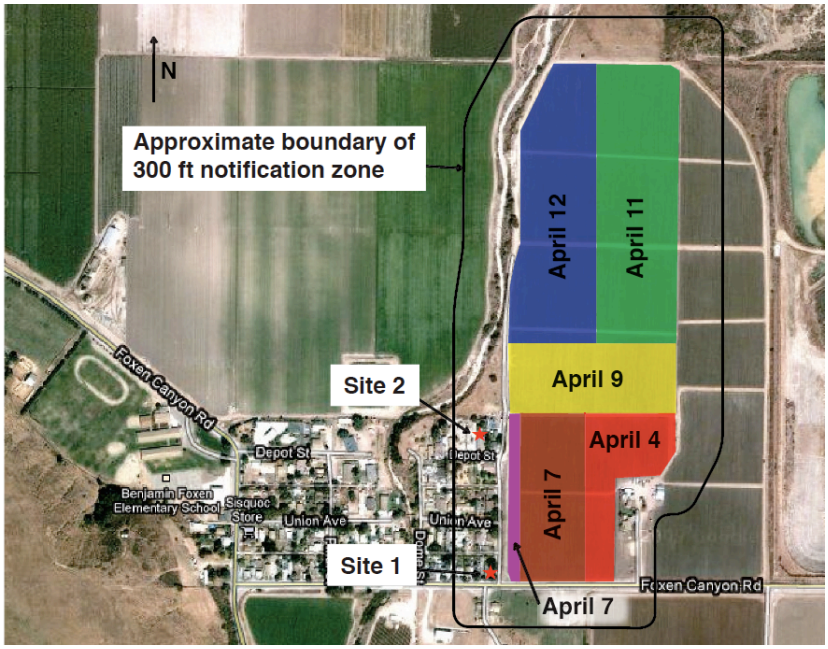
Air Monitoring in Sisquoc, California April 2–22, 2008

Technical Report



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June 22, 2010

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Support for air monitoring in Sisquoc was generously provided by the Cedar Tree Foundation.

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List of Abbreviations

ARB	Air Resources Board, the California agency in charge of regulating air pollution in the state.
ATSDR	Agency for Toxic Substances and Disease Registry, the agency within the US Department of Health and Human Services that "performs specific functions concerning the effect on public health of hazardous substances in the environment."
DPR	Department of Pesticide Regulation, the California agency in charge of regulating pesticides in the state.
FQPA	The Federal Food Quality Protection Act. Passed in 1996, this law substantially revised the way U.S. EPA evaluates pesticides for registration, requiring them to account for the special vulnerability of children and women of child-bearing age.
LD_{50}	A dose that is lethal to 50% of test animals of a given species. Commonly expressed in units of mg/kg, LD_{50} values are used to rank the acute toxicity of chemicals.
LOQ	Limit of Quantitation, the lowest concentration at which a laboratory can reliably measure the amounts of a pesticide present in a sample. See **Calculations** section for details.
MDL	Method Detection Limit, the lowest concentration that can reliably be detected for a sample collected and analyzed according to a specific method. See **Calculations** section for details.
NIOSH	National Institute for Occupational Safety and Health, the federal agency that oversees worker safety.
NOAEL	No Observable Adverse Effect Level, the toxicological dose of a chemical below which no adverse effects are anticipated from exposure to that chemical alone, usually in units of mg/kg-day.
REL	Reference Exposure Level, the concentration of a chemical in air, derived from the U.S. EPA-selected NOAEL and EPA-designated uncertainty factors, below which no adverse effects are anticipated from inhalation exposure to that chemical alone, given in units of ng/m^3. RELs can be adjusted for different age groups by using typical breathing rates and body weights. See **Calculations** section for details. A REL represents a level of concern for inhalation exposure analogous to the Reference Dose U.S. EPA uses to assess levels of concern for dietary exposure.
RfC	Reference Concentration, the concentration of a chemical in air, derived from the U.S. EPA-selected NOAEL and EPA-designated uncertainty factors, below which no adverse effects are anticipated from inhalation exposure to that chemical alone for an adult male, given in units of ng/m^3.
SOP	Standard Operating Procedure, a written method for conducting sampling, analysis and other laboratory protocols. See Appendix 3 for an example.
TWA	Time-weighted-average. Used in this report to calculate an average concentration of chloropicrin over a given time period or an average breathing rate over a lifetime.
U.S. EPA	United States Environmental Protection Agency, the federal agency charged with regulating pesticides, air, water, hazardous waste sites, and more.
USDA-ARS	United States Department of Agriculture-Agricultural Research Service, the research arm of the USDA. One part of their work is to evaluate the fate and transport of pesticides in the environment.
USGS	United States Geological Survey, a federal agency that, among other activities, evaluates airborne pesticides as a source of water pollution.

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Air Monitoring in Sisquoc, California, April 2–22, 2008

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Executive Summary

This report presents the results of an air monitoring experiment in Sisquoc, California. Between April 2 and April 22, 2008, a total of 57 samples were collected at two sites in Sisquoc adjacent to a chloropicrin application. Of the samples collected, 51% contained chloropicrin above the limit of quantitation (LOQ) of 0.05 μg of chloropicrin per sample. The LOQ is equivalent to an air concentration of 0.35 μg/m^3 for a 24-hour sample and 0.70 μg/m^3 for a 12-hour sample.

The highest concentration of chloropicrin observed for a 12-hour period was 6.1 μg/m^3 and for a 24-hour period was 14.5 μg/m^3 on April 7, 2008. The time-weighted-average concentration for the 19 days sampled was 1.44 μg/m^3 at Site 1 and 2.40 μg/m^3 at Site 2. Results from the air monitoring in Sisquoc are summarized in Tables 2 and 3 on pages 13 and 14 and in Figures 3 and 4 on page 15.

The results of this study indicate that for one day at each site, the air concentration of chloropicrin exceeded acute levels of concern for children set forth by the California Department of Pesticide Regulation (DPR). The time-weighted-average chloropicrin concentration of 2.40 μg/m^3 at Site 2 exceeds both U.S. EPA's short- and intermediate-term level of concern of 1.8 μg/m^3 and DPR's seasonal child level of concern of 2.3 μg/m^3, indicating an unacceptably high risk of adverse effects for people who spent significant time in the vicinity of that site. The highest observed 24-hour concentration of 14.5 μg/m^3 at Site 2 is more than twice as high as DPR's 24-h level of concern of 6.2 μg/m^3 for children. None of the samples exceeded the U.S. EPA, DPR, or OEHHA 1-hour or 8-hour levels of concern.

Age-adjusted cancer risks were higher than the standard acceptable level of one additional cancer per million people by a factor ranging from 23 (for a child exposed from birth to 2 years) to 151 (for an adult exposed for a lifetime). Buffer zone mitigations that will be required by US EPA in 2011 would not have protected the community from exposures exceeding levels of concern.

Comparison of the chloropicrin concentrations measured in Sisquoc with concentrations measured by the California Air Resources Board (ARB) near fumigation sites indicate that the levels observed in Sisquoc are relatively low compared to worst-case scenarios. Figures 5 and 6 on pages 28 and 30 provide a graphical comparison of Sisquoc concentrations to prior ARB studies for both application site monitoring and ambient community monitoring.

In Sisquoc, methyl bromide was applied concurrently with chloropicrin; however, due to resource limitations, only chloropicrin was monitored. Given the high levels of chloropicrin observed, community residents almost certainly experienced some co-exposure to methyl bromide during the monitoring period. The effects of combined exposure to methyl bromide and chloropicrin have not

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been evaluated, but it is likely that the potential for adverse effects increases with exposures to multiple chemicals.

Chloropicrin is used as a soil fumigant prior to planting crops. In California, 5.5 million pounds of chloropicrin were reported used in 2008, the latest year for which data are available. Use has increased steadily over the last ten years; in 1998, only 3.0 million pounds of chloropicrin were used in the state. In California, chloropicrin is used primarily on strawberries, soil pre-plant applications and in nurseries.

Over the course of the last several years, chloropicrin has been the cause of over 1,000 poisonings in California. Two of the largest incidents occurred in Kern County in 2003 and Monterey County in 2005. The details of these and other chloropicrin-related poisonings in California are summarized in Table 11 on page 33. Chloropicrin is highly irritating in low concentrations, highly acutely toxic, a carcinogen, and causes developmental and reproductive toxicity in animal studies. Symptoms of acute poisoning include eye and respiratory irritation, difficulty breathing, nausea and vomiting. Chronic effects include permanent lung damage, kidney damage, and cancer.

Exposures calculated from the measured air concentrations should be viewed as estimates. In the case of the Sisquoc study, these concentrations do not represent a worst-case exposure scenario, and do not necessarily represent the precise exposure individuals may experience. Variability in actual exposures and the effects that may be experienced by individuals are governed by breathing rates and activity levels, time spent in areas where pesticide exposure can occur, as well as individuals' ability to detoxify chemicals.

