress makes the pesticide carbaryl more deadly to gray treefrog tadpoles (*Hyla versicolor*). Proc. Nat. Acad. Science 98(5):2491-2496.

Relyea, R.A., and N. Diecks, 2008. An unforeseen chain of events: lethal effects of pesticides at sublethal concentrations. Ecol. Appl. 18(7):1728–1742.

Richmond, M.L., C.J. Henny, R.L. Floyd, R.W. Mannan, D.M. Finch, and L.R. DeWeese. 1979. Effects of Sevin 4-oil, Dimilin, and Orthene on forest birds in northeastern Oregon. USDA, Pacific SW Forest and Range Experiment Station, Research Paper PSW–148.

Rohr, J.R., Elskus, A.A., Shepherd, B.S., Crowley, P.H., McCarthy, T.M., Niedzwiecki, J.H., Sager, T., Sih, A., and B.D. Palmer. 2003. Lethal and sublethal effects of atrazine, carbaryl, endosulfan, and octylphenol on the streamside salamander (*Ambystoma barbouri*). Env. Toxicol. Chem. 22(10): 2385-2392.

Sanders, H.O., Finley, M.T. and J.B. Hunn. 1983. Acute toxicity of six forest insecticides to three aquatic invertebrates and four fishes. Tech. Pap. No. 110, U. S. Fish Wildl. Serv., Washington, DC.

Schafer, E.W., Bowles, W.A., and J. Hurlbut. 1983. The acute oral toxicity, repellency, and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environ. Contam. Toxicol. 12: 355–382.

Shea, T.B. and E. S. Berry. 1983. Toxicity and intracellular localization of carbaryl and 1-Naphthol in cell cultures derived from goldfish. Bul. Environ. Contam. Toxicol. 30:99-104.

Shepard, M. and W. Sterling. 1972. Effects of early season applications of insecticides on beneficial insects and spiders in cotton. Texas Agricultural Experimental Station Bulletin MP–1045.

Smith, D.I., Lockwood, J.A., Latchininsky, A.V., and D.E. Legg. 2006. Changes in non-target populations following applications of liquid bait formulations of insecticides for control of rangeland grasshoppers. Internat. J. Pest Mgt. 52(2):125-139.

Solomon, K.E., and R.J. Robel. 1980. Effects of carbaryl and carbofuran on bobwhite energetics. J. Wildl. Manage. 44:682-686.

Teske, M.E., and T.B. Curbishley. 2003. AgDisp Version 8.07 User's Manual. Continuum Dynamics Tech. Note No. 02–06.

Teske, M.E., and H.W. Thistle. 2004. Aerial application model extension into the far field. Biosystems Engr. 89(1): 29–36.

Teske, M.E., and H.W. Thistle. 2003. Release height and far-field limits of Lagrangian aerial spray models. Tran.ASABE. 46(4): 977–983.

Teske, M.E., Thistle, H.W., and R.E. Mickle. 2000. Modeling finer droplet aerial spray drift and deposition. Appl. Engr. Agric. 16(4): 351–357.

Tessenderlo Kerley, Inc. 2012. Label for SEVIN® XLR plus (EPA Reg. No.61842-37), 39 pp.

Tessenderlo Kerley, Inc. 2015. Safety Data Sheet for SEVIN® XLR plus Safety, issue date: May 8, 2015, 7 pp.

Tilak, K.S., Rao, D.M., Devi, A.P., and A.S. Murty. 1981. Toxicity of carbaryl and 1-naphthol to four species of freshwater fish. J. Biosci. 3(4):457-461.

Thistle, H.W., Thompson, D.G., Richardson, B., Bird, S., and R. Karsky. 2008. Deposition of aerially released Bt over a 2-km sampling grid: near field model comparison. Presented at the 2008 American Society of Agricultural and Biological Engineers Annual Meeting as Paper No. 084124.

United States Department of Agriculture, Animal and Plant Health Inspection Service (USDA APHIS). 2002. Rangeland Grasshopper and Mormon Cricket Suppression Program Final Environmental Impact Statement, 283 pp.

USDA APHIS. 2008. Grasshopper Guidebook Provisional, January 2008, 120 pp.

USDA APHIS. 2015a. Grasshopper Mormon Cricket Background, Available at: https://www.aphis.usda.gov/aphis/ourfocus/planthealth/plant-pest-and-disease-programs/pests-and-diseases/grasshopper-mormon-cricket/ct_background/, last modified April 6, 2015, last accessed August 31, 2017.

USDA APHIS. 2015b. Biological Assessment for the APHIS Rangeland Grasshopper and Mormon Cricket Suppression Program, March 2015.

USDA APHIS. 2016. APHIS Rangeland and Grasshopper/Mormon Cricket Suppression Program Aerial Application Statement of Work, March 2016, 41 pp.

USDA APHIS. 2017. APHIS Rangeland Grasshopper and Mormon Cricket Suppression Program FY-2017 Treatment Guidelines, Version 2/17/2017, 16 pp.

USDA APHIS. 2018. Grasshopper and Mormon Cricket Suppression Program for Southern Idaho, Environmental Assessment ID-18-01 April 19, 2018, 40 pp, available at: https://static1.squarespace.com/static/564b8c9ae4b0459b2b8187a3/t/5ae7315d70a6adc56154851a/1525100898608/ID-2018-EA-GHMC_FINAL.pdf, last accessed June 11, 2018.

USDA Forest Service (FS). 2008. Carbaryl Human Health and Ecological Risk Assessment Revised Final Report, SERA TR-052-01-05a, dated February 9, 2008, 276 pp.

USDA FS. 2013. Wildland Firefighter Smoke Exposure, 26 pp.

U.S. Department of the Interior. 1992. Crystalline silica primer. Special Publication. Washington, DC: U.S. Department of the Interior, Branch of Industrial Minerals. 29 pp.

U.S. Department of Labor, Occupational Safety and Health Administration. 2002. OSHA carbon monoxide poisoning. OSHA Fact Sheet. 2 pp.

United States Environmental Protection Agency (USEPA). 2003. Environmental fate and ecological risk assessment for re-registration of carbaryl.

