UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
CHEMICAL SAFETY AND
POLLUTION PREVENTION

**MEMORANDUM**

**Date:** 16-DEC-2019

**SUBJECT:** **1,3-Dichloropropene (Telone)**: Draft Human Health Risk Assessment for Registration Review

**PC Code:** 029001
**Decision No.:** 556379
**Petition No.:** NA
**Risk Assessment Type:** Single chemical/Aggregate
**TXR No.:** NA
**MRID No.:** NA

**DP Barcode:** D454712
**Registration No.:** NA
**Regulatory Action:** Registration Review
**Case No.:** 328
**CAS No.:** 542-75-6
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This memorandum serves as HED's human health draft risk assessment (DRA) of the dietary, occupational, and residential exposure; and aggregate risk to support the registration review of 1,3-dichloropropene (1,3-D) also known as Telone®. The most recent quantitative human health risk assessment was performed in 2008 (*1,3-Dichloropropene: Proposed New Use for Drip Irrigation in Vineyards: Revised HED Human Health Risk Assessment*; 24-JAN-2008, C. Olinger, D347789) which provided additional characterization to the previous assessment in 2007 (*1,3-Dichloropropene: HED Human Health Risk Assessment for Phase 5*; 12-APR-2007, C. Olinger, D337328). The following risk assessment updates have been made for Registration Review:

- Updated human-equivalent concentrations (HECs) were calculated for the inhalation risk assessment.
- An updated cancer classification was incorporated into the risk assessment.
- An updated drinking water and dietary assessment was incorporated/conducted to incorporate available data.
- An updated ambient air quantitative assessment was conducted to incorporate available data including:
    - Ambient air monitoring studies conducted since 2007, and
    - Soil Fumigant Exposure Assessment (SOFEA) version 4.1.4 modeling software.
- An updated occupational exposure assessment for the registered uses was completed reflecting recent updates to the inhalation risk assessment HECs.

A summary of the findings and an assessment of human risk resulting from the registered uses of 1,3-D are provided in this document.

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## 1.0 Executive Summary

HED has conducted a human health DRA to evaluate all existing registrations of the active ingredient 1,3-dichloropropene (1,3-D), which is a non-selective soil fumigant with nematicidal properties. This assessment was conducted as part of Registration Review.

*Use Profile*
1,3-D is a soil fumigant containing approximately equal proportions of the *cis* and *trans* isomers formulated as a pressurized gas, liquid ready to use, emulsifiable concentrate, or a flowable concentrate. The liquid concentrates are intended for direct metering into drip irrigation systems, and other formulations are used in more conventional soil fumigation applications (i.e., shank/chisel/plow-sole pre-plant soil treatments). The concentrations of the active ingredients in the end-use products range from 29.2 to 97.5%. 1,3-D is registered for pre-plant and for post-plant applications to control nematodes and/or garden symphylans. Registered products for pre-plant applications may be applied by drip irrigation and soil injection, including row and broadcast applications. The only post-plant use registered is for drip irrigation to established vineyards. Several 1,3-D products also contain the fumigant chloropicrin to enhance plant pathogen and weed control. The human health risk assessment of chloropicrin is addressed in a separate document.

1,3-D is registered for pre-plant use on soils being prepared for the production of a variety of agricultural crops including vegetables, fruits, nuts, forage crops, tobacco, fiber crops, golf course turf and nursery crops. When used as a pre-plant fumigant there is no reasonable expectation of residues in food or animal feed, so tolerances are not needed for these uses. Only the post-plant use in vineyards is considered a food-use, since there is a potential for residues in foods resulting from that use. 1,3-D has no residential uses but is registered for pre-plant non-agricultural uses including golf course turf, ornamental and/or shade trees, ornamental herbaceous and non-flowering plants, and ornamental woody shrubs and vines. 1,3-D flux is well known, and the buffers/reentry intervals preclude direct exposure; therefore, the established buffers and their durations address bystander exposures for all uses where there is potential for non-occupational exposure. HED used the Biological and Economics Analysis Division (BEAD) Label Data System to identify all agricultural and non-agricultural uses of 1,3-D. For specific labeled information, see the Pesticide Label Use Summary (PLUS) Report[1].

*Exposure Profile*
Humans may be exposed to 1,3-D in food and drinking water since 1,3-D is approved for post-plant irrigation drip irrigation use in vineyards, and soil applications may result in 1,3-D reaching ground sources of drinking water. There are no uses of 1,3-D resulting in direct residential exposures; however, there is the potential for off-site/off-field bystander inhalation exposure of 1,3-D as it volatilizes off a treated area. In occupational settings, applicators may be exposed while handling the pesticide prior to application, as well as during fumigation activities, post-fumigation tasks, and direct handing tasks during application.

Based on the expected exposure pattern and physicochemical properties of 1,3-D, the Agency does not anticipate dermal exposure resulting from typical use. As a result, HED is not

[1] *(029001) PLUS – Maximum Use Scenario Report.xlsx*, 14-DEC-2018

conducting a quantitative dermal risk assessment at the present time. However, due to the acute dermal Toxicity Category II (detailed in Appendix A) handlers are required by the current labels to wear baseline attire such as loose fitting or well-ventilated long-sleeved shirt, long pants, shoes plus socks and varying levels of dermal personal-protective equipment (PPE) (e.g., chemical-resistant gloves when in contact with liquid 1,3-D).

Potential inhalation exposures from fumigant emissions, including those from 1,3-D, outside of occupational exposures can be categorized in two distinct manners: 1) acute exposures to bystanders from single applications (near field sources); and 2) short-/intermediate-term (ST/IT) exposures from many applications within a region (hereon discussed as ambient sources).

*Hazard Characterization & Dose Response Assessment*
The toxicology database is complete to support the existing uses of 1,3-D. 1,3-D showed moderate acute toxicity by the oral and dermal exposure routes (Toxicity was Category II), was moderately irritating to the eye and skin, and was a dermal sensitizer in guinea pigs. It is classified as Toxicity Category IV for acute inhalation toxicity and produced tremors, convulsions, salivation, lacrimation, diarrhea, lethargy, and death at concentrations 647 ppm or higher.

Most studies in the 1,3-D toxicological database were conducted via the inhalation route given the high volatility of 1,3-D. Consistent with the irritant properties of 1,3-D, there was evidence of degenerative changes in the nasal olfactory epithelium and histopathological changes of the respiratory epithelium in rats and mice after subchronic inhalation exposure. Histopathological findings in the non-glandular stomach and generalized systemic toxic effects were also observed. There was no evidence of increased sensitivity to the young in developmental and reproduction toxicity studies. In male mice, benign bronchioloalveolar adenomas were observed in the mouse carcinogenicity study via the inhalation route.

In oral studies, increased histopathological findings of the non-glandular stomach, increased liver weights, microcytic anemia, increased hematopoietic activity, and body weight decrements were observed. Liver adenomas were observed in the chronic/carcinogenicity study in male rats via dietary administration.

In 2019, the Cancer Assessment Review Committee (CARC) reevaluated the carcinogenic potential of 1,3-D (26-SEP-2019, G. Akerman, TXR 0057949). In accordance with the EPA's Final Guidelines for Carcinogen Risk Assessment (March 2005), the CARC classified 1,3-Dichloropene (Telone) as "Suggestive Evidence of Carcinogenic Potential" based on the presence of liver tumors by the oral route in male rats only. Given this finding, quantification of human cancer risk is not required. The CARC recommended using a non-linear approach [i.e., reference dose/concentration (RfD/RfC)] that will adequately account for all chronic toxicity including carcinogenicity, that could result from exposure to 1,3-D.

The Food Quality Protection Act (FQPA) Safety Factor (SF) can be reduced to 1X because the toxicological database is complete, there is no evidence of neurotoxicity or increased sensitivity to the young, and the exposure databases are complete or are estimated based on data that reasonably account for potential exposures.

Endpoints and PODs were only selected to evaluate chronic dietary and various durations of inhalation exposures. Since the last risk assessment, there have been no changes to the studies selected for selecting endpoints or points of departure (PODs), except to combine the ST/IT endpoint to evaluate inhalation exposures. For chronic dietary, a total uncertainty factor (UF) of 100X was applied (10X interspecies extrapolation, 10X intraspecies variability, and 1X FQPA SF). For inhalation exposures, the HECs have been recalculated using the RfC methodology for acute, ST/IT, and long-term (LT) scenarios according to current practices to account for pharmacokinetic interspecies differences. As a result, the interspecies UF can be reduced to 3X. Therefore, for inhalation exposures, a total UF of 30X was applied (3X interspecies extrapolation, 10X intraspecies variability, and 1X FQPA SF when applicable).

*Dietary Exposure Assessment*

Pre-Plant Soil Fumigant Uses

1,3-D's volatility in the environment and results of metabolism studies in soil and plants indicate that there is no reasonable expectation of finite residues to be incurred in/on any raw agricultural commodity when these products are applied according to label directions. Therefore, for pre-plant soil fumigant uses, this fumigant does not require tolerances in/on food and animal feed.

Post-Plant Drip Irrigation Use in Vineyards

There is no study with a single dose and endpoint appropriate for the acute dietary scenario. A chronic dietary-exposure and risk assessment was conducted using the Dietary Exposure Evaluation Model (DEEM-FCID, Version 3.16) which uses 2003-2008 food consumption data from the U.S. Department of Agriculture's (USDA's) National Health and Nutrition Examination Survey, What We Eat in America, (NHANES/WWEIA). The unrefined chronic analysis assumed modeled drinking water estimates provided by Environmental Fate and Effects Division (EFED), 100% crop treated (CT), and tolerance-level residues for grapes. The resulting chronic food plus drinking water exposure estimates are not of concern to HED (<100% chronic population-adjusted dose (cPAD) for all population subgroups of interest). The resulting chronic exposure estimate for all infants (<1-year-old; the subgroup with the greatest exposure) was 40% of the cPAD.

*Residential Exposure and Risk Assessment*

1,3-D is a currently classified as a restricted use pesticide and is not used in residential settings; therefore, there are no direct residential handler or post-application exposures to assess as part of Registration Review outside of the bystander assessments described below.

*Aggregate Risk Assessment*

In accordance with the FQPA, HED must consider and aggregate (add) pesticide exposures and risks from three major sources: food, drinking water, and residential exposures. There are no residential uses of 1,3-D; therefore, aggregate assessments are equivalent to the dietary risk assessments.

*Non-Occupational Spray Drift Assessment*

Based on the chemical/physical properties of 1,3-D, as well as the application methods used for 1,3-D there is no potential for spray drift to occur in accordance with HED's standard operating procedures and this exposure pathway has not been quantitatively assessed.

*Non-Occupational Bystander Volatilization Assessment*
Chemical-specific and application-specific studies have been submitted and reviewed to address volatilization from single application events from the soil fumigant uses of 1,3-D. These reviews and resulting risk estimates have been presented in previous assessments[2]. Because both the acute inhalation HEC (ST/IT and LT exposure durations are not expected for this type of exposure) and the expected exposures (e.g., application rates, use sites, etc.) have not changed since the previous assessments, the non-occupational bystander volatilization assessment has not been revisited as part of Registration Review.

*Non-Occupational Ambient Inhalation Exposure Assessment*
The potential for inhalation exposure from ambient air have been reassessed for acute, ST/IT and LT exposure durations for 1,3-D. The current assessment relies in part on monitoring data and introduces a new modeling-based approach. The monitoring data used were developed by the California Department of Pesticide Regulation (CDPR) Air Resources Board (ARB) Toxic Air Contaminant (TAC) Program, and the CDPR Air Monitoring Network (AMN). For each data source, acute air concentrations (represented as 24-hour measurements), ST/IT air concentrations (represented as a range of 4-week to 13-week rolling averages or 90$^{th}$ percentile air concentrations) and LT air concentrations (either 6-month to 1-year averages) were used as reported by CDPR. HED did not conduct an analysis of the raw data but used the values as reported by the investigators. To satisfy an outstanding Generic Data Call-In (GDCI: 029001-1397) requiring ambient air monitoring at four geographical regions including those outside of California, the Agency received one monitoring study from Dow AgroSciences LLC (DAS) conducted in Merced County, California which was used empirically to conduct a preliminary analysis of the performance of an updated version of the "Soil Fumigant Exposure Assessment" (SOFEA) ambient air modeling software to model. SOFEA was also empirically used to predict ambient air levels in the remaining three geographic regions where 1,3-D use is prevalent (Pacific Northwest, Upper Midwest, and Southeast). The validation and review of the recently updated SOFEA model is ongoing. Finally, the HED has summarized the risk estimates from the EPA National Air Toxics Assessment (NATA) which resulted in no identified risks of concern for 1,3-D at the highest reported concentration.

*Occupational Exposure and Risk Assessment*
Occupational inhalation exposures were previously evaluated using 1,3-D-specific handler monitoring data and summarized in 2007 (12-APR-2007, C. Olinger, D337328). This addressed activities like, bulk loading, mini-bulk loading, and drum loading; post-fumigation tasks such as irrigation system maintenance, rock removal, and bed shaping; and direct handling tasks during application such as fumigant rig operators, drivers, shovelers, and tarp cutters/sealers. However, occupational HECs were revised in this assessment so the 1,3-D occupational handler inhalation risk assessment was updated for Registration Review to incorporate those changes.
Occupational dermal exposures are not expected given the high vapor pressure of 1,3-D. Therefore, dermal exposures have not been quantitatively assessed. Entry restrictions into treated fields for activities such as tarp cutting, and tarp removal remain unchanged.

[2] (24-JAN-2008, C. Olinger; D347789) and (12-APR-2007, C. Olinger, D337328)

*Environmental Justice*
Potential areas of environmental justice concerns, to the extent possible, were considered in this human health risk assessment, in accordance with U.S. Executive Order 12898, "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations.[3]"

*Human Studies*
This risk assessment relies in part on data from studies in which adult human subjects were exposed to 1,3-D to determine their exposure. Appendix D provides additional information on the review of human research used to complete the risk assessment. There is no regulatory barrier to continued reliance on these studies, and all applicable requirements of EPA's Rule for the Protection of Human Subjects of Research (40 CFR Part 26) have been satisfied see Appendix D).

## 2.0 Risk Assessment Conclusions

*Dietary Exposures:*
There are no dietary risks of concern. The unrefined chronic analysis assumed 100% CT and tolerance-level residues for all commodities. The resulting chronic risk estimates are not of concern to HED. The most highly exposed population was all infants with a risk estimate of 40% of the cPAD.

*Ambient Air Exposures:*
With regard to potential exposures from ambient air, the acute risks associated with all of the monitoring stations considered are not of concern (MOEs range from 680 to 780,000, LOC = 30). Most ST/IT risks are also not of concern with two exceptions out of all sample periods collected since 2007 (greater than 2,000 total samples) (MOEs range from 27 to 8,600, LOC = 30):

- 90-day highest rolling average concentration resulting in a risk of concern (MOE = 28) in Parlier, CA in 2018, and
- 2-month highest rolling average concentration resulting in a risk of concern (MOE = 27) near Delhi, CA in 2011
    - It should be noted that in 2017, CDPR revised permit conditions which eliminated 1,3-D use in the month of December and restricted the total allotted application amount within a 6x6 square mile area to a maximum of 136,000 adjusted pounds (i.e., township cap) in a calendar year. Both CDPR imposed restrictions would be expected to refine the highest 24-hour air concentrations for this data point as the peak concentration was detected in December 11-14, 2011, where 193,138 lbs. of 1,3-D was applied in that township over the course of the year (57,138 over the future township cap).

The LT (chronic) risks from ambient air for all monitoring periods considered are not of concern (MOEs range from 52 to 2,100, LOC = 30).

[3] https://www.epa.gov/laws-regulations/summary-executive-order-12898-federal-actions-address-environmental-justice

*Occupational Exposures:*
Various levels of PPE are required for the inhalation route depending on potential for contact with liquid fumigant, application equipment/method, time since fumigations took place, and concentration levels of 1,3-D. For these scenarios occupational handlers may be required to wear National Institute for Occupational Safety and Health (NIOSH) filtering half-face respirators, engineering controls (i.e., enclosed cab equipped with a vapor adsorptive filter containing activated charcoal), self-contained breathing apparatus (SCBA) or supplied-air respirators depending on the labeled activity.

Acute risks to occupational handlers are not of concern for all sample points considered without a respirator (MOEs range from 320 to 19,000). Conversely, many ST/IT risks to handlers are of concern without a respirator (MOEs range from 1 to 380). With the addition of a PF 10 respirator (present on many labels), only one of the ST/IT handler scenarios (bulk loading for pre-plant broadcast applications) has risks which are of concern (MOEs range from 5.3 to 3,800). Results are similar if a PF 50 respirator is considered (also present on many labels), the bulk loading for pre-plant broadcast applications scenario remains of concern (MOEs range from 27 to 19,000).

## 2.1 Data Deficiencies

Although no new data deficiencies have been identified in this Registration Review memo, it is noted that there is an outstanding Data Call-in for ambient air monitoring of 1,3-D (GDCI: 029001-1397). The registrant submitted the updated SOFEA model and a study waiver in lieu of submitting a study; the waiver will be addressed when the SOFEA validation has been completed.

## 2.2 Tolerance Considerations

### 2.2.1 Enforcement Analytical Method

DAS has submitted a gas chromatography/mass spectroscopy (GC/MS) method, Method GRM 99.09.R1, for the determination of residues of *cis*-1,3-dichloropropene and *trans*-1,3-dichloropropene and metabolites in/on grape. The LOQ is 0.003 ppm for each analyte in grape. The submitted methods have undergone successful independent laboratory validation (ILV) and are acceptable for data collection and enforcement.

The multiresidue methods described in FDA PAM I are unsuitable for enforcement of 1,3-D, and its metabolites; however, a multiresidue method is available for 1,3-D[4].

### 2.2.2 Recommended & Established Tolerances

Permanent tolerances have been established in 40 CFR §180.636 residues of 1,3-D. A summary of established tolerances is summarized in Table 2.2.2.

[4] http://ejournal.cvuas.de/docs/cvuas_ejournal_201501.pdf

| Table 2.2.2. Tolerance Summary for 1,3-D (40 CFR §180.636). | | | |
|---|---|---|---|
| Commodity/ **Correct Commodity Definition** | **Established Tolerance (ppm)** | **Recommended Tolerance (ppm)** | **Comments *Correct Commodity Definition*** |
| **Grape** | 0.3 | 0.3 | |

### 2.2.3 Revisions to Established Tolerances

The tolerance expression for 1,3-D residues needs to be updated to reflect current Agency practice: "Tolerances are established for residues of 1,3-dichloro-1-propene, including its metabolites and degradates, in or on the commodities in the table below. Compliance with the tolerance levels specified below is to be determined by measuring only the sum of cis- and trans-1,3-dichloro-1-propene and its metabolites *cis*- and *trans*-3-chloro-2-propenoic acid, and *cis*- and *trans*-3-chloro-2-propen-1-ol, calculated as the stoichiometric equivalent of 1,3-dichloro-1-propene, in or on the following commodities:"

### 2.2.4 International Harmonization

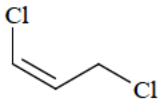
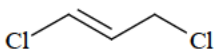
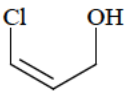
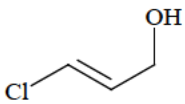
There are no established Codex maximum residue limits (MRLs) for residues of 1,3-D. Canadian MRLs are harmonized with the corresponding U.S. tolerances (Appendix G). Therefore, issues of compatibility with respect to U.S. tolerances and Canadian and Codex MRLs do not exist.

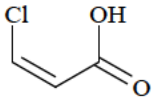
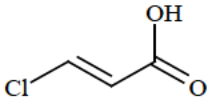
## 2.3 Label Recommendations

There are no specific label recommendations.

## 3.0 Introduction

## 3.1 Chemical Identity

| Table 3.1. Test Compound and Regulated Metabolite Nomenclature. | | |
|---|---|---|
| Compound Structure |  |  |
| Common name | cis-1,3-Dichloropropene | trans-1,3-Dichloropropene |
| Company experimental name | 1,3-D | |
| IUPAC name | (*EZ*)-1,3-Dichloropropene | |
| CAS name | 1,3-Dichloro-1-propene | |
| CAS registry number | 542-75-6 | |
| End-use product (EP) | Cordon™, Telone™ | |
| **Metabolite/Degradate** | | |
| Compound Structure |  |  |
| Common name | cis-CAAL | trans-CAAL |
| IUPAC name | (EZ)-3-Chloroprop-2-en-1-ol | |

| Table 3.1. Test Compound and Regulated Metabolite Nomenclature. | | |
|---|---|---|
| CAS name | 3-Chloro-2-propen-1-ol | |
| CAS registry number | 4643-05-4 | 4643-06-5 |
| Compound Structure |  |  |
| Common name | cis-CAAC | trans-CAAC |
| IUPAC name | (*EZ*)-3-Chloroprop-2-enoic acid | |
| CAS name | 3-Chloro-2-propenoic acid | |
| CAS registry number | 1609-93-4 | 2345-61-1 |

## 3.2 Physical/Chemical Characteristics

*Cis* and *trans* 1,3-D (MW = 110.97 g/mol) are liquids with boiling point of 104 and 112.6 °C, respectively. These have high water solubility (2 g/L) and high vapor pressure (34.3 mm Hg for *cis* isomer and 23.0 mm Hg for *trans* isomer at 25°C). More detailed physicochemical properties of 1,3-D are summarized in Appendix C.

## 3.3 Pesticide Use Pattern

1,3-D is a soil fumigant containing approximately equal proportions of the *cis* and *trans* isomers formulated as a pressurized gas, liquid ready to use, emulsifiable concentrate, or a flowable concentrate. The liquid concentrates are intended for direct metering into drip irrigation systems, and other formulations are used in more conventional soil fumigation applications (i.e., shank/chisel/plowsole pre-plant soil treatments). The concentrations of the active ingredients in the end-use products range from 29.2 to 97.5%. 1,3-D is registered for pre-plant and for post-plant applications to control nematodes and/or garden symphylans. Registered products for pre-plant applications may be applied by drip irrigation, soil injection and row and broadcast applications. The only post-plant use registered is for drip irrigation to established vineyards. Several 1,3-D products also contain the fumigant chloropicrin to enhance plant pathogen and weed control. The human health risk assessment of chloropicrin is addressed separately.

1,3-D is registered for pre-plant use on soils being prepared for the production of a variety of agricultural crops including vegetables, fruits, nuts, forage crops, tobacco, fiber crops, golf course turf, and nursery crops. Only the post-plant use in vineyards is considered a food-use, since the potential for residues in foods results from that use.

1,3-D has no residential uses but is registered for non-agricultural uses on golf course turf (pre-plant/direct to soil) where there is potential for non-occupational exposure. Other non-agricultural uses include ornamental and/or shade trees, ornamental herbaceous and non-flowering plants, and ornamental woody shrubs and vines.

1,3-D labels currently require occupational handlers to wear baseline attire defined as loose fitting long-sleeve shirt, long pants, shoes plus socks and a variety of additional dermal PPE due to the acute dermal Toxicity Category II (detailed in Appendix A) including chemical-resistant aprons, footwear, gloves, headgear, and suits, coveralls, and protective eyewear. Additional PPE

is required which varies by label/product for the inhalation route depending on potential for contact with liquid fumigant, application equipment/method, time since fumigations took place, and concentration levels of 1,3-D. For these scenarios occupational handlers may be required to wear NIOSH filtering half-face respirators, engineering controls (i.e., enclosed cab equipped with a vapor adsorptive filter containing activated charcoal), SCBA or supplied-air respirators depending on the labeled activity.

Preplant soil use in agriculture accounts for most of the use of 1,3-D. Typical use consists of making one application per year prior to planting to sterilize the soil. Individually, strawberries (35.3 %), peppers (19 %), tobacco (16.1 %), carrots (13.5 %), potatoes (11.4 %), and tomatoes (4.6 %) were the crops with the highest percentage of their overall acreage treated (%CT) according to the most recent BEAD Usage and Benefits for Soil Fumigants report[5]. Usage averaged approximately 24,435,000 pounds of 1,3-D applied annually on approximately 272,000 acres treated between 2001-2005. However, over the course of 2013 to 2017, there were on average 33,755,000 lbs of 1,3-D active ingredient applied per year corresponding to an average of approximately 320,000 acres treated per year from 2013 to 2017. Overall, growers are treating more acres using more pounds of 1,3-D than in previous years (2013-2017 vs. 2001-2005).

As part of Registration Review, HED has queried the recent years of CDPR's monitoring for 1,3-D. A query of the California Pesticide Information Portal (CALPIP) Pesticide Use Reporting (PUR) database[6] for 2017 (most recent year reported) shows that approximately 17.44 million pounds of active ingredient (1,3-D) were applied in the State of California.

A compilation of the registered uses of 1,3-D was created by BEAD and is current for the purposes of Registration Review (*(029001) PLUS – Maximum Use Scenario Report.xlsx*, 14-DEC-2018).