Introduction

In early October 2007, residents of Sisquoc, California, received notification of an impending application of methyl bromide to an adjacent field used to grow strawberries. Members of this small community were concerned about the use of such a highly toxic chemical just a few hundred feet from their homes. These fears were confirmed on October 5 when, following the fumigation earlier in the day, high winds ripped the tarps off the 20-acre field, releasing the chemical into the community. Following the incident, residents reported that several children became ill with symptoms of fever, restlessness, and in some cases vomiting.[1]

The October incident motivated the community to try to prevent or at least monitor future applications. In November, scientists from PANNA met with several members of the community, and provided them with air monitoring equipment ("Drift Catchers") and detailed training. In late March of 2008, residents received notification that methyl bromide and chloropicrin would be applied to the field adjacent to their homes starting in the first week of April. They used Drift Catchers to monitor these applications, and the results are reported herein.

The goal of this study was to characterize the levels of chloropicrin in ambient air in the community during and following the fumigation of the adjacent field. While PANNA and the community members were also interested in measuring methyl bromide levels, this chemical was not monitored due to resource limitations.

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Site Description and Application Details

Sisquoc, California, is an unincorporated town in Santa Barbara County that is home to about 350 people. This tiny community, measuring just an eighth of a mile from north to south and a quarter mile from east to west, has an elementary school, a church, a firehouse, and a general store (see Figure 1). Directly to the south of the town is a vineyard, to the west are hills that are not in agricultural production, and to the north is a field typically planted with broccoli, cauliflower, or corn. According to local residents, the field of approximately 70 acres bordering the eastern edge of the community had traditionally been used to grow alfalfa for fodder and gladiolas for bulbs. Only recently had it been planted with peppers, strawberries, and tomatillos. This is the field that was fumigated during the October 2007 methyl bromide incident mentioned above, and it is also the site of the chloropicrin and methyl bromide application monitored in this report.

Brooks Street (Figure 1) forms the boundary between the application site and the Sisquoc community; Drift Catchers were placed at homes on the southern and northern ends of this street in areas where people were likely to spend time outside. At Site 1, a Drift Catcher was located in the yard of a private home on the corner of Brooks and Foxen Canyon Road, 35 feet from the fence that runs along Brooks Street. At Site 2, a Drift Catcher was located in the yard of another home near the corner of Brooks and Depot Streets, 94 feet from the fence line. Both sites were located on properties within 300 feet of the application site, and thus received notification of the impending fumigation, as required by California regulations.

Records obtained from the County of Santa Barbara Office of the Agricultural Commissioner show that 42 acres of this field were fumigated in six blocks between April 4 and April 14. The dates that the individual blocks were treated are indicated on the map in Figure 1. All applications were scheduled to begin at 6 a.m. The product, Tri Con 57/43 (57% methyl bromide and 43% chloropicrin), was applied at a rate of 300 pounds per acre, and the listed application method was "3bii"—presumably the tarped/shallow/broadcast method described in 3 CCR § 6447.3(a)(3)(b)(ii).[2] The tarp type is listed as "Covalence." The records indicate the field was fumigated in preparation for planting with peppers.

Information on the sizes of the application blocks and the approximate distances from Sites 1 and 2 to the edge of each block are provided in Table 1. The distances are estimates based on the poorly reproduced permit obtained from the Santa Barbara County Agricultural Commissioner's Office.

Table 1. Fumigation Dates and Sizes of Fumigation Blocks

Block	Date of Fumigation	Size (Acres)	Approximate Distance from Site 1	Approximate Distance from Site 2
1	April 4	6	390 ft	450 ft
2	April 7	5[a]	130 ft	190 ft
3	April 9	6	840 ft	170 ft
4	April 11	12	>1000 ft	730 ft
5	April 12	12	>1000 ft	430 ft
6	April 14	1	70 ft	140 ft

[a] In both the original and revised permits, the area of Block 2 is listed as 6 acres, however, the revised permit adds a new Block 6, and a note in the margin says, "Amend: 1 acre from Day 3 [i.e. Block 2] not completed," so it is clear that Block 2 was 5 acres. This is corroborated by residents' observations.

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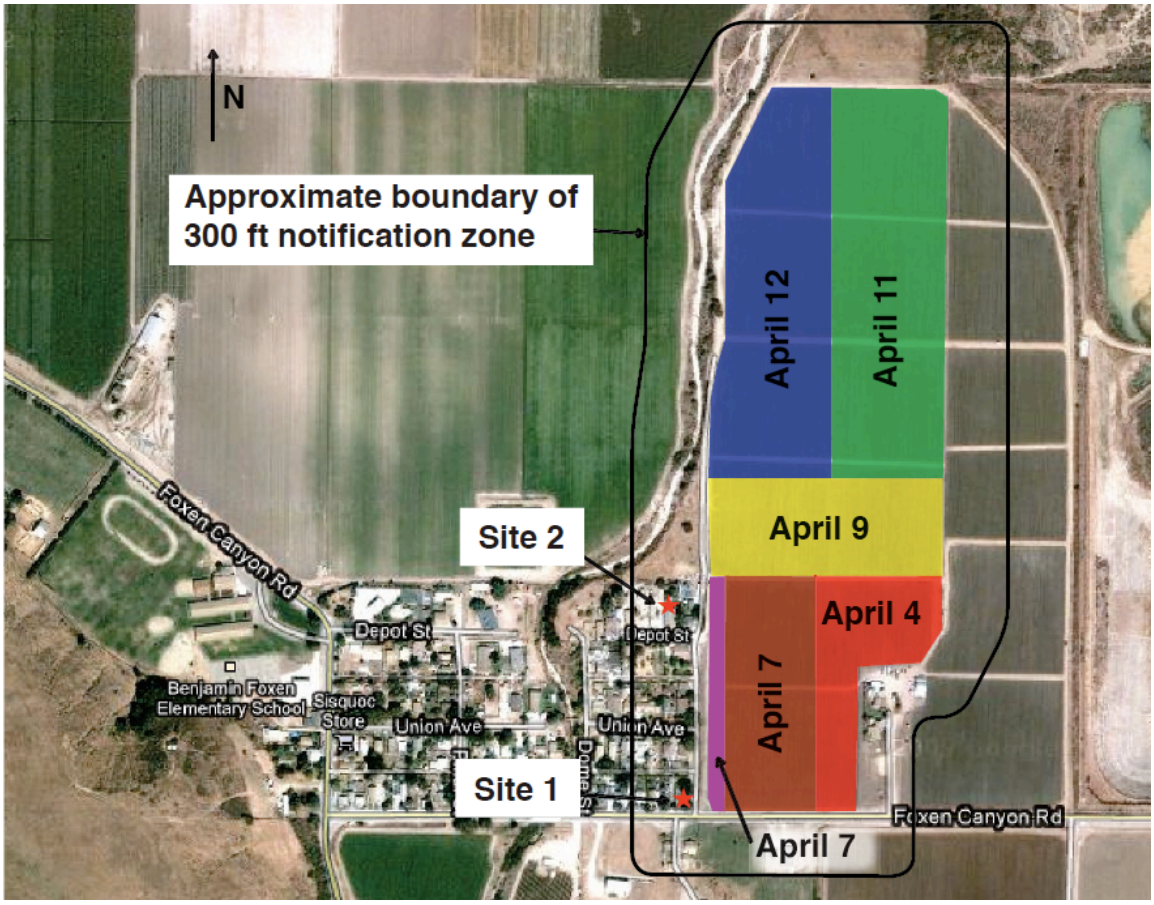

Figure 1. Approximate locations of fumigation blocks and the 300-foot notification zone. The sketch is based on the Methyl Bromide Work Site Plan submitted to the County of Santa Barbara Office of the Agricultural Commissioner. Dates of fumigation for each block are shown. All boundaries are approximate. Underlying map image is from Google Maps, © 2010.

Methods

Sample Collection

Samples were collected by pumping air through XAD-4 resin tubes at a rate of approximately 0.10 L/min. Sample tubes were obtained from SKC Inc. (#226-175, 8 x 150 mm, 400/200 mg in front/rear beds, respectively), and were generally changed every 12 or 24 hours. This sampling method was based on that employed by the California Air Resources Board (ARB) in its monitoring of fumigant applications. The ARB employed sample tubes of the same dimensions and with the same amount of XAD-4 resin, utilized the same or similar flow rates (0.09–0.10 L/min), and collected each sample over a similar duration (8–24 hrs).[3, 4, 5]

The air sampling device consists of a vacuum pump (McMaster-Carr No. 41675K41) connected with 3/8" Teflon tubing and compression fittings to a manifold equipped with two Cajon-type, vacuum-tight Teflon fittings (Beco Mfg.) as tube holders (Figure 2). Flow controller valves for each sample

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allowed for adjustment of airflow to each tube independently. To prevent overheating of the pump, a bleed valve was installed between the pump and the manifold so that a large air flux could be maintained through the pump while restricting the flow through the manifold and sample tubes to the low flow rates required for chloropicrin monitoring.