USEPA. 2004. Finalization of Interim Reregistration Eligibility Decisions (IREDs) and Interim Tolerance Reassessment and Risk Management Decisions (TREDs) for the Organophosphate Pesticides, and Completion of the Tolerance Reassessment and Reregistration Eligibility Process for the Organophosphate Pesticides, dated July 31, 2006.

USEPA. 2005. User's Guide T-REX Version1.2.3 (Terrestrial Residue Exposure Model).

USEPA. 2006. Reregistration Eligibility Decision for Propylene Glycol and Diprophylene Glycol, EPA-739-R-06-002, September 2006, 80 pp.

USEPA. 2007a. Risks of Carbaryl Use to the Federally Listed Endangered Barton Springs Salamander (*Eurycea sosorum*), Pesticide Effects Determination Environmental Fate and Effects Division Office of Pesticide Programs Washington, D.C. 20460, dated September 19, 2007, 602 pp.

USEPA. 2007b. Reregistration Eligibility Decision (RED) for Carbaryl. Prevention, Pesticides and Toxic Substances (7508P), EPA-738R07-018, September 2007, 47 pp.

USEPA. 2007c. Propylene glycol/dipropylene glycol: AD's Risk Assessment for Issuance of the Reregistration Eligibility Decision (RED) Document. Reregistration Case No.: 3126. PC Codes: 068603, 068604. CAS Registry No.: Propylene Glycol, 57-55-6; Dipropylene Glycol, 25265-71-8, February 5, 2007, 44 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2006-0831-0003, last accessed Oct. 25, 2017.

USEPA. 2007d. Memorandum - Propylene/Dipropylene Glycol Revised Ecological Hazard and Environmental Risk Characterization Chapter for the Reregistration Eligibility Decision (RED) Document, Case 3126, February 14, 2006, 16 pp, available at:

https://www.regulations.gov/document?D=EPA-HQ-OPP-2006-0831-0010, last accessed Oct. 25, 2017.

USEPA. 2007e. Memorandum – Propylene glycol/dipropylene glycol: Revised Toxicology Chapter in Support of Issuance of the Reregistration Eligibility Decision (RED) Document. PC Code for Propylene Glycol: 068603; PC Code for Dipropylene Glycol: 068604. CAS Registry Number for Propylene Glycol: 57-55-6; CAS Registry Number for Dipropylene Glycol: 25265-71-8. Reregistration Case Number: 3126. DP #: 327061, February 5, 2007, 18 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2006-0831-0004, last accessed Oct. 25, 2017.

USEPA. 2010a. Memorandum – Carbaryl. Human Health Assessment Scoping Document in Support of Registration Review, dated August 26, 2010, 21 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0005, last accessed Aug. 9, 2017.

USEPA. 2010b. Memorandum – Registration review – preliminary problem formulation for ecological risk and environmental fate, endangered species, and drinking water assessments for carbaryl, September 3, 2010, 56 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0004, last accessed Aug. 9, 2017.

USEPA. 2010c. Memorandum – Carbaryl: Review of Human Incidents, dated May 20, 2010, 62 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0008, last accessed Aug. 9, 2017.

USEPA. 2012. Benchmark Dose Technical Guidance. Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC.

USEPA. 2015. Memorandum: EDSP Weight of Evidence Conclusions on the Tier 1 Screening Assays for the List 1 Chemicals, EDSP: Weight of Evidence Analysis of potential interactions with the estrogen, androgen or thyroid pathways, chemical: carbaryl, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0029, last accessed April 6, 2016.

USEPA. 2016. Overview of risk assessment – human health risk assessment (https://www.epa.gov/risk/human-health-risk-assessment), website last updated Oct. 3, 2016, last accessed 8/1/2017.

USEPA. 2017a. Memorandum - Carbaryl: Draft Human Health Risk Assessment in Support of Registration Review, March 30, 2017, 113 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0034, last accessed March 26, 2018.

USEPA. 2017b. Memorandum - Carbaryl: Tier I Update Review of Human Incidents and Epidemiology for Draft Risk Assessment, March 30, 2017. 60 pp, available at: https://www.regulations.gov/document?D=EPA-HQ-OPP-2010-0230-0035, last accessed March 26, 2018.

USEPA. 2017a. Memorandum – Carbaryl. Occupational and Residential Exposure Assessment in Support of Registration Review, March 30, 2017, 126 pp.

USEPA. 2018. Occupational Pesticide Handler Unit Exposure Surrogate Reference Table, June 2018. 17 pp, available at: https://www.epa.gov/sites/production/files/2018-06/documents/opp-hed-pesticide-handler-surrogate-unit-exposure-table-june-2018.pdf, last accessed June 25, 2018.

Weiland, R.T., Judge, F.D., Pels, T., and A.C. Grosscurt. 2002. A literature review and new observations on the use of diflubenzuron for control of locusts and grasshoppers throughout the world. J. Orthoptera Res. 11(1):43-54.

Wilbur-Ellis Company. 2012. Label for 2% SEVIN BAIT (EPA Reg. No.2935-556), 4 pp.

Wilbur-Ellis Company. 2014. Label for SEVIN 5 BAIT (EPA Reg. No.2935-366), 7 pp.

Wilbur-Ellis Company LLC. 2016a. Safety Data Sheet for 2% SEVIN BAIT, 1141 Version #: 01, issue date: 02-23-2016, 6 pp.

Wilbur-Ellis Company LLC. 2016b. Safety Data Sheet for SEVIN 5 BAIT, 1654 Version #: 03, Issue date: 05-01-2015, Revision date: 02-23-2016, 6 pp.

Woodward, D.F., and W.L. Mauck. 1980. Toxicity of five forest insecticides to cutthroat trout and two species of aquatic invertebrates. Bul. Environ. Contam. Toxicol. 25:846–853.

Zaga, A., E.E. Little, C.E. Rabeni, and M.R. Ellersieck. 1998. Photoenhanced toxicity of a carbamate insecticide to early life stage anuran amphibians. Environ. Toxicol. and Chem. 17:2543–2553.

Zinkl, J.G., C.J. Henny, and L.R. DeWeese. 1977. Brain cholinesterase activities of birds from forests sprayed with trichlorfon (Dylox) and carbaryl (Sevin 4-oil). Bull. Environ. Contam. Toxicol. 17:379–386.