### 3.4 Anticipated Exposure Pathways

Humans may be exposed to 1,3-D in food as 1,3-D may be applied as a post-plant use in vineyards. Humans may also be exposed to 1,3-D through the inhalation route. There are no pesticidal residential uses of 1,3-D; however, in non-occupational settings, there is the potential for off-site inhalation exposure of 1,3-D as it volatilizes off a treated area. In an occupational setting, applicators may be exposed while handling the pesticide prior to application, as well as, during application however there is little potential for post-application exposure for workers re-entering treated fields because all activities associated with the application are considered handler related, even those which occur days later such as tarp cutting and removal.

Based on the expected exposure pattern and physicochemical properties of 1,3-D, the Agency does not anticipate dermal exposure resulting from typical use. As a result, HED is not conducting a quantitative dermal risk assessment. However, it should be noted that handlers are

[5] B. Chism *et.al.*, 21-AUG-2019; *Usage and Benefits for Soil Fumigants: Chloropicrin (081501), Dazomet (035602), 1,3-Dichloropropene (029001), Furfural (043301), Metam Sodium (039003), Metam Potassium (039002), and Methyl Bromide (053201)*

[6] https://calpip.cdpr.ca.gov/main.cfm

required by the current labels to wear baseline attire such as loose fitting or well-ventilated long-sleeved shirt, long pants, shoes plus socks and varying levels of dermal PPE (e.g., chemical-resistant gloves when in contact with liquid 1,3-D). These vary depending on potential for contact with liquid 1,3-D, application method used, time since fumigation, and potential for high concentration exposures. See 1,3-D PLUS report for additional details[7].

Releases of fumigants, such as 1,3-D, can be categorized in two distinct manners including bystander exposures from known, single application sites (i.e., treated farm fields hereon discussed as near-field sources) and exposures via ambient air where exposures could result from many applications within a region (hereon discussed as ambient sources). Because there is a potential for fumigants to move off-site following field applications, exposures to bystanders both near treated areas and farther away from treated areas (ambient air) have been quantified.

In an occupational setting, applicators may be exposed while handling the pesticide prior to application, as well as during an application; therefore, occupational handler acute and ST/IT inhalation exposures are expected and have been evaluated. Entry restrictions into treated fields for activities such as tarp cutting, and tarp removal remain unchanged from the Phase 5 RED.

Risk assessments have been previously prepared for the existing uses of 1,3-D. This risk assessment considers all the aforementioned exposure pathways based on the existing uses.

### 3.5 Consideration of Environmental Justice

Potential areas of environmental justice concerns, to the extent possible, were considered in this human health risk assessment, in accordance with U.S. Executive Order 12898, "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations," (https://www.archives.gov/files/federal-register/executive-orders/pdf/12898.pdf). As a part of every pesticide risk assessment, OPP considers a large variety of consumer subgroups according to well-established procedures. In line with OPP policy, HED estimates risks to population subgroups from pesticide exposures that are based on patterns of that subgroup's food and water consumption, and activities in and around the home that involve pesticide use in a residential setting. Extensive data on food consumption patterns are compiled by the USDA's NHANES/WWEIA and are used in pesticide risk assessments for all registered food uses of a pesticide. These data are analyzed and categorized by subgroups based on age and ethnic group. Additionally, OPP is able to assess dietary exposure to smaller, specialized subgroups and exposure assessments are performed when conditions or circumstances warrant. Whenever appropriate, non-dietary exposures based on home use of pesticide products and associated risks for adult applicators and for toddlers, youths, and adults entering or playing on treated areas post-application are evaluated. Further considerations are also currently in development as OPP has committed resources and expertise to the development of specialized software and models that consider exposure to other types of possible bystander exposures and farm workers as well as lifestyle and traditional dietary patterns among specific subgroups. This includes the extensive work conducted to capture the potential near-field bystander exposures as well as exposure from area-wide air concentrations in agricultural areas where 1,3-D is applied.

[7] *(029001) PLUS – Maximum Use Scenario Report.xlsx*, 14-DEC-2018.

## 4.0 Hazard Characterization and Dose-Response Assessment

No new toxicity studies were required for registration review (05-SEP-2013, I. Negron-Encarnacion, D410115); but the registrant submitted additional data in support of the cancer re-classification. Most of the toxicology data presented in this document were described in the risk assessment used to support the new use on grapes (24-JAN-2008, C. Olinger; D347789) and from a Scoping Document in Support of Registration Review (05-SEP-2013, I. Negron-Encarnacion, D410115).

### 4.1 Toxicology Studies Available for Analysis

The toxicity database for 1,3-D is currently considered complete, and available studies are adequate for selecting endpoints and PODs for risk assessment. The acute and 90-day neurotoxicity battery was waived, and a dermal toxicity study was not required due to the highly volatile nature of the chemical and anticipated very low dermal exposure relative to inhalation (23-APR-2013, J. Van Alstine, TXR 0056626). The available studies were mostly conducted via the inhalation route of exposure given 1,3-D is highly volatile. The following studies are available:

- Subchronic oral study in rats and mice
- 30-Day inhalation studies in mice
- 90-Day inhalation study in rats
- Developmental toxicity studies in rats and rabbits (inhalation)
- Multi-generation reproduction study in rats (inhalation)
- Chronic oral toxicity studies in dogs (microcapsule)
- National Toxicology Program (NTP) combine chronic/carcinogenicity study in rat (Fischer) & mice (B6C3F1) (oral gavage)
- Combined chronic/Carcinogenicity studies in mice (B6C3F1) and rats (Fischer) (dietary)
- Combined chronic/Carcinogenicity studies in mice (B6C3F1) and rats (Fischer) (inhalation)
- Immunotoxicity study in rats (dietary)
- Metabolism and pharmacokinetic studies in rats (oral & inhalation)
- Battery of mutagenicity studies (bacterial and mammalian model systems)
- Mechanistic studies of tumorigenicity in rats and mice

As part of registration review for 1,3-D, a broad survey of the literature was conducted to identify studies that report toxicity following exposure to 1,3-D via exposure routes relevant to human health pesticide risk assessment not accounted for in the Agency's 1,3-D toxicology database. The search strategy employed terms restricted to the name of the chemical plus any common synonyms, and common mammalian models to capture as broad a list of publications as possible for the chemical of interest. The search strategy returned 6 studies from the literature, which included chronic/carcinogenicity studies conducted by the NTP. During the title/abstract and/or full text screening of the remaining studies, none of the studies were deemed to contain potentially relevant information (either quantitative or qualitative) for the 1,3-D human health risk assessment. Appendix B has detailed information regarding the literature review.

## 4.2 Absorption, Distribution, Metabolism and Excretion (ADME)

Pharmacokinetics studies were conducted in Fischer 344 rats and B6C3F1 mice *via* the oral route. The primary route of excretion for both species was the urine. Following oral administration, most of the radiolabel was found in the stomach and gastrointestinal tract with lesser amounts in the kidneys, liver, urinary bladder, skin, fat, blood and carcass. Oral administration also depleted the non-protein-sulfhydryl contents of several tissues including the non-glandular stomach (both time- and dose-dependent). Dose-related increases in macromolecular bindings were noted in several organs with the highest binding sites being found in the non-glandular stomach. The two major urinary metabolites were identified as 1,3-DCP-mercapturic acid and its sulfoxide (or sulfone) derivative.

In another study with Fischer 344 rats, gavage administration of 1,3-D for 14 days resulted in rapid absorption from the gastrointestinal tract with distribution to all tissues examined. Highest concentrations appeared in the non-glandular stomach and urinary bladder. There was rapid elimination in the urine, as carbon dioxide in expired air, and small amounts in the feces. Nine metabolites were isolated from urine with two being identified as 1,3-D-mercapturic acid and the sulfoxide derivative. No parent compound was present in the urine.

Additional pharmacokinetics studies with inhalation exposure were also conducted; these studies were performed with the objective of providing data on mechanistic insights for tumorigenic analysis. In one study, B6C3F1 mice were administered a single, acute inhalation dose to determine the respiratory response and systemic absorption/bioavailability. The results indicated a concentration-related decrease in respiratory rate and correlated decreases in minute volume. In another acute inhalation study, F344 rats were administered 1,3-D with nose only exposure. Respiratory frequency and tidal volumes were measured. As in mice, there was a significant decrease in breathing rate with the lower resultant minute volume. In a third study, steady-state pharmacokinetics of 1,3-D in male mice was explored. In this study, male B6C3F1 mice were administered 1,3-D (vapors) with repeated nose-only exposures for 15 days. A concentration-related decrease in mean respiratory rate and in minute volume was seen. Most importantly, the data indicated a relationship between 1,3-D concentration in male mice blood and inhalation exposure was non-linear (and non-proportional) at exposure concentration levels of 40 ppm and above. This information was critical in analyzing the benign bronchiole alveolar adenomas found in the 60-ppm males (26-SEP-2019, G. Akerman, TXR 0057949).

## 4.3 Toxicological Effects

The major routes of exposure to 1,3-D are the inhalation and oral (food and drinking water) routes; however, most studies were conducted via the inhalation route given the high volatility of 1,3-D. The pattern of toxicity attributed to 1,3-D exposure via the inhalation route includes histopathology findings in the nasal cavity (e.g., degeneration of the olfactory epithelium) and non-glandular stomach, as well as generalized systemic toxic effects (body-weight, body-weight gain, and food consumption decrements). There were no reproductive effects in a two-generation reproduction study in rats via inhalation route. No developmental effects were found in the rabbit developmental toxicity study via inhalation route. In a developmental toxicity study in rats via inhalation, a decrease of ossification of the vertebral was observed at a relative high concentration (120 ppm). No quantitative/qualitative susceptibility was found with *in-utero* or

post-natal exposure. In male mice, benign bronchioloalveolar adenomas were observed in the mouse carcinogenicity study via inhalation (see Section 4.5.3).

In oral studies, increased histopathological findings of the non-glandular stomach, increased liver weights, microcytic anemia, increased hematopoietic activity, and body weight decrements were observed. Liver adenomas were observed in the chronic/carcinogenicity study in male rats via dietary administration (see Section 4.5.3).

1,3-D showed moderate acute toxicity by the oral and dermal exposure routes (Toxicity was Category II), was moderately irritating to the eye and skin, and was a dermal sensitizer in guinea pigs. It is classified as Toxicity Category IV for acute inhalation toxicity and produced tremors, convulsions, salivation, lacrimation, diarrhea, lethargy and death at concentrations 647 ppm (approximately 3 mg/L) or higher.

In addition to the parent compound (1,3-D), two degradates were identified, 3- chloroallyl alcohol and 3- chloroacrylic acid. These degradates are assumed to have toxicity equal to the parent compound. Consequently, the risk assessment for the parent compound will be protective of the potential toxic effects elicited by the two degradates.

## 4.4 Safety Factor for Infants and Children (FQPA SF)[8]

Based on the hazard and exposure data, the 1,3-D risk assessment team has recommended that the FQPA SF be reduced to 1X. There is a complete toxicity database for 1,3-D. There is no evidence of susceptibility following *in utero* and/or postnatal exposure in the developmental inhalation toxicity studies in rats or rabbits, and in the two-generation inhalation rat reproduction study. There is no neurotoxicity concern. Finally, the exposure data are complete or are estimated based on data that reasonably account for potential exposures.

### 4.4.1 Completeness of the Toxicology Database

The toxicology database is complete and sufficient for assessing susceptibility to infants and children as required by FQPA. Developmental toxicity studies in rats and rabbits and a reproduction toxicity study in rats are available for 1,3-D.

### 4.4.2 Evidence of Neurotoxicity

There was limited evidence of neurotoxicity and neurobehavioral effects following repeat oral and inhalation exposure in the 1, 3-D toxicity database. The apparent neurotoxic effects were observed following acute inhalation exposures to high doses only and are likely a result of irritation due to the corrosive nature of 1, 3-D. The acute and 90-day neurotoxicity battery was waived for 1,3-D (23-APR-2013, J. Van Alstine, TXR 0056626).

[8] HED's standard toxicological, exposure, and risk assessment approaches are consistent with the requirements of EPA's children's environmental health policy (https://www.epa.gov/children/epas-policy-evaluating-risk-children).

### 4.4.3 Evidence of Sensitivity/Susceptibility in the Developing or Young Animal

There was no evidence for increased quantitative and/or qualitative susceptibility after *in utero* or postnatal exposure to 1,3-D in developmental toxicity studies in rats and rabbits, or a reproduction study in rats.

### 4.4.4 Residual Uncertainty in the Exposure Database

There are no residual uncertainties in the exposure database: 1) the dietary assessment is based on partially-refined assumptions including 100% crop treated, anticipated residues in foods taking into account residue dissipation, and drinking water values from groundwater monitoring studies; 2) the non-occupational bystander and ambient assessments are based on monitoring data and conservative models; and 3) there are no residential uses of 1,3-D.

## 4.5 Toxicity Endpoint and Point of Departure (POD) Selection

The toxicity endpoints have not changed since the last risk assessment for 1,3-D except one endpoint was selected to evaluate occupational ST/IT inhalation. Additionally, human equivalent concentrations (HECs) were recalculated using current practices. The cancer classification has been reconsidered after submission of additional toxicity, genotoxicity, and ADME studies.

**Acute Dietary Exposure (all populations)**

No appropriate endpoint attributable to a single exposure (dose) was identified from oral toxicity studies.

**Chronic Dietary Exposure**

The chronic toxicity endpoint and POD were selected from the **c**ombined chronic toxicity/carcinogenicity study in B6C3F1 mice following oral administration (MRID 43757901). A NOAEL of 2.5 mg/kg/day was selected as the POD and the toxicity endpoint was based on decreased body weight and increased incidence of basal cell hyperplasia of non-glandular stomach mucosa at 12.5 mg/kg/day. The study is appropriate for the route and duration of exposure. A total UF of 100X was applied (10X interspecies extrapolate, 10X intraspecies variability, and 1X FQPA SF) to derive the cPAD of 0.025 mg/kg/day.

**Incidental Oral Exposure (short- and intermediate-term)**

1,3-D is not used in residential settings, so incidental oral exposures are not expected. Also, the volatile nature of 1,3-D reduces the potential for significant residues of 1,3-D and its degradates in the upper layer of soil that may be consumed by young children.

**Dermal Exposure (short-, intermediate- and long-term)**

Dermal toxicity studies have not been submitted for 1,3-D. The dermal toxicity study was not required due to the highly volatile nature of the chemical and anticipated very low dermal

exposure relative to inhalation (23-APR-2013, J. Van Alstine, TXR 0056626). Since 1,3-D is formulated as a liquid there is some potential for dermal and eye contact. The use of mitigation controls such as PPE and closed transfer systems minimizes the potential but does not eliminate it. Although 1,3-D may be irritating to the skin and eyes, no dermal endpoints of concern were selected for risk assessment purposes. PPE for dermal protection should be based on the acute toxicity of the end-use product as described in the Worker Protection Standard and mitigation measures for dermal exposure (i.e. PR Notice 93-7, 1995 label amendments, 30-SEP-1998 agreement with Dow).

**Inhalation Exposure (acute, short-, intermediate- and long-term)**

The critical effects of 1,3-D exposure via the inhalation route are clinical signs and decreased body weight for acute exposures, and histopathological lesions in the olfactory region of the nasal cavity for longer term exposures. Inhalation endpoints have been selected for acute, ST/IT, and LT durations. HECs were then calculated using the RfC methodology[9], which accounts for pharmacokinetic (not pharmacodynamic) interspecies differences. As a result, the interspecies UF is reduced from 10X to 3X. The level of concern (LOC) for all durations is 30X (3X interspecies extrapolation, 10X intraspecies variability, and 1X FQPA when applicable). Due to differences between anticipated human exposures, HECs were calculated for occupational, residential indoor post-application, and residential bystander scenarios (see Tables 4.5.3.3 – 4.5.3.5). The toxicity endpoints selected for inhalation risk assessment are presented below.

**a. Acute Inhalation Exposure**

For the acute inhalation scenario, two acute inhalation rat studies were selected for establishing the toxicity endpoints for risk assessment. In one acute inhalation study (MRID 00032985), mortality was 30% or greater at doses ≥647 ppm following 4 hours of exposure (whole body). Clinical signs (tremors, convulsions, salivation, lacrimation, diarrhea, lethargy) were also observed at these doses. In a second study (MRID 41672201), decreased body weights were observed at the lowest dose tested (583 ppm) following 4 hours of exposure (whole body); however, no animals died at this dose. Increased mortality was observed at ≥ 771 ppm. A no observed adverse effect concentration (NOAEC) of 454 ppm was selected based on the results of both studies. The studies are appropriate for the route and durations of exposure. Since the effects are considered systemic, the regional gas dose ratio (RGDR) used for HEC calculation is 1. Daily duration adjustments were applied from the rat inhalation study (4 hours) to anticipated human exposures.

**a. Short- and Intermediate-Term Inhalation Exposure**

For both ST/IT exposures, a 13-Week inhalation toxicity in rats was selected with a NOAEC of 10 ppm. Histopathological lesions in the nasal turbinates were observed at the LOAEC of 30 ppm. The study is appropriate for the route and duration of exposure. The study is also protective of effects observed in the rat developmental toxicity study via the inhalation route at considerably higher concentrations. The RGDR applied for HEC calculation is 0.168 based on

[9] USEPA. (1994). Methods for derivation of inhalation reference concentrations and applications of inhalation dosimetry.

the extrathoracic effects. Duration adjustments (daily and weekly) were applied from the rat inhalation study (6 hours/day, 5 days/week) to anticipated human exposures.

#### a. Long-Term Inhalation Exposure

For LT inhalation exposure assessments, the mouse chronic toxicity/carcinogenicity study via inhalation route of administration was selected with a NOAEC of 5 ppm. Nasal histopathological findings (hypertrophy/hyperplasia of the nasal respiratory mucosa) were observed at the LOAEC of 20 ppm. The study is appropriate for the route and duration of exposure. The RGDR applied for HEC calculation is 0.03 based on the extrathoracic effects. Duration adjustments (daily and weekly) were applied from the rat inhalation study (6 hours/day, 5 days/week) to anticipated human exposures.

### 4.5.1 Recommendation for Combining Routes of Exposure for Risk Assessment

A dermal endpoint was not selected. The oral and inhalation exposures should not be combined since the points of departure are based on different adverse effects.

### 4.5.2 Cancer Classification and Risk Assessment Recommendation

In 2019, the CARC evaluated the carcinogenic potential of 1,3-D (26-SEP-2019, G. Akerman, TXR 0057949). In total, six carcinogenicity studies were considered in this evaluation: oral gavage studies in the mouse and rat, oral dietary studies in the mouse and rat, and inhalation studies in the mouse and rat. The NTP rodent cancer bioassays conducted with 1,3-D containing epichlorohydrin are not included in this evaluation. The CARC concluded that the interpretation of the findings in those studies are confounded by the presence of a known mutagen and determined that the database of studies conducted with 1,3-D without epichlorohydrin was adequate and appropriate for assessing the carcinogenic potential of 1,3-D.

The CARC considered the following in its weight-of-evidence determination of the carcinogenic potential of 1,3-D:

**Rats**

With dietary administration, a statistically significant ($p<0.05$) increase in hepatocellular adenomas was observed at the high dose (25 mg/kg/day) in F344 male rats. Although there was no clear evidence of supporting non-neoplastic lesions, the incidence of these benign tumors at the high dose exceeded the historical control range. The doses tested were considered to be adequate and not excessive for assessing the carcinogenic potential of 1,3-D in both sexes. The CARC concluded that the liver adenomas are treatment-related. There were no treatment-related tumors in female rats in the dietary study.

In an oral (gavage) combined chronic/carcinogenicity study in Sprague Dawley (SD) rats, CARC determined that although the animals could have tolerated higher doses, the committee concluded that the high dose (25 mg/kg/day) was adequate to assess carcinogenicity. There were no treatment-related tumors in male or female rats in this study.

In a combined chronic/carcinogenicity study in F343 rats with inhalation exposure, CARC

determined that the concentrations tested were adequate and not excessive for assessing carcinogenicity. There were no treatment-related tumors observed in male or female F343 rats via the inhalation route.

**Mice**

In a 2-year dietary carcinogenicity study in B6C3F1 mice, CARC concluded that the animals likely could have tolerated a higher dose; however, the committee concluded that the doses evaluated are adequate to access the carcinogenic potential of 1,3-D in mice. There were no treatment-related tumors in male or female mice.

In an oral gavage carcinogenicity study in CD-1 mice, the CARC determined the doses tested to be adequate and not excessive. There were no treatment-related tumors seen in male or female mice.

In an inhalation carcinogenicity study in B6C3F1mice, there was a statistically significant trend ($p<0.001$) and pairwise ($p<0.05$) increase in benign bronchioloalveolar adenomas in male mice at the highest test concentration (60 ppm). The CARC considered the concentrations tested to be adequate and not excessive based on toxicological endpoints; however, the registrant conducted a toxicokinetic study evaluating the relationship between 1,3-D concentration in male mice blood and inhalation exposure. These data indicated that the relationship was non-linear at exposure levels of 40 ppm and above. Based on these findings, the CARC concluded that there were no treatment-related tumors in male mice at doses below the kinetically derived maximum tolerated dose/concentration (KMD/KMC). There was no evidence of carcinogenicity in female mice.

Based on a weight-of-evidence of the available genotoxicity studies, the CARC concluded that there is low concern for mutagenicity in vivo.

**In accordance with the EPA's Final Guidelines for Carcinogen Risk Assessment (March, 2005), the CARC classified 1,3-Dichloropene (Telone) as "Suggestive Evidence of Carcinogenic Potential" based on the presence of liver tumors by the oral route in male rats only.**

Quantification of human cancer risk is not required. The CARC recommends using a non-linear approach [i.e., RfD/RfC] that will adequately account for all chronic toxicity including carcinogenicity, that could result from exposure to 1,3-D.

### 4.5.3 Summary of Points of Departure and Toxicity Endpoints Used in Human Risk Assessment

**Table 4.5.3.1. Summary of Toxicological Dose and Endpoints for Use in 1,3-D Dietary Risk Assessments.**

| Exposure/ Scenario | POD | Uncertainty/ FQPA Safety Factors | LOC for Risk Assessment RfD, PAD | Study and Toxicological Effects |
|---|---|---|---|---|
| Acute Dietary Exposure (any Subpopulation) | No endpoint attributable to a single oral exposure was identified. | | | |

| Table 4.5.3.1. Summary of Toxicological Dose and Endpoints for Use in 1,3-D Dietary Risk Assessments. | | | | |
|---|---|---|---|---|
| **Exposure/ Scenario** | **POD** | **Uncertainty/ FQPA Safety Factors** | **LOC for Risk Assessment RfD, PAD** | **Study and Toxicological Effects** |
| Chronic Dietary Exposure | NOAEL = 2.5 mg/kg/day | $UF_A$ = 10X<br>$UF_H$ = 10X<br>FQPA SF = 1X | Chronic PAD = 0.025 mg/kg/day | 2-year combined chronic toxicity/ carcinogenicity study (rat)<br>LOAEL = 12.5 mg/kg/day based on decreased body weight and increased incidence of basal cell hyperplasia of nonglandular stomach mucosa |
| Cancer (oral) | CARC classified 1,3-D as "Suggestive Evidence of Carcinogenic Potential" based on the presence of liver tumors by the oral route in male rats only and recommended using a non-linear approach [i.e., RfD] that would adequately account for all chronic toxicity including carcinogenicity, that could result from exposure to 1,3-D (26-SEP-2019, G. Akerman, TXR 0057949). | | | |

Point of departure (POD) = A data point or an estimated point that is derived from observed dose-response data and used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures. LOAEL = lowest-observed adverse-effect level. NOAEL = no-observed adverse-effect level. LOC = level of concern. UF = uncertainty factor. $UF_A$ = extrapolation from animal to human (interspecies). $UF_H$ = potential variation in sensitivity among members of the human population (intraspecies). FQPA SF = FQPA Safety Factor. cPAD = chronic population-adjusted dose.

| Table 4.5.3.2. Summary of Toxicological Dose and Endpoints for Use in 1,3-D Human Health Inhalation Risk Assessment (Non-Occupational and Occupational). | | | | |
|---|---|---|---|---|
| **Exposure Scenario** | **POD** | **Uncertainty/ FQPA Safety Factors** | **LOC** | **Study and Toxicological Effects** |
| Acute Inhalation | NOAEC = 454 ppm | $UF_H$ = 10X<br>$UF_A$ = 3X<br>FQPA = 1X when applicable | LOC=30 | Acute (4-hour) inhalation studies (rat)<br>LOAEC = 583 ppm, based on clinical signs (tremors, convulsions, salivation, lacrimation, diarrhea, lethargy) and decreased body weight (mortality observed at ≥647 ppm) |
| Short- (1-30 day) Intermediate -Term Inhalation (1-6 months) | NOAEC = 10 ppm | $UF_H$ = 10X<br>$UF_A$ = 3X<br>FQPA = 1X when applicable | LOC = 30 | 13-week inhalation study (rat)<br>LOAEC = 30 ppm, based on nasal histopathology |
| Long-Term Inhalation (>6 months) | NOAEC = 5 ppm | $UF_H$ = 10X<br>$UF_A$ = 3X<br>FQPA = 1X when applicable | LOC =30 | Chronic toxicity/carcinogenicity study (mouse)<br>LOAEC = 20 ppm, based on nasal histopathology |
| Cancer (inhalation) | The Cancer Assessment Review Committee (CARC) classified 1,3-D as "Suggestive Evidence of Carcinogenic Potential" based on the presence of liver tumors by the oral route in male rats only and recommended using a non-linear approach [i.e., RfD] that would adequately account for all chronic toxicity including carcinogenicity, that could result from exposure to 1,3-D (26-SEP-2019, G. Akerman, TXR 0057949). | | | |

UF = uncertainty factor; NOAEC = no observed adverse effect concentration; LOAEC = lowest observed adverse effect concentration; $UF_A$ = extrapolation from animal to human (interspecies). $UF_H$ = potential variation in sensitivity among members of the human population (intraspecies). NA = Not Applicable

## Summary of Updated Inhalation HECs

| Table 4.5.3.3. Acute Inhalation HECs. | | | | | |
|---|---|---|---|---|---|
| **Population** | **Scenario** | **Toxicity duration adjustment** | | **HEC** | |
| | | **Daily** | **Weekly** | **mg/L** | **mg/m3** |
| Occupational | Handler | 0.5 | 1.00 | 1.030 | 1030 |
| Residential | Bystander | 0.167 | 1.00 | 0.343 | 343 |

NOAEC = 2.06 mg/L. RGDR = 1 for systemic effects. Daily duration adjustment from 4 hours from acute inhalation toxicity studies to anticipated human exposure duration (occupational handler = 8 hours, residential post-application=16 or 18 hours, and 24 hours for residential bystander). No weekly adjustments made from acute inhalation studies.

| Table 4.5.3.4. Short-/Intermediate-Term Inhalation HECs. | | | | | |
|---|---|---|---|---|---|
| **Population** | **Scenario** | **Toxicity duration adjustment** | | **HEC** | |
| | | **Daily** | **Weekly** | **mg/L** | **mg/m3** |
| Occupational | Handler | 0.75 | 1 | 0.006 | 5.68 |
| Residential | Bystander | 0.25 | 0.71 | 0.001 | 1.35 |

NOAEC = 0.045 mg/L. RGDR = 0.168 for extrathoracic effects (using 0.241 kg body weight based on average body weights observed for both sexes in 13-week inhalation toxicity study with F344 rats). Daily duration adjustment from 6 hours/day from 13-week inhalation toxicity study to anticipated human exposure duration (occupational handler = 8 hours, residential post-application=16 or 18 hours, and 24 hours for residential bystander). Weekly adjustment from 5 days/week from 13-week inhalation study to anticipated human exposure duration (occupational handler = 5 days/week, residential = 7 days/week).

| Table 4.5.3.5. LT Inhalation HECs. | | | | | |
|---|---|---|---|---|---|
| **Population** | **Scenario** | **Toxicity duration adjustment** | | **HEC** | |
| | | **Daily** | **Weekly** | **mg/L** | **mg/m3** |
| Occupational | Handler | 0.75 | 1 | 0.003 | 2.91 |
| Residential | Bystander | 0.25 | 0.71 | 0.001 | 0.69 |

NOAEC = 0.023 mg/L. RGDR = 0.168 for extrathoracic effects (using 0.03 kg body weight based on average body weights observed for both sexes in the mouse chronic/carcinogenicity study with B6C3F1 mice). Daily duration adjustment from 6 hours/day from mouse chronic/carcinogenicity study to anticipated human exposure duration (occupational handler = 8 hours, residential post-application=16 or 18 hours, and 24 hours for residential bystander). Weekly adjustment from 5 days/week from mouse chronic/carcinogenicity study to anticipated human exposure duration (occupational handler = 5 days/week, residential = 7 days/week).