Pre-labeled sample tubes were attached to the manifold, which stood at 1.5 meters in height. Flow rates were measured with a 0.05–0.5 L capacity rotameter (SKC Inc., Cat. #320-2A05) pre-calibrated with a Bios Defender 510 flow meter (SKC Part #717-510L). The initial flow rate through each of the tubes was set to 0.10 liters per minute. The flow rate was set at the beginning of the sampling run and then measured at the end to check for any changes. If the difference between the start and stop flow rates was less than 25%, these two values were averaged together to calculate an average flow rate for the sampling period. If the ending flow rate differed by more than 25% from the starting flow rate, then the greater flow rate was used, providing a conservative estimate of the final pesticide concentration.

Sample tubes were covered with mylar light shields during the sampling period to prevent any photolytically catalyzed degradation of the sample. Sample identification, start and stop times, and flow rates were recorded on a Sample Log Sheet (see Appendix 4). In addition, wind speed and direction, as well as temperature, weather conditions and any additional observations were noted at the beginning and end of each sampling period. At the end of each sampling period, labeled tubes were capped and placed in a zip-lock plastic bag with the completed log sheet.

Within 10 minutes of removal from the sampling manifold, samples were placed into either a 10°C freezer or into a cooler at 0°C for transport to freezer storage. After storage for no more than two weeks, samples were shipped from the field to PANNA at -10 to 0°C by overnight express mail for analysis. At PANNA, data from sample log sheets were entered into a database (see Appendix 6: Sample Log Database Screen Shot) and stored in a -20°C freezer prior to being shipped by overnight express mail to a commercial laboratory for analysis. A chain of custody form accompanied each batch of samples during handling and transport. In the laboratory, samples were stored in a -20°C freezer prior to processing and analysis, which occurred within one month of receipt in the laboratory. Not more than 8 weeks passed between sample collection and analysis. Prior sample storage stability assessments conducted by the ARB indicate that chloropicrin is stable on XAD-4 resin for at least 4 weeks under these conditions.[3,4,5]

All other sampling details are identical to those described in our report on air monitoring in Hastings, Florida.[6, 7]

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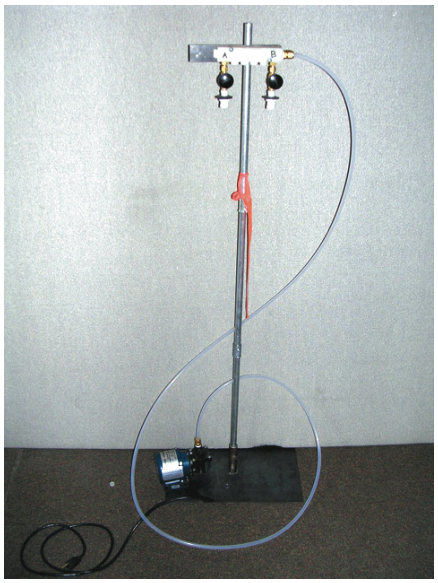
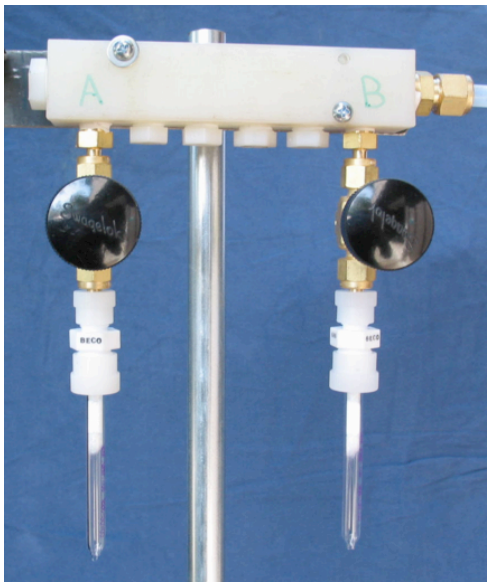
(a)  (b) 

Figure 2: (a) The Drift Catcher™ air monitoring device. (b) Close-up of manifold with flow control valves and sample tubes attached. The design is based on sampling equipment used by the California Air Resources Board. This design has been evaluated by a Scientific Advisory Committee comprised of scientists from the California Department of Pesticide Regulation, the California Air Resources Board, U.S. EPA Region 9, the US Geological Survey, and the California Department of Health Services.

Sample Analysis and Quality Assurance

Samples were analyzed for chloropicrin by Environmental Micro Analysis, Inc. (Woodland, California) using GC with electron capture detection according to OSHA method PV 2103.[8,9] Samples were desorbed with 3 mL of ethyl acetate rather than 1 mL as specified in the method. The lab's reporting limit was 0.05 µg/tube, which corresponds to an air concentration of 0.35 µg/m^3 for a 24 h sample collected at 0.1 L/min. In addition to the field samples, three trip blank samples and two spiked samples were sent to the lab for analysis. The lab was unaware of which samples were field samples and which were blanks or spikes. No chloropicrin was detected in any of the blanks nor in field samples collected before fumigation commenced, and recoveries were 97% and 80% for the tubes fortified with 1.00 µg and 3.00 µg of chloropicrin, respectively. The front and rear beds of the sample tubes were analyzed separately. In no cases were pesticides detected in the rear bed, indicating that there was no breakthrough from the front to rear bed, and that the sample tubes were not overloaded. The lab did not test the samples for any other chemicals.

Weather Monitoring

Meteorological data (wind speed and direction) were obtained from the California Irrigation Management Information System (CIMIS), which maintains a weather station in Sisquoc.[10] The meteorological data for the sampling period are provided in Appendix 1.

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Results

A total of 28 samples (spikes and blanks excluded) were collected at Site 1, and 29 at Site 2 from April 2–22, 2008. As described in the **Methods** section above, flow audits were performed at the beginning and end of each sampling period. For most samples, the starting and ending flow rates differed by < 25%, and the average value was used to calculate the sample volume. For "Wave," "Hill," "Ruby," "Knee," "Needle," "Thread," "Ant," "Razor," "Fork," "Pepper," and "Bird" the difference in flow rates exceed 25%, so the total sample volume was calculated based solely on the greater flow rate so as to over-estimate the sample volume, and thus provide a conservative estimate of the airborne pesticide concentrations. The reported pesticide concentrations for these samples should therefore be considered as minimum values. Such flow rate instability has been noted in prior chloropicrin sampling conducted by the California Air Resources Board and has been attributed to moisture in the resin cartridges from rain or fog in the air altering the permeability of the resin.[4] Complete results are provided in Tables 2 and 3, and plots of chloropicrin concentrations over time are presented in Figures 3 and 4.

Chloropicrin was detected in 13 (46%) and 16 (55%) of the field samples from Sites 1 and 2, respectively. Sample "Ant", collected at Site 2 on April 7–8, had the highest observed concentration of chloropicrin: 14.5 $\mu g/m^3$.

Several field samples are background samples, collected before the fumigation began. According to the permit, fumigation was scheduled to begin in Block one at 6 am on April 4. Notes taken by the Drift Catcher operators indicate that the fumigators began laying down tarp between approximately 6:45 and 7:45 a.m. Thus for Site 1, the background samples are "Sock," "Wind," and "Earth." The first two were collected on April 2 and 3, and "Earth" was started on the evening of April 3 and stopped at 7:38 a.m. on April 4, and had little if any overlap with fumigation operations. For Site 2, background samples are "Stone," collected April 2–3, and "Car", which began on the evening of April 3 and ended at 8:10 a.m. on April 4, and therefore overlapped very little with the application that took place that morning. Chloropicrin was not detected in any of these samples nor in any of the three trip blanks ("Badger," "Petal," and "Banana.")

Time Weighted Average (TWA) chloropicrin concentrations were also calculated for the sampling period excluding background samples. Thus, these TWA concentrations cover the 19-day period from April 4–22, and were 1.44 $\mu g/m^3$ and 2.40 $\mu g/m^3$ at Sites 1 and 2, respectively.