Zinkl, J.G., Shea, P.J., Nakamoto, R.J., and J. Callman. 1987. Brain cholinesterase activity of rainbow trout poisoned by carbaryl. Bull. Environ. Contam. Toxicol. 38:29–35.

APPENDIX A

Risk Evaluation for Rangeland Firefighters from Potential Exposure to Pyrolysis Products of Carbaryl

A risk evaluation for rangeland firefighters from exposure to pyrolysis products of carbaryl was performed by comparing potential levels of carbaryl, and its pyrolysis products, in wildfire-associated smoke to human health hazard benchmarks. The Occupational Safety and Health Administration (OSHA) occupation safety standards for carbaryl and its potential pyrolysis products were used as safe exposure levels for firefighters. The USEPA inhalation point of departure (POD) for carbaryl (USEPA, 2017) was also used as a no adverse effect level for inhalation exposure in the risk evaluation. A POD is a dose that is considered to be in the range of observed responses, without significant extrapolation (USEPA, 2012).

Estimates of exposure for carbaryl and its pyrolysis products

The USDA APHIS grasshopper and Mormon cricket suppression program may use carbaryl bait (Sevin® 5 Bait, EPA Reg. No. 2935-366, and 2% Sevin® Bait, EPA Reg. No. 2935-556) and spray (Sevin® XLR Plus, EPA Reg. No. 61842-37) by aerial or ground application to suppress rangeland grasshoppers during outbreaks (USDA APHIS, 2008; 2016). The application rates for carbaryl baits are 0.5 lb a.i./acre for the conventional rate and 0.2 lb a.i./acre for the reduced agent area treatment (RAAT) rate. The application rates for the carbaryl spray formulation are 0.5 lb a.i./acre for the conventional rate and 0.25 lb a.i./acre for the RAAT rate. The USDA APHIS program applies carbaryl once per season, and typically uses RAAT rates (APHIS, 2018a).

APHIS reviewed safety data sheets (SDS) for the carbaryl bait and spray formulation used by the program and conducted a literature search to identify carbaryl pyrolysis products. The SDS for the bait formulation (Wilbur-Ellis Company, 2017a, b) states that hazardous gases may be formed during fire without listing specific pyrolysis products of carbaryl. The SDS for the spray formulation (Tessenderlo Kerley, 2018) states that nitrogen oxides and carbon dioxide can be released from fire. Trace amounts of methyl isocyanate were listed under hazardous decomposition products along with carbon and nitrogen oxides. Toxic combustion products that may be released in a fire involving carbaryl include oxides of nitrogen, methylamine, and carbon monoxide (NIH, 2009). Specific studies of carbaryl pyrolysis products from a wildfire do not appear to be available in the literature.

APHIS used several conservative assumptions to estimate carbaryl exposure to wildfire firefighters. APHIS assumed that a wildfire event would occur immediately after application of a bait and that no degradation of carbaryl or removal by grasshoppers would occur after treatment. APHIS used a mixing height of 400 meters to calculate potential residues in the atmosphere from carbaryl and any known degradates that would occur as a result of a wildfire. A mixing height of 400 meters in the air represents a conservative exposure scenario because it is the approximate smoke-plume height of the most intensive smoke particulates during initial stage

of a prescribed fire using a ground-based scanning lidar (Kovalev, et al., 2015). Under stagnant conditions, a smoke mixing height is 518 meters or less (Auburn University, No Date). Under the 400-meter mixing height scenario, 0.5 and 0.2 pounds of carbaryl produce 0.14 mg/m^3 and 0.06 mg/m^3 of total combustion products, respectively. For trace amounts of methyl isocyanate (less than 1%) in the combustion products, the estimated levels are 0.0014 mg/m^3 and 0.0006 mg/m^3, respectively. The calculations are included in attachment A.

Risk evaluation to wildfire firefighters

The U.S. occupational health standards (i.e. OSHA permissible exposure limits (PELs)) were used as no adverse health effect levels for comparison to determine risk. The PELs are the highest levels of exposure that workers may be exposed to for 8 hours a day without incurring adverse health effects. The OSHA PELs for carbaryl, methyl isocyanate, methylamine, and carbon oxide are an eight-hour time-weighted average (TWA) of 5 mg/m^3, 0.05 mg/m^3, 12 mg/m^3, and 55 mg/m^3, respectively (CDC, 2018a, b, c, d). The OSHA PEL for nitrogen dioxide is a ceiling level of 9 mg/m^3 (CDC, 2018e).

The comparison results (table 1) show that the estimated potential exposed levels for carbaryl and its possible pyrolysis products under a 400-meter mixing height scenario, the estimated potential exposed levels for carbaryl and its possible pyrolysis products under the conventional or RAAT application rates were below the occupational health standards.

Table 1. Comparison of estimated exposure levels for carbaryl and pyrolysis products to OSHA PELs.

Carbaryl and Its Possible Pyrolysis Products	Estimated Exposure Levels (mg/m^3) (0.5 lb a.i./acre)	Estimated Exposure Levels (mg/m^3) (0.2 lb a.i./acre)	OSHA PELs (mg/m^3)
Carbaryl	0.14	0.06	5 TWA
Methyl Isocyanate[1]	0.0014	0.0006	0.05 TWA
Methylamine[2]	0.14	0.06	12 TWA
Carbon monoxide[2]	0.14	0.06	55 TWA
Nitrogen dioxide[2]	0.14	0.06	9 Ceiling

[1] 1% of the total amount of applied carbaryl.
[2] The potential exposure levels to carbaryl from a wildfire were also used for methylamine, nitrogen dioxide, and carbon monoxide.