# 5.0 Dietary Exposure and Risk Assessment

## 5.1 Residues of Concern Summary and Rationale

The qualitative nature of the residue in plants is adequately understood based on soybean, tomato, and sugar beet metabolism studies with 1,3-D as a pre-plant soil fumigant. In studies

with tomatoes and soybean, no parent or metabolites were detected, and incorporation into natural plant constituents was demonstrated. In the study with sugar beets, parent and metabolites were also not detected, and the parent compound was shown to have been metabolized and incorporated into sucrose. An acceptable confined rotational crop study was conducted with wheat, lettuce, carrots, and radishes. The results were in agreement with those from primary plant metabolism studies, showing extensive incorporation of radiolabeled residues into natural plant biochemical constituents. No plant-back restriction is required. There are no livestock feed items associated with 1,3-D; therefore, metabolism livestock is not relevant.

The *cis* and *trans* isomers of CAAC and CAAL are environmental degradates and exaggerated rate residue trials conducted in grapes showed very low residues of these degradates when 1,3-D is applied during the growing season. The acute and subchronic toxicity studies for CAAC and CAAL indicate that the toxicity of the degradates is within the same order of magnitude as the parent compound and generally exhibit similar effects at high doses. For these reasons, the degradates are included in the chronic dietary exposure and risk assessments and assumed to have equal toxicity to parent 1,3-D.

**Table 5.1. Summary of Metabolites and Degradates to be Included in the Risk Assessment and Tolerance Expression.**

| Matrix | | Residues Included in Risk Assessment | Residues Included in Tolerance Expression |
|---|---|---|---|
| Plants | Primary Crop | *cis* and *trans* isomers of the parent, 3-chloroallyl alcohol, and 3-chloroacrylic acid | *cis* and *trans* isomers of the parent, 3-chloroallyl alcohol, and 3-chloroacrylic acid. |
| | Rotational Crop | Not Applicable | Not Applicable |
| Livestock | Ruminant | Not Applicable | Not Applicable |
| | Poultry | Not Applicable | Not Applicable |
| Drinking Water | | *cis* and *trans* isomers of the parent, 3-chloroallyl alcohol, and 3-chloroacrylic acid | Not Applicable |

### 5.2 Food Residue Profile

1,3-D is registered for pre-plant use on soils being prepared for the production of a variety of agricultural crops including vegetables, fruits, nuts, forage crops, tobacco, fiber crops, golf course turf and nursery crops. Only the post-plant use in vineyards is considered a food-use, since the potential for residues in foods results from that use. Thirteen grape field trials were conducted at ~5X exaggerated rates. Most of the pre-harvest intervals ranged from 6-30 days, which is considerably shorter than the labeled 60-day pre-harvest interval (PHI). The analytical methods used were appropriate for the parent and metabolites and showed good recoveries. The residue data are supported by adequate storage stability data. Residues of the parent (*cis* and *trans* isomers) were non-detectable (at an LOD of approximately 0.9 ppb) in all trials with a pre-harvest interval exceeding 21 days. Residues of the metabolites CAAC and CAAL were generally non-detectable at most sites at all pre-harvest intervals with the exception of one trial in Washington and one trial in California. HED does not expect quantifiable residues when 1,3-

D is used in accordance with the use directions; the tolerance level is set at the combined limits of quantitation for all of the residues of concern (0.018 ppm). The residue data show that the actual residues are likely to be considerably lower. Processing studies are not available. However, none are required as exaggerated rate data showed that is unlikely that residues at the proposed PHI will be detectable. Due to the volatile nature of the residues of concern, residues are likely to dissipate during processing. However, should residues concentrate during processing, they will be below the recommended tolerance.

### 5.3 Water Residue Profile

Unlike the majority of the soil fumigants, 1,3-D underwent a chemical-specific drinking water assessment for Registration Review (Memo, 10-OCT-2019, A. Shelby *et al.*, D454589). Due to the fumigants' volatile nature (i.e., vapor pressure, Henry's Law Constant), the methods used for their application, and the use of tarps, water seals, and buffer zones, implemented since the last drinking water assessments were conducted, to reduce exposure to volatile emissions as well as transport of any residues in runoff, the potential for fumigants to be present in runoff from a treated field, or in surface waters that serve as sources of drinking water, is expected to be very low.

EFED has conducted a refined drinking water assessment (DWA) that assumes typical application rates, one application every three years, and model parameterization that is heavily reliant upon a single prospective groundwater (PGW) monitoring study. The total-toxic residue (TTR) approach was used for this assessment that combines the expected exposures from parent 1,3-D and its degradates of toxic concern, identified as CAAC and CAAL. Incorporating a 100-foot buffer to drinking water sources which is currently required by product labels, EFED recommends that 257 ppb be used as the peak estimated drinking water concentration (EDWC) and 184 ppb be used as the chronic EDWC (Table 5.3). EDWCs are estimated using Pesticides in Water Calculator (PWC) Version 1.52. Water residues were incorporated in the DEEM-FCID into the food categories "water, direct, all sources" and "water, indirect, all sources."

**Table 5.3. EDWCs for 1,3-D.**

| Type of Exposure and Estimate Source | Highest Peak EDWC (ppb) | Highest Chronic EDWC (ppb) |
|---|---|---|
| Ground water -100 feet off-site – modeled | 257[1,2] | 184[1,2] |
| Ground water - 200 feet off-site - Wisconsin PGW | 36 | 7.4 |
| Surface water –PRZM-EXAMS | 65[3] | 0.59[3] |

[1] Wisconsin Corn groundwater scenario at potato typical rate of 145 lbs ai/A.
[2] Assumes typical lateral flow velocity of 0.2 ft/day.
[3] North Carolina Sweet Potato surface water scenario at potato typical rate of 145 lbs ai/A. The 1-in-10-year annual average for surface water is 1.3 ppb. Idaho potato scenario produced lower EDWCs.

### 5.4 Dietary Risk Assessment

There is no study with a single dose and endpoint appropriate for the acute scenario and a non-linear approach (i.e., RfD) adequately accounts for all chronic toxicity including carcinogenicity, that could result from exposure to 1,3-D. Therefore, acute and cancer risk assessments were not performed.

An unrefined chronic analysis was performed using DEEM-FCID (ver. 3.16) which estimates the dietary exposure of the U.S. population and various population subgroups. The results reported are for the general U.S. population, all infants (<1 year old), children 1-2 years old, children 3-5 years old, children 6-12 years old, youth 13-19 years old, females 13-49 years old, adults 20-49 years old, and adults 50-99 years old.

The unrefined chronic analysis assumed 100% CT and tolerance-level residues for all commodities. The resulting chronic risk estimates are not of concern to HED. The most highly exposed population was infants with a risk estimate of 40% of the cPAD. The majority of the exposure is from drinking water. See Table 5.4 below for a summary of the results of the dietary assessment.

**Table 5.4. Chronic Dietary (Food and Drinking Water) Exposure and Risk for 1,3-D.**

| Population Subgroup | Chronic Dietary | |
|---|---|---|
| | Dietary Exposure (mg/kg/day) | % cPAD* |
| General U.S. Population | 0.003863 | 16 |
| All Infants (<1 year old)* | **0.009955** | **40** |
| Children 1-2 years old | 0.005616 | 22 |
| Children 3-5 years old | 0.004717 | 19 |
| Children 6-12 years old | 0.003391 | 14 |
| Youth 13-19 years old | 0.002814 | 11 |
| Adults 20-49 years old | 0.003851 | 15 |
| Adults 50-99 years old | 0.003808 | 15 |
| Females 13-49 years old | 0.003837 | 15 |

*The subpopulation(s) with the highest risk estimates.

## 6.0 Residential Exposure/Risk Characterization

1,3-D (Telone) is currently classified as a Restricted Use Pesticide[10] and is not used in residential settings; therefore, residential handler and post-application exposures are not expected and have not been quantitatively assessed.

## 7.0 Aggregate Exposure/Risk Characterization

In accordance with the FQPA, HED must consider and aggregate (add) pesticide exposures and risks from three major sources: food, drinking water, and residential exposures. In an aggregate assessment, exposures from relevant sources are added together and compared to quantitative estimates of hazard (e.g., a NOAEL or PAD), or the risks themselves can be aggregated. When aggregating exposures and risks from various sources, HED considers both the route and duration of exposure. There are no residential uses of 1,3-D; therefore, the aggregate assessments are equivalent to the dietary risk assessments (see Section 5.4). Furthermore, the inhalation and oral points of departure are based on different adverse effects so it would be inappropriate to aggregate. Non-occupational and ambient exposures are not typically aggregated with dietary exposures because the former is isolated and sporadic in nature, and the likelihood of having a significant food exposure occurring concurrently with a significant non-occupational exposure is negligible.

[10] https://www.epa.gov/sites/production/files/2019-10/documents/rup-report-oct2019.pdf

## 8.0 Non-Occupational Spray Drift Exposure and Risk Estimates

A spray drift assessment was not completed for 1,3-D. The application practices for 1,3-D are not reflected in the standard spray drift assessment as outlined in the Residential SOP Addenda 1: *Consideration of Spray Drift*. Due to the high vapor pressure and physical state of 1,3-D, there is negligible likelihood that residues (i.e., sprays) would be present to drift onto nearby or adjacent areas. Therefore, spray drift exposures have not been quantitatively assessed. However, non-occupational bystander inhalation exposures are expected, and an assessment was completed using the most appropriate methodology to assess the off-site and off-field transport of 1,3-D application related exposures. See Section 9 below for additional information.

## 9.0 Non-Occupational Bystander Post-Application Inhalation Exposure and Risk Estimates

A quantitative non-occupational bystander volatilization assessment has been conducted for 1,3-D. Because of the potential for fumigants to move off-site following field applications, exposure to bystanders near specific treated areas and to people through ambient air has been quantified based on application specific data as well as area-wide monitoring measurements, when available.

Non-occupational bystander exposure to 1,3-D depends on two main factors: (1) the rate at which 1,3-D comes off of a treated field (described as the emission or flux) and (2) how those resulting 1,3-D emissions are dispersed in the air over and around the field. Emission rates are affected primarily by the amount of 1,3-D applied, the application methods and equipment (i.e., drip irrigation and soil injection, including row and broadcast applications, etc.), and the sealing technologies (i.e., un-tarped, type of tarp used, water sealing, etc.). Additional details on tarps, buffer zones are available on the Agency website[11]. Also, there are a variety of soil factors that can potentially have an effect on the magnitude of the concentration of fumigant coming off of a treated field. Some of these include soil type, soil moisture, soil temperature, and organic content of the soil.

In the years following the Phase 5 RED (12-APR-2007, C. Olinger, D337328), the Agency has worked with the grower community and the registrant to implement various emissions reduction technologies. Some of these approaches are outlined in *Updated Health Effects Division Recommendations for Good Agricultural Practices and Associated Buffer Credits* (Memo, 14-MAY-2009, C. Smith, D362369) and *Second Update to Health Effects Division Recommendations for Good Agricultural Practices and Associated Buffer Credits* (Memo, J. Dawson, 11-JAN-2011, D385314). There are a number of 1,3-D specific studies that have been previously used to quantitatively determine flux and the associated bystander inhalation assessment which are relied upon as part of registration review.

To address near-field exposures to 1,3-D associated with specific applications, the registrants has submitted application and chemical-specific flux data that have been reviewed by HED. Much of these data were reviewed as part of the Phase 5 RED (12-APR-2007, C. Olinger, D337328). Since the completion of the Phase 5 Red, HED has reviewed additional studies and evaluated

[11] https://www.epa.gov/soil-fumigants/tarps

proposed label amendments based on these additional studies. This information is discussed below in Section 9.1*:* Non-Occupational Bystander Exposure and Risk Estimates from Near Field Sources.

To address the potential for exposure from ambient air to 1,3-D, the Agency required ambient monitoring data to quantify potential exposures of 1,3-D from multi/non-point sources over time. These concentrations are intended to represent anticipated exposures of 1,3-D in high use areas for periods up to a year. Along with the available monitoring data generated by the registrants, the assessment also used existing monitoring data for 1,3-D from several publicly available data sources (i.e., CDPR ARB, TAC Program, and AMN)[12], and the Soil Fumigant Exposure Assessment (SOFEA) version 4.1.4 modeling software which was submitted to the Agency to satisfy the ambient air Generic Data Call-In in lieu of generating monitoring data for all required sampling locations (GDCI: 029001-1397). These data and the modeling approaches are discussed below in Section 9.2*:* Non-Occupational Ambient Exposure and Risk Estimates from Near Field Sources. The Agency has also included risk estimates from EPA 2018 National Air Toxics Assessment (NATA)[13] in Section 9.3. NATA is EPA's ongoing review of air toxics developed as a screening tool for state, local, and tribal air agencies to help identify which pollutants, emissions sources, and places may need further study to better understand any possible risks to public health from air toxics.

## 9.1 Non-occupational Bystander Exposure and Risk Estimates from Near Field Sources

The *1,3-Dichloropropene: HED Human Health Risk Assessment for Phase 5* (12-APR-2007, C. Olinger, D337328) first established the methodology by which near-field non-occupational bystander exposures of 1,3-D would be quantitatively assessed (*Phase 5 Section 6.1.1)*. The 2007 document quantitatively assessed these exposures based on a series of flux (i.e., field volatility) studies and the use of the Probabilistic Exposure and Risk model for Fumigants (PERFUM) to calculate distances at which target concentrations are achieved at varied percentiles of exposure. The distances determined based on a target concentration defined by the inhalation HEC adjusted by an UF may be used as a basis for determining "buffer zones" to establish for reducing potential risk.

Following this assessment, the registrant submitted additional studies that represented additional cultural practices of the grower community as well as the emissions reduction practices and technologies for 1,3-D referenced above in Section 9.0. A compilation of the available chemical and application specific studies reviewed by the Agency is provided in Appendix E. The Agency has reviewed and incorporated these data as they were submitted to the Agency using the same practices and assumptions as outlined in the Phase 5 RED (2007). There have been no changes to the toxicological PODs relevant to the near field bystander exposures or to the methodologies used to assess this scenario. The previously reviewed and assessed data will not be revisited as part of this document.

[12] California Department of Pesticide Regulation (CDPR) Air Resources Board (ARB) Toxic Air Contaminant (TAC) Program, and the CDPR Air Monitoring Network (AMN)
[13] https://www.epa.gov/national-air-toxics-assessment

In the previous risk assessment, PERFUM results (i.e., buffer zone distances) were presented based on a range of inputs for each modeled parameter, including multiple sources of weather data, field sizes, application rates, and percentile of exposure for risk management purposes. A complete review of the modeling inputs is available in the Phase 5 risk assessment (12-APR-2007, C. Olinger, D337328) and its associated 2008 new-use/addendum which incorporated a post-plant drip irrigation flux study on vineyard grapes (24-JAN-2008, C. Olinger; D347789). The results of the previous assessments show that bystander inhalation exposure to 1,3-D after a soil fumigation application can vary depending on a variety of factors such as application method, agricultural tarps, water seals, and soil parameters like moisture and organic content.

The typical application rates currently used for 1,3-D range from 30 to 330 lbs ai/A depending on the crop. Application rates ranging from 17.5 lbs ai/A to 362 lbs ai/A were used in the previous residential bystander exposure PERFUM modeling analyses. The application parameters and control measures used in the previous assessments are still representative of the currently registered uses of 1,3-D. Therefore, the previous analyses are representative of the currently registered use pattern of 1,3-D.

A list of 1,3-D specific flux study data and other applicable documents are included below for reference.

- The *1,3-Dichloropropene: HED Human Health Risk Assessment for Phase 5* (12-APR-2007, C. Olinger, D337328)
- *1,3-Dichloropropene: Proposed New Use for Drip Irrigation in Vineyards: Revised HED Human Health Risk Assessment* (24-JAN-2008, C. Olinger *et al*., D347789)
    - Review *for post-plant drip irrigation field volatility study on vineyard grapes* (MRID #45296101)
- *The Health Effects Division's Review of the Determination of Buffer Zones for Chloropicrin when Co-applied with 1,3-Dichloropropene in 18 inch Deep Untarped Broadcast Applications (dated 04/13/2009)* (Memo, C. Smith, 11-JAN-2011, D348640)

Using these previously reviewed flux data representing all major application methods, meteorological data in key production regions, and representative ranges of field sizes; PERFUM modeling results for all 1,3-D products indicated that acute risks do not exceed HED's level of concern at 0 meters from treated fields as presented most recently in 2008 (24-JAN-2008, C. Olinger *et al*., D347789).

## 9.2 Non-occupational Ambient Exposure Assessment from Non-Point Sources

Bystanders who live or work near fumigated fields are potentially exposed to fumigant emissions that travel off-site. There is the potential for inhalation exposure to 1,3-D via ambient air resulting from multiple agricultural soil applications across large regions.

These assessments have been updated to reflect the recent available monitoring data and inhalation HECs which have been updated for ST/IT, and LT inhalation exposures according to current practices. The resulting HECs along with the additional years of ambient air monitoring data accumulated since the Phase 5 RED were incorporated into this assessment.

### 9.2.1 Ambient Air Monitoring Risk Estimates from Non-Point Sources

As part of the *1,3-Dichloropropene: HED Human Health Risk Assessment for Phase 5* (12-APR-2007, C. Olinger, D337328), results from existing California Air Resources Board (CARB) data were used to assess risks from acute (single day), ST/IT, and LT (chronic) exposures. The data included monitoring fourteen sites in three counties (Kern, Monterey, and Santa Cruz) during high 1,3-D use seasons over seven- to nine-week study periods between 2000 to 2001. As a supplement to the ambient air monitoring data previously presented in the Phase 5 RED, HED has queried any recent existing data that captures the ambient concentrations of 1,3-D and has compared that data to the appropriate inhalation PODs for use in risk assessment. This assessment has conducted the non-occupational ambient bystander analysis based solely on the monitoring results available since 2007 and has not re-assessed the data presented in the Phase 5 RED, to reflect current practices.

As part of Registration Review, HED has queried the recent years of CDPR's monitoring for 1,3-D. A query of the California Pesticide Information Portal (CALPIP) Pesticide Use Reporting (PUR) database[14] for 2017 (most recent year reported) shows that approximately 17.44 million pounds of active ingredient (1,3-D) were applied in the State of California.

Exposures from ambient air that occur from multiple regional sources of 1,3-D were estimated from monitoring data collected to represent conditions at a regional level. The California Air Resources Board (CARB) and California Department of Pesticide Regulation (CDPR) generated most of the data considered in this analysis. CARB and CDPR are widely recognized institutions for these types of programs and are part of the California Environmental Protection Agency. CARB conducts air monitoring studies for various types of chemicals throughout California through the Toxic Air Contaminants (TAC) program. DCPR utilizes the Air Monitoring Network (AMN) specifically to measure pesticides in various high-use agricultural communities.

For ease and clarity, the HED has opted by convention to describe the available ambient bystander data used in this assessment as follows:

- CDPR AMN Data: monitoring data generated by CDPR's AMN since 2011, intended as a multi-year statewide air monitoring program focusing on agricultural areas with high pesticide use (encompasses multiple pesticides).
- CARB TAC Agricultural Data: monitoring data generated by CARB's TAC Network since 2010 specifically focused on agricultural areas with high 1,3-D and methyl bromide use.
- CARB TAC Urban Data: monitoring data from CARB's TAC Network since 2002 for 1,3-D that quantifies background levels in non-agricultural, urban environments.
- CDPR Merced County Township Data: monitoring data generated since 2016 by CDPR intended to evaluate the effectiveness of township caps and permit conditions in two high 1,3-D use communities which were not already included in other CDPR or CARB studies.
- DAS Merced County Data: 14-months of continuous 1,3-D monitoring data generated by DAS since 2010 spanning nine townships (4 with high 1,3-D use and 5 with low to moderate 1,3-D use) for SOFEA modeling software validation purposes.

[14] https://calpip.cdpr.ca.gov/main.cfm

These data sources represent the 1,3-D ambient air monitoring sources identified in recent years by the Agency at the time of Registration Review. The compilation of data sources below provides a risk picture of 1,3-D ambient air exposures in high-use agricultural and urban environments and illustrates the evolution of 1,3-D use over a long duration.

*CDPR AMN Program Data*

In February 2011, CDPR implemented a multi-year statewide air monitoring program, the air monitoring network (AMN), to measure pesticides in various agricultural communities. The AMN originally provided monitoring for three communities, but with the passing of the Budget Act of 2016, it was expanded to include a total of eight sites for a two-year period. Four sites were operational in 2017, while the other four were added to the AMN in 2018. The four operational AMN monitoring sites were in the communities of Shafter (Kern County), Santa Maria (Santa Barbara County), Watsonville (Monterey County), and Chualar (Monterey County). At each sampling site location, one 24-hour (h) air sample set was collected on a weekly basis. Monitoring data on 1,3-D are available for 2011-2018[15]. This assessment has conducted the non-occupational ambient bystander analysis based on the 2011 through 2018 monitoring results to reflect the current practices used in California. Air concentrations from previous years would not illustrate the current agricultural use practices of 1,3-D as well as the emissions reductions practices and technologies referenced above in Section 9.0.

For the ambient bystander exposure assessment, HED has queried the AMN[16] data analysis for ambient concentrations of 1,3-D. All data were used as reported by CDPR (an analysis of the raw data was not conducted; values as reported were used). In 2018, of the 333 possible 1,3-D detection samples taken, a total of 37 quantifiable detections were identified (11.1%). HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 $mg/m^3$ = 343,330,000 $ng/m^3$). Risks from ST/IT exposures were calculated using the highest 4-week rolling average concentration (or 13-week depending on the study site) for each location and the ST/IT ambient HEC for the inhalation POD (1.35 $mg/m^3$ = 1,353,000 $ng/m^3$). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 $mg/m^3$ = 692,000 $ng/m^3$). All HEC durations were converted to $ng/m^3$ in Table 9.2.1.1 below to remain consistent with the concentration units reported in the study.