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Table 2: Chloropicrin Concentrations at Site 1, Sisquoc, California, April 2–22, 2008

Sample Name	Start Date	Start Time	Stop Date	Stop Time	Total Time (min.)	Total Sample Volume (m^3)	Chloropicrin Concentration ($\mu g/m^3$)	Notes[a]
Sock	4/2/08	11:43 AM	4/2/08	6:56 PM	433	0.043	0	Background
Wind	4/2/08	7:13 PM	4/3/08	7:33 AM	740	0.074	0	Background
Earth	4/3/08	6:49 PM	4/4/08	7:38 AM	769	0.077	0	Background
Paper	4/4/08	7:49 AM	4/4/08	7:01 PM	672	0.064	0	
Sun	4/4/08	7:16 PM	4/5/08	7:46 AM	750	0.075	0	
String	4/5/08	7:58 AM	4/5/08	6:45 PM	647	0.071	0	
Nail	4/5/08	6:53 PM	4/6/08	7:28 PM	1475	0.148	0	
Egg	4/6/08	7:40 AM	4/7/08	7:02 AM	1402	0.140	0	
Glue	4/7/08	7:10 AM	4/7/08	6:44 PM	694	0.076	1.40	
Wave	4/7/08	6:54 PM	4/8/08	7:26 AM	752	0.075	10.85	MV
Moon	4/8/08	7:35 AM	4/9/08	7:40 AM	1445	0.152	4.19	
Hill	4/9/08	7:51 AM	4/10/08	7:56 AM	1445	0.145	0.54	MV
Ruby	4/10/08	8:01 AM	4/10/08	7:38 PM	697	0.098	6.79	MV
Girl	4/10/08	7:50 PM	4/11/08	7:59 AM	729	0.145	0.99	
Dad	4/11/08	8:15 AM	4/12/08	7:44 AM	1409	0.141	1.30	
Knee	4/12/08	7:52 AM	4/13/08	08:04 AM	1452	0.203	3.01	MV
Dance	4/13/08	8:09 AM	4/13/08	07:18 PM	669	0.067	4.47	
Wire	4/13/08	7:24 PM	4/14/08	08:02 AM	758	0.076	0	
Alpha	4/14/08	8:02 AM	4/14/08	06:44 PM	642	0.083	0	
Badger	4/14/08	6:41 PM	-	-	-	-	0	Trip Blank
Red	4/14/08	6:51 PM	4/15/08	7:56 AM	785	0.075	2.23	
Needle	4/15/08	8:01 AM	4/16/08	7:51 AM	1430	0.200	2.74	MV
Chair	4/16/08	7:56 AM	4/17/08	7:50 AM	1434	0.143	1.53	
Thread	4/17/08	7:57 AM	4/18/08	8:19 AM	1462	0.146	1.29	MV
Button	4/18/08	8:23 AM	4/19/08	7:58 AM	1415	0.127	0	
Mitt	4/19/08	8:03 AM	4/20/08	7:17 AM	1394	0.139	0	
Candy	4/20/08	7:22 AM	4/21/08	8:06 AM	1484	0.134	0	
Bat	4/21/08	7:15 AM	4/22/08	8:51 AM	1536	0.169	0	
Light	4/22/08	8:56 AM	4/23/08	8:00 AM	1384	0.125	0	
Petal	4/30/08	9:35 PM	-	-	-	-	0	Trip Blank

[a] MV = minimum value (see text)

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Table 3: Chloropicrin Concentrations at Site 2, Sisquoc, California, April 2–22, 2008

Sample Name	Start Date	Start Time	Stop Date	Stop Time	Total Time (min.)	Total Sample Volume (m3)	Chloropicrin Concentration (µg/m3)	Notes[a]
Stone	4/2/08	7:32 PM	4/3/08	7:18 AM	706	0.071	0	Background
Car	4/3/08	7:10 PM	4/4/08	8:10 AM	780	0.070	0	Background
Hat	4/4/08	8:23 AM	4/4/08	7:15 PM	652	0.072	0	
Valley	4/4/08	7:37 PM	4/5/08	7:47 AM	730	0.069	0.72	
Coat	4/5/08	8:09 AM	4/5/08	7:37 PM	688	0.069	0	
Cable	4/7/08	7:54 AM	4/7/08	7:04 PM	670	0.074	0	
Ant	4/7/08	7:20 PM	4/8/08	7:23 PM	1443	0.202	14.50	MV
Razor	4/8/08	7:33 PM	4/9/08	8:08 AM	755	0.098	3.79	MV
Hand	4/9/08	8:24 AM	4/9/08	6:09 PM	585	0.076	0	
Brick	4/9/08	6:14 PM	4/10/08	7:35 PM	1521	0.167	5.69	
Fork	4/10/08	7:42 PM	4/11/08	8:15 AM	753	0.094	2.86	MV
Them	4/11/08	8:33 AM	4/11/08	7:42 PM	669	0.067	1.57	
Snow	4/11/08	7:52 PM	4/12/08	8:12 AM	740	0.074	0	
Pony	4/12/08	8:22 AM	4/12/08	7:35 PM	673	0.067	2.73	
Pepper	4/12/08	7:47 PM	4/13/08	7:46 AM	719	0.072	4.58	MV
Orange	4/13/08	7:56 AM	4/13/08	7:34 PM	698	0.070	3.15	
Bird	4/13/08	7:45 PM	4/14/08	8:19 AM	754	0.075	0.94	MV
Sunset	4/14/08	8:29 AM	4/14/08	7:17 PM	648	0.062	0	
Banana	4/14/08	8:16 AM	-	-	-	-	0	Trip Blank
Sage	4/14/08	7:26 PM	4/15/08	8:06 AM	760	0.068	4.37	
Cactus	4/15/08	8:17 AM	4/15/08	7:27 PM	670	0.067	3.88	
Desert	4/15/08	7:35 PM	4/16/08	8:26 AM	771	0.073	4.27	
Lizard	4/16/08	8:35 AM	4/16/08	7:25 PM	650	0.072	1.41	
Phone	4/16/08	7:37 PM	4/17/08	7:28 PM	1431	0.129	2.59	
Glass	4/17/08	5:36 PM	4/18/08	6:50 PM	1514	0.136	2.45	
Ball	4/18/08	6:54 PM	4/19/08	7:16 PM	1462	0.146	0	
Drop	4/19/08	7:21 PM	4/20/08	7:10 PM	1429	0.157	0	
Dew	4/20/08	7:15 PM	4/21/08	6:22 PM	1507	0.136	0	
Stalk	4/21/08	6:30 PM	4/22/08	6:50 PM	1460	0.131	0	
Monkey	4/22/08	6:53 PM	4/23/08	6:03 PM	1390	0.132	0	

[a] MV = minimum value (see text)

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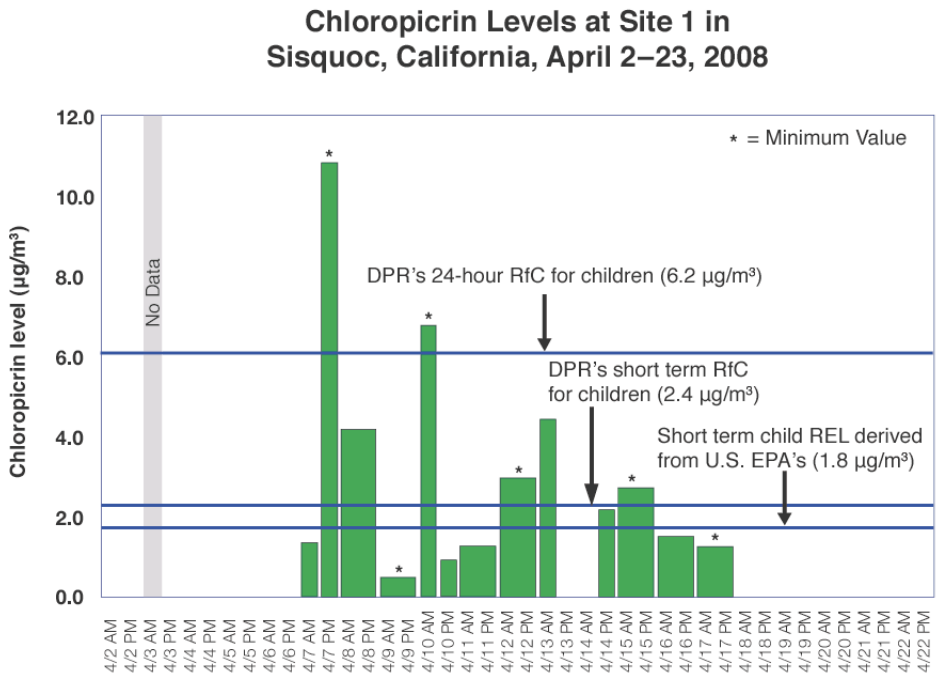

Figure 3: Chloropicrin concentrations in air at Site 1 in Sisquoc, CA, April 2–22, 2008.