APHIS further evaluated firefighter risks by comparing the exposure doses estimated from the exposure levels in table 1 to the USEPA's occupational inhalation POD (1.0 mg/kg/day) for carbaryl (USEPA, 2017a). The occupational inhalation POD is a human-equivalent dose of no adverse effect based on an acute inhalation toxicity study in rats. The potential exposure dose for a wildland fire fighter assumes a body weight of 80 kg (USEPA, 2017a), a breathing rate of 24 liter per minutes, and an average of 13.6 hours per daily shift (Navarro, et al., 2019). The estimated exposure dose levels are below the POD for the RAAT application suggesting no adverse effects (table 2). The estimated margin of exposure (MOE) under the mixing height of

400 meters for the conventional and RAAT application rates are higher than the USEPA's level of concern of 30 for the inhalation exposure, which indicates that there is no concern. A MOE is a numerical value that characterizes the amount of safety to a toxic chemical. The actual mixing heights could be greater than 400 meters during an actual wildfire and less bait would be available due to consumption by grasshoppers and Mormon crickets. Inhalation POD values are not available for pyrolysis products of carbaryl.

Table 2. Comparison of estimated potential exposed dose levels to the USEPA POD for carbonyl.

Scenarios	Estimated Exposure Levels (mg/m^3)	Estimated Exposure Doses (mg/kg/day)	Inhalation POD (mg/kg/day)	Margin of Exposure* (mg/kg/day)
Conventional application (0.5 lb a.i./acre)	0.14	0.03	1.0	33
RAAT application (0.2 lb a.i./acre)	0.06	0.01	1.0	100

Note:
MOE–a ratio of a toxicological endpoint (usually a NOAEL) to exposure. The calculated MOE is an inhalation POD divided by exposure dose.

References

Auburn University. No Date. Smoke Dispersion, Mixing Height, Online at: http://www.auburn.edu/academic/forestry_wildlife/fire/smoke_guide/smoke_dispersion.htm, Last accessed March 28, 2019.

Beauvais, G. P. and J. Struttman. 2003. Potential effects of pesticide applications on Preble's meadow jumping mouse (*Zapus hudsonius preblei*) and mountain plover (*Charadrius montanus*) in southeast Wyoming. Wyoming Natural Diversity Database, University of Wyoming, Laramie, Wyoming, 23 pp.

Burling, I., Yokelson, R., Griffith, D., Johnson, T., Veres, P., Roberts, J., Warneke, C., Urbanski, S., Reardon, J., Weise, D., Hao, W., and J. de Gouw. 2010. Laboratory measurements of trace gas emissions from biomass burning of fuel types from the southeastern and southwestern United States. Atmospheric Chemistry and Physics 10: 11115-111130.

Bytnerowicz, A., Arbaugh, M., Riebau, A., and C. Andersen. (Eds.). 2009. Wildland fires and air pollution. Developments in Environmental Science 8. Oxford, UK: Elsevier.

Carratt, S. A., Flayer, C. H., Kossack, M. E., and J. A. Last. 2017. Pesticides, wildfire suppression chemicals, and California wildfires: A human health perspective. Current Topics in Toxicology. Vol. 13, 1-12.

Centers for Disease Control and Prevention (CDC), 2018a. The National Institute for Occupational Safety and Health (NIOSH) Pocket Guide to Chemical Hazards. Carbaryl. Available at: https://www.cdc.gov/niosh/npg/npgd0100.html, Page last reviewed November 29, 2018, last accessed March 25, 2019.

CDC, 2018b. NIOSH Pocket Guide to Chemical Hazards. Methyl Isocyanate. Available at: https://www.cdc.gov/niosh/npg/npgd0423.html, Page last reviewed November 29, 2018, last accessed March 25, 2019.

CDC. 2018c. NIOSH Pocket Guide to Chemical Hazards. Methylamine. Available at: https://www.cdc.gov/niosh/npg/npgd0398.html, Page last reviewed November 29, 2018, last accessed March 25, 2019.

CDC. 2018d. NOISH Pocket Guide to Chemical Hazards. Carbon Oxide. Available at: https://www.cdc.gov/niosh/npg/npgd0105.html, Page last reviewed November 29, 2018, last accessed April 11, 2019.

CDC. 2018e. NOISH Pocket Guide to Chemical Hazards. Nitrogen dioxide. Available at: https://www.cdc.gov/niosh/npg/npgd0454.html, Page last reviewed November 29, 2018, last accessed April 11, 2019.

Foster, R.N. and J.A. Onsager. 2001a. A review of chemical sprays in cooperative rangeland control programs. Chap. II.4 in D.H. Branson and B. Redlin (editors). Grasshoppers: their biology, identification and management. USDA Agricultural Research Service.

Foster, R.N and J.A. Onsager. 2001b. Sprays versus baits. Chapter II.3 in D.H. Branson and B. Redlin (editors). Grasshoppers: their biology, identification and management. USDA Agricultural Research Service.

Kovalev, V., Petkow, A., Wold, C., Urbanski, S., and Hao, W.M. 2015. Determination of the smoke-plume heights and their dynamics with ground-based scanning lidar. Applied Optics 54(8):2011-2017.

National Institutes of Health (NIH). 2009. HSDB: Carbaryl, CASRN: 63-25-2, U.S. National Library of Medicine, Toxnet, available at: https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~xEEdG9:3, last revision date: 2009-06-26, last field update on 2012-03-26, last accessed on Feb. 21, 2018.

Navarro, Kathleen M., Kleinman, Michael T., Mackay, Chris E., Reinhardt, Timothy E.,

Balmes, John R., Broyles, George A., Ottmar, Roger D., Naher, Luke P., and Joseph W. Domitrovich. 2019. Wildland firefighter smoke exposure and risk of lung cancer and cardiovascular disease mortality. Environmental Research 173:462-468.

Reinhardt, T. and R. Ottmar. 2004. Baseline measurements of smoke exposure among wildland firefighters. Journal of Occupational and Environmental Hygiene 1: 593-606.

Reisen, F. and S. Brown. 2009. Australian firefighters' exposure to air toxics during bushfire burns of autumn 2005 and 2006. Environment International 35: 342-352.

Tessenderlo Kerley, Inc. 2018. Safety Data Sheet for SEVIN® XLR plus Safety, issue date: July 25, 2018, 7 pp.

United States Department of Agriculture, Animal and Plant Health Inspection Service (USDA, APHIS). 2008. Grasshopper Guidebook Provisional, January 2008, 120 pp.

USDA APHIS. 2015. Biological Assessment for the APHIS Rangeland Grasshopper and Mormon Cricket Suppression Program, March, 2015.