For the acute 24-hour exposures, the risk estimates range from 130 to 780,000 (LOC = 30). For the ST/IT rolling 4- and 13-week average exposures, the risk estimates range from 54 to 8,600 (LOC = 30). For the LT annual average exposures, the risk estimates range from 100 to 21,000 (LOC = 30). See Table 9.2.1.1 for the concentrations and the risk estimates.

[15] https://www.cdpr.ca.gov/docs/emon/airinit/air_network_results.htm.
[16] https://www.cdpr.ca.gov/docs/emon/airinit/air_network.htm

**Table 9.2.1.1. Ambient Air Concentration and Risk Estimates for 1,3-D; CDPR AMN 2011-2018 Results[1].**

| CDPR Monitoring site[2] | Year collected | Concentrations (ng/m³) [3,4,5] | | | Inhalation MOE[6] | | |
|---|---|---|---|---|---|---|---|
| | | 24-hour | 4-week | 1-year | Acute (LOC= 30) | ST (LOC = 30) | LT (LOC = 30) |
| Chualar | 2018 | 460 | 370* | 120 | 750,000 | 3,700 | 5,800 |
| Oxnard | | 450 | 240* | NA[6] | 760,000 | 5,600 | NA[7] |
| San Joaquin | | 3,359 | 468* | NA[6] | 100,000 | 2,900 | NA[7] |
| Santa Maria | | 2,200 | 440* | 280 | 160,000 | 3,100 | 2,500 |
| Shafter | | 230,000 | 25,000* | 6,900 | 1,500 | 54 | 100 |
| Watsonville | | 1,200 | 430* | 210 | 290,000 | 3,100 | 3,300 |
| Chualar | 2017 | 1,996 | 398 | 252 | 170,000 | 3,400 | 2,700 |
| Santa Maria | | 2,450 | 1,152 * | 366 | 140,000 | 1,200 | 1,900 |
| Shafter | | 3,394 | 4,812 * | 486 | 100,000 | 280 | 1,400 |
| Watsonville | | 1,860 | 904 | 397 | 180,000 | 1,500 | 1,700 |
| Salinas | 2016 | 1,561 | 1,245 | 187 | 220,000 | 1,100 | 3,700 |
| Shafter | | 45,323 | 4,678 * | 1,559 | 7,600 | 290 | 440 |
| Ripon | | 2,917 | 2,127 | 390 | 120,000 | 640 | 1,800 |
| Salinas | 2015 | 3,643 | 1,812 | 201 | 94,000 | 750 | 3,400 |
| Shafter | | 9,713 | 2,176 * | 800 | 35,000 | 620 | 870 |
| Ripon | | 4,074 | 2,711 | 380 | 84,000 | 500 | 1,800 |
| Salinas | 2014 | 440 | 158 | 33 | 780,000 | 8,600 | 21,000 |
| Shafter | | 9,251 | 10,119 * | 909 | 37,000 | 130 | 760 |
| Ripon | | 3,511 | 1,740 | 302 | 98,000 | 780 | 2,300 |
| Salinas | 2013 | 4,319 | 2,611 | 407 | 79,000 | 520 | 1,700 |
| Shafter | | 36,969 | 9,190 * | 2,589 | 9,300 | 150 | 270 |
| Ripon | | 14,745 | 7,993 | 883 | 23,000 | 170 | 780 |
| Salinas | 2012 | 3,430 | 1,082 | 259 | 100,000 | 1,300 | 2,400 |
| Shafter | | 3,643 | 594* | 453 | 94,000 | 2,300 | 1,500 |
| Ripon | | No Detect | | | No Detect | | |
| Salinas | 2011 | 1,072 | 2,743 | 695 | 320,000 | 490 | 1,000 |
| Shafter | | No Detect | | | No Detect | | |
| Ripon | | 12,250 | 4,022 | 784 | 28,000 | 340 | 880 |

1. https://www.cdpr.ca.gov/docs/emon/airinit/air_network.htm
2. Salinas and Ripton were not reported in the AMN report starting in 2017 due to a change in the design plan where the sites were replaced by Watsonville, Chualar, and Santa Maria (with the addition of Oxnard and San Joaquin with quantifiable concentrations in 2018).
3. Air concentrations reflect 24-hr highest air concentrations; 4 – and 13-*week highest rolling average concentrations; 1-year average concentrations.
4. For 1,3-D, CDPR defines the level of detection (LOD) to be equivalent to the level of quantitation (LOQ). CDPR used two labs to process samples: the LOD/LOQ at ARB-OLS = 454 ng/m³; the LOD/LOQ at DCFA CAC = 45.4 ng/m³.
5. Total possible detection samples collected in 2018 = 333; 2017 = 195; 2016 = 156; 2015=155; 2014=157; 2013=157; 2012=156; 2011=141 (total=1450)

* Highest 13-week rolling average concentrations were reported for these sites.

6. Acute Inhalation MOEs = Inhalation HEC (343.3 mg/m³ = 343,330,000 ng/m³) / reported 24-hour air concentration (mg /m³); ST/IT Inhalation MOEs = Inhalation HEC (1.353 mg/m³ = 1,353,000 ng/m³) / reported 4-week air concentration

(mg /$m^3$); LT Inhalation MOEs = Inhalation HEC (0.692 mg/$m^3$ = 692,000 ng/$m^3$) / reported 1-year average air concentration (mg/$m^3$). [1 mg/$m^3$ × 1,000,000 ng/1 mg = ng/$m^3$ (reported units in study)].

7. NA: 12 months of monitoring data at the sampling location were not available; therefore, no 1-year average air concentration was determined at the Oxnard and San Joaquin sites due to their start date in the AMN of 14-AUG-2018 and 4/26/2019 respectively.

*CARB TAC Program Monitoring Data in High Use Agricultural Environments*

In September 2010, as part of the California Toxic Air Contaminant (TAC) program, CDPR submitted a request to the California Air Resources Board (CARB) for monitoring of two pesticide fumigants, methyl bromide and 1,3-D. Because most large-scale pesticide applications are seasonal and occur in agricultural areas, ARB conducts monitoring in areas of high use, and at times when use is at its peak. This worst-case information can help determine the ambient exposures of people living in all areas where the pesticide is used. 1,3-D was monitored from 2010 to 2017/2018 in three sites in central and southern California [Oxnard (Ventura County), Santa Maria (Santa Barbara County), and Watsonville (Santa Cruz County)]. As of 2018, the only monitoring location remaining in the TAC program was Oxnard as the sampling sites in Santa Maria and Watsonville were transitioned into CDPR's AMN in 2017. Monitoring at the Oxnard monitoring location began in October 2011 and remained in operation as a TAC site until August 14, 2018 when it was also transitioned into DPR's AMN. The TAC Monitoring Results for Methyl Bromide and 1,3-D 2011-2018 report[17] only includes results for monitoring from Oxnard for the 2010-2018 calendar years as the most recent AMN report now incorporates Santa Maria and Watsonville. All data were used as reported by CDPR (an analysis of the raw data was not conducted; values as reported were used).

For the ambient bystander exposure assessment, HED has queried the TAC data analysis for ambient concentrations of 1,3-D. HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 mg/$m^3$ = 75,646 ppb). Risks from ST/IT exposures were calculated using the highest 4-week (or 13-week depending on the study site) rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 mg/$m^3$ = 298 ppb). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 mg/$m^3$ = 152 ppb). All HEC durations were converted to ppb in Table 9.2.1.2 below to remain consistent with the concentration units reported in the study.

The available TAC ambient air concentrations did not result in risk estimates of concern for acute, ST/IT, or LT exposures. The acute ambient MOEs range from 8,700 to 190,000 (LOC = 30). The ST/IT ambient MOEs range from 420 to 4,300 (LOC = 30). The LT ambient MOEs range from 730 to 1,700 (LOC = 30). See Table 9.2.1.2 for the concentrations and the risk estimates.

[17] https://www.cdpr.ca.gov/docs/emon/airinit/tac_results_methyl_bromide_1,3-d.pdf

| Table 9.2.1.2. Highest Ambient Air Concentration and Risk Estimates for 1,3-D; CDPR ARB TAC 2011-2018 Results[1]. | | | | | | | |
|---|---|---|---|---|---|---|---|
| CDPR Monitoring site[1] | Year collected | Concentrations (ppb) [2,3,4] | | | Inhalation MOE[5] | | |
| | | 24-hour | 4-week | 1 year | Acute (LOC= 30) | ST (LOC = 30) | LT (LOC = 30) |
| Oxnard | 2018 | 0.4 | 0.07 | * | 190,000 | 4,300 | * |
| | 2017 | 1.2 | 0.18 | 0.11 | 63,000 | 1,700 | 1,400 |
| | 2016 | 2.9 | 0.27 | 0.11 | 26,000 | 1,100 | 1,400 |
| | 2015 | 8.7 | 0.71 | 0.21 | 8,700 | 420 | 730 |
| | 2014 | 2.2 | 0.19 | 0.09 | 34,000 | 1,600 | 1,700 |
| | 2013 | 3 | 0.54 | 0.17 | 25,000 | 550 | 900 |
| | 2012 | 6.4 | 0.62 | 0.19 | 12,000 | 480 | 800 |
| | 2011 | No Detect | | | No Detect | | |

1. Oxnard was the only site summarized in the most recent 2018 report as Santa Maria and Watsonville are now incorporated into AMN (see Table 9.2.1.1).
2. Air concentrations available at https://www.cdpr.ca.gov/docs/emon/airinit/tac_results_methyl_bromide_1,3-d.pdf.
3. Reporting Limit = 0.10 ppb.
4. (n) = 450 possible detection samples taken in Oxnard from 2011 to 2018
5. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (ppb) ÷ concentration (ppb). Conversion to ppb = (1,000 ppb/1ppm) × ((mg/m$^3$) * 24.45/ (molecular weight (110.97 g/mol)). https://www.cdc.gov/niosh/docs/2004-101/calc html

* 12 months of monitoring data at the sampling location were not available; therefore, no 1-year average air concentration was determined.

*CARB TAC Program Monitoring Data in Urban Environments*

In addition to the agricultural area monitoring sites described above, TAC monitoring sites are also located throughout urban areas in California, such as Long Beach, Burbank, Los Angeles, and San Francisco[18]. Air concentrations collected from these sites are used to represent exposure in urban environments. All data were used as reported by CDPR (an analysis of the raw data was not conducted; values as reported were used).

For the ambient bystander exposure assessment in urban environments, HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 mg/m$^3$ = 75,646 ppb). Risks from ST/IT exposures were calculated using the highest 4-week (or 13-week depending on the study site) rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 mg/m$^3$ = 298 ppb). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 mg/m$^3$ = 152 ppb). Means shown on ARB's toxics pages are means of monthly means. Using the mean of monthly means compensates for the uneven distribution of samples over the 12 months of the year. All HEC durations were converted to ppb in Table 9.2.1.3 and 9.2.1.4 below to remain consistent with the concentration units reported in the study.

[18] https://www.arb.ca.gov/adam/toxics/sitesubstance html

The majority of reported sites and years of available ambient air concentrations did not result in quantifiable concentrations above the level of detection (monitoring LOD = 0.10 ppb). Sites/reported years with quantifiable concentrations above the LOD did not result in risk estimates of concern for acute, ST/IT, or LT exposures for either the cis- or trans-isomer of 1,3-D. The acute ambient MOEs range from 69,000 to 760,000 (LOC = 30), the ST/IT ambient MOEs range from 2,700 to 6,000 (LOC = 30), and the LT ambient MOEs are all 3,000 (LOC = 30) (Table 9.2.1.3 and 9.2.1.4).

**Table 9.2.1.3. Results of Urban Ambient Monitoring for Cis 1,3-D using CDPR TAC Program[1,2].**

| Site | Year | Highest Year Concentration of Cis Isomer (ppb)** LOD = 0.10 ppb | | | Acute MOE (LOC = 30)[3] | Short- and Intermediate-Term MOE (LOC = 30)[4] | Long-Term MOE (LOC = 30)[5] |
|---|---|---|---|---|---|---|---|
| | | Mean | 90th Percentile | Max | | | |
| **Southern California** | | | | | | | |
| Azusa | 2002-17 | <LOD | | | | | |
| Burbank | 2002-14 | <LOD | | | | | |
| Calexico-Ethyl Street | 2002-17 | <LOD | | | | | |
| Chula Vista | 2002-17 | <LOD | | | | | |
| El Cajon-Floyd Smith Drive | 2014-16 | <LOD | | | | | |
| El Cajon-Lexington Elementary | 2016-17 | <LOD | | | | | |
| El Cajon-Redwood Ave | 2002-13 | <LOD | | | | | |
| Los Angeles – North Main Street | 2002-17 | <LOD | | | | | |
| North Long Beach | 2002-13 | <LOD | | | | | |
| Riverside-Rubidoux | 2002-17 | <LOD | | | | | |
| Simi Valley-Cochran Street | 2002-17 | <LOD | | | | | |
| **Northern California** | | | | | | | |
| Bakersfield | 2008 | 0.11 | 0.02 | 1.1 | 69,000 | 2,700 | 3,000 |
| Chico-East Avenue | 2012-17 | <LOD | | | | | |
| Chico-Manzanita Avenue | 2011 | 0.05 | 0.05 | 0.1 | 760,000 | 6,000 | 3,000 |
| Freemont-Chapel Way | 2002-10 | <LOD | | | | | |
| Fresno-1st Street | 2006 | 0.08 | 0.05 | 0.7 | 110,000 | 3,700 | 3,000 |
| Fresno-Garland | 2015 | 0.08 | 0.2 | 0.3 | 250,000 | 3,700 | 3,000 |
| Roseville-N Sunrise Blvd | 2006 | 0.05 | 0.05 | 0.2 | 380,000 | 6,000 | 3,000 |
| San Francisco-Arkansas St | 2006 | 0.05 | 0.05 | 0.1 | 760,000 | 6,000 | 3,000 |
| San Jose-4th St | 2002 | <LOD | | | | | |
| San Jose-Jackson St | 2002-17 | <LOD | | | | | |
| Stockton-Hazelton St | 2015 | 0.07 | 0.1 | 0.3 | 250,000 | 4,300 | 3,000 |

1. Air concentrations available at https://www.arb.ca.gov/adam/toxics/sitelists/cdcpsites.html
2. Values below the LOD are assumed to be ½ the LOD 0.05 ppb).
3. Acute MOE = Acute ambient (24-hour exposure) HEC for the inhalation POD (75,656 ppb)/maximum concentration (ppb). LOC = 30.
4. ST/IT MOE = ST/IT ambient HEC for the inhalation POD (298 ppb)/mean concentration (ppb). LOC = 30.
5. LT MOE = LT ambient HEC for the inhalation POD (152 ppb)/median concentration (ppb). LOC = 30.

** Conversion to ppb = (1,000 ppb/1ppm) × ((mg/m$^3$) * 24.45/ (molecular weight (110.97 g/mol)).

| Table 9.2.1.4. Results of Urban Ambient Monitoring for Trans 1,3-D using CDPR TAC Program[1,2]. | | | | | | | |
|---|---|---|---|---|---|---|---|
| Site | Year | Highest Year Concentration of Trans Isomer (ppb)** LOD = 0.10 ppb | | | Acute MOE (LOC = 30)[3] | Short- and Intermediate-Term MOE (LOC = 30)[4] | Long-Term MOE (LOC = 30)[5] |
| | | Mean | 90th Percentile | Max | | | |
| **Southern California** | | | | | | | |
| Azusa | 2002-17 | <LOD | | | | | |
| Burbank | 2002-14 | <LOD | | | | | |
| Calexico-Ethyl Street | 2005 | 0.08 | 0.05 | 0.7 | 110,000 | 3,700 | 3,000 |
| Chula Vista | 2002-17 | <LOD | | | | | |
| El Cajon-Floyd Smith Drive | 2014-16 | <LOD | | | | | |
| El Cajon-Lexington Elementary | 2016-17 | <LOD | | | | | |
| El Cajon-Redwood Ave | 2002-13 | <LOD | | | | | |
| Los Angeles – North Main Street | 2002-17 | <LOD | | | | | |
| North Long Beach | 2002-13 | <LOD | | | | | |
| Riverside-Rubidoux | 2002-17 | <LOD | | | | | |
| Simi Valley-Cochran Street | 2012 | * | 0.05 | 0.2 | 380,000 | * | 3,000 |
| **Northern California** | | | | | | | |
| Bakersfield | 2008 | 0.09 | 0.2 | 0.9 | 84,000 | 3,300 | 3,000 |
| Chico-East Avenue | 2012-17 | <LOD | | | | | |
| Chico-Manzanita Avenue | 2002-12 | <LOD | | | | | |
| Freemont-Chapel Way | 2002-10 | <LOD | | | | | |
| Fresno-1st Street | 2006 | 0.09 | 0.05 | 0.6 | 130,000 | 3,300 | 3,000 |
| Fresno-Garland | 2012-15 | 0.06 | 0.05 | 0.2 | 380,000 | 5,000 | 3,000 |
| Roseville-N Sunrise Blvd | 2013 | 0.05 | 0.05 | 0.3 | 250,000 | 6,000 | 3,000 |
| San Francisco-Arkansas St | 2012 | 0.05 | 0.05 | 0.1 | 760,000 | 6,000 | 3,000 |
| San Jose-4th St | 2002 | <LOD | | | | | |
| San Jose-Jackson St | 2002-17 | <LOD | | | | | |
| Stockton-Hazelton St | 2015 | 0.06 | 0.05 | 0.2 | 380,000 | 5,000 | 3,000 |

1. Air concentrations available at https://www.arb.ca.gov/adam/toxics/sitelists/tdcpsites.html
2. Values below the LOD are assumed to be ½ the LOD 0.05 ppb).

** Conversion to ppb = (1,000 ppb/1ppm) × ((mg/m$^3$) * 24.45/ (molecular weight (110.97 g/mol)).

3. Acute MOE = Acute ambient (24-hour exposure) HEC for the inhalation POD (75,656 ppb)/maximum concentration (ppb). LOC = 30.
4. ST/IT MOE = ST/IT ambient HEC for the inhalation POD (298 ppb)/mean concentration (ppb). LOC = 30.
5. LT MOE = LT ambient HEC for the inhalation POD (152 ppb)/median concentration (reported as 0.05 ppb for all sites and detectable years). LOC = 30.
6. "*" indicates insufficient data or no data to determine the value.

*CDPR Delhi (Merced County) and Parlier (Fresno County) Township Cap Monitoring Data*[19]

In 2017, CDPR revised permit conditions which eliminated 1,3-D use in the month of December and restricted the total allotted application amount within a 6x6 square mile area to a maximum of 136,000 adjusted pounds (i.e., township cap) in a calendar year. In November of 2016, CDPR initiated an air monitoring study to evaluate the effectiveness of current township caps and permit conditions in two communities (Delhi in Merced County, and Parlier in Fresno County) characterized by relatively high levels of 1,3-D use which were not already included in other CDPR or ARB studies. All data were used as reported by CDPR (an analysis of the raw data was not conducted; values as reported were used).

HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 mg/m$^3$ = 75,646 ppb). Risks from ST/IT exposures were calculated using the highest 90-day rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 mg/m$^3$ = 298 ppb). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 mg/m$^3$ = 152 ppb). All HEC durations were converted to ppb in Table 9.2.1.5 below to remain consistent with the concentration units reported in the study.

The majority of available ambient air concentrations did not result in risk estimates of concern for acute, ST/IT, or LT exposures, However, one ST/IT ambient exposure sample in Parlier in 2018 resulted in a risk of concern with an MOE of 28 (LOC = 30). The highest concentration sample was collected on October 9, 2018 which contained the highest measured 1,3-D detection from this study to date (111 ppb). Through further investigation, CDPR determined that the heightened concentration was most likely the result of five subsequent 1,3-D applications made during the sample period with field boundaries ranging from 0.1 to 1 mile from the Parlier monitoring site. All other sample concentrations in Parlier for 2018 were less than or equal to 9.08 ppb, while all sample concentrations in Parlier for 2017 were less than or equal to 15.96 ppb.

In 2018, 101 valid primary samples were collected from the two sites with 50 from Delhi and 51 from Parlier. During this period, 1,3-D was detected in 76% of air samples collected from Delhi (68% or 34 out of 50 samples) and Parlier (84% or 43 out of 51 samples). Quantifiable detections in Delhi ranged from 0.012 to 1.80 ppb with an annual mean of 0.19 ppb. Quantifiable detections in Parlier ranged from 0.011 to 111 ppb with a median of 0.109 ppb and a mean of 2.94 ppb. Due to how maximum subchronic and chronic exposures are calculated by CDPR, the presence of the 111-ppb detection in the calculations increased the subchronic average concentration from 1.97 ppb to 10.53 ppb. Similarly, this high 111 ppb value almost quadrupled the 1-yr chronic concentration from 0.76 ppb to 2.94 ppb.

The acute ambient MOEs range from 680 to 71,000 (LOC = 30). The ST/IT ambient MOEs range from 28 to 1,000 (LOC = 30). The LT ambient MOEs range from 52 to 1,200 (LOC = 30). See Table 9.2.1.3 for the concentrations and the risk estimates.

[19] https://www.cdpr.ca.gov/docs/emon/airinit/monitoring_1,3-d_merced_fresno.pdf

**Table 9.2.1.3. Highest Ambient Air Concentration and Risk Estimates for 1,3-D; CDPR Township Monitoring Caps 2017-2018 Results[1].**

| CDPR Monitoring site | Year collected[2] | Concentrations (ppb)[3] | | | Inhalation MOE[4] | | |
|---|---|---|---|---|---|---|---|
| | | 24 hour | 90 day | 1 year | Acute (LOC= 30) | ST/IT (LOC = 30) | LT (LOC = 30) |
| Delhi | 2018 | 1.8 | 0.48 | 0.19 | 42,000 | 620 | 800 |
| Parlier | | 111.29 | 10.53 | 2.94 | 680 | **28** | 52 |
| Delhi | 2017 | 1.06 | 0.29 | 0.13 | 71,000 | 1,000 | 1,200 |
| Parlier | | 15.96 | 1.83 | 0.62 | 4,700 | 160 | 250 |

1. Air concentrations available at https://www.cdpr.ca.gov/docs/emon/airinit/monitoring_1,3-d_merced_fresno.pdf
2. Monitoring study began in November of 2016.
3. Air concentrations reflect 24- highest air concentrations; 90-day highest rolling average concentrations; 1-year average concentrations.
4. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (ppb)/concentration (ppb). ** Conversion to ppb = (1,000 ppb/1ppm) × ((mg/m3) * 24.45/ (molecular weight (110.97 g/mol)).

*Merced County, CA – DAS Ambient Air Monitoring Study (MRID 49318301)*

In 2014, HED reviewed an ambient air monitoring study (*Monitoring of Cis- and Trans-l,3-Dichloropropene in Air in 9 High 1,3-Dichloropropene Use Townships Merced County, California.* MRID 49318301) submitted to satisfy the Southwest regional requirement (California) and provide a dataset to compare against the SOFEA modeling software for validation purposes (10-DEC-2014, M. Lloyd, D418726). HED determined that the submission satisfied the Southwest region data requirement which will allow for the completion of a non-occupational bystander inhalation exposure assessment for acute, ST/IT, and LT quantitative assessment.

The purpose of the study was to measure ambient air concentrations of 1,3-D in nine townships of Merced County, CA over an ~14-month period (Oct 14, 2010 to Jan 1, 2012). The region was selected as a test site because four of the townships have historically high 1,3-D use, and the surrounding five sites were included because of their historically low to moderate 1,3-D use. A total of 1,060,543 lbs of 1,3-D was applied in the nine townships during the study time frame. The study encompassed three high-use periods: 1) Fall (Nov-Dec) of 2010 (279,795 lbs total usage), 2) Spring (Mar-May) of 2011 (379,548 lbs total usage), and 3) Fall (Nov-Dec) of 2011 (308,220 lbs total usage). Meteorological conditions during the application and monitoring period were reported to be typical both regionally and seasonally.

In addition to the individual 72-hr concentration samples, monthly mean concentrations were calculated by averaging the 72-hr concentrations within the month. Using the monthly mean concentrations, 2-month running averages (for ST and IT) and 6-month running averages (for LT) were also calculated. Approximately 20% of the residues were below the LOQ, with most of these values occurring in the summer (Jun-Aug) of 2011.

A sub-study compared the 72-hr time weighted averages (TWA) air concentrations with a subset of 24-hr samples collected over the course of the study. A comparison of 72-hr TWA concentrations calculated from the 24-hr samples and the 72-hr samples show that concentrations were similar, although the 3 X 24-hr concentrations were consistently slightly higher. A paired

t-test conducted by the study author shows that the results were not statistically different ($p<0.05$).

HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 72-hour air concentrations for each modeled location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 mg/m$^3$). Risks from ST/IT exposures were calculated using the highest 2-month rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 mg/m$^3$). Risks from LT exposures were calculated using the 6-month average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 mg/m$^3$).