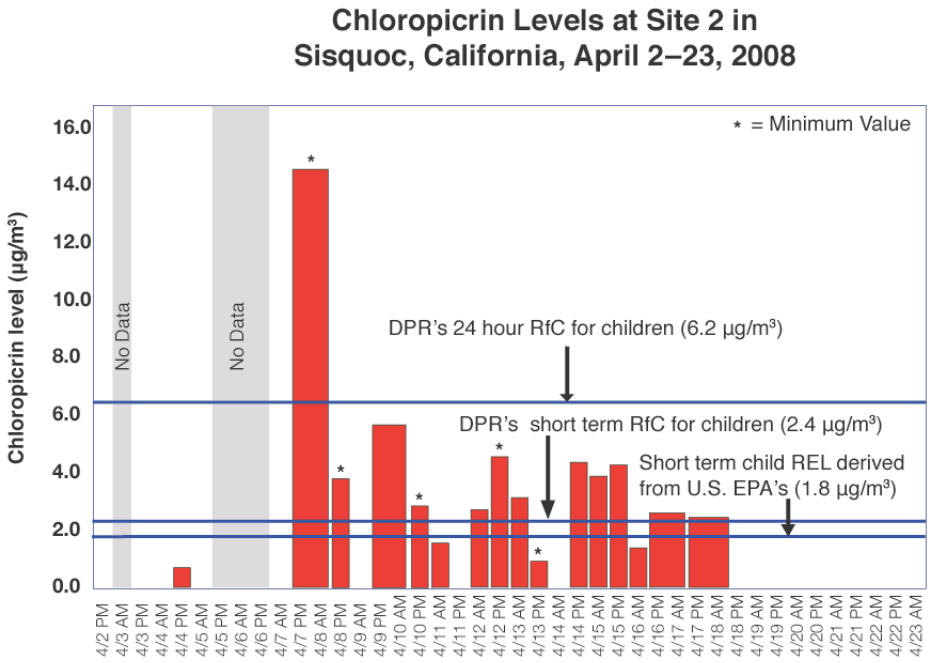

Figure 4: Chloropicrin concentrations in air at Site 2 in Sisquoc, CA, April 2–22, 2008.

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Discussion

Meteorology and Timing of Fumigations

Fumigation operations on April 4, 7, 9, 11, 12, and 14 were scheduled to begin around 6 a.m. Weather station data indicate that during the monitoring, winds in Sisquoc generally blew strongly (7–12 mph) from the west in the afternoon/early evening (noon–8 pm) and at lower speeds (1–3 mph) from the east late at night and early in the morning (11 pm–8 am). The exception to this pattern is the prolonged period of winds from the west on April 18–20. Figures A-1 through A-4 in Appendix 1 show the hourly average wind speed and direction for April 2–8, April 9–15, April 16–22, and April 23–30, 2008.

The Drift Catcher data are consistent with these observations. Compared to daytime samples that were started on the mornings of fumigations, chloropicrin levels were generally higher in samples that were started on evenings following fumigations due to lower wind speeds and the general tendency of the winds to come from the east at night, blowing the fumigant from the field towards the sampling sites. For example, at Site 2, the highest chloropicrin concentrations were in samples "Ant," "Brick," and "Pepper", all of which were overnight samples started on the evenings of April 7, 9, and 12, respectively. These dates correspond to the mornings in which Blocks 2, 3, and 5 were fumigated. At Site 1, the highest concentration was observed in sample "Wave," started the evening of April 7. Consistent with the change in wind pattern on April 18–20 (no wind from the east), chloropicrin was not present in samples beginning on or after April 18. On the previous day with easterly winds, the concentrations of chloropicrin were significantly higher. Thus, on the evenings of April 18 and 19, the westerly winds (Figure A-5) considerably reduced the concentration of chloropicrin in the air near the residences (see Figure 1). This occurrence of prolonged wind from the west for two days toward the end of the chloropicrin application likely protected the community from additional exposures.

Somewhat surprisingly, although Block 1 was fumigated on April 4, chloropicrin was not detected at Site 1 until April 7—the day Block 2 was fumigated—despite overnight winds blowing generally from the fumigated block towards the site.[11] We cannot fully explain this observation, but note that compared to the cumulative amount of chloropicrin applied to the field, the amount "in the ground" from April 4–6 is rather low: only 774 lbs out of the total of 5,418 lbs that were applied through April 14.

The highest-concentration samples were taken overnight starting on April 7, after the application to Block 2 (one of the closest blocks to the sampling sites), when winds were blowing from the field to the neighborhood at very low speeds.

Distances from Fumigated Blocks

At 2.40 $\mu g/m^3$, the TWA chloropicrin concentration for April 4–22 at Site 2 was greater than the TWA for Site 1 during the same period (1.44 $\mu g/m^3$). This is consistent with Site 1 being on the corner of the fumigated area and Site 2 being located at the midpoint of the field edge. Also, while Site 2 was about 60 ft further away from the field edge than Site 1, it was substantially closer to the large blocks (Nos. 3–5) comprising the northern sector of the fumigated area (see Figure 1 and Table 1).

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At both sites, the highest chloropicrin concentration was observed in samples started on the evening of April 7. These data likely represent drift from Block 2, which was fumigated earlier in the day. Block 6, fumigated April 14, was closer to the sampling sites than Block 2, yet chloropicrin levels observed on April 14 and later are lower than levels observed at other times. This is likely due to the fact that, at one acre, Block 6 was significantly smaller than all other blocks (see Table 1, above).

Comparison to Levels of Concern

Chloropicrin is ranked by U.S. EPA as Category I (highly acutely toxic), and California listed it as a Toxic Air Contaminant in April 2010.[12] It is extremely irritating to the eyes and respiratory system. In the lungs, the medium and small bronchi and the alveoli are damaged when exposed to chloropicrin, which results in pulmonary edema at high exposures.[13] It reacts systemically with hemoglobin and interferes with oxygen transport. It is also highly electrophilic, and as a result is genotoxic and carcinogenic. Detecting chloropicrin in the air in a residential area is thus troubling.

To assess whether the levels of chloropicrin observed in this study constitute a significant risk to the people exposed, we compared the measured concentrations to levels of concern used by authoritative government sources. As discussed in greater detail in Appendix 2, a level of concern is a concentration of a contaminant in air above which the risk of adverse effects is unacceptably high (although perhaps still quite small in absolute terms), and below which the risk of harm is deemed acceptably small. It is not a threshold level above which adverse effects are guaranteed or even expected, nor are concentrations below the level of concern necessarily safe.

In general, levels of concern are derived from toxicological studies in which laboratory animals (usually rats, mice, or rabbits) are exposed to a chemical in a controlled environment. For health effects other than cancer, it is assumed that there is a toxicity threshold, and only exposure to levels above the threshold will result in adverse health effects. To assess these effects, scientists determine the highest dose that test animals can tolerate without any detectable signs of illness or distress, the so-called “No Observable Adverse Effect Level” (NOAEL). Regulators then derive a level of concern by dividing the NOAEL by a series of uncertainty factors to account for differences between the test animals and humans and also variability between humans. See Appendix 2 for more detail about levels of concern.

In contrast, for carcinogenic substances it is assumed that cancer risk is a non-threshold event, with risk proportional to exposure. As long as there is some exposure, there is a non-zero probability of developing cancer. Cancer risk approaches zero as exposure approaches zero, but as long as a person is exposed, there is some small chance of the exposure leading to cancer. As discussed in greater detail below, to assess cancer risk scientists use animal studies to determine the relationship between exposure level and the probability of developing cancer. Regulators then apply this relationship to the human exposure scenarios and determine the probability that such exposures will result in cancer. Usually, if a scenario is associated with a risk of cancer of less than one in a million, the risk is considered negligible and the exposure is considered acceptable. Scenarios with greater risk of cancer generally trigger action to reduce exposure.

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Non-Cancer Levels of Concern

In this study, we use levels of concern for acute and short-term exposure derived by the U.S. EPA, DPR, and OEHHA. These are: Reference Concentrations (RfCs) derived by DPR in their 2010 chloropicrin risk assessment,[14] Reference Exposure Levels (RELs) determined by OEHHA,[15] and RELs derived from the Human Equivalent Concentrations (HECs) in U.S. EPA's 2009 risk assessment.[16] These levels of concern represent air concentration in micrograms of pesticide per cubic meter of air ($\mu g/m^3$) equivalent to a dose in milligrams of pesticide per kilogram of body weight (mg/kg) below which the risk of adverse effects is anticipated to be negligible, assuming exposure to chloropicrin alone. U.S. EPA, DPR, and OEHHA use somewhat different assumptions and in some cases had different data available to them, hence the differing values for RfCs and RELs covering the same exposure periods. The DPR and OEHHA levels of concern are quoted directly from agency documents, and we use them without modification. EPA utilized a "Margin of Exposure" approach in its assessment, and we have converted the Agency's target MOEs into RELs**,** as described in the **Calculations** section.

The levels of concern used in this report and their underlying data are summarized in Table 4. A comprehensive discussion of how to interpret air monitoring results is presented in Appendix 2.