USDA APHIS. 2016. APHIS Rangeland and Grasshopper/Mormon Cricket Suppression Program Aerial Application Statement of Work, March 2016, 41 pp.

USDA APHIS. 2018a. Rangeland Grasshopper and Mormon Cricket Suppression Program Draft Environmental Impact Statement, December 2018, 126 pp.

USDA APHIS. 2018b. Grasshopper and Mormon Cricket Suppression Program for Southern Idaho Environmental Assessment ID-18-01, April 19, 2018, 84 pp.

U.S. Department of the Interior. 1992. Crystalline silica primer. Special Publication. Washington, DC: U.S. Department of the Interior, Branch of Industrial Minerals. 29 pp.

U.S. Department of Labor, Occupational Safety and Health Administration. 2002. OSHA carbon monoxide poisoning. OSHA Fact Sheet. 2 pp.

United States Environmental Protection Agency (USEPA). 2010. Memorandum – Registration review – preliminary problem formulation for ecological risk and environmental fate, endangered species, and drinking water assessments for carbaryl, September 3, 2010, 56 pp.

USEPA. 2012. Terminology Services (TS), Vocabulary Catalog, Pesticides Glossary, last updated: May 9, 2012. Online source: https://ofmpub.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?details=&vocabName=Pesticides%20Glossary&filterTerm=point%20of%20departur

e&checkedAcronym=false&checkedTerm=false&hasDefinitions=false&filterTerm=point%20of%20departure&filterMatchCriteria=Contains, last accesses April 11, 2019.

USEPA. 2017a. Memorandum – Carbaryl. Occupational and Residential Exposure Assessment in Support of Registration Review, March 30, 2017, 126 pp.

USEPA. 2017b. Memorandum – Carbaryl Drinking Water Assessment for Registration Review, March 20, 2017, 39 pp.

USDA Forest Service (FS). 2013. Wildland Firefighter Smoke Exposure, 26 pp.

Volker, K. 2016. Pyrolysis of carbaryl and potential hazard to firefighters-Director of Development, TKI NovaSource, National Grasshopper Management Board, January 20-21, 2016

Wilbur-Ellis Company LLC. 2017a. Safety Data Sheet for 2% SEVIN BAIT, 1141 Version #: 02, revision date: 09-26-2017, issue date: 02-23-2016, 6 pp.

Wilbur-Ellis Company LLC. 2017b. Safety Data Sheet for SEVIN 5 BAIT, 1654 Version #: 04, Issue date: 05-01-2015, Revision date: 09-26-2017, 7 pp.

Attachment A

Assumptions in estimating potential exposure levels of carbaryl, and associated pyrolysis products, to wildfire firefighters

Potential exposed concentration = Application rate x 1 acre cube

Potential exposure dose level = (Potential exposed concentration x Breathing rate x Daily shift hours x Conversion factors)/Body weight

Carbaryl application rate (lb/acre) (APHIS, 2015)		
Conventional	0.5	
RAAT	0.2	
Mixing height (meter)	400	(Kovalev, et al. 2015)
1 acre and 400 meter cube (m3)	1618744	
Conversion factors	1 acre = 4046.86 m^2	
	1 lb = 453592 mg	
	1 liter = 0.001 m^3	
Potential exposed concentration (mg/m^3)		
Conventional	0.140	
RAAT	0.06	
Body weight (kg)	80	(USEPA, 2017a)
Breathing rate (liter per minute)	24	(Navarro, et al., 2019)
Average hours per daily shift	13.6	(Navarro, et al., 2019)
Potential exposure dose level (mg/kg/day)		
Conventional	0.03	
RAAT	0.01	

OSHA Safety Levels

Carbaryl	5 mg/m^3 TWA (CDC, 2018a)
Methyl isocyanate	0.05 mg/m^3 TWA (CDC, 2018b)
Methylamine	12 mg/m^3 TWA (CDC, 2018c)
Carbon monoxide	55 mg/ m^3 TWA (CDC, 2018d)
Nitrogen dioxide	9 mg/m^3 (Ceiling) (CDC, 2018e)

USEPA Inhalation Point of Departure

Carbaryl	1.0 mg/kg/day

Appendix B-1. Carbaryl acute aquatic fish toxicity values

Test Organism	Endpoint/Length	Toxicity Value	Reference
Salmo salar	96-hour LC_{50}	250 µg/L	Mayer and Ellersiek, 1986
Perca flavescens	96-hour LC_{50}	350 µg/L	Mayer and Ellersiek, 1986
Salvelinus fontinalis	96-hour LC_{50}	680 µg/L	Mayer and Ellersiek, 1986
Salvelinus namaycush	96-hour LC_{50}	690 µg/L	Mayer and Ellersiek, 1986
Oncorhynchus mykiss	96-hour LC_{50}	780 µg/L	Mayer and Ellersiek, 1986
Oncorhynchus clarki	96-hour LC_{50}	970 µg/L	Mayer and Ellersiek, 1986
Oncorhynchus kisutch	96-hour LC_{50}	1,150 µg/L	Mayer and Ellersiek, 1986
Acipenser brevirostrum	96-hour LC_{50}	1,810 µg/L	Dwyer et al., 2005
Ptychocheilus lucius	96-hour LC_{50}	1,300 µg/L	Beyers et al., 1994
Oncorhynchus apache	96-hour LC_{50}	1,540 µg/L	Dwyer et al., 2005
Oncorhynchus clarki stomias	96-hour LC_{50}	1,550 µg/L	Dwyer et al., 2005
Fundulus similis	96-hour LC_{50}	1,600 µg/L	Mayer, 1987
Lepomis macrochirus	96-hour LC_{50}	1,800 µg/L	Mayer and Ellersiek, 1986
Salmo trutta	96-hour LC_{50}	2,000 µg/L	Mayer and Ellersiek, 1986
Etheostoma lepidum	96-hour LC_{50}	2,014 µg/L	Dwyer et al., 2005
Etheostoma fonticola	96-hour LC_{50}	2,020 µg/L	Dwyer et al., 2005
Gilea elegans	96-hour LC_{50}	2,020 µg/L	Beyers et al., 1994
Oncorhynchus clarki henshawi	96-hour LC_{50}	2,250 µg/L	Dwyer et al., 2005
Oncorhynchus tshawytscha	96-hour LC_{50}	2,400 µg/L	Mayer and Ellersiek, 1986
Pomoxus nigromeculatus	96-hour LC_{50}	2,600 µg/L	Mayer and Ellersiek, 1986
Cyprinodon variegatus	96-hour LC_{50}	2,600 µg/L	USEPA, 2003
Hybopsis monacha	96-hour LC_{50}	3,410 µg/L	Dwyer et al., 2005
Xyrauchen texanus	96-hour LC_{50}	4,350 µg/L	Dwyer et al., 2005
Notropis mekistocholas	96-hour LC_{50}	4,510 µg/L	Dwyer et al., 2005
Cyprinodon bovinus	96-hour LC_{50}	4,540 µg/L	Dwyer et al., 2005
Cyprinus carpio	96-hour LC_{50}	5,280 µg/L	Mayer and Ellersiek, 1986
Micropterus salmoides	96-hour LC_{50}	6,400 µg/L	Mayer and Ellersiek, 1986
Cyprinodon macularius	96-hour LC_{50}	7,710 µg/L	Dwyer et al., 2005
Pimepheles promelas	96-hour LC_{50}	7,770 µg/L	Mayer and Ellersiek, 1986
Ictalurus puncatus	96-hour LC_{50}	7,790 µg/L	Mayer and Ellersiek, 1986
Lepomis cyanellas	96-hour LC_{50}	9,460 µg/L	Mayer and Ellersiek, 1986
Carassius auratus	96-hour LC_{50}	12,800 µg/L	Mayer and Ellersiek, 1986
Mystis vittatus	96-hour LC_{50}	17,500 µg/L	Arunachalam et al., 1980
Amelurus melas	96-hour LC_{50}	20,000 µg/L	Mayer and Ellersiek, 1986