The majority of available ambient air concentrations did not result in risk estimates of concern for acute, ST/IT, or LT exposures, However, one ST/IT ambient exposure sample at the 07S11E site during the Fall (Nov-Dec) 2011 high-use period resulted in a risk of concern with an MOE of 27 (LOC = 30). The highest concentration sample was collected for the period encompassing December 11-14, 2011, which contained the highest measured 1,3-D detection from this study (0.452 mg/m$^3$), was noted to be in close proximity to treated fields. Additionally, the study notes that on December 11$^{th}$, 2011, this monitoring device had to be relocated <1/4 mile from the original site due to theft of the sampling equipment.

In 2017, CDPR revised permit conditions which eliminated 1,3-D use in the month of December and restricted the total allotted application amount within a 6x6 square mile area to a maximum of 136,000 adjusted pounds (i.e., township cap) in a calendar year. Both CDPR imposed restrictions would be expected to refine the highest 24-hour air concentrations for the study site (07S11E) as the peak concentration was detected in December 11-14, 2011, where 193,138 lbs of 1,3-D was applied in that township over the course of the year (57,138 over the future township cap). Additionally, an ongoing study accounting for these restrictions has been conducted in Delhi (within 9 miles of the 07S11E monitoring station) starting in 2016 which resulted in a highest 24-hour concentration of 1.8 ppb (or 0.0068 mg/m$^3$) (summarized in the previous study section "*CDPR Delhi (Merced County) and Parlier (Fresno County) Township Cap Monitoring Data"* above).

The acute ambient MOEs range from 760 to 74,000 (LOC = 30). The ST/IT ambient MOEs range from 27 to 1,100 (LOC = 30). The LT ambient MOEs range from 42 to 1,200 (LOC = 30). See Table 9.2.1.4 for the study concentrations and the risk estimates.

**Table 9.2.1.4. Results of 14-Month Continuous Air Monitoring Using 72-hr Samples in Nine Contiguous Townships of Merced County California.**

| Study Site[5] | Total 1,3-D Usage[1] | Concentrations (mg/m$^3$) [3] [date range] | | | Inhalation MOE[4] | | |
|---|---|---|---|---|---|---|---|
| | | Max 72 Hr[2] | Max 2-mo[3] | Max 6-mo[4] | Acute (LOC= 30) | ST/IT (LOC = 30) | LT (LOC = 30) |
| 06S10E | 62,032 | 0.0126 [Dec 23-26, 2011] | 0.00343 [Nov-Dec, 2011] | 0.00123 [Jul-Dec, 2011] | 27,000 | 390 | 560 |

**Table 9.2.1.4. Results of 14-Month Continuous Air Monitoring Using 72-hr Samples in Nine Contiguous Townships of Merced County California.**

| Study Site[5] | Total 1,3-D Usage[1] | Concentrations (mg/m$^3$)[3] [date range] | | | Inhalation MOE[4] | | |
|---|---|---|---|---|---|---|---|
| | | Max 72 Hr[2] | Max 2-mo[3] | Max 6-mo[4] | Acute (LOC= 30) | ST/IT (LOC = 30) | LT (LOC = 30) |
| 06S101E | 209,891 | 0.341 [Dec 8-11, 2011] | 0.0285 [Nov-Dec, 2011] | 0.00958 [Jul-Dec, 2011] | 1,000 | 47 | 72 |
| 06S12E | 165,164 | 0.0109 [Dec 29, 2011-Jan 1, 2012] | 0.00367 [Nov-Dec, 2011] | 0.00135 [Jul-Dec, 2011] | 31,000 | 370 | 510 |
| 07S10E | 39,195 | 0.0148 [Mar 16-19, 2011] | 0.00375 [Nov-Dec, 2011] | 0.00153 [Dec, 2010-May, 2011] | 23,000 | 360 | 450 |
| 07S11E | 193,138 | **0.452** [Dec 11-14, 2011] | **0.0497** [Nov-Dec, 2011] | **0.0166** [Jul-Dec, 2011] | 760 | **27** | 42 |
| 07S12E | 256,712 | 0.0749 [Dec 11-14, 2011] | 0.0145 [Nov-Dec, 2011] | 0.00510 [Jul-Dec, 2011] | 4,600 | 93 | 140 |
| 08S10E | 0 | 0.00692 [Nov 1-4, 2010] | 0.00125 [Oct-Nov 2010] | 0.00059 [Oct, 2010-Mar, 2011] | 50,000 | 1,100 | 1,200 |
| 08S11E | 101,258 | 0.0242 [Nov 1-4, 2010] | 0.00357 [Oct-Nov 2010] | 0.00185 [Oct, 2010-Mar, 2011] | 14,000 | 380 | 370 |
| 08S12E | 33,153 | 0.00462 [Dec 17-20, 2011] | 0.00169 [Nov-Dec, 2011] | 0.00079 [Jul-Dec, 2011] | 74,000 | 800 | 880 |

1. Total 1,3-D Usage is the amount of 1,3 D applied in each township during the study duration (Oct 25, 2010 to Dec 31, 2011 for Site 08S10E, Oct 26, 2010 to Dec 31, 2011 for Site 08S11E, and Oct 14, 2010 to Dec 31, 2011 for remaining sites). Three high use seasons were identified during the study duration: 1) Fall (Nov-Dec) of 2010 (279,795 lbs total usage), 2) Spring (Mar-May) of 2011 (379,548 lbs total usage), and 3) Fall (Nov-Dec) of 2011 (308,220 lbs total usage).
2. The maximum 72-hr. concentration represents the maximum average 72-hr. concentration observed during the study duration.
3. Maximum 2-mo. running concentration is the maximum average 2-month concentration calculated by averaging monthly means for each consecutive 2-month period. The date presented is the 2-month period where this maximum occurred.
4. Maximum 6-mo. running concentration is the maximum average 6-month concentration calculated by averaging monthly means for each consecutive 6-month period. The date presented is the 6-month period where this maximum occurred.
5. The month of October is a partial month. Sampling did not start until October 25, 2010 for Site 08S10E, October 26, 2010 for Site 08S11E, and October 14, 2010 for the remaining sites.

### 9.2.2 SOFEA 4 Modeling Software Summary, Validation, and Initial Risk Estimates

On September 24, 2014, the Agency required an ambient air monitoring study for 1,3-D (GDCI-029001-1397), that addressed ambient exposures to non-occupational bystanders in proximity to agricultural fumigations from high use areas. The GDCI was the result of HED's

recommendations for fumigant data to refine exposure assessments identified in 2008[20]. To fulfill the GDCI, ambient air monitoring data from four separate geographic regions of high 1,3-D use were required: Southeast (e.g. Florida/Georgia/Virginia), Mid-Atlantic to Upper Mid-west (e.g., Michigan/Wisconsin), Pacific Northwest (e.g., Idaho/Washington), and Southwest (e.g. California/Arizona). Subsequently, in an October 30, 2014 document (MRID# 49503106), DAS requested a waiver for the required ambient air monitoring study. The document proposed that realistic concentrations of 1,3-D in ambient air could be effectively derived from modeling using the **So**il **F**umigant **E**xposure **A**ssessment (SOFEA) modeling system as opposed to the required monitoring at all four sites.

The SOFEA modeling software was originally developed and reviewed in the 2004 USEPA Scientific Advisory Panel (SAP) meeting[21] to evaluate and manage human inhalation exposure potential associated with agricultural applications of fumigants. SOFEA calculates fumigant concentrations in air arising from volatility losses from treated fields for entire agricultural regions using multiple sources (treated fields), GIS information, agronomic specific variables, user specified buffer zones and field intervals. The original SOFEA model used a modified version of ISCST3 as the air dispersion model to predict ST/IT and LT pesticide concentrations in air for large air sheds resulting from representative agronomic practices. It would also use ISCST3 in tandem with Monte Carlo techniques to vary the following parameters: weather information, field size, application date, application rate, application method, pesticide degradation rates in air, sealing method, field re-treatment, and buffer setbacks. Multi-year, multiple field simulations can be conducted with SOFEA using random field placement in all agricultural areas or by selectively placing fields in historical or prospective use areas. Regional land use information can be used to refine the placement of treated fields, dispersion calculations, and exposure assessments.

The SOFEA model has several advantages over monitoring studies; primarily the ability to predict concentrations at many locations and at a much greater temporal frequency than could be practically accomplished through monitoring studies. Additionally, the receptor density and specific locations where 1,3-D concentrations in ambient air are simulated can also be varied. The uncertainties in estimating exposure associated with spatially and temporally sporadic air measurements, such as those collected in the AMN program, are easily overcome with the use of an air dispersion model that takes into account spatial and temporal variability in product use patterns as well as weather variability.

SOFEA was updated in 2014 (v2.0) to run on a Windows 7 operating system and enable the model to be run in 'validation' mode, which allows the user to specify specific field locations where applications are made and specific receptor locations where fumigant concentrations are measured. Other enhancements to the model, added in response to the EPA soil fumigant SAP in September 2004, included the option of importing results from a soil physics model (CHAIN_2D) to generate flux inputs in lieu of flux obtained from field experiments.

[20] See HED's memo, *J. Dawson, 01-JUL-2008, "Recommendations for Fumigant Data to Refine Exposure Assessments" D353724* for additional information.
[21] https://www.epa.gov/sap/fifra-scientific-advisory-panel-historical-meetings

In 2018, SOFEA (v4.0) was modified to use the American Meteorological Society (AMS)/EPA Regulatory Model (AERMOD), the USEPA's recommended air dispersion model. AERMOD possesses more explicit parameterization for land surface characteristics (e.g., surface roughness length, albedo, and Bowen Ratio) and develops vertical wind scaling parameters (e.g., Monin Obukhov Length, friction velocity, and convective velocity scale) to calculate boundary layer heat flux to improve discretization of mixing regimes. This scheme replaces previous stability classes used in ISCST3 and has become the EPA's Office of Air's preferred model for near field releases.

The latest version of SOFEA (v4.1.4) includes the following updates:

1. Inclusion of a Graphical User Interface (GUI).
2. Inclusion of a modified version of AERMOD which supports buffer zones and netCDF POSTFILE output for high performance post processing. The buffer zone modifications are based on code written by Roger Brode of Pacific Environmental Services (a core AERMOD system developer) for buffer zone support in ISCST3, which was readily adapted to AERMOD. netCDF is a standard format used by numerous other air quality models, including EPA's Community Multiscale Air Quality (CMAQ) model.
3. Calculation of concentration distributions using the P2 algorithm (Jian and Chlamtac, 1985). Previously, a binning method was used where the number of concentrations in certain specified ranges were counted. This method was necessary because it was not possible to store all of the values in memory. The P2 algorithm allows for a distribution to be constantly updated based on new values, though removing the need to store them in memory. The P2 algorithm allows for a more accurate and precise estimate of distribution percentiles.
4. Inclusion of an analysis module that includes high performance, single pass algorithms for summary statistics, percentile estimation, moving averages (MAs), cumulative distribution function (CDF), and probability density function (PDF) generation.

*SOFEA v4.1.4 Preliminary Modeled Results for the Southeast, Mid-Atlantic, and Pacific Northwest Regions*

Preliminary modeled results of SOFEA v4.1.4 were submitted to the Agency using three high-use areas. Modeling runs were completed for the Southeast (Florida), Mid-Atlantic (North Carolina), and Pacific Northwest regions (Washington) using reported application details (including field GPS location, lbs ai applied, date applied, and depth applied) provided by DAS for each modeled area. **NOTE:** These model outputs have been recently made available to the Agency and will be further reviewed and incorporated through the Registration Review process as appropriate. For the current purposes of Registration Review, HED has calculated preliminary risk estimates using the modeled concentrations as they were submitted to the Agency for acute, ST/IT, and LT durations with the updated inhalation HECs derived by the Agency which are detailed in Section 4.5. The model inputs (e.g., reported chemical use data, meteorological data, flux data, etc.) and summary of all modeled percentiles are presented in Appendix F.

HED evaluated different durations of exposure including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each modeled location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 mg/m$^3$ = 75.6 ppm). Risks presented from ST/IT exposures were calculated using the 95$^{th}$ percentile 28-day and 90-day rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 mg/m$^3$ = 0.30 ppm). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 mg/m$^3$ = 0.152 ppm).

The 100$^{th}$ percentile acute ambient MOEs range from 230 to 970 (LOC = 30). Based on the 95$^{th}$ percentile 28-day moving average, ST/IT MOEs range from 40 to 370 (LOC = 30); The 95$^{th}$ percentile 90-day moving average, ST/IT MOEs range from 110 to 980 (LOC = 30). Starting at the 99$^{th}$ percentile some ST/IT MOEs are less than the LOC of 30 depending on the duration (28-day and 90-day) and region for ST/IT durations and are further detailed in Appendix F. The LT 1-year average ambient MOEs range from 650 to 8,200 (LOC = 30). See Table 9.2.2.1 below for the summary of concentrations and the risk estimates. A full distribution of concentrations at all reported percentile ranges is available in Appendix F Tables F.1 to F.4.

**Table 9.2.2.1. Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4[1].**

| Modeled Region (state) | Modeled Year | Concentrations (ppm) [2,3] | | | | Inhalation MOE[4] | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 24-hour | 28-day | 90-day | 1-year[5] | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | | (LOC = 30 for all durations) | | | |
| Pacific Northwest (Washington) | 2015/16 | 8.85E-02 | 1.04E-03 | 4.28E-04 | 2.84E-05 | 860 | 290 | 700 | 5,400 |
| | 2016/17 | 1.05E-01 | 8.03E-04 | 3.04E-04 | 1.85E-05 | 720 | 370 | 980 | 8,200 |
| Mid-Atlantic (North Carolina) | 2015/16 | 7.80E-02 | 4.66E-03 | 1.55E-03 | 1.24E-04 | 970 | 64 | 190 | 1,200 |
| | 2016/17 | 1.96E-01 | 4.27E-03 | 1.43E-03 | 1.02E-04 | 390 | 70 | 210 | 1500 |
| Southeast (Florida) | 2015/16 | 1.16E-01 | 7.51E-03 | 2.63E-03 | 2.33E-04 | 650 | 40 | 110 | 650 |
| | 2016/17 | 1.05E-01 | 6.34E-03 | 2.24E-03 | 2.00E-04 | 720 | 47 | 130 | 760 |

1. Full air concentration percentile ranges, MOEs, and data inputs detailed in Appendix F
2. Air concentrations using 24 hour averaging periods reflect 100$^{th}$ percentile 24-hour air concentrations; 95$^{th}$ percentile 28-day and 90-day rolling average concentrations; 1-year average concentrations.
3. Modeled 1,3-D concentrations in µg/m$^3$ were converted to ppm, where 1 µg/m$^3$ of 1,3-D = 0.000222 ppm. [1 µg/m$^3$ = (0.001mg/m$^3$) * 24.45/ (molecular weight (110.97 g/mol = 0.000222 ppm] https://www.cdc.gov/niosh/docs/2004-101/calc html
4. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (ppm)/highest 24-hour concentration (ppm).
5. Arithmetic mean of annual average concentrations for all receptors

## 9.3 EPA 2018 National Air Toxics Assessment (NATA)[22]

In August 2018, EPA released the most recent update to the National Air Toxics Assessment (NATA). NATA is EPA's ongoing review of air toxics in the United States, and was developed as a screening tool for state, local, and tribal air agencies. NATA's results help these agencies identify which pollutants, emissions sources, and places they may wish to study further to better

[22] https://www.epa.gov/national-air-toxics-assessment

understand any possible risks to public health from air toxics. These data aren't intended to provide precise exposures and risks for a specific person, and are best applied to larger areas (counties, states, and the nation as a whole).

The most recent NATA[23] uses emissions data from 2014 to estimate health risks from toxic air pollutants. HED used the highest concentration of 1,3-D reported by NATA in Santa Cruz, CA (0.0157 mg/m$^3$) to calculate a screening level MOE[24] of approximately 44 (LOC = 30), which is not of concern. This finding is consistent with the finding from EPA's NATA, where calculated hazard quotients (HQs) were not of concern. In an air toxics risk assessment, the potential for non-cancer effects in humans is typically quantified by calculating a ratio of the inhalation exposure concentration to the toxicity reference concentration (RfC); the 1,3-D chronic RfC used in the NATA assessment was 0.02 mg/m$^3$ (LT HEC of 0.69 mg/m$^3$ is used by OPP for risk assessment is considered equivalent to the NATA RfC[25]). This ratio is referred to as the HQ. For a given air toxicant, HQs of 1 or less are not likely to be associated with adverse health effects. Using the data collected for all sites, NATA reported the respiratory hazard quotients (the route of concern for 1,3-D) for 1,3-D are all ≤ 0.7154 and are not of concern.

## 10.0 Cumulative Exposure/Risk Characterization

Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, EPA has not made a common mechanism of toxicity finding as to 1,3-D and any other substances and 1,3-D does not appear to produce a toxic metabolite produced by other substances. For the purposes of this action, therefore, EPA has not assumed that 1,3-D has a common mechanism of toxicity with other substances. In 2016, EPA's OPP released a guidance document entitled, *Pesticide Cumulative Risk Assessment: Framework for Screening Analysis* [https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/pesticide-cumulative-risk-assessment-framework]. This document provides guidance on how to screen groups of pesticides for cumulative evaluation using a two-step approach beginning with the evaluation of available toxicological information and if necessary, followed by a risk-based screening approach. This framework supplements the existing guidance documents for establishing common mechanism groups (CMGs)[26] and conducting cumulative risk assessments (CRA)[27]. During Registration Review, the Agency will utilize this framework to determine if the available toxicological data for 1,3-D suggests a candidate CMG may be established with other pesticides. If a CMG is established, a screening-level toxicology and exposure analysis may be conducted to provide an initial screen for multiple pesticide exposure.

## 11.0 Occupational Exposure/Risk Characterization

This section of the risk assessment focuses on potential exposures and risk to occupational handlers, to occupational reentry workers who could be exposed when entering 1,3-D treated

[23] https://www.epa.gov/national-air-toxics-assessment/2014-nata-assessment-results#about
[24] MOE = HEC (0.69 mg/m$^3$) ÷ Exposure (0.0157 mg/m$^3$) = 44
[25] LT Bystander HEC = 0.69 mg/m$^3$ ~ 0.02 mg/m$^3$ RfC (NATA RfC = HEC ÷ 30 UF)
[26] *Guidance For Identifying Pesticide Chemicals and Other Substances that have a Common Mechanism of Toxicity* (USEPA, 1999)
[27] *Guidance on Cumulative Risk Assessment of Pesticide Chemicals That Have a Common Mechanism of Toxicity* (USEPA, 2002)

areas to perform crop-production tasks, and to occupational bystanders who could be exposed when performing crop-production tasks near (but not inside) 1,3-D-treated areas. These scenarios were assessed as part of the *HED Human Health Risk Assessment for Phase 5* (12-APR-2007, C. Olinger, D337328).

As part of Registration Review an update to the occupational exposures has been conducted to address the updates to the inhalation ST/IT and LT HECs. As no new handler data has been submitted since the Phase 5 RED, the study information, including air concentrations which represent potential worker exposure, has been carried forward with no changes and reanalyzed solely to incorporate the updated toxicological information (HECs).

It is important to consider that in this assessment worker exposure monitoring data have been used directly for risk assessment purposes. In a typical pesticide handler assessment, HED uses normalized estimates of exposures based on similar equipment and with similar levels of protective equipment or clothing. Additionally, in typical post-application worker assessments, exposures are scaled based on how residues decay over time. These approaches have not been used in the occupational assessments presented below due to methodological issues. For example, it is not clear how changes in various parameters or conditions (e.g., temperature, emission reduction methods such as tarps or application methods) may impact exposures. It is also not clear how time after application can be used for scaling exposures from one day to the next because worker exposures may be inherently related to the conditions of the field under which monitoring has occurred. Entry restrictions into treated fields for activities such as tarp cutting, and tarp removal remain unchanged.

## 11.1 Acute and Short-/Intermediate-Term Occupational Handler Exposure and Risk Estimates

For the soil uses of 1,3-D, occupational handlers may be exposed while handling the pesticide prior to application, as well as during application. The potential for 1,3-D exposure was only considered for the inhalation route of exposure. Dermal exposures are not expected given the high vapor pressure of 1,3-D, and based on the delivery systems, packaging (i.e., pressurized cylinders), and emission reduction techniques (e.g., tarping) used. While no data are available to assess drip irrigation applicator exposure, the application takes place using a closed system and exposure is expected to be negligible. Therefore, dermal exposures have not been quantitatively assessed. Because 1,3-D is currently classified as a Restricted Use Pesticide, it may only be applied by a certified applicator or under the direct supervision of a certified applicator. Occupational acute and ST/IT inhalation exposures are expected from the registered uses of 1,3-D. Continuous LT exposures for more than 6 months per year are not expected based on the seasonal nature of 1,3-D use.

While an updated handler assessment has been completed to incorporate updates to the 1,3-D ST/IT inhalation HEC, there have been no new worker exposure data submitted since the summaries provided in the Phase 5 RED (12-APR-2007, C. Olinger, D337328). The concentrations for each presented duration of exposure have been pulled forward from that document unaltered for use in this assessment. For 1,3-D, handler exposure estimates were based on chemical-specific handler studies that examined 1,3-D exposures to handlers involved in

applications. 1,3-D worker exposure data are available, but the data did not cover the entire range of possible exposure scenarios. Occupational exposures studies (MRIDs) used in this assessment are listed below. The tasks monitored include:

- MRID 42946201 and 42845602: drum and bulk loaders and applicators, irrigation maintenance worker, rock removers, bed shapers;
- MRID 43880401: mini-bulk loaders and applicators;
- MRID 45120702: pre-bed row applications (Yetter rig), shovel men, operators, drivers.

At this time, HED has no worker exposure data to assess potential risks to occupational handlers or reentry workers resulting from the pre-plant drip irrigation uses, the post-plant vineyard use, or the turf uses of 1,3-D. The Phase 5 RED utilized field volatility data available to address off-site exposure for the drip irrigation and golf course uses. For these application techniques, the ISCST3 and PERFUM models were previously used as surrogate data to estimate occupational bystander exposures following/during a single 1,3-D application to outdoor agricultural fields. As these surrogate data runs inherently rely on the acute endpoint (which was not impacted by the HEC updates) with outputs encompassing 24-hour durations, and there were no risks of concern identified for any modeled meteorological condition or application method at that time (12-APR-2007, C. Olinger, D337328; Table 17), they were not updated for the purposes of this assessment.

Much of the handler exposure monitoring data used in the occupational exposure estimates reflect the use of some engineering controls such as tarps, tractor cabs, deep injection, or other devices. Agricultural practices or application technology may have changed since the submission of these data; additionally, there may be application activities that were not captured in the worker monitoring studies submitted. 1,3-D labels currently require occupational handlers to wear baseline attire defined as long-sleeve shirt, long pants, shoes plus socks and a variety of additional PPE including chemical-resistant aprons, footwear, gloves, headgear, and suits, coveralls, and protective eyewear. Additional PPE is required which varies by label/product for the inhalation route depending on potential for contact with liquid fumigant, application equipment/method, time since fumigations took place, and concentration levels of 1,3-D. For these scenarios occupational handlers may be required to wear NIOSH filtering half-face respirators, engineering controls (i.e., enclosed cab equipped with a vapor adsorptive filter containing activated charcoal), SCBA or supplied-air respirators depending on the labeled activity (for additional details, see *(029001) PLUS – Maximum Use Scenario Report.xlsx*, 14-DEC-2018).

Acute risks to handlers are less than HED's LOC of 30 for all of the sample points at baseline attire without a respirator (MOEs range from 320 to 19,000). Many ST and IT risks to handlers are less than HED's LOC of 30 for occupational activities at baseline attire without a respirator (MOEs range from 1 to 380). However, with the addition of a PF 10 respirator, all but one of the ST and IT handler risks are greater than HED's LOC (MOEs range from 5.3 to 3,800). With the addition of a PF 50 respirator (e.g., full-face/recirculated air respirator system), all but one of the ST and IT handler risks are greater than HED's LOC (MOEs range from 27 to 19,000). The updated non-cancer risk estimates are based on the existing worker monitoring data and are provided in Table 11.1.1 below.