U.S. Environmental Protection Agency (EPA) Levels of Concern

U.S. EPA recently completed a multi-year re-evaluation of chloropicrin, during which it repeatedly revised its level of concern for acute (1-hour) exposure to chloropicrin. The assessment of risk from acute exposures was based on a human study in which healthy adults, ages 18–35, were exposed to various concentrations of chloropicrin vapor in a chamber for 30–50 minutes and rated eye, nose, and throat irritation. Certain physiological parameters related to respiration were also recorded periodically. Observed effects were eye irritation, increased nasal nitric oxide (nNO), and differential effects on inspiratory and expiratory flow.[17] Individuals with respiratory problems or chronic illness were excluded from the study.

In US EPA's 2006 Preliminary Risk Assessment for chloropicrin,[18] the Agency used a level of concern of 49 $\mu g/m^3$. This was derived from a benchmark concentration ($BMCL_{10}$) of 490 $\mu g/m^3$ and an interspecies uncertainty factor of one (because it was a human study) and an intraspecies uncertainty factor of 10 to account for potential differences in susceptibility to chloropicrin between different individuals. The U.S. EPA's Human Studies Review Board encouraged the Health Effects Division (HED) to utilize additional uncertainty factors for the protection of children and other vulnerable populations;[17] HED did not take this advice. The Agency also applied this level of concern to 24-hour exposures, even though it was based on a study that exposed subjects for only 30–50 minutes.[19]

In a later phase of the reevaluation process, U.S. EPA issued a Revised Risk Assessment in April of 2007.[20] This assessment removed the intraspecies uncertainty factor of 10, and a value of 490 $\mu g/m^3$ was proposed as the level of concern. The document noted that "73 ppb [490 $\mu g/m^3$] represents a level at which upper respiratory changes and irritation (eyes, nose) would not be expected to occur." Interestingly, U.S. EPA determined that the human study participants differed in their ability to detect eye irritation caused by chloropicrin by a factor of 42 between the 10^{th} and 90^{th} percentile of the population, substantially higher than the factor of 10 they had removed.

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U.S. EPA finalized the chloropicrin risk assessment in May 2009.[16] In this document, EPA indicated that in some cases, acute exposures of up to twice the level of concern (i.e. 980 μg/m^3) would be "acceptable", i.e. an MOE of 0.5.

The final risk assessment also identifies 54 μg/m^3 as the endpoint appropriate for assessing the risk of non-occupational short-term (1–30 day) and intermediate-term (1–6 month) exposure based on significant increases in nasal lesions (rhinitis) in a 13-week mouse study. As this endpoint is from an animal study, the assessment specifies the use of a 30-fold uncertainty factor. This results in a REL of 1.8 μg/m^3 for non-occupational short- and intermediate-term (1 day–6 month) exposure. Since this endpoint is a port-of-entry effect rather than systemic effect, U.S. EPA did not calculate different levels of concern for children and adults.[16]

California Department of Pesticide Regulation (DPR) Levels of Concern

The levels of concern developed by CA DPR in its February 2010 draft risk assessment are lower (i.e. more health-protective) than those used by EPA.[14] DPR derived an acute (1 hour) Reference Concentration (RfC) for chloropicrin for adults and children of 30 μg/m^3 from the same human study used by U.S. EPA described above. DPR also derived 8-hour RfCs for children and adults of 18 μg/m^3 and 39 μg/m^3, respectively, and 24-hour RfCs of 6.2 μg/m^3 and 13 μg/m^3, respectively. DPR's seasonal/subchronic RfCs for children and adults are 2.3 μg/m^3 and 4.9 μg/m^3.

California Office of Environmental Health Hazard Assessment (OEHHA) Levels of Concern

OEHHA has also determined a REL for acute (1 hour) exposure to chloropicrin of 29 μg/m^3, based on decreased respiratory rates observed in mice exposed to chloropicrin vapor for 10 minutes.[21] This REL is more protective than both U.S. EPA's and DPR's levels of concern for a one-hour exposure. This REL incorporates a 30-fold uncertainty factor (3-fold interspecies and 10-fold intraspecies). OEHHA has not determined RELs for longer acute exposures or for subchronic/seasonal exposure.

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Table 4: Summary of Toxicity Information Used To Calculate RELs[a]

Exposure Scenario (Timeframe)	Critical endpoint expressed as a Human Equivalent Concentration (HEC)[b] (µg/m^3)	Effects at LOAEL	Uncertainty Factors	REL or RfC[b] (µg/m^3)
U.S. EPA Acute Adult (1–24 hour)	$BMCL_{10}$ = 490	Eye irritation, increased nasal nitric oxide, altered breathing	1 (interspecies: 1X intraspecies: 1X)	490 (73 ppb)
U.S. EPA Short- and Intermediate-Term (1 day to 6 months) (Adult and Child)	HEC = 54	Rhinitis	30 (interspecies: 10X intraspecies: 3X)	1.8 (0.26 ppb)
DPR Acute (1 hour)	$BMCL_{10}$ = 296	Increased NO concentration in nasal air	10 (interspecies: 1X intraspecies: 10X)	30 (4.4 ppb)
DPR Acute (1–8 hour)	Child: HEC = 1,800 Adult: HEC = 3,900	Nasal discharge, reduced food consumption and body weights, and mortalities during the first few days of exposure in rabbits	100 (interspecies: 10X intraspecies: 10X)	Child: 18 (2.7 ppb) Adult: 39 (5.8 ppb)
DPR Acute (8–24 hour)	Child: HEC = 620 Adult: HEC = 1,300	Nasal discharge, reduced food consumption and body weights, and mortalities during the first few days of exposure	100 (interspecies: 10X intraspecies: 10X)	Child: 6.2 (0.92 ppb) Adult: 13 (1.9 ppb)
DPR Seasonal (7 days to 6 months)	Child: HEC = 230 Adult: HEC = 490	Rhinitis in female rats	100 (interspecies: 10X intraspecies: 10X)	Child: 2.3 (0.35 ppb) Adult: 4.9 (0.73 ppb)
OEHHA Acute (1 hour)	RD_{05} = 890[c]	5 % decrease in respiratory rate	30 (interspecies: 3X intraspecies: 10X)	29 (4.3 ppb)

[a] Data in table is from references 14, 15, and 16.
[b] When calculating HECs for children, DPR uses the breathing rate of 1-year-old infant of 0.59 m^3/kg/day.
[c] In contrast to the other entries in this column, the OEHHA RD_{05} of 890 µg/m^3 is not an HEC, but is instead a concentration (Reference Dose or RD) expected to cause a 5% decrease in respiratory rate in rats exposed for one hour.

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Comparison of Sisquoc Results to Non-Cancer Levels of Concern

The results of this study indicate that acute levels of concern are exceeded at each site at least once during the two and a half week sampling period. Furthermore, the time-weighted-average at Site 2 for the period beginning on the day that the fumigation started and ending 19 days later exceeds both the U.S. EPA and DPR short-term levels of concern. While exceedances of levels of concern are not necessarily anticipated to cause symptoms of acute poisoning, they do represent a potential health concern—the larger the exceedance, the higher the probability of adverse effects from pesticide exposure. When estimated exposures exceed levels of concern, U.S. EPA normally takes action to reduce exposures to below levels of concern.

None of the samples exceeded the U.S. EPA, DPR, or OEHHA 1-hour or 8-hour levels of concern, but the highest observed concentrations do exceed DPR's 24-h level of concern of 6.2 $\mu g/m^3$ for children. These are samples "Wave" and "Ruby" from Site 1 and "Ant" from Site 2. The duration of "Ant" is 24.1 h, but "Wave" and "Ruby" are 12-h samples, so comparing these samples to a 24-h RfC is not necessarily appropriate. However meaningful comparisons can be made to the time-weighted-average (TWA) of these samples with those immediately proceeding or following them. For "Wave" and the sample preceding it ("Glue"), the TWA chloropicrin concentration is 6.3 $\mu g/m^3$ for the 24.1-h period, which exceeds DPR's 24-h child RfC. Using instead the sample that follows it ("Moon"), the TWA chloropicrin concentration for the 36.6-h period is 6.5 $\mu g/m^3$, which also exceeds this level of concern. For "Ruby," using the sampling interval either immediately before or after both yield TWA concentrations that are less than the RfC. Thus, at both sites there is one period of at least 24 hours that exceeds DPR's 24-h child RfC.

DPR's 24-h adult RfC of 13 $\mu g/m^3$ was exceeded in one 24-h sample: "Ant," collected at Site 2 on April 7-8, with a concentration of 14.5 $\mu g/m^3$.

The TWA chloropicrin concentrations of 1.44 $\mu g/m^3$ and 2.40 $\mu g/m^3$ at Sites 1 and 2, respectively, were also calculated. The concentration at Site 2 exceeds both U.S. EPA's short- and intermediate-term REL of 1.8 $\mu g/m^3$ and DPR's seasonal child RfC of 2.3 $\mu g/m^3$, indicating an unacceptably high risk of adverse effects for people who spent significant time in the vicinity of that site.