Appendix B-2. Carbaryl acute aquatic invertebrate toxicity values

Test Organism	Endpoint/Length	Toxicity Value	Reference
Chironomus riparius	24-hour LC_{50}	1.2 µg/L	Karnak and Collins, 1974
Paneaus aztecus	48-hour LC_{50}	1.5 µg/L	Mayer, 1987
Pteronarcella badia	96-hour LC_{50}	1.7 µg/L	USEPA, 2003
Isogenus sp.	96-hour LC_{50}	3.6 µg/L	USEPA, 2003
Pteronarcys californica	96-hour LC_{50}	4.8 µg/L	Mayer and Ellersiek, 1986
Paleomenetes kadiankesis	96-hour LC_{50}	5.6 µg/L	Mayer and Ellersiek, 1986
Classenia sabulosa	96-hour LC_{50}	5.6 µg/L	USEPA, 2003
Daphnia magna	48-hour EC_{50}	5.6 µg/L	USEPA, 2003
Mysidopsis bahia	96-hour LC_{50}	5.7 µg/L	USEPA, 2003
Daphnia pulex	48-hour EC_{50}	6.4 µg/L	Mayer and Ellersiek, 1986
Chironomus tentans	24-hour LC_{50}	7.0 µg/L	Karnak and Collins, 1974
Simocephalus serrulatus	48-hour EC_{50}	7.6 µg/L	Mayer and Ellersiek, 1986
Chironomus plumosus	96-hour LC_{50}	10 µg/L	Sanders et al., 1983
Cynigma sp.	96-hour LC_{50}	11.1 µg/L	Peterson et al., 2001a
Calineura californica	96-hour LC_{50}	17.3 µg/L	Peterson et al., 2001a
Ameletus sp.	96-hour LC_{50}	24 µg/L	Peterson et al., 2001b
Gammarus lacustrus	96-hour LC_{50}	22 µg/L	Mayer and Ellersiek, 1986
Metapenaeus monoceros	96-hour LC_{50}	24.6 µg/L	Reddy and Rao, 1992
Gammarus fasciatus	96-hour LC_{50}	26 µg/L	Mayer and Ellersiek, 1986
Paleomenetes pugio	48-hour LC_{50}	28 µg/L	Mayer, 1987
Lepidistoma unicolor	96-hour LC_{50}	29.0 µg/L	Peterson et al., 2001b
Psyglypha sp.	96-hour LC_{50}	30.3 µg/L	Peterson et al., 2001b
Paneaus duorarum	48-hour EC_{50}	32 µg/L	Mayer, 1987
Brachycentrus americanus	96-hour LC_{50}	41.2 µg/L	Peterson et al., 2001b
Pseudochinus magellanicus	96-hour EC_{50}	92.5 µg/L	Hernandez et al., 1990
Cypridopsis vidua	48-hour EC_{50}	115 µg/L	Mayer and Ellersiek, 1986
Xanthocnemis zealandica	48-hour LC_{50}	156 ppb	Hardersen and Wratten, 2000
Aselius bravicaudus	96-hour LC_{50}	280 µg/L	Mayer and Ellersiek, 1986
Callinectes sapidus	48-hour LC_{50}	320 µg/L	Mayer, 1987
Procambarus sp.	96-hour LC_{50}	1900 µg/L	Mayer and Ellersiek, 1986
Crassostrea virginica	48-hour EC_{50}	2900 µg/L	USEPA, 2003
Corbicula striatella	96-hour LC_{50}	5100 µg/L	Jadhav et al., 1996
Mytilus edulis	96-hour LC_{50}	22,700 µg/L	CA DFG, 1998