**Table 11.1.1. 1,3-D Handler and Post-application MOEs Calculated from Study Point Estimates for Preplant Agricultural Field Fumigation.[1]**

| Study Site | Job Description (sample duration) | (n) | 1,3-D Concentrations (ppm)[2] | | | Baseline MOEs[3] | | PF 10 Respirator MOEs[4] | | PF 50 Respirator MOEs | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Median | Max | Mean | Acute | ST-IT | Acute | ST-IT | Acute | ST-IT |
| **MRID 43880401** | | | | | | | | | | | |
| **Pre-plant broadcast and row applications** | | | | | | | | | | | |
| WA, AZ | Bulk Loading[a] (4 hours) | 10 | 0.14 | 1.29 | 0.35 | 180 | **4** | 1800 | 36 | 9000 | 180 |
| WA, AZ | Bulk Loading[a] (task only) | 10 | 1.05 | 7.05 | 2.35 | 32 | **1** | 320 | **5** | 1600 | **27** |
| NC | Mini-bulk Loading[a] (task only) | 12 | 0.1 | 0.26 | 0.1 | 870 | **13** | 8700 | 130 | 43500 | 650 |
| WA, AZ, NC | Bulk, Mini-bulk, and Drum Application[b] (4 hours and task) | 28 | 0.25 | 1.43 | 0.29 | 160 | **4** | 1600 | 43 | 8000 | 215 |
| **MRID 42946201** | | | | | | | | | | | |
| **Workers performing tasks post-fumigation** | | | | | | | | | | | |
| WA | Irrigation system maintenance | 5 | 0.02 | 0.03 | 0.02 | 7600 | 63 | 76000 | 630 | 380000 | 3150 |
| AZ | rock removal | 5 | 0.01 | 0.02 | 0.01 | 11000 | 130 | 110000 | 1300 | 550000 | 6500 |
| AZ | bed shapers | 5 | 0.13 | 0.22 | 0.13 | 1000 | **10** | 10000 | 96 | 50000 | 480 |
| **MRID 454002-02** | | | | | | | | | | | |
| **Pre-bed row application equipment (Yetter rig)** | | | | | | | | | | | |
| FL | Shovel men[c] ((265-299 minutes) | 15 | NA | 0.278 | 0.0033 | 820 | 380 | 8200 | 3800 | 41000 | 19000 |
| FL | Operators[d] (269-287 minutes) | 15 | NA | 0.079 | 0.0048 | 2900 | 260 | 29000 | 2600 | 145000 | 13000 |
| FL | Drivers (255-299 minutes) | 9 | NA | 0.0122 | 0.0049 | 19000 | 260 | 190000 | 2600 | 950000 | 13000 |

a With use of dry disconnects.
b With use of end-row spill control.
c Shovel men anchored the plastic and drip tape when the tarping machine started out, worked within the field to fix any problems in the tarp, and made seams in the tarp or drip tape as necessary.
d Operators sat on the back of the tarping machine and ensured that the plastic and drip tape were laid properly.
1 This table was presented in D337328 and has been revised to reflect the change in the ST/IT inhalation HEC only. All study information, including air concentrations has remained the same as previously presented.
2 Conversion to $mg/m^3$ = (ppm) * (molecular weight (110.97 g/mol) / (24.45)): conversion to ppm = ($mg/m^3$) * 24.45/ (molecular weight (110.97 g/mol). https://www.cdc.gov/niosh/docs/2004-101/calc.html
3 Acute handler MOEs were calculated using an HEC of 226.94 ppm / max air concentration (ppm)); ST/IT Inhalation MOEs = ST/IT Inhalation POD (1.25 ppm) / (geometric mean air concentration (ppm)); highlighted MOEs reflect MOEs < LOC.
4 PF10 Respirator MOEs reflect a 10X reduction in exposure due to the use of a PF10 respirator. It is noted that many of the fumigant application labels may have various exposure mitigation measures in place, including respirators.

While HED has no worker exposure data to assess potential risks to occupational handlers or reentry workers resulting from the pre-plant drip irrigation uses, the post-plant vineyard use, or the turf uses of 1,3-D, Appendix E of the Phase 5 RED (Summary of 1,3-D Bystander Exposure

from Known Area Sources Estimated Using the Monitoring Method) utilized field volatility data based on a previous bystander exposure review of 1,3-D (01-NOV-2002, S. Weiss, D284547) as a surrogate data source to estimate occupational bystander risk. In summary, twenty studies[28], with field volatility data collected near 1,3-D treated fields, were submitted to the Agency since 1989. Some of the studies reflect application methods that are no longer used or are covered under the available occupational handler studies detailed in Table 11.1.1 (i.e., broadcast and row applications), and are therefore not included in this assessment. Several studies (MRID#s 447956-02, 451129-02, 452961-01) are used to estimate use of drip application methods. Bystander inhalation exposure estimates for the pre-plant turf uses of 1,3-D are based on air concentration measurements reported in field volatility studies, MRID 451207-01 and 451207-02. One field volatility study is available to address off-site exposure resulting from the post-plant vineyard use. Bystander inhalation exposure estimates are based on air concentration measurements reported in this study, (MRID 452961-01).

Due to the updates for the 1,3-D ST/IT inhalation HEC, HED has reassessed these scenarios using the previously reported highest seven-day air concentrations as reported by the study, and the label maximum application rate. Consistent with the referenced review of bystander exposure (Memo, 01-NOV-2002, S. Weiss, D284548), the consecutive seven-day average air concentration were also estimated from each field volatility study. The highest average seven-day average in each direction is compared to the updated ST/IT HEC to estimate ST risk for bystanders.

In the field volatility studies, 1,3-D peak off gassing occurs one to three days after application. Additionally, since 1, 3-D products are used only one to two times per field each year, the majority of bystander exposure resulting directly from treatment of agricultural fields is expected to be acute or ST. As the acute POD has not changed, and chronic exposure is not expected since it is unlikely that bystanders will be continually exposed to significant concentrations of 1,3-D for 6 consecutive months or longer, a quantitative acute and chronic (LT) re-assessment was not conducted.

HED evaluated risks from ST/IT exposures using the highest 7-day rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (5.68 mg/m$^3$ = 1.25 ppm). The ST/IT ambient MOEs range from 50 to 250 (LOC = 30). See Table 11.1.2 below for the summary of concentrations and the risk estimates.

**Table 11.1.2. Screening Assessment of Short- and Intermediate Term 1,3-D Risk for Occupational Handlers Based on Field Volatility Data[1]**

| | | | | | Application Rate | | Highest 7-day Air concentration | | ST/IT MOE[2] | |
|---|---|---|---|---|---|---|---|---|---|---|
| # | Study Location/ | MRID# | Formulation | Distance (m) | study (g/A) | label max (g/A) | study (ppm) | label max (ppm) | study | label max |
| **Drip Irrigation Applications** | | | | | | | | | | |
| | Rio Grande Valley, TX 1998 | 447956-02 | Telone EC | 30 | 8.65 | 18 | 0.012 | 0.025 | 100 | 50 |

[28] Study descriptions are available in Appendix A of the *REVISED Post-application Non-occupational Bystander Risk Estimates for Proposed Label Change from 300 to 100 foot Buffer Zone for Telone II, Telone C-17, and Telone C-35*, (01-NOV-2002, S. Weiss, D284547)

**Table 11.1.2. Screening Assessment of Short- and Intermediate Term 1,3-D Risk for Occupational Handlers Based on Field Volatility Data[1]**

| # | Study Location/ | MRID# | Formulation | Distance (m) | Application Rate | | Highest 7-day Air concentration | | ST/IT MOE[2] | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | study (g/A) | label max (g/A) | study (ppm) | label max (ppm) | study | label max |
| 15 | Douglas, GA; 2000 | 451129-02 | In-Line | 30 | 24.6 | 20.5 | 0.015 | 0.012 | 83 | 100 |
| 8 | Rio Grande Valley, TX 1998 | 447956-02 | Telone EC | 91 | 8.65 | 18 | 0.005 | 0.011 | 250 | 110 |
| 15 | Douglas, GA; 2000 | 451129-02 | In-Line | 91 | 24.6 | 20.5 | 0.006 | 0.005 | 210 | 250 |
| 20 | Gilroy, CA, 1998 | 452961-01 | Telone II | 91 | * | * | 0.0003 | | 4,200 | * |

1. Drip irrigation application data inputs (other than re-calculated ST/IT MOEs) sourced from Phase 5 RED (12-APR-2007, C. Olinger, D337328) Appendix E Table E.2.
2. ST/IT MOE = HEC (5.68 5.68 mg/m3 = 1.25 ppm)/highest 7-day air concentration. Conversion to ppm = ($mg/m^3$) * 24.45/ (molecular weight (110.97 g/mol). https://www.cdc.gov/niosh/docs/2004-101/calc.html

## 11.2 Acute and Short-/Intermediate-Term Occupational Post-Application Exposure and Risk Estimates

Occupational dermal post-application exposures are not expected given the high vapor pressure of 1,3-D. In addition, emission reduction techniques used (e.g., tarping) reduce potential exposures. Therefore, dermal exposures have not been quantitatively assessed. Occupational acute, ST/IT inhalation post-application exposures are expected from the registered uses of 1,3-D; LT exposures, or continuous exposures for more than 6 months per year, are not expected based on the seasonal nature of 1,3-D use.

Based on the high vapor pressure, application methods, and practices of 1,3-D, all activities associated with the application are handler activities. There is no expectation of soil or foliar dermal exposure to 1,3-D following an application. There is potential for inhalation exposure to 1,3-D following an application; however, all activities associated with the application are considered handler activities and are assessed/discussed Section 11.1. Entry restrictions into treated fields for activities such as tarp cutting, and tarp removal remain unchanged.

## 12.0 Incident and Epidemiological Data Review

A summary report listing incidents for 1,3-D reported to the OPP Incident Data System (IDS) has been conducted for this assessment (02-DEC-2019, S. Recore *et al*, D455269).

1,3-D incidents were last reviewed in May 2013 (15-MAY-2015, E. Evans and S. Recore, D411762). In 2013, HED prepared a preliminary Tier I human incident review of 1,3-D human incident reports by consulting the OPP Incident Data System (IDS) for reports of poisoning incidents. In 2013, based on the low frequency and severity of incident cases reported for 1,3-D in both IDS and NIOSH Sentinel Event Notification System for Occupational Risk (SENSOR)-Pesticides, further investigation was not warranted.

For the 1,3-D Tier II Incident and Epidemiology Report, HED found the majority of incidents involving 1,3-D were low in severity (82% in IDS and 89% in SENSOR-Pesticides). Most of the incidents reviewed for this memorandum reported that individuals experienced minor symptoms

such as burning eyes and coughing. These are symptoms, which were minimally traumatic and resolved rapidly, and are likely the result of chloropicrin exposure. Chloropicrin is used as a warning agent with other more toxic active ingredients, such as 1,3-D, because it has a strong odor and causes respiratory and eye irritation.

In IDS (71%), SENSOR-Pesticides (83%), and California's Pesticide Incident Surveillance Program (PISP) (96%) exposure from drift/volatilization was responsible for the most reported 1,3-D incidents. These incident events, often involving multiple cases, resulted from off-target drift or volatilization of the a 1,3-D product onto nearby farms, fields and residential areas. These events exposed workers and residents of neighboring communities.

Epidemiological studies investigating the association between 1,3-D and health outcomes available in the open literature were reviewed. Overall, there was insufficient evidence to suggest a clear associative or causal relationship exists between 1,3-D exposure and the health outcomes investigated in the studies reported here. The Agency will continue to monitor the epidemiology data, and -- if a concern is triggered – then additional analysis will be conducted.

## 13.0 References

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# Appendix A. Toxicity Profile and Executive Summaries

## A.1 Toxicology Data Requirements

The requirements (40 CFR 158.500) for food uses for 1,3-D are in Table 1. Use of the new guideline numbers does not imply that the new (1998) guideline protocols were used.

| Study | | Technical | |
|---|---|---|---|
| | | Required | Satisfied |
| 870.1100 | Acute Oral Toxicity | yes | yes |
| 870.1200 | Acute Dermal Toxicity | yes | yes |
| 870.1300 | Acute Inhalation Toxicity | yes | yes |
| 870.2400 | Primary Eye Irritation | yes | yes |
| 870.2500 | Primary Dermal Irritation | yes | yes |
| 870.2600 | Dermal Sensitization | yes | yes |
| 870.3100 | Oral Subchronic (rodent) | yes | yes |
| 870.3150 | Oral Subchronic (nonrodent) | yes | yes |
| 870.3200 | 21-Day Dermal | yes | no[A] |
| 870.3250 | 90-Day Dermal | CR | - |
| 870.3465 | 90-Day Inhalation | yes | yes |
| 870.3700a | Developmental Toxicity (rodent) | yes | yes |
| 870.3700b | Developmental Toxicity (nonrodent) | yes | yes |
| 870.3800 | Reproduction | yes | yes |
| 870.4100a | Chronic Toxicity (rodent) | yes | yes |
| 870.4100b | Chronic Toxicity (nonrodent) | yes | yes |
| 870.4200a | Oncogenicity (rat) | yes | yes |
| 870.4200b | Oncogenicity (mouse) | yes | yes |
| 870.5100 | Mutagenicity—Gene Mutation - bacterial | yes | yes |
| 870.5300 | Mutagenicity—Gene Mutation - mammalian | yes | yes |
| 870.5xxx | Mutagenicity—Structural Chromosomal Aberrations | yes | yes |
| 870.6100a | Acute Delayed Neurotoxicity (hen) | no | no |
| 870.6100b | 90-Day Neurotoxicity (hen) | no | no |
| 870.6200a | Acute Neurotoxicity Screening Battery (rat) | yes | waived[B] |
| 870.6200b | 90-Day Neurotoxicity Screening Battery (rat) | yes | waived[B] |
| 870.6300 | Develop. Neurotoxicity | CR | - |
| 870.7485 | General Metabolism | yes | yes |
| 870.7600 | Dermal Penetration | CR | - |
| 870.7800 | Immunotoxicity | yes | yes |

[A]: highly volatile chemical; very low dermal exposure anticipated relative to inhalation; realistic quantification of dermal risk is not possible (study not required).

[B] :HASPOC TXR 0056626;

CR = Conditionally Required.

## A.2 Acute Toxicology

### Acute Profile of 1,3-Dichloropropene (1,3-D)

**Table A2.1. Acute Toxicity profile of 1,3-Dichloropropene**

| Guideline No. | Study Type | MRID # | Results | Toxicity Category |
|---|---|---|---|---|
| 870.1100 | Acute Oral Toxicity – rat | 40220901 | $LD_{50}$ = 224 mg/kg (F)<br>$LD_{50}$ = 300 mg/kg (M) | II |
| 870.1200 | Acute Dermal Toxicity - rat | 40220902 | $LD_{50}$ = 333 mg/kg (M and F) | II |
| 870.1300 | Acute Inhalation Toxicity – rat | 40220903<br>40543301 | 3.88 mg/L > $LC_{50}$ < 4.69 mg/L (M)<br>$LC_{50}$ = 4.10 (F) | IV |
| 870.2400 | Acute Eye Irritation - rabbit | 40220904 | Severe irritant | II |
| 870.2500 | Acute Dermal Irritation - rabbit | 40220905 | Slight irritant | III |
| 870.2600 | Skin Sensitization - guinea pig | 40220906 | Sensitizer | NA |