It should be noted that methyl bromide was applied concurrently with chloropicrin and that the Drift Catcher operators also noted a helicopter spraying fields near the fumigated area during the sampling. However only chloropicrin was monitored in this study. Given the high levels of chloropicrin observed, it is reasonable that there was also some co-exposure to methyl bromide during the monitoring period. Exposure to the chemical(s) applied aerially to the other field is also a possibility. The effects of combined exposure to methyl bromide and chloropicrin have not been evaluated, but it is likely that the potential for adverse effects increases with exposures to additional chemicals.

Cancer Risks from Chloropicrin Exposure in Sisquoc

U.S. EPA does not consider chloropicrin to be carcinogenic by inhalation exposure. The Agency did find, however, evidence for mutagenicity in bacterial cells, as well as conflicting evidence of carcinogenicity by the oral route, but did not evaluate all available data, noting that "possible increased incidence of mammary fibroadenoma in the high-dose females in a two-year gavage study (MRID 43744301) in rats has not been fully evaluated."[20]

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In contrast, DPR's more recent evaluation concluded that, "the weight of evidence was sufficient to do a quantitative assessment of the carcinogenic risk using a linear approach," and the Department derived a cancer potency factor of 2.2 $(mg/kg\text{-}day)^{-1}$ for chloropicrin.[14] This value is higher than that for ethylene oxide, a Known carcinogen according to the International Agency for Research on Cancer and the US National Institutes of Health, although lower than that for benzidine, a highly potent carcinogen used to synthesize dyes. Table 5 provides the cancer potency factors of other common chemicals for comparison.

Table 5: Cancer Potency Factors for Common Chemicals

Chemical	Use	Cancer Potency by Inhalation $(mg/kg\text{-}day)^{-1}$
Methylene chloride	Industrial solvent	0.0035[a]
Propylene oxide	Fumigant pesticide for stored nuts and fruit, polymer precursor	0.013[a]
Pentachlorophenol	Wood preservative	0.018[a]
Formaldehyde	Resin component in particle board, glues	0.021[a]
1,3-Dichloropropene	Fumigant pesticide	0.04[b]
Perchloroethylene	Dry cleaning solvent	0.051[a]
Benzene	Industrial solvent	0.1[a]
Metam sodium	Fumigant pesticide	0.185[b]
Ethylene oxide	Hospital sterilant	0.31[a]
Chloropicrin	Fumigant pesticide	2.2[b]
Benzidine	Dye precursor	500[a]

[a] Reference 22.
[b] Reference 23.

A cancer potency factor can be used to determine cancer risk, which is defined as the probability of a person developing cancer during a lifetime as a result of the exposure. The cancer risk is expressed as the number of people who are likely to get cancer per million people. Cancer risks exceeding one in one million represent risks of concern. Below, we use DPR's cancer potency factor for chloropicrin to calculate the cancer risk associated with exposure to the chloropicrin levels observed in this study. Cancer risks are evaluated for a variety of scenarios using several different assumptions about the length of residence in the exposed community. Cancer risks for children are also presented. In all cases, cancer risks from the chloropicrin exposure in Sisquoc were found to exceed the one in one million level of concern, ranging from five to 80 times the acceptable risk level.

Lifetime Cancer Risk

Cancer risk is most often calculated assuming exposure occurs over the course of a 70-year lifetime, using the average daily exposure and the potency factor to estimate risk. Lifetime cancer risk estimates were developed for chloropicrin exposure scenarios based on the monitoring data from Sisquoc.

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For the 19-day period from April 4–22, the TWA chloropicrin concentrations were 1.44 μg/m^3 and 2.40 μg/m^3 at Sites 1 and 2, respectively, and it was assumed that this represents total annual chloropicrin exposure (i.e., there is no additional exposure beyond these 19 days/year, but that this exposure happens every year). The results indicate that the lifetime cancer risk exceeds the level of concern of one excess cancer per million people by a factor of 46 at Site 1 and 77 at Site 2 (see Table 6). The methodology employed is identical to that used by DPR in its chloropicrin risk assessment (see the **Calculations** section for full details).[14]

Table 6: Lifetime Cancer Risk Estimates for Sites 1 and 2 in Sisquoc, CA

Parameter	Site 1	Site 2
Average concentration during monitoring period (μg/m^3)	1.44	2.40
Exposure frequency as a percent of a year	5.2%	5.2%
Average annual concentration (μg/m^3)	0.075	0.125
Annual exposure[a] (mg/kg-day)	2.84×10^{-5}	4.73×10^{-5}
Cancer potency factor (mg/kg-day)$^{-1}$	2.2	2.2
Lifetime Cancer Risk (excess cancers per million people)	46	77

[a] For an adult breathing rate of 0.28 m^3 per kilogram per day, representing the predominant breathing rate for a 70-year life span.

Childhood Cancer Risks

Children are more susceptible to cancer risk because they are growing and developing. To assess this increased risk, OEHHA has devised a method for calculating cancer risks for early life exposure to carcinogens.[24, 25] We applied this method to chloropicrin exposure scenarios based on the chloropicrin air concentrations observed in this study, and calculated the resulting cancer for children. In doing this analysis, we utilized time-weighted-average (TWA) breathing rates for different life stages, as given in the Exposure Factors Handbook.[26] The scenarios examined are postnatal exposure from birth through weaning (0 to 2 years), juvenile exposure (2 to 16 years), and adult life exposure (16-70 years). The results summarized are summarized in Table 7 below; see the **Calculations** section for details.

Table 7: Childhood Cancer Risk Estimates for Sites 1 and 2

Exposure Scenario	Cancer Risk per Million Site 1	Cancer Risk per Million Site 2
Infant (birth to 2 years)	23	39
Juvenile (2–16 years)	38	62
Adult (16–70 years)	30	50
Lifetime (birth–70 years)[a]	91	151

[a] Using TWA breathing rates for different life stages. The TWA breathing rates are slightly different from the standard adult breathing rate of 0.28 m^3/kg-day, which results in a slightly different lifetime cancer risk compared to the values in Table 6.

In all cases, cancer risks exceed the one in a million level of concern. Even for the scenario of relatively brief exposure early in life, significant cancer risk is predicted: 23–38 excess cancers per million for exposure during infancy (2 years). The risks calculated for juveniles between two and 16 years of age are 38–62, and for lifetime exposure range from 91–151, approximately double the risk calculated without accounting for age-specific sensitivity. The OEHHA method gives more weight

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to exposures occurring early in life than to those taking place later in life, which reflects the increased sensitivity of developing organisms to carcinogens, and also the fact that the earlier in life the exposure occurs, the more time there is available for cancer to manifest.

Less-Than-Lifetime Cancer Risk

The lifetime cancer risks calculated in the section above exceed U.S. EPA and DPR's level of concern of one excess cancer per million people. However, the calculation assumes 70 years of exposure, an unlikely amount of time to spend living in the same place and/or for fumigations with the same chemical to be taking place. In fact, OEHHA recommends 9 and 30 years as the central tendency and high-end estimates of the typical length of residency at a home, respectively.[26] Therefore, in this section, we calculate the cancer risks associated with the more likely scenarios of spending birth until age 9 and birth through age 30 exposed annually to the levels of chloropicrin observed in Sisquoc.

U.S. EPA and DPR do not typically calculate less-than-lifetime cancer risks, therefore we employed the methodology developed by OEHHA. As discussed in greater detail in the **Calculations** section, this methodology essentially adjusts the standard lifetime cancer risk calculation used in the preceding section by incorporating a multiplier equal to the number of years exposed divided by 70, i.e. the lifetime risk is multiplied by the fraction of life exposed. As in the calculation of age-adjusted risks, we used age-adjusted TWA breathing rates to estimate exposure during different life stages. The results, shown in Table 8 indicate that even for these abbreviated exposure durations, the risk ranges from 42 to 89 excess cancers per million people.

Table 8: Less Than Lifetime Cancer Risk Estimates for Sites 1 and 2

Exposure Scenario (Sites 1 and 2 averaged)	Cancer Risk per Million People Site 1	Cancer Risk per Million People Site 2
9-year residency (birth to age 9)[a]	42	70
30-year residency (birth to age 30)[a]	54	89

[a] Using TWA breathing rates for different life stages.

Exposure Assumptions

In the above sections, we document exceedences of non-cancer levels of concern for acute and subchronic exposure as well as levels of concern for carcinogenicity. We therefore conclude that residential exposure to chloropicrin is unacceptably high. A concern previously expressed about community air monitoring results is that samplers were stationed outside, but residents do not spend 24 hours per day outside; instead, people spend significant time indoors, where contaminant levels are assumed to be lower. Residents may also leave the community entirely, for example, to work or attend school in a different area. Sometimes these factors may contribute to reduced exposures; however, the data indicate that the exposure assumptions used in the calculations are realistic for some fraction of the population, in consideration of the following:

- There is little actual evidence to support the presumption that pesticide concentrations indoors will be lower than the corresponding outdoor concentrations. Few studies have been conducted that compare indoor to outdoor pesticide exposures. However, two of those studies found that indoor air concentrations were equal to or higher than outdoor concentrations.