Appendix B-3. Carbaryl acute sublethal and chronic aquatic toxicity values

Test Organism	Endpoint/Length	Toxicity Value	Reference
Oncorhynchus clarki	6-hr NOEC (predator avoidance)	200 µg/L	Labenia et al., 2007
Oncorhynchus clarki	6-hr NOEC (swimming performance)	500 µg/L	Labenia et al., 2007
Oncorhynchus mykiss	96-hour NOEC (swimming capacity)	100 µg/L	Little et al., 1990
Oncorhynchus mykiss	96-hour NOEC (swimming activity)	100 µg/L	Little et al., 1990
Oncorhynchus mykiss	96-hour NOEC (Daphnia consumed)	100 µg/L	Little et al., 1990
Cyprinodon variegatus	96-hour NOEC	1,100 µg/L	USEPA, 2003
Pimephales promelas	7-day NOEC (growth)	250 µg/L	Pickering et al., 1996
Mysidopsis bahia	96-hour NOEC	3.2 µg/L	USEPA, 2003
Pimepheles promelas	35-day NOEC (reproduction)	210 µg/L	USEPA, 2003
Gilea elegans	32-day NOEC	650 µg/L	Beyers et al., 1994
Ptychocheilus lucius	32-day NOEC	445 µg/L	Beyers et al., 1994
Daphnia magna	21 day NOEC (reproduction)	1.5 µg/L	USEPA, 2003
Chironomus riparius	28-day NOEC (emergence/development)	500 µg/L	USEPA, 2003

Appendix C

Risk Estimates for Accidental Worker Exposure for Applications (Ground and Aerial), and during Mixing and Loading

Dermal or Inhalation Doses:

Dermal Doses = (Application Rate x Area Treated Daily x Dermal Unit Exposure x Conversion Factor) / Body Weight
(lb a.i./acre x acre/day x µg/lb a.i. x 0.001 mg/µg)/kg

Inhalation Doses = (Application Rate x Area Treated Daily x Inhalation Unit Exposure x Conversion Factor) / Body Weight
(lb a.i./acre x acre/day x µg/lb a.i. x 0.001 mg/µg) / kg

Hazard Quotient (HQ) **Dose/Reference Dose (mg/kg-day)/(mg/kg-day)**
Cancer Risk **Lifetime Average Daily Dose x Q1 (mg/kg-day)/(mg/kg-day)**

Margin of Exposure (MOE) = Point of Departure (POD) ÷ Exposure Dose
Aggregate Risk Index (ARI) = 1÷ [(Dermal LOC ÷ Dermal MOE) + (Inhalation LOC ÷ Inhalation MOE)]

Note: A dermal absorption factor of 4.5% applied to the cancer calculations when estimating a dermal dose.

1) Mixing and Loading (10,000 acres per day)

Input Parameters	Upper	Unit	Sources
Application Rate			
Maximum	0.5	lb a.i./acre	USDA APHIS, 2015b, 0.5 lb a.i. per acre for APHIS conventional rate
Average	0.25	lb a.i./acre	USDA APHIS, 2015b, 0.25 lb a.i. per acre for APHIS RAATs rate
Area Treated	10,000	acre/day	Assumed mixing and loading for the program aerial application of 10,000 acres per day based on the highest recent actual acreage of 16,953 acres in 2 days applied in 2016.
Unit Exposure			
Dermal	37.6	µg/lb ai	single layer, gloves for the mixing/loading liquids exposure scenario (USEPA, 2018)
	8.6	µg/lb ai	Engineering control (closed loading system) for the mixing/loading liquids exposure scenario (USEPA, 2018)
Inhalation	0.219	µg/lb ai	No respirator for the mixing/loading liquids exposure scenario (USEPA, 2018)

	0.083	µg/lb ai	Engineering control (closed loading system) for the mixing/loading liquids exposure scenario (USEPA, 2018)
Conversion Factor	0.001	mg/µg	
Body Weight	80	kg	Body weight
Dermal absorption Factor	0.045		
Days per year of exposure	30	days	
Days per year	365	days	A commercial applicator scenario (USEPA, 2017c) is used for worker's exposure.
Years per lifetime of exposure	35	years	
Lifetime expectancy	78	years	
Dermal Dose (non-cancer)			
Maximum	2.4E+00	mg/kg-day	calculated
Average	1.2E+00	mg/kg-day	calculated
Closed system	5.4E-01	mg/kg-day	calculated
Dermal Dose (cancer)			
Maximum	1.1E-01	mg/kg-day	calculated
Average	5.3E-02	mg/kg-day	calculated
Closed system	2.4E-02	mg/kg-day	calculated
Inhalation Dose			
Maximum	1.4E-02	mg/kg-day	calculated
Average	6.8E-03	mg/kg-day	calculated
Closed system	5.2E-03	mg/kg-day	calculated
Dermal Reference Dose	0.86	mg/kg-day	An estimated human dermal POD of 86 mg/kg divided by the dermal LOC of 100 (10x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF), USEPA 2017c
Inhalation Reference Dose	0.033	mg/kg-day	An inhalation POD of 1.0 mg/kg divided by the inhalation LOC of 30 (3x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF), USEPA 2017c
Q1	8.75×10^{-4}	$(mg/kg\text{-}day)^{-1}$	
Dermal HQ			
Maximum	2.7		calculated
Average	1.4		calculated
Closed system	0.6		calculated
Inhalation HQ			
Maximum	0.4		calculated
Average	0.2		calculated

	Closed system	0.2	calculated
Combined HQ			
	Maximum	3	calculated
	Average	2	calculated
	Closed system	0.8	calculated
Dermal Lifetime Average Daily Dose			
	Maximum	3.9E-03	calculated
	Average	2.0E-03	calculated
	Closed system	8.9E-04	calculated
Inhalation Lifetime Average Daily Dose			
	Maximum	5.0E-04	calculated
	Average	2.5E-04	calculated
	Closed system	1.9E-04	calculated
Cancer Risk			
	Maximum	3.9E-06	calculated
	Average	1.9E- 06	calculated
	Closed system	9.5E-07	calculated
POD (mg/kg-day)			
	Dermal	86	USEPA, 2017c
	Inhalation	1	USEPA, 2017c
MOE (=POD/Exposure Dose)			
Dermal	Maximum	37	calculated
	Average	73	calculated
	Closed system	160	calculated
Inhalation	Maximum	73	calculated
	Average	146	calculated
	Closed system	193	calculated
ARI			
	Maximum	0.3	calculated
	Average	0.6	calculated
	Closed system	1.3	calculated

2) Ground application with a mechanical spreader (500 acres per day)