## A.3 Profile for Subchronic, Chronic, and Other Toxicity Studies for 1,3-D.

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| Acute inhalation studies employed for toxicity endpoint and point of departure (POD) selection | | |
| Acute inhalation studies in rats (Single dose) | 00032985 (1976)<br>454, 647, 699, 762, 832, or 958 ppm. Whole body 4 hrs. exposure<br><br>41672201 (1990)<br>583, 771, or 1020 ppm<br>Whole body 4 hrs. exposure | The results of these two studies were considered together to yield the following results and used to establish POD:<br>NOAEC = 454 ppm.<br>LOAEC = 583 ppm based on decreased body weights. Body weight decreases observed at or ≥ 583 ppm were the outcome of a single exposure to 1,3-D during the acute inhalation toxicity study (MRID 41672201). These decreases were first manifested on day 2 of the study (one day after cessation of exposure) and persisted for 7 days. The clinical signs and deaths were seen at or ≥647 ppm. |
| Subchronic Toxicity Studies | | |
| 870.3100<br>90-Day oral toxicity rodents<br>[Fischer 344 rats] | 42954802 (1993)<br>Acceptable/Guideline<br>0, 5, 15, 50, or 100 mg/kg/day in diet | NOAEL = 5 mg/kg/day.<br>LOAEL = 15 mg/kg/day based on hyperkeratosis and/ or basal cell hyperplasia in the non-glandular portion of the stomach (both sexes). |
| 870.3100<br>90-Day oral toxicity rodents<br>[B6C3F1 mice] | 42954801 (1993)<br>Acceptable/Guideline 0,15, 50, 100, or 175 mg/kg/day in diet | NOAEL = 15 mg/kg/day.<br>LOAEL = 50 mg/kg/day based on decreased body weights and body-weight gain (both sexes). |
| 870.3100<br>90-Day oral toxicity nonrodent | See 870.4100b, below | |
| 870.3465<br>30-Day inhalation toxicity rodent [Fischer 344 rats] | 00039685 (1978)<br>Acceptable/Guideline<br>0, 3, 10, or 30 ppm (0, 0.0136, 0.045, or 0.136 mg/L) 6 hours/day, 5 days/week | NOAEL = 30 ppm (0.136 mg/L), highest dose tested.<br>LOAEL => 30 ppm. |
| 870.3465<br>30-Day inhalation toxicity rodent [CD-1 mice] | 00039685 (1978)<br>Acceptable/Guideline<br>0, 3, 10, or 30 ppm (0, 0.0136, 0.045, or 0.136 mg/L) 6 hours/day, 5 days/week | NOAEL = 30 ppm (0.045 mg/L), highest dose tested.<br>LOAEL => 30 ppm (0.136 mg/L). |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| 870.3465<br>90-Day inhalation toxicity rodent [Fischer 344 rats] | 00146461 (1984)<br>Acceptable/Guideline<br>0, 10, 30, 90, or 150 ppm (0, 0.045, 0.136, 0.408, or 0.680 mg/L) 6 hours/day, 5 days/week | NOAEL = 10 ppm (0.045 mg/L).<br>LOAEL = 30 ppm (0.136 mg/L) based on histopathological lesions in the nasal turbinates. |
| 870.3465<br>30-Day inhalation toxicity rodent [B6C3F1 mice] | 00146461 (1984)<br>Acceptable/Guideline<br>0, 10, 30, 90, or 150 ppm (0, 0.045, 0.136, 0.408, or 0.680 mg/L) 6 hours/day, 5 days/week | NOAEL = 10 ppm (0.045 mg/L).<br>LOAEL = 30 ppm (0.136 mg/L) based on histopathological lesions in the nasal turbinates. |
| Developmental and Reproductive Toxicity Studies | | |
| 870.3700a<br>Prenatal developmental in rodents<br>[Fischer 344 rats] | 00144715, 00152848 (1983)<br>Acceptable/Guideline<br>0, 20, 60, or 120 ppm (0, 0.091, 0.272, or 0.545 mg/L) by inhalation 6 hours/day during gestation days 6 through 15 | Maternal NOAEL = <20 ppm (0.091 mg/L).<br>LOAEL = 20 ppm (0.091 mg/L) based on decreased body-weight gain and food consumption.<br>Developmental NOAEL <120 ppm (0545 mg/L), highest concentration tested.<br>LOAEL =120 ppm (0545 mg/L) based on increased delay in ossification of the vertebral centra. |
| 870.3700b<br>Prenatal developmental in nonrodents<br>[New Zealand White Rabbit] | 00144715, 00152848 (1983)<br>Acceptable/Guideline<br>0, 20, 60, or 120 ppm (0, 0.091, 0.272, or 0.545 mg/L) by inhalation 6 hours/day during gestation days 6 through 18 | Maternal NOAEL = 20 ppm (0.091 mg/L).<br>LOAEL = 60 ppm (0.272 mg/L) based on decreased body-weight gain.<br>Developmental NOAEL 120 ppm (0545 mg/L), highest concentration tested.<br>LOAEL >120 ppm (0545 mg/L). |
| 870.3800<br>Reproduction and fertility effects<br>[Fischer 344 rats] | 40312401, 40835301(1987)<br>Acceptable/Guideline<br>0, 10, 30, or 90 ppm (0, 0.045, 0.136, or 0.408 mg/L) by inhalation 6 hours/day, 5 days/week<br>(premating) 6 hours/day, 7 days/week ($F_0$ breeding at weeks 11-13, during gestation and lactation; $F_{1a}$ and $F_{1b}$, dams from gestation day 20 until postpartum day 5; $F_1$ ♂♀ parents after weaning and continued for 12 weeks, 5 days/week | Parental/Systemic NOAEL = 30 ppm (0.136 mg/L).<br>LOAEL = 90 ppm (0.408 mg/L) based on decreased body-weight gain, microscopic non-glandular stomach lesions and hyperplasia of the nasal respiratory epithelium with focal degeneration of the olfactory tissue.<br><br>Reproductive NOAEL = 90 ppm (0.408 mg/L), highest concentration tested.<br>LOAEL >90 ppm (0.408 mg/L).<br><br>Offspring NOAEL = 90 ppm (0.408 mg/L), highest concentration tested.<br>LOAEL >90 ppm. |
| Chronic Toxicity Studies | | |
| 870.4100b<br>Chronic toxicity nonrodent<br>[Beagle dog] | 42441001, 42922301 (1992)<br>Acceptable/Guideline<br>0, 0.5, 2.5, or 15 mg/kg/day microcapsules by **dietary** admix | NOAEL = 2.5 mg/kg/day.<br>LOAEL = 15 mg/kg/day based on decreased body-weight gain, microcytic anemia, an increase in hematopoietic activity in both sexes and possible increased liver weights in males. |
| 870.4300<br>Combined Chronic Oral Toxicity/Carcino-genicity for 2-year rat study<br>[Fischer 344 rats] | 42912001, 43763501 (1995)<br>Acceptable/Guideline<br>0, 2.5, 12.5, or 25 mg/kg/day microcapsules by **dietary** admix | Chronic Toxicity NOAEL = 2.5 mg/kg/day.<br>LOAEL = 12.5 mg/kg/day based on decreased body-weight gain and an increase in the incidence of basal cell hyperplasia of the non-glandular mucosa of the stomach.<br>Carcinogenicity: Increased incidence of rats with primary hepatocellular adenomas: 0, 2.5, 12.5 and 25 mg/kg/day = ♂ 2/50, 1/50, 6/50 and 9/50; ♀ 0/50, 0/50, 0/50 and 4/50. |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| 870.4300<br>Combined Chronic Toxicity/Carcinogenicity (104 week)<br>[Fischer 344 rats] | 00141492, 00144714, 00146469 (1985)<br>NTP study<br>Acceptable/Guideline<br>0, 25, or 50 mg/kg/day by **oral gavage** 3 times/week for 104 weeks | Chronic Toxicity NOAEL = not established.<br>LOAEL = 25 mg/kg/day based on increased tumor incidence<br>Carcinogenicity: Increased incidence of squamous cell papillomas of the forestomach: 0, 25 and 50 mg/kg/day = ♂ 1/52, 1/52 and 9/52; ♀ 0/52, 2/52, 3/52. Squamous cell carcinomas: ♂ 0/52, 0/52 and 4/52. Neoplastic nodules of the liver: ♂ 1/52, 6/52 and 7/52: ♀ 6/52, 6/52, 10/52.<br>NTP concluded that there was "clear evidence of carcinogenicity" in males and "some evidence" of carcinogenicity in females. |
| 870.4300<br>Combined Chronic Toxicity/Carcinogenicity (104 week)<br>[B6C3F1 mice] | 43757901 (1995)<br>Acceptable/Guideline<br>0, 2.5, 25, or 50 mg/kg/day microcapsules by **dietary** admix | Chronic toxicity NOAEL = 2.5 mg/kg/day.<br>LOAEL = 25 mg/kg/day based on lower body weights and decreased body-weight gain (both sexes).<br><br>Carcinogenicity: No evidence of carcinogenicity but study not adequate for assessment due to several deficiencies in conduct. |
| 870.4300<br>Combined Chronic Toxicity/Carcinogenicity (104 week)<br>[B6C3F1 mice] | 00141492, 00144714, 00146469 (1985)<br>NTP study<br>Acceptable/Guideline<br>0, 25, or 50 mg/kg/day by **oral gavage** 3 times/week for 104 weeks | Chronic Toxicity NOAEL = not established.<br>LOAEL = 25 mg/kg/day based on increased mortality in males<br>Carcinogenicity: Increased incidence of squamous cell papillomas of the forestomach: 0, 25, and 50 mg/kg/day = ♀ 0/50, 1/50, 2/50. Squamous cell carcinomas of thee forestomach: ♀ 0/50, 0/50, 2/50. Transitional cell carcinomas of the urinary bladder: ♀ 0/50, 8/50, 21/50.<br>Alveolar/bronchiolar adenomas: ♀ 0/50, 3/50, 8/50.<br>In ♂, study was inadequate for carcinogenicity.<br>NTP concluded that there was "clear evidence of carcinogenicity" in females. |
| 870.4300<br>Combined Chronic Toxicity/Carcinogenicity (2 years)<br>[Fischer 344 rats] | 40312201 (1987)<br>Acceptable/Guideline<br>0, 5, 20, or 60 ppm (0, 0.023, 0.091, or 0.272 mg/L) by **inhalation** 6 hours/day, 5 days /week for 509 days | Chronic Toxicity NOAEL = 20 ppm (0.091 mg/L).<br>LOAEL = 60 ppm (0.272 mg/L) based on histopathological changes in nasal tissue (males and females) and a suggestion of decreased body-weight gain (first year of the study only).<br><br>There was no evidence of carcinogenicity. |
| 870.4300<br>Combined Chronic Toxicity/Carcinogenicity (2 years)<br>[B6C3F1 mice] | 40312301 (1987)<br>Acceptable/Guideline<br>0, 5, 20, or 60 ppm (0, 0.023, 0.091, or 0.272 mg/L) by **inhalation** 6 hours/day, 5 days /week for 510 days | Chronic Toxicity NOAEL = 5 ppm (0.023 mg/L).<br>LOAEL = 20 ppm (0.091 mg/L) based urinary bladder hyperplasia, and hypertrophy/hyperplasia of the nasal respiratory mucosa.<br><br>Carcinogenicity: Increased incidence of bronchioloalveolar adenomas: 0, 5, 20, or 60 ppm = ♂ 9/50, 6/50, 13/50, or 22/50. Although the lung tumors were benign, tumor induction was concentration dependent, the tumor incidence was dose dependent, the tumor incidence was outside of the historical controls, and the tumor type was seen in the mouse oral bioassay. |
| Genotoxicity Studies | | |
| Gene Mutation<br>870.5300<br>*In vitro* mammalian cell in culture gene mutation assay<br>Chinese hamster ovary (CHO) cells | 00159679 (1986)<br>Acceptable/Guideline<br>50-250 µM -S9<br>50-200 µM +S9 | Negative up to cytotoxicity (≥200 µM -S9) or the highest dose tested +S9. |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| Gene Mutation<br>870.5300<br>*Drosophila melanogaster* sex-linked recessive lethal mutations | 00146469 (1985)<br>Acceptable/Guideline<br>0, 5750 ppm/feeding | Positive: Induction of sex-linked recessive lethal mutations but negative for the induction of reciprocal translocations at 5750 ppm. National Toxicology Program (NTP); Valencia *et al.*, Environ Mutagenesis 7:325-348. |
| Gene Mutation<br>870.5300<br>Host Mediated assay | 00039688 (1978)<br>Acceptable/Guideline<br>0, 30, 60 mg/kg (oral gavage administration at 1, 2, & 3 hrs) | Negative up to the highest dose tested. |
| Cytogenetics<br>870.5375<br>*In vitro* mammalian cytogenetics assay<br>CHO cells | NTP (1989)<br>Acceptable/Guideline<br>4.91-49.1 µg/mL -S9 (Trial 1)<br>50-100 µg/mL -S9 (Trial 2)<br>10-50 µg/mL +S9 (Trial 1 only) | Negative up to concentrations causing 50% reduction in cell confluency (≥50 µg/mL ± S9). NTP: Loveday *et al.*, Environ Mutagenesis 13:6–94. |
| Cytogenetics<br>870.5395<br>*In vivo* mouse micronucleus assay | 00146468 (1985)<br>Acceptable/Guideline<br>0, 38, 115, 380 mg/kg | Negative up to a lethal dose (380 mg/kg). |
| Cytogenetics<br>870.5450<br>Dominant Lethal Mutation in Sprague Dawley Rats | 44302801 (1997)<br>Acceptable/Guideline<br>0, 10, 60, or 150 ppm, 7 day/wk, 10 wks (whole body inhalation) | Negative up to the LOAEL of 150 ppm, based on adverse effects on body weight. |
| Other Effects<br>870.5500<br>Bacterial DNA repair *Bacillus subtilis* H15 & M45 | 00039688 (1978)<br>Acceptable/Guideline<br>50-1,250 µg/well | Positive: Preferential inhibition of the DNA repair deficient strain at 1250 µg/well. |
| Other Effects<br>870.5550<br>Unscheduled DNA Synthesis Primary rat hepatocytes | 00146467 (1985)<br>Acceptable/Guideline<br>$3x10^{-3}$ to $1x10^{-6}$ M | Negative up to a cytotoxic level ($3 \times 10^{-4}$ M). |
| Other Effects<br>870.5900<br>*In vitro* sister chromatid exchange (SCE)<br>CHO cells | NTP (1989)<br>Acceptable/Guideline<br>0.995-29.900 µg/mL -S9 (Trial 1)<br>30-50 µg/mL -S9 (Trial 2)<br>2.990-29.900 µg/mL +S9 (Trial 1 only) | Positive: Significant and concentration-related ↑in SCE induction at 30-50 µg/mL -S9 & 10 -30 µg/mL +S9. These levels were not cytotoxic. |
| METABOLITES OF 1,3-D | | |
| Gene Mutation<br>870.5100<br>*In vitro* bacterial reverse gene mutation assay *Salmonella typhimurium* TA98, TA100, TA1535, TA1537<br>*Escherichia coli* WP2 *(uvrA)* | 44940327 (1999)<br>3-Chloroacrylic acid<br>Acceptable/Guideline<br>500 - 5000 µg/plate ±S9 | Negative in independently performed preincubation assays up to the limit concentration. |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| Gene Mutation<br>870.5100<br>*In vitro* bacterial reverse gene mutation assay *Salmonella typhimurium* TA98, TA100, TA1535, TA1537<br>*Escherichia coli* WP2 *(uvrA)* | 44940326 (1999)<br>3-Chloroallyl alcohol<br>Acceptable/Guideline<br>33.3 - 5000 µg/plate ±S9 | Negative in independently performed preincubation assays up to the limit concentration. |
| Gene Mutation<br>870.5300<br>*In vitro* mammalian cell in culture gene mutation assay<br>mouse lymphoma L5178Y | 44940311 (1999)<br>3-Chloroallyl alcohol<br>Acceptable/Guideline<br>Trial 1: 12.5- 925 µg/mL -S9; 1.5-100 µg/mL +S9<br>Trial 2: 12.5- 500 µg/mL -S9; 3-100 µg/mL +S9 | Positive: Dose-related and reproducible ↑ MF at 400 and 500 µg/mL -S9 & 75 and 100 µg/mL +S9: no difference in the induction of small or large mutant colonies. |
| Cytogenetics<br>870.5395<br>*In vivo* mouse (CD-1) micronucleus assay | 44940312 (1999)<br>3-Chloroacrylic acid<br>Acceptable/Guideline<br>62.5-250 mg/kg (♂)<br>62.5-200 mg/kg (♀) | Negative up to overtly toxic (death and/or decreased activity) highest doses. |
| Immunotoxicity<br>870.7800<br>(sheep red blood cell assay) | 49074801 (2010)<br>28-day dietary<br>male F344/DuCrl Rats<br>0, 2.5, 7.5, or 25 mg/kg/day (time-weighted average doses of 2.8, 8.4, and 26.7 mg/kg). | Systemic toxicity NOAEL = 8.4 mg/kg/day. LOAEL = 26.7 mg/kg/day based on reduced body-weight gain and food consumption.<br><br>Immunotoxicity: No effect was found.<br>NOAEL = 26.7 mg/kg/day (HDT). |
| MECHANISM STUDIES | | |
| Gene Mutation<br>*In vitro* bacterial reverse gene mutation assay *Salmonella typhimurium* TA100 plus mouse lung homogenate | 44460501 (1998)<br>Unacceptable/Non-guideline<br>100-450 µg/plate - metabolic activation;<br>150-1000 µg/plate + untreated mouse lung homogenate - glutathione (GSH)or 75-1000 µg/plate +GSH; 75-1000 µg/plate ± GSH + mouse lung S9 (pretreated with 63 ppm 1,3-D, 5da/wk, 2.5 wks) | Negative at any concentration ± GSH and ± untreated or 1,3-D lung S9 but under conditions that would not favor a mutagenic response -S9 (no epoxidized soybean stabilizer, purification through silicic column and storage at 5°C under $N_2$) or +S9 because of low level microsomal/oxidative proteins in the lung preparation. |
| Gene Mutation<br>*In vivo* with transgenic Big Blue B6C3F1 mice gene mutation assay<br>target gene *(lacI)* | 44470501 (1998)<br>Unacceptable/Non-guideline<br>0, 10, 60, 150 ppm (via whole body inhalation) 6 hrs/da, 5 da/wk, 2 wks | Negative in lung and liver tissue but test system uncertainties weaken the understanding of the negative response. |
| Other Mutagenic Effects<br>*In vitro* DNA binding assays | 44446301 (1997)<br>Unacceptable/Non-guideline<br>11 mM reacted w calf thymus DNA ± S9 (Aroclor 1254-induced rat liver) ± GSH | Inconclusive -S9; negative +S9 ± GSH but uncertainties regarding use of optimum conditions. |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| Mechanism of Tumorigenicity ♂B6C3F1 mice & ♂ Fischer 344 rats | 44446302 (1997) Unacceptable/Non-guideline 0, 5,12.5, 25, 100 mg/kg (oral gavage-rats) 3, 12, 26 days 0, 10, 30, 60, 150 ppm (whole body inhalation- mice) 6 hrs/da, 5 da/wk, 2 wks | RATS: No mortality or clinical signs S ↓GSH at 25 & 100 mg/kg (adaptive process) but liver tumors were seen in the 2-yr bioassay at 12.5 mg/kg. No conclusion possible for apoptosis or cell proliferation because of variability in data. No conclusion possible for DNA adduct formation because of variability in data & small sample size.<br><br>MICE: No mortality or clinical signs. Data show conjugation of 1,3-D w GSH in lung tissue, no clear effect on cell proliferation or apoptosis in bronchiole epithelium or bladder transitional cells or DNA adduct formation in lungs, but extreme variability and small sample size compromised the findings.<br><br>Concerns regarding whether a biological effective dose was achieved. |
| GSH Activity in Several Mammalian Cell Lines: ♂B6C3F1 mice & ♂ Fischer 344 rats primary rat hepatocytes, CHO cells, Chinese hamster lung cells, *Salmonella typhimurium* | 44460503 (1998) Unacceptable/Non-guideline GSH measurements in cell lines reacted with various substrates: $^{13}$C-1,3-D; 4-chloro-1,3-dinitrobenzene; para-nitro-phenylethylbromide; trans-4-phenyl-3-buten-2-one | No conclusions relative to the correlation between physiological levels of GSH and mitigation of mutagenicity.<br><br>Low level GSH activity with *S. typhimurium* but conflicting results with various mammalian cell lines (*i.e.*, high & low level GSH activity with cell lines producing negative mutagenicity data and high GSH activity with 2 cell lines that were positive in standard mutagenicity assays). |
| Bioavailabity of Microencapsulated Telone II in Female Rats | 44460502 (1996) Unacceptable/Non-guideline Phase I: 25 mg/kg $^{13}$C-1,3D co-administered w 25 mg/kg microencapsulated 1,3-D sampled at 1,3,5,10,15, 20, 30,40,50, or 60 min. Phase II: 25 or 50 mg/kg $^{13}$C-1,3D + microencapsulated 1,3-D; 25 or 50 mg/kg $^{13}$C-1,3D + 7.5 or 15 mg/kg microencapsulated 1,3-D | No conclusions because of unclear study design, technical deficiencies and biased treatment of the data. |
| Initiation-Promotion: Mechanism of Mouse Lung Tumors ♂A/J mice | 45897502 (2003) B&D Unacceptable/Non-guideline 16 mg/kg vinyl carbamate (VC) (initiator) ± 0, 60 ppm 1,3-D (whole body inhalation 6 hrs/da, 5 da/wk, 25 wks) | Lung adenomas in 1,3-D alone 26% vs 10% in air control suggest initiating event. Lack of ↑total tumors for VC-treated alone vs. VC + 1.3-D does not support a promoter role for 1,3-D. |
| Initiation-Promotion: Mechanism of Rat Liver Tumors ♂ Fischer 344 rats | 45897502 (1998) C&D Unacceptable/Non-guideline 100 mg/kg diethylnitrosamine (DEN, initiator) + 0, 25 mg/kg/day 1,3-D; 80 mg/kg phenobarbital (PB, promoter); or 5-10 mg/kg 2-acetylaminofluorene (2-AAF, complete carcinogen) | Data do not support a promotional role for 1,3-D. |

| Guideline No./ Study Type | MRID No./ Classification /Doses | Results |
|---|---|---|
| Inhalation and steady-state pharmacokinetics in B6C3F1 mice (Nose-only, 6hr/day for 15 days). | 50715302 (2018)<br>Acceptable/Non-guideline<br>0, 10, 20, 40, 60, 90, or 120 ppm. On Day 15, blood samples were collected and processed to determine the concentrations of *cis*- and *trans*-1,3-dichloropropene. | Dose-related decrease in mean respiratory rate and minute volumes as concentrations increased from 20 to 120 ppm on day 1. From the analyses of the 3-parameter model, there was high confidence that the relationship between 1,3-D concentration in male mice blood and inhalation exposure was non-linear (and non-proportional) at exposure levels of 40 ppm and above. The study report also presented results from the ratio analysis of CIS indicated the relationship between blood concentration and exposure dose was not proportional at 60 ppm and greater exposure level (non-proportional relationship could start at a lower exposure level, but there was no evidence). However, the results of ratio analyses of *trans* and *total* indicated the relationship was non-proportional at 40 ppm and above exposure concentrations. |
| Inhalation and steady-state pharmacokinetics in B6C3F1 mice (Nose-only, 6hr/day for 15 days). | 50715302 (2018)<br>Acceptable/non-guideline<br>0, 10, 20, 40, 60, 90, or 120 ppm. On Day 15, blood samples were collected and processed to determine the concentrations of cis- and trans-1,3-D. | Dose-related decrease in mean respiratory rate and minute volumes as concentrations increased from 20 to 120 ppm on day 1. From the analyses of the 3-parameter model, there was high confidence that the relationship between 1,3-D concentration in male mice blood and inhalation exposure was non-linear (and non-proportional) at exposure levels of 40 ppm and above. The study report also presented results from the ratio analysis of CIS indicated the relationship between blood concentration and exposure dose was not proportional at 60 ppm and greater exposure level (non-proportional relationship could start at a lower exposure level, but there was no evidence). However, the results of ratio analyses of *trans* and *total* indicated the relationship was non-proportional at 40 ppm and above exposure concentrations. |
| Metabolism | MRID 00155846 (TXR 0005734)<br><br>Single oral gavage dose of 1 or 50 mg/kg in rats<br><br>Single oral gavage of 1 or 100 mg/kg in mice | The primary route of excretion for both species was the urine. Following oral administration, most of the radiolabel was found in the stomach and gastrointestinal tract with lesser amounts in the kidneys, liver, urinary bladder, skin, fat, blood and carcass. Oral administration also depleted the non-protein-sulfhydryl contents of several tissues including the non-glandular stomach (both time- and dose-dependent). Dose-related increases in macromolecular bindings were noted in several organs with the highest binding sites being found in the non-glandular stomach. The two major urinary metabolites were identified as 1,3-DCP-mercapturic acid and its sulfoxide (or sulfone) derivative. |
| Metabolism | MRID 40959801 (TXR 0007131)<br><br>5 mg/kg as single or repeated (14-day) oral gavage | Single or repeated doses rapidly absorbed, distributed to all examined tissues, and completely metabolized to at least 9 metabolites found in urine. Radioactivity cleared from tissues within 48 hours postdosing. No sex-related differences. |

## Appendix B. Literature Search Details

Date and Time of Search: 08/07/2018; 02:49 pm
Date and Time of Search: 02/21/2019; 08:08 am (NO CHANGE in Hits)

Search Details:
((*Telone*) OR (*Telone 1,3-D*)) AND (rat OR mouse OR dog OR rabbit OR monkey OR mammal)

PubMed* hits: 8
Number of Swift** Articles: 6 for Animal
Number of Swift Articles: 6 for Human
Number of Swift Articles: 0 for No Tag

All studies identified in the PubMed search were screened when the citation list was ≤100. Screening of larger citations lists (>100 citations) was conducted after prioritization in SWIFT-Review and focused on studies identified with the "Animal" and/or "Human" tag.

Conclusion of Literature Search: Following title/abstract and/or full text screening, no studies were identified as containing potentially relevant information (either quantitative or qualitative) for the 1,3-D human health registration review risk assessment.

*PubMed is a freely available search engine that provides access to life science and biomedical references predominantly using the MEDLINE database.
**SWIFT-Review is a freely available software tool created by Sciome LLC that assists with literature prioritization. SWIFT-Review was used to prioritize studies identified in the PubMed search based on the model of interest in the study (e.g., human, animal, *in vitro*, etc.). Studies could have resulted in multiple tags which would account for citations identified in PubMed not matching the number of tagged citations."

## Appendix C. Physical/Chemical Properties

| Table C.1. Physicochemical Properties of the Technical Grade Test Compound. | | |
|---|---|---|
| **Parameter** | **Value/Description** | **Reference** |
| Physical State | Near colorless, oily liquid | |
| Melting point/range | -69.2°C | Merck Index |
| Boiling point/range | 112°C at 757 mm H | Merck Index |
| Density | 1.7 g/ml at 25°C | 09-OCT-2004, K. Dockter, D268927 |
| Water Solubility | at 25 °C: 2,180 mg/L (cis isomer), 2,320 mg/L (trans isomer) | USEPA-738-R-98-016 |
| Solvent Solubility | 70.5 g/mL at 25°C in acetone, ethyl alcohol, hexane, and xylene | 09-OCT-2004, K. Dockter, D268927 |
| Vapor Pressure | 23.8 mm Hg at 25°C | 09-OCT-2004, K. Dockter, D268927 |
| Dissociation constant ($pK_a$) | Not applicable, nonpolar | 09-OCT-2004, K. Dockter, D268927 |
| Octanol/water partition coefficient | Log ($K_{OW}$) = 2.58 | 09-OCT-2004, K. Dockter, D268927 |

## Appendix D. Review of Human Research

The chemical-specific studies used to develop the previous occupational handler assessment have been reviewed for compliance with required ethical standards for conducting human studies.

The ethic reviews covering each study MRID are as follows:

- M. Arling. 02-DEC-2019; Ethics Review of Applicator and Worker Exposure Monitoring Study Involving Telone (1,3-Dichloropropene)
    - Houtman, B. An Evaluation of 1,3-Dichloropropene Worker Exposures Associated with TELONE Soil Fumigant Loading, Application and Re-entry – Final Report. September 29, 1993. Unpublished Report sponsored by DowElanco. 76p. (MRID 42946201)
    - Houtman, B. An Evaluation of 1,3-Dichloropropene Worker Exposures Associated with TELONE Soil Fumigant Loading, Application and Re-entry – Phase 3. Interim Report. July 14, 1993. Unpublished Report sponsored by DowElanco. 55p. (MRID 42945602)
- M. Arling. 02-DEC-2019; Ethics Review of Applicator and Worker Exposure Monitoring Study Involving Telone (1,3-Dichloropropene)
    - Dupuis, M.R. *et al.* An Evaluation of Worker Exposure to Airborne Concentrations of 1,3-Dichloropropene During Loading and Application of Telone Soil Fumigants Packaged in Traveler Cylinders. August 30, 1995. Unpublished Report sponsored by DowElanco. 76p. (MRID 43880401)
- M. Arling. 02-DEC-2019; Ethics Review of Worker Exposure Monitoring Study Involving 1,3-Dichloropropene
    - Rotondaro, A. 1,3-Dichloroproene Exposure to Workers Tarping Beds During Pre-Bed Fumigation with 1,3-D Soil Fumigants. April 17, 2001. Unpublished Report sponsored by Dow AgroSciences. 115p. (MRID 45400202)

## Appendix E. 1,3-D Application Specific Flux Studies

| MRID | Study Title |
|---|---|
| 45296101 | Beard, K.; Mueller, J.; Stolz, E.; Dolder, S. (1998) 1,3-Dichloropropene Air Monitoring; Post-Plant Drip Application in Grapes. 40p. |
| 47813901 | Ajwa, H.; Sullivan, D.; Chellemi, D. (2009) Monitoring 1,3-Dichloropropene, Chloropicrin and Methyl Isothiocyanate Emissions from Shank Applications at Three Sites Near Duette, Florida. Project Number: 2009A. Unpublished study prepared by University of California, Sullivan Environmental Consulting and JRF America. 114 p. |
| 48085701 | Ajwa, H.; Sullivan, D.; Chellemi, D. (2010) Monitoring 1,3-Dichloropropene and Cloropicrin Emissions from Solid-Tarp Shank Injections at Two Sites Near Fort Pierce, Florida. Project Number: 2009H. Unpublished study prepared by University of California and Sullivan Environmental Consulting. 146 p. |
| 48888301 | Sullivan, D.; Ajwa, H.; Sullivan, R.; et al. (2012) Monitoring of Flux from Soil Injection Application Method of 1,3 Dichloropropene and Chloropicrin for Four Different Tarping Materials. Project Number: SEC2012A. Unpublished study prepared by Sullivan Environmental Consulting, University of California and AJK Consultancy. 247p. |
| 48917601 | Ajwa, H.; Sullivan, D. (2012) Soil Fumigant Emissions Reduction Using EVAL Barrier Resin Film (VaporSafe) and Evaluation of Tarping Duration Needed to Minimize Fumigant Total Mass Loss. Project Number: HA2011A/OCR, 263977. Unpublished study prepared by Sullivan Environmental Consulting and University of California. 404p. |
| 49031901 | Sullivan, D.; Ajwa, H.; Sullivan, R.; et al. (2013) Monitoring of Flux from Soil Injection Application Method of 1,3 Dichloropropene and Chloropicrin for Four Different Tarping Materials. Project Number: SEC2011B. Unpublished study prepared by Sullivan Environmental Consulting , University of California, Davis. 221p. |

## Appendix F. SOFEA 4 Data Assumptions for Acute and ST/IT Risk Estimate Percentiles

The following language and data assumptions were submitted by DAS and are summarized as presented in "*SOFEA modeling of 24-hour, 28-day and annual average 1,3-D concentrations in ambient air in intensive 1,3-D use areas in Washington, North Carolina, and Florida in 2015/16 and 2016/17 - Final Report (Revised)*" (MRID 50594901).

Since the exact field locations and 1,3-D application parameters were known the SOFEA model was used in "retrospective mode." The model ran for two consecutive one-year simulations using equally spaced receptors (500m receptor spacing) in the study area where hourly 1,3-D concentrations in ambient air were predicted.

**Regional Selections and Data Inputs**

*Pacific Northwest (PNW):*

>15% of 1,3-D sold in the USA is applied annually in WA, primarily to annual crops such as potatoes and onions, with some tree and vine and vegetable crop uses as well. A study area spanning approximately 1900 square miles, to the southeast of the town of Quincy, WA contains a significant amount of agricultural land planted in potato and onion that is regularly fumigated with 1,3-D and is considered representative of the PNW region.

During the 2015/16 use season, growers in the WA study area applied a total of 1,110,559 kg (2,448,782 pounds) of 1,3-D in the WA study area with 138 applications on a total of 6015 hectares (ha) (14,857 acres) of land. The average treated field area was approximately 43.6 ha (~107 acres). During the 2016/17 use season, growers applied a total of 849,446 kg (1,873,028 pounds) of 1,3-D in the WA study area with 107 applications on a total of 4,784 ha (11,768 acres) of land. The average treated field area was approximately 44.5 ha (~110 acres). The product use data used to parameterize SOFEA for 2015/16 and 2016/17 years of modeling in WA is shown in the Excel worksheet titled *WA_Year1_data.csv* and *WA_Year2_data.csv*. SOFEA reads the product use data directly from the Excel spreadsheets. The 1,3-D use by month during the 2015/16 and 2016/17 study seasons is consistent with historical use patterns in Washington, with major use in the fall (Oct/Nov) and the spring (March), and no use between April and July.

The SOFEA simulations in the Quincy, WA study area were conducted on a 70 km x 70 km land area (1,892 square miles) containing 19,881 receptors spaced 500m apart. The simulation spanned two calendar years (6/1/2015 through 5/31/2016 and 6/1/2016 through 5/31/2017), with each year simulated individually. SOFEA simulated concentrations and probability distributions of 24-hour, 28-day, 90-day moving average (MA) and annual average 1,3-D concentrations for the WA study area, for 2015/16 and 2016/17 are shown in Table F.2.

*Mid-Atlantic states:*

>10% of 1,3-D sold in the USA is applied annually in North Carolina, primarily to annual crops such as tobacco and market vegetables including sweet potatoes. A study area spanning approximately 160 square miles in Wilson County, NC contains a significant amount of fumigated land and 1,3-D use and is representative of the 'mid- Atlantic states'.

During the 2015/16 use season, growers made 84 applications of 1,3-D on 806 ha (~1,991 acres) of land totaling 72,684 kg (160,268 pounds) in the NC study area. The average treated field size was 9.6 ha (~24 acres). The Excel worksheet containing the 1,3-D application information for NC in 2015/16 is titled *NC_Year1_drip_Apps.csv* and *NC_Year1_shank_Apps.csv*. During the 2016/17 use season, growers made 71 applications of 1,3-D on 654 ha (~1615 acres) of land totaling 40,029 kg (88,063 pounds) in the NC study area. The Excel worksheet containing the 1,3-D application information for NC in 2016/17 is titled *NC_Year2_drip_Apps.csv* and *NC_Year2_shank_Apps.csv*. The 1,3-D use by month during the 2015/16 and 2016/17 study season reflects the extensive use of 1,3-D for fumigating tobacco fields in March and April.

The SOFEA simulations in the Wilson, NC study area were conducted on a 23 km x 18 km land area (160 square miles) containing 1,656 receptors spaced 500m apart. The simulations spanned an entire year of use, starting on 6/1/2015 and ending on 5/31/2016 for the first year and starting on 6/1/2016 and ending on 5/31/2017 for the second year. The 1,3-D use by month during the 2015/16 and 2016/17 study seasons is consistent with historical use patterns in North Carolina, with major use in the spring (March/April). SOFEA concentrations and probability distributions of 24-hour, 28-day, 90-day MA and annual average 1,3-D concentrations for the NC study area, for 2015/16 and 2016/17 are shown in Table F.2 (drip applications) and F.3 (shank applications).

*Georgia-Florida coastal plain:*
Approximately 20% of 1,3-D sold in the USA are applied annually in Georgia and Florida, primarily to soil being prepared for annual crops such as potatoes, peppers, tomatoes and with some tree and vine use. A study area spanning approximately 270 square miles of intensive agricultural land bounded by the town of St. Johns, FL to the north and Palatka, FL to the south, that contains a significant amount of fumigated land and 1,3-D use, is representative of agricultural production in GA/FL and was selected for modeling.

During the 2015/16 use season, growers made 206 applications of 1,3-D on 5,504 ha (~12,408 acres) of land totaling 323,971 kg (714,356 pounds) in the FL study area. The average treated field size was 24.5 ha (~60 acres). The Excel worksheet containing the 1,3-D application information for FL in 2015/16 is titled *FL_Year1_data.csv*. During the 2016/17 use season, growers made 205 applications of 1,3-D on 4401 ha (~10,828 acres) of land totaling 251,959 kg (554,311 pounds) in the FL study area. The Excel worksheet containing the 1,3-D application information for NC in 2015/16 is titled *FL_Year2_data.csv*.

The SOFEA simulations in the FL study area were conducted on a 20 km x 35 km (269 square mile) land area containing 2911 receptors spaced 500m apart. The simulations spanned an entire year of use, starting on 6/1/2015 and ending on 5/31/2016 for the first year and starting on 6/1/2016 and end ing on 5/31 /2017 for the second year. SOFEA simulated concentrations and probability distributions of 24-hour, 28-day, 90-day MA and annual average 1,3-D concentrations for the FL study area, for 2015/16 and 2016/17 are shown in Table F.4.

| Mass of 1,3-D Applied and Land Area Treated in the WA, NC, and FL Modeled Study Areas[1] | | | | |
|---|---|---|---|---|
| **Region** | **Year** | **# of 1,3-D Applications** | **Treated Area (acres)** | **1,3-D Applied (lbs)** |
| Washington | 2015-16 | 138 | 14,857 | 2,448,782 |
| | 2016-17 | 107 | 11,768 | 1,873,028 |
| North Carolina | 2015-16 | 84 | 1,991 | 160,268 |
| | 2016-17 | 71 | 1,615 | 136,810 |
| Florida | 2015-16 | 206 | 12,409 | 714,356 |
| | 2016-17 | 205 | 10,775 | 555,571 |

1. Data inputs compiled from Excel Use Data (i.e.., WA_Year1_data.csv, WA_Year2_data.csv, NC_Year1_drip_Apps.csv, NC_Year2_drip_Apps.csv, NC_Year1_shank_Apps.csv, NC_Year2_shank_Apps.csv, FL_Year1_data.csv, FL_Year2_data.csv)

1,3-D Flux Files

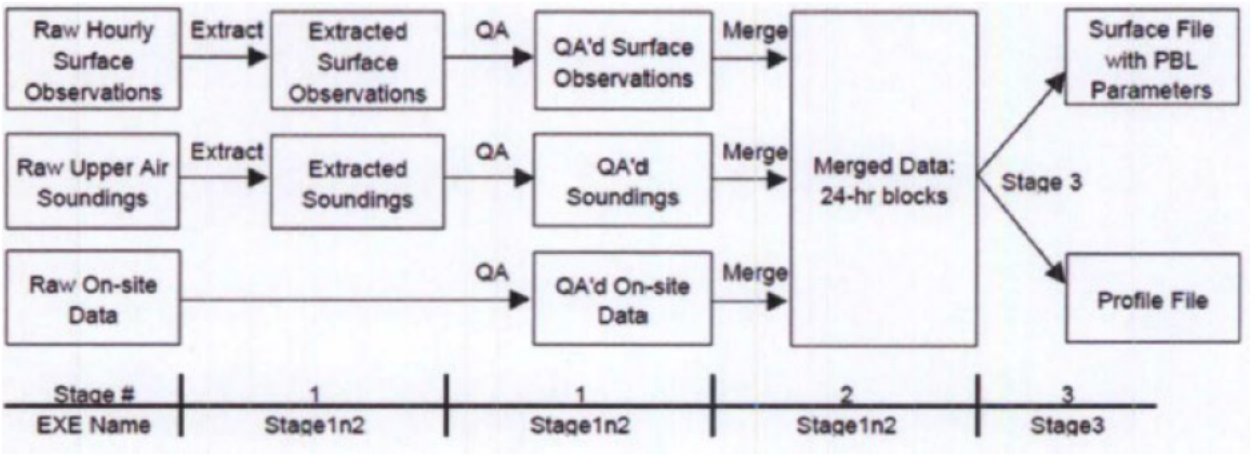
Flux files characterize the rate of 1,3-D flux ($g/m^2/h$) from the soil as a function of time from field volatility studies. A study conducted by Knuteson *et al.* (1992)[29] near Salinas, CA was used to parameterize the flux from shank injection applications, while a study conducted by Knuteson *et al.* (1999)[30], also near Salinas was used to parameterize the flux from drip applications. The use of these flux profiles, developed from field studies conducted in the late 90s forms a conservative basis for the flux since improved application techniques, new formulations and flux emission mitigation technologies such as low permeability agricultural films result in significantly lower emissions of 1,3-D and other fumigants. For example, a significant portion of the 1,3-D applied by coastal strawberry producers is applied as PicClor60 (nominally 40% 1,3-D and 60% chloropicrin) has 'Totally Impermeable Film' (TIF) placed over the beds after the 1,3-D is injected which has been shown in field studies to reduce total 1,3-D emissions to under 10%, and the maximum flux reduced to 10 $\mu g/m^2/sec$ if the TIF tarp is left in place for 10 days. As new application technologies and flux mitigation technologies enter the market, emissions will be further reduced, and therefore the use of the flux profiles from Kunetson *et al.* (1992, 1999) conservatively estimates ambient air concentrations.

Weather Files

Weather data were obtained from a weather station in close proximity to the study site in each region. The weather files were processed using AERMET pre-processor (v. 15181) using the process shown below.

[29] Knuteson, J., Petty, D., Shurdut, B. (1992) Field Volatility of 1,3-Dichloropropene in Salinas Valley California: Lab Project Number: Dow Elanco unpublished report Number: GH-C 2917. (MRID 42545101).

[30] Knuteson, J., Dolder, S. (1999) Field Volatility of 1,3-Dichloropropene and Chloropicrin from Shallow Drip Irrigation Application of Telone C-35 (InLine) to Strawberry Beds Covered with VIF Tarp. Dow AgroSciences unpublished report Number: GH-C 5075.




The AERMET surface file with PBL parameters, and profile weather files for each study area are shown in the table below.

| **AERMET Surface File and Parameters:** | | | | | | |
|---|---|---|---|---|---|---|
| **Study Area** | **Nearest Town** | **Lat/Long** | **Upper Air File ID** | **Surface File ID** | **Surface File** | **Profile File** |
| Washington | Ephrata | 47.308N 119.515W | 04106 | 727900 | Ephrata.SFC | Ephrata.PFL |
| North Carolina | Raleigh | 35.892N 78.782W | 13723 | 13722 | Raleigh.SFC | Raleigh.PFL |
| Florida | Jacksonville | 30.459N 81.694W | 13723 | 13880 | Jackson.SFC | Jackson.PFL |

HED evaluated different durations of exposure using the SOFEA v4.1.4 modeled 24-hour averaging period including single day (acute) exposures, ST/IT exposures, and LT exposures. Risks from acute exposures were calculated using the highest 24-hour air concentrations for each modeled location and the acute ambient (24-hour exposure) HEC for the inhalation POD (343.33 $mg/m^3$ = 343,330 $\mu g/m^3$). Risks from ST/IT exposures were calculated using the highest 28-day and 90-day rolling average concentration for each location and the ST/IT ambient HEC for the inhalation POD (1.35 $mg/m^3$ = 1,353 $\mu g/m^3$). Risks from LT exposures were calculated using the 1-year average air concentration for each location and the LT ambient HEC for the inhalation POD (0.69 $mg/m^3$ = 692 $\mu g/m^3$).

**Table F.1. Washington: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³) [2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| **2015/2016** | | | | | | | | |
| 10 | 1.48 | 0.19 | 0.10 | 0.024 | 230,000 | 7,100 | 13,000 | 29,000 |
| 20 | 2.17 | 0.29 | 0.15 | 0.035 | 160,000 | 4,700 | 8,800 | 20,000 |
| 30 | 3.02 | 0.43 | 0.22 | 0.048 | 110,000 | 3,100 | 6,300 | 14,000 |
| 40 | 4.05 | 0.66 | 0.33 | 0.069 | 85,000 | 2,100 | 4,100 | 10,000 |
| 50 | 5.19 | 0.88 | 0.45 | 0.089 | 66,000 | 1,500 | 3,000 | 7,800 |
| 60 | 6.72 | 1.10 | 0.56 | 0.11 | 51,000 | 1,200 | 2,400 | 6,400 |
| 70 | 8.95 | 1.41 | 0.70 | 0.13 | 38,000 | 960 | 1,900 | 5,200 |
| 80 | 12.85 | 1.91 | 0.90 | 0.17 | 27,000 | 710 | 1,500 | 4,100 |
| 90 | 22.01 | 2.92 | 1.30 | 0.25 | 16,000 | 460 | 1,000 | 2,800 |
| 95 | 37.82 | 4.75 | 1.95 | 0.36 | 9,100 | 280 | 700 | 1,900 |
| 99 | 122.93 | 16.36 | 5.57 | 0.94 | 2,800 | 83 | 240 | 740 |
| 99.9 | 269.36 | 30.66 | 9.88 | 1.60 | 1,300 | 44 | 140 | 430 |
| 100 | 402.14 | 45.52 | 14.92 | 2.37 | 850 | 30 | 91 | 290 |
| **2016/2017** | | | | | | | | |
| 10 | 1.74 | 0.23 | 0.11 | 0.022 | 200,000 | 5,900 | 12,000 | 31,000 |
| 20 | 2.43 | 0.33 | 0.16 | 0.030 | 140,000 | 4,100 | 8,300 | 23,000 |
| 30 | 3.16 | 0.43 | 0.21 | 0.039 | 110,000 | 3,200 | 6,500 | 18,000 |
| 40 | 3.99 | 0.54 | 0.25 | 0.047 | 86,000 | 2,500 | 5,300 | 15,000 |
| 50 | 4.93 | 0.65 | 0.31 | 0.056 | 70,000 | 2,100 | 4,400 | 12,000 |
| 60 | 6.18 | 0.79 | 0.36 | 0.067 | 56,000 | 1,700 | 3,700 | 10,000 |
| 70 | 8.04 | 1.00 | 0.45 | 0.081 | 43,000 | 1,400 | 3,000 | 8,500 |
| 80 | 11.22 | 1.33 | 0.57 | 0.10 | 31,000 | 1,000 | 2,400 | 6,900 |
| 90 | 19.31 | 2.17 | 0.86 | 0.15 | 18,000 | 620 | 1,600 | 4,700 |

**Table F.1. Washington: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³) [2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| 95 | 33.20 | 3.65 | 1.38 | 0.23 | 10,000 | 370 | 980 | 3,000 |
| 99 | 109.23 | 14.04 | 4.52 | 0.65 | 3,100 | 96 | 300 | 1,100 |
| 99.9 | 242.01 | 27.54 | 8.93 | 1.26 | 1,400 | 49 | 150 | 550 |
| 100 | 475.54 | 48.12 | 15.06 | 2.03 | 720 | **28** | 90 | 340 |

1. Specific use inputs detailed in Excel spreadsheets (WA_Year1_data.csv, WA_Year2_data.csv)
2. Air concentrations using 24 hour averaging periods reflect highest modeled receptor/average for: 24-hour air concentrations; 28-day and 90-day rolling average concentrations; and 1-year average concentrations.
3. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (µg/m3) ÷ corresponding concentration (µg/m3).

**Table F.2. North Carolina: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³) [2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| **2015/2016** | | | | | | | | |
| 10 | 7.52 | 0.99 | 0.42 | 0.11 | 46,000 | 1,400 | 3,200 | 6,300 |
| 20 | 10.26 | 1.37 | 0.53 | 0.16 | 33,000 | 990 | 2,500 | 4,500 |
| 30 | 12.78 | 1.72 | 0.66 | 0.20 | 27,000 | 790 | 2,100 | 3,500 |
| 40 | 16.37 | 2.18 | 0.82 | 0.24 | 21,000 | 620 | 1,700 | 2,900 |
| 50 | 20.70 | 2.79 | 1.02 | 0.31 | 17,000 | 490 | 1,300 | 2,300 |
| 60 | 26.95 | 3.58 | 1.30 | 0.39 | 13,000 | 380 | 1,000 | 1,800 |
| 70 | 36.47 | 4.91 | 1.78 | 0.52 | 9,400 | 280 | 760 | 1,300 |
| 80 | 52.68 | 7.08 | 2.43 | 0.73 | 6,500 | 190 | 560 | 950 |
| 90 | 91.15 | 12.20 | 4.15 | 1.17 | 3,800 | 110 | 330 | 590 |
| 95 | 144.42 | 21.20 | 7.07 | 1.98 | 2,400 | 64 | 190 | 350 |
| 99 | 246.21 | 46.16 | 15.91 | 4.55 | 1,400 | **29** | 85 | 150 |
| 99.9 | 347.51 | 65.90 | 21.74 | 6.44 | 990 | **21** | 62 | 110 |

**Table F.2. North Carolina: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³) [2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| 100 | 354.77 | 73.51 | 25.15 | 6.74 | 970 | **18** | 54 | 100 |
| **2016/2017** | | | | | | | | |
| 10 | 2.91 | 0.26 | 0.13 | 0.033 | 120,000 | 5,200 | 11,000 | 21,000 |
| 20 | 5.29 | 0.54 | 0.25 | 0.064 | 65,000 | 2,500 | 5,500 | 11,000 |
| 30 | 8.05 | 0.88 | 0.38 | 0.10 | 43,000 | 1,500 | 3,500 | 6,900 |
| 40 | 10.53 | 1.36 | 0.51 | 0.15 | 33,000 | 1,000 | 2,700 | 4,600 |
| 50 | 13.87 | 1.76 | 0.64 | 0.20 | 25,000 | 770 | 2,100 | 3,400 |
| 60 | 18.11 | 2.34 | 0.85 | 0.27 | 19,000 | 580 | 1,600 | 2,600 |
| 70 | 25.46 | 3.34 | 1.16 | 0.38 | 13,000 | 400 | 1,200 | 1,800 |
| 80 | 38.62 | 5.48 | 1.86 | 0.61 | 8,900 | 250 | 730 | 1,100 |
| 90 | 71.02 | 10.27 | 3.44 | 0.98 | 4,800 | 130 | 390 | 700 |
| 95 | 126.19 | 19.41 | 6.40 | 1.82 | 2,700 | 70 | 210 | 380 |
| 99 | 227.05 | 47.83 | 16.09 | 4.55 | 1,500 | **28** | 84 | 150 |
| 99.9 | 347.59 | 65.97 | 22.86 | 6.51 | 990 | **21** | 59 | 110 |
| 100 | 891.68 | 165.67 | 53.52 | 13.98 | 390 | **8** | **25** | 50 |

1. Specific use inputs detailed in Excel spreadsheets (NC_Year1_drip_Apps.csv, NC_Year2_drip_Apps.csv, NC_Year1_shank_Apps.csv, NC_Year2_shank_Apps.csv)
2. Air concentrations using 24 hour averaging periods reflect highest modeled receptor/average for: 24-hour air concentrations; 28-day and 90-day rolling average concentrations; and 1-year average concentrations.
3. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (µg/m3) ÷ corresponding concentration (µg/m3).

**Table F.3. North Carolina – Drip Shank Separate: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³)[2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| **2015/2016** | | | | | | | | |
| 10 | 0.12 | 0.0069 | 0.0022 | 0.00073 | 2,900,000 | 200,000 | 620,000 | 940,000 |
| 20 | 0.25 | 0.014 | 0.0046 | 0.0015 | 1,400,000 | 95,000 | 300,000 | 450,000 |
| 30 | 0.46 | 0.027 | 0.0086 | 0.0028 | 740,000 | 50,000 | 160,000 | 250,000 |
| 40 | 0.93 | 0.057 | 0.0181 | 0.0059 | 370,000 | 24,000 | 75,000 | 120,000 |
| 50 | 2.51 | 0.18 | 0.0564 | 0.0168 | 140,000 | 7,500 | 24,000 | 41,000 |
| 60 | 13.20 | 2.25 | 0.77 | 0.24 | 26,000 | 600 | 1,800 | 2,800 |
| 70 | 19.89 | 3.70 | 1.24 | 0.36 | 17,000 | 370 | 1,100 | 1,900 |
| 80 | 29.89 | 5.43 | 1.84 | 0.54 | 11,000 | 250 | 740 | 1,300 |
| 90 | 55.17 | 10.73 | 3.62 | 1.03 | 6,200 | 130 | 370 | 670 |
| 95 | 97.30 | 18.46 | 6.33 | 1.74 | 3,500 | 73 | 210 | 400 |
| 99 | 261.36 | 44.67 | 14.97 | 4.07 | 1,300 | 30 | 90 | 170 |
| 99.9 | 445.44 | 72.86 | 23.92 | 6.42 | 770 | **19** | 57 | 110 |
| 100 | 1452.94 | 177.20 | 56.95 | 14.03 | 240 | **8** | **24** | 49 |
| **2016/2017** | | | | | | | | |
| 10 | 0.02 | 0.0012 | 0.00039 | 0.00013 | 18,000,000 | 1,100,000 | 3,500,000 | 5,100,000 |
| 20 | 0.04 | 0.0022 | 0.00068 | 0.00022 | 8,900,000 | 620,000 | 2,000,000 | 3,200,000 |
| 30 | 0.07 | 0.0042 | 0.0013 | 0.00042 | 4,600,000 | 320,000 | 1,000,000 | 1,600,000 |
| 40 | 0.17 | 0.0096 | 0.0030 | 0.0010 | 2,000,000 | 140,000 | 450,000 | 730,000 |
| 50 | 1.09 | 0.06 | 0.02 | 0.0059 | 320,000 | 21,000 | 67,000 | 120,000 |
| 60 | 13.02 | 2.20 | 0.75 | 0.24 | 26,000 | 620 | 1,800 | 2,900 |
| 70 | 19.73 | 3.67 | 1.24 | 0.36 | 17,000 | 370 | 1,100 | 1,900 |
| 80 | 29.65 | 5.43 | 1.84 | 0.54 | 12,000 | 250 | 740 | 1,300 |
| 90 | 54.89 | 10.73 | 3.62 | 1.03 | 6,300 | 130 | 370 | 670 |
| 95 | 97.30 | 18.46 | 6.33 | 1.74 | 3,500 | 73 | 210 | 400 |
| 99 | 261.36 | 44.67 | 14.97 | 4.07 | 1,300 | 30 | 90 | 170 |

**Table F.3. North Carolina – Drip Shank Separate: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³)[2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| 99.9 | 445.44 | 72.86 | 23.92 | 6.42 | 770 | **19** | 57 | 110 |
| 100 | 1452.94 | 177.20 | 56.95 | 14.03 | 240 | **8** | **24** | 49 |

1. Specific use inputs detailed in Excel spreadsheets (NC_Year1_drip_Apps.csv, NC_Year2_drip_Apps.csv, NC_Year1_shank_Apps.csv, NC_Year2_shank_Apps.csv)
2. Air concentrations using 24 hour averaging periods reflect highest modeled receptor/average for: 24-hour air concentrations; 28-day and 90-day rolling average concentrations; and 1-year average concentrations.
3. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (µg/m3) ÷ corresponding concentration (µg/m3).

**Table F.4. Florida: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³)[2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| **2015/2016** | | | | | | | | |
| 10 | 12.98 | 2.63 | 0.96 | 0.28 | 26,000 | 510 | 1,400 | 2,500 |
| 20 | 16.94 | 3.41 | 1.22 | 0.36 | 20,000 | 400 | 1,100 | 1,900 |
| 30 | 20.40 | 4.29 | 1.55 | 0.46 | 17,000 | 320 | 870 | 1,500 |
| 40 | 24.78 | 5.29 | 1.94 | 0.58 | 14,000 | 260 | 700 | 1,200 |
| 50 | 29.46 | 6.38 | 2.36 | 0.70 | 12,000 | 210 | 570 | 990 |
| 60 | 36.06 | 7.57 | 2.84 | 0.87 | 9,500 | 180 | 480 | 800 |
| 70 | 47.30 | 9.34 | 3.48 | 1.06 | 7,300 | 140 | 390 | 660 |
| 80 | 68.35 | 13.14 | 4.90 | 1.42 | 5,000 | 100 | 280 | 490 |
| 90 | 132.04 | 23.18 | 8.48 | 2.34 | 2,600 | 58 | 160 | 300 |
| 95 | 192.43 | 34.14 | 11.96 | 3.27 | 1,800 | 40 | 110 | 210 |
| 99 | 319.51 | 57.63 | 20.88 | 5.63 | 1,100 | **23** | 65 | 120 |
| 99.9 | 446.12 | 82.35 | 31.42 | 8.86 | 770 | **16** | 43 | 78 |
| 100 | 527.15 | 94.01 | 36.93 | 9.84 | 650 | **14** | 37 | 70 |

**Table F.4. Florida: Highest Modeled Ambient Air Concentration and Risk Estimates for 1,3-D; SOFEA Ambient Air Modeling Runs V4.1.4.[1]**

| Percentile (%) | Concentrations (µg/m³) [2] | | | | Inhalation MOE[3] | | | |
|---|---|---|---|---|---|---|---|---|
| | 24-hour | 28-day | 90-day | Annual Ave | Acute | ST/IT (28-day) | ST/IT (90-day) | LT |
| | | | | | (LOC = 30 for all durations) | | | |
| 2016/2017 | | | | | | | | |
| 10 | 10.94 | 1.76 | 0.61 | 0.22 | 31,000 | 770 | 2,200 | 3,200 |
| 20 | 14.06 | 2.48 | 0.85 | 0.29 | 24,000 | 540 | 1,600 | 2,400 |
| 30 | 17.48 | 3.13 | 1.10 | 0.37 | 20,000 | 430 | 1,200 | 1,900 |
| 40 | 21.28 | 3.75 | 1.38 | 0.48 | 16,000 | 360 | 980 | 1,400 |
| 50 | 26.57 | 4.61 | 1.73 | 0.60 | 13,000 | 290 | 780 | 1,200 |
| 60 | 33.06 | 5.79 | 2.17 | 0.74 | 10,000 | 230 | 620 | 940 |
| 70 | 42.86 | 7.23 | 2.72 | 0.94 | 8,000 | 190 | 500 | 740 |
| 80 | 61.81 | 9.95 | 3.73 | 1.26 | 5,600 | 140 | 360 | 550 |
| 90 | 110.60 | 17.65 | 6.61 | 1.99 | 3,100 | 77 | 200 | 350 |
| 95 | 163.06 | 28.80 | 10.19 | 2.98 | 2,100 | 47 | 130 | 230 |
| 99 | 254.92 | 48.40 | 16.73 | 4.78 | 1,300 | **28** | 81 | 140 |
| 99.9 | 363.83 | 65.97 | 24.43 | 6.62 | 940 | **21** | 55 | 100 |
| 100 | 477.81 | 76.84 | 30.84 | 8.38 | 720 | **18** | 44 | 83 |

1. Specific use inputs detailed in Excel spreadsheets (FL_Year1_data.csv, FL_Year2_data.csv)
2. Air concentrations using 24 hour averaging periods reflect highest modeled receptor/average for: 24-hour air concentrations; 28-day and 90-day rolling average concentrations; and 1-year average concentrations.
3. Acute MOE = Duration Specific (Acute, ST/IT, and LT) HEC for the inhalation POD (µg/m3) ÷ corresponding concentration (µg/m3).

## Appendix G. International Residue Limit Status Sheet

1,3-D (029001)

| Summary of U.S. and International Tolerances and Maximum Residue Limits | | | | |
|---|---|---|---|---|
| *Residue Definition*: | | | | |
| U.S. - 40 CFR 180.636: Tolerances are established for the combined residues of the fungicide *cis*- and *trans*-1,3-dichloropropene and its metabolites *cis*- and *trans*-3-chloroacrylic acid, and *cis*- and *trans*-3-chloroallyl alcohol in or on the following commodities. | | | | |
| Canada - 1,3-dichloro-1-propene, including the metabolites (2Z)-3-chloro-2-propen-1-ol, (2E)-3 -chloro-2-propen-1-ol, (2Z)-3-chloro-2-propenoic acid, and (2E)-3-chloro-2-propenoic acid | | | | |
| Codex - | | | | |
| Other[1] - | | | | |
| *Commodity* | *Tolerance (ppm)/Maximum Residue Limit (mg/kg)* | | | |
| | U.S. | Canada | Codex | Other |
| Grape | 0.018 | 0.018 | | |
| | | | | |
| Completed: G. Kramer; 11/04/2019 using Global MRL | | | | |