Input Parameters	Upper	Unit	Sources
Application Rate			
Maximum	0.5	lb a.i./acre	USDA APHIS, 2015b, 0.5 lb a.i. per acre for APHIS conventional rate
Average	0.2	lb a.i./acre	USDA APHIS, 2015b, 0.2 lb a.i. baits per acre for APHIS RAATs rate
Area Treated	500	acre/day	Assumed the program ground application of 500 acres per day
Unit Exposure			
Dermal	9.9	µg/lb ai	single layer, no gloves for the applicator with open cab solid broadcast spreader exposure scenario (USEPA, 2018)
Inhalation	1.2	µg/lb ai	No respirator for the applicator with open cab solid broadcast spreader exposure scenario (USEPA, 2018)
Conversion Factor	0.001	mg/µg	
Body Weight	80	kg	body weight
Dermal absorption Factor	0.045		
Days per year of exposure	30	days	A commercial applicator scenario (USEPA, 2017c) is used for worker's exposure.
Days per year	365	days	
Years per lifetime of exposure	35	years	
Lifetime expectancy	78	years	
Dermal Dose (non-cancer)			
Maximum	3.1E-02	mg/kg-day	calculated
Average	1.2E-02	mg/kg-day	calculated
Dermal Dose (cancer)			
Maximum	1.4E-03	mg/kg-day	calculated
Average	5.6E-04	mg/kg-day	calculated
Inhalation Dose			
Maximum	3.8E-03	mg/kg-day	calculated
Average	1.5E-03	mg/kg-day	calculated
Dermal Reference Dose	0.86	mg/kg-day	An estimated human dermal POD of 86 mg/kg divided by the dermal LOC of 100 (10x for interspecies extrapolation, 10x for intraspecies extrapolation,

				and 1x for FQPA SF), USEPA 2017c
Inhalation Reference Dose		0.033	mg/kg-day	An inhalation POD of 1.0 mg/kg divided by the inhalation LOC of 30 (3x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF), USEPA 2017c
Q1		8.75×10^{-4}	$(mg/kg\text{-}day)^{-1}$	
Dermal HQ				
	Maximum	0.04		calculated
	Average	0.01		calculated
Inhalation HQ				
	Maximum	0.1		calculated
	Average	0.05		calculated
Combined HQ				
	Maximum	0.1		calculated
	Average	0.06		calculated
Dermal Lifetime Average Daily Dose				
	Maximum	5E-05		calculated
	Average	2E-05		calculated
Inhalation Lifetime Average Daily Dose				
	Maximum	1E-04		calculated
	Average	6E-05		calculated
Combined Cancer Risk				
	Maximum	2E-07		calculated
	Average	7E-08		calculated
POD (mg/kg-day)				
	Dermal	86		USEPA, 2017c
	Inhalation	1		USEPA, 2017c
MOE (POD/Exposure Dose)				
Dermal	Maximum	2780		calculated
	Average	6949		calculated
Inhalation	Maximum	267		calculated
	Average	667		calculated
ARI				
	Maximum	6.7		calculated
	Average	16.8		calculated

3) Aerial application with a mechanical spreader (10,000 acres per day based on the highest recent actual acreage of 16,953 acres in 2 days applied in 2016)

Input Parameters	Upper	Unit	Sources
Application Rate			
Maximum	0.5	lb a.i./acre	USDA APHIS, 2015b, 0.5 lb a.i. per acre for APHIS conventional rate
Average	0.25	lb a.i./acre	USDA APHIS, 2015b, 0.25 lb a.i. per acre for APHIS RAATs rate
Area Treated	10,000	acre/day	Assumed the program aerial application of 10,000 acres per day based on the highest recent actual acreage of 16,953 acres in 2 days applied in 2016.
Unit Exposure			
Dermal	2.8	µg/lb ai	Aerial applicator with fixed-wing, liquid, and engineering control of enclosed cockpit exposure scenario (USEPA, 2018)
Inhalation	0.0049	µg/lb ai	Aerial applicator with fixed-wing, liquid, and engineering control of enclosed cockpit exposure scenario (USEPA, 2018)
Conversion Factor	0.001	mg/µg	
Body Weight	80	kg	Body weight
Dermal absorption Factor	0.045		
Days per year of exposure	30	days	A commercial applicator scenario (USEPA, 2017c) is used for worker's exposure.
Days per year	365	days	
Years per lifetime of exposure	35	years	
Lifetime expectancy	78	years	
Dermal Dose (non-cancer)			
Maximum	1.3E-01	mg/kg-day	calculated
Average	6.5E-02	mg/kg-day	calculated
Dermal Dose (cancer)			
Maximum	5.9E-03	mg/kg-day	calculated
Average	2.9E-03	mg/kg-day	calculated
Inhalation Dose			
Maximum	3.1E-04	mg/kg-day	calculated
Average	1.5E-04	mg/kg-day	calculated

Dermal Reference Dose		0.86	mg/kg-day	An estimated human dermal POD of 86 mg/kg divided by the dermal LOC of 100 (10x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF), USEPA 2017c
Inhalation Reference Dose		0.033	mg/kg-day	An inhalation POD of 1.0 mg/kg divided by the inhalation LOC of 30 (3x for interspecies extrapolation, 10x for intraspecies extrapolation, and 1x for FQPA SF), USEPA 2017c
Q1		8.75×10^{-4}	$(mg/kg\text{-}day)^{-1}$	
Dermal HQ				
	Maximum	0.15		calculated
	Average	0.076		calculated
Inhalation HQ				
	Maximum	0.009		calculated
	Average	0.005		calculated
Combined HQ				
	Maximum	0.2		calculated
	Average	0.08		calculated
Dermal Lifetime Average Daily Dose				
	Maximum	2E-04		calculated
	Average	1E-04		calculated
Inhalation Lifetime Average Daily Dose				
	Maximum	1E-05		calculated
	Average	6E-06		calculated
Cancer Risk				
	Maximum	2E-07		calculated
	Average	1E-07		calculated
POD (mg/kg-day)				
	Dermal	86		USEPA, 2017c
	Inhalation	1		USEPA, 2017c
MOE (POD/Exposure Dose)				
Dermal	Maximum	662		calculated
	Average	1323		calculated
Inhalation	Maximum	3265		calculated
	Average	6531		calculated
ARI				

Maximum	6.2	calculated
Average	12.5	calculated