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Pesticide Research Institute monitored a fumigation with 1,3-dichloropropene (1,3-D) in August 2007 in Moss Landing, CA and found that "The highest 12-hour concentration of 1,3-D was measured *indoors* at 10963 Potrero Road overnight on August 22–23, 140 feet from the nearest edge of the fumigated field, Block 4.[27] No windows were open inside the home, and the door was only opened twice, once to place the canister in the house and again to pick it up at the end of the sampling period. The measured concentration outside the house for the same time period was nearly identical at 136 $\mu g/m^3$. This observation demonstrates that, at least for poorly insulated homes, being inside offers no protection from drifting fumigants."

A study conducted by the California Air Resources Board in Arvin, California for the fumigant MITC indicated that concentrations indoors were sometimes higher than outdoors, sometimes lower, and other times nearly the same.[28]

- Particularly in the summer and in hot, humid areas such as California's agricultural valleys, it is extremely unlikely that homeowners would not employ some measures to reduce indoor temperatures. They are likely to use either air conditioners or "swamp coolers" or simply open windows and doors and possibly turn on a fan. Regardless of the method, there will be significant exchange between indoor and outdoor air.

- Staying inside with windows and doors shut may be an effective defense against plumes of air contaminants that are likely to dissipate in relatively short timeframes—hence the logic of "shelter in place" warnings for refinery fires and other short-term toxic releases. But when an airborne contaminant is present in the air over a sustained period of time—as chloropicrin was in this study—it will end up indoors. Homes are not hermetically sealed.

- Finally, while it is true that most members of a community leave their homes regularly for work, school, or other reasons, this is not the case for everyone. Many people spend all or nearly all of their time within their own home or neighborhood, including retirees, people who work at home, stay-at-home parents and their children, children on summer break, and those who are sick. In fact, in this study, one Drift Catcher operator was a retiree, and another worked out of her home.

Newly Mandated Buffer Zones Would Not Have Protected Sisquoc Residents

The U.S. EPA recently completed a comprehensive assessment of all fumigant pesticides, including chloropicrin. This "Fumigant Cluster Assessment" concluded that the use of fumigants poses significant risks to human health and the environment, and mandated a number of new restrictions on their use to mitigate some of these risks. These risk mitigation measures will be phased in by 2012. Buffer zones between fumigated fields and occupied structures (e.g. homes) are one such measure that is being required to protect people who live and work around fumigated fields.[29]

The newly mandated buffer zones would not have mitigated the chloropicrin exposure documented in this study. The buffer zone distances that would have been required had the new regulation been in place at the time are shown in Table 9, below. Approximate distances from Sites 1 and 2 to the edge of each block are given as well. These buffer zones were calculated according to the amended REDs for chloropicrin and methyl bromide, and are based on the size of each application block, the application method, and the application rate. In this study, we monitored a fumigation with Tri Con

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57/43, which is 57% methyl bromide and 43% chloropicrin, at a total application rate of 300 lbs/acre. Buffer zones were thus calculated assuming application rates of 171 lbs/acre for methyl bromide and 129 lbs/acre for chloropicrin. The amended chloropicrin RED[16] specifies that for such mixtures, the buffer zone should be based on component of the mixture in highest concentration, which in this case is methyl bromide.

As shown in Table 9, for each block in the April 2008 Sisquoc fumigation, the newly mandated buffer zone is less than the actual distance between the edge of that fumigated block and either of the Drift Catcher sites. In other words, the new buffer zones would not have changed the application monitored in this study. In fact, fumigations substantially closer to homes than this one will still be permitted. These data indicate that U.S. EPA's new buffer zones are not adequately protective of people who live and work near fields.

Table 9: U.S. EPA-Required Buffer Zones To Be Implemented for Chloropicrin in 2011

Block	Buffer Zone based on methyl bromide RED[a] (ft)	Buffer Zone based on chloropicrin RED[b] (ft)	Approximate Distance of Block Border from Site 1 (ft)	Approximate Distance of Block Border from Site 2 (ft)
1	124	25	390	450
2	105	25	130	190
3	124	25	840	170
4	264	46	>1000	730
5	264	46	>1000	430
6	25	25	70	140

[a] Based on Table 5 of the Amended Methyl Bromide RED at page 53.[30] Block sizes and application rate were rounded up to closest value listed in the table. No buffer zone credits were applied.
[b] Based on Table 7 of the Amended Chloropicrin RED at page 53.[31] Block sizes and application rate were rounded up to closest value listed in the table. No buffer zone credits were applied.

The amended chloropicrin RED states that, "Based on several factors including the severity and reversibility of the effect and also the quality of the hazard database, *the goal of the buffer zone distances in the July 2008 RED was to reach an air concentration of 0.073 ppm which equates to an MOE of 1*."[31]

We have serious doubts that a target MOE of one is sufficiently protective of human health. While we see the logic in removing the interspecies uncertainty factor since this assessment is based on a human study, it is inappropriate to also remove the intraspecies uncertainty factor. The test subjects were healthy adults, with no chronic or acute respiratory disease, such as asthma. There is no indication that they performed any exercises or tasks during their controlled exposure that would have elevated their breathing rates. There was also substantial variability between the human subjects—for eye irritation, the intra-subject variability between the 10th and 90th percentile subject was a factor of 42; for the odor threshold, variability was a factor of 1.9. Thus, it is inappropriate to assume that an MOE of 1 will be protective of children, the elderly, the sick, or other individuals with potentially increased sensitivity to respiratory chemical insult, or to individuals who are exerting themselves physically. U.S. EPA even admits that these buffer zones will not achieve the target MOE in all situations: "if the target MOE was not reached, at minimum half of the target (MOE 0.5), which corresponds to minor, reversible effects, was achieved at high percentiles of [modeled exposure]."[16]

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In contrast to US EPA, DPR employed an intraspecies uncertainty factor in its determination of an RfC from this human study. Thus DPR's acute Level of Concern is more protective.

Comparison of Sisquoc Data to Other Air Monitoring Studies

As part of the implementation of the California Toxic Air Contaminant Act, the California Air Resources Board (ARB) has monitored many pesticide applications, providing information on acute (short-term) exposure to pesticides via drift.[32] In these studies, air sampling stations are generally set up between 25 and 500 feet from the borders of the field on all sides. All pesticide applications monitored by the ARB were carried out according to label instructions. Therefore, their monitoring results represent a best-case scenario in terms of applicator compliance with best practices to reduce drift. Three such application studies have been conducted for chloropicrin.

The ARB has also conducted air monitoring in regions of high pesticide use, but some distance from application sites to provide information on longer-term, seasonal exposures. In these seasonal, ambient air monitoring studies, sampling stations are generally located atop government buildings such as schools, firehouses, and offices. Two seasonal monitoring studies have been conducted for chloropicrin. The results of ARB's application and seasonal monitoring studies for chloropicrin are summarized below.

Application Site Monitoring Studies for Chloropicrin

The three chloropicrin applications monitored by ARB took place between 2001 and 2005 in Monterey,[33] Santa Cruz,[3] and Santa Barbara counties.[34] The details of these studies are summarized in Table 10, and Figure 5 shows the maximum 12- and 24-hour chloropicrin concentrations measured in these studies.

Table 10: Chloropicrin Application Monitoring Conducted by ARB

Location of Application	Application Method and Rate	Field Size (acres)	Distance of Samplers from Field (feet)	Range of Concentrations Observed ($\mu g/m^3$)	Reference
Monterey County, 2001	Shank tarped bed; 50:50 chloropicrin: MeBr @ 125 lbs/acre each	22	870	2–39	33
Santa Cruz County, 2003	Shallow shank tarped bed; 50:50 chloropicrin: MeBr @ 150 lbs/acre each	4.8	160	0.084–270	3
Santa Barbara County, 2005	Drip tarped bed; 94% chloropicrin @ 200 lbs/acre	8.2	60	0.3–415	34

The highest concentrations adjacent to a fumigation were observed in Santa Barbara County in 2005 at 252 $\mu g/m^3$ (24 hours) and 415 $\mu g/m^3$ (12 hours). The maximum concentrations observed in the Monterey County study, 28 $\mu g/m^3$ (24-hour) and 39 $\mu g/m^3$ (12-hour), were the lowest of the three studies. The peak concentrations observed in Sisquoc were somewhat lower: 10.8 $\mu g/m^3$ and

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14.5 µg/m^3, for the 24-hour measurements at Sites 1 and 2, respectively, and 6.79 µg/m^3 and 4.58 µg/m^3 for the 12-hour samples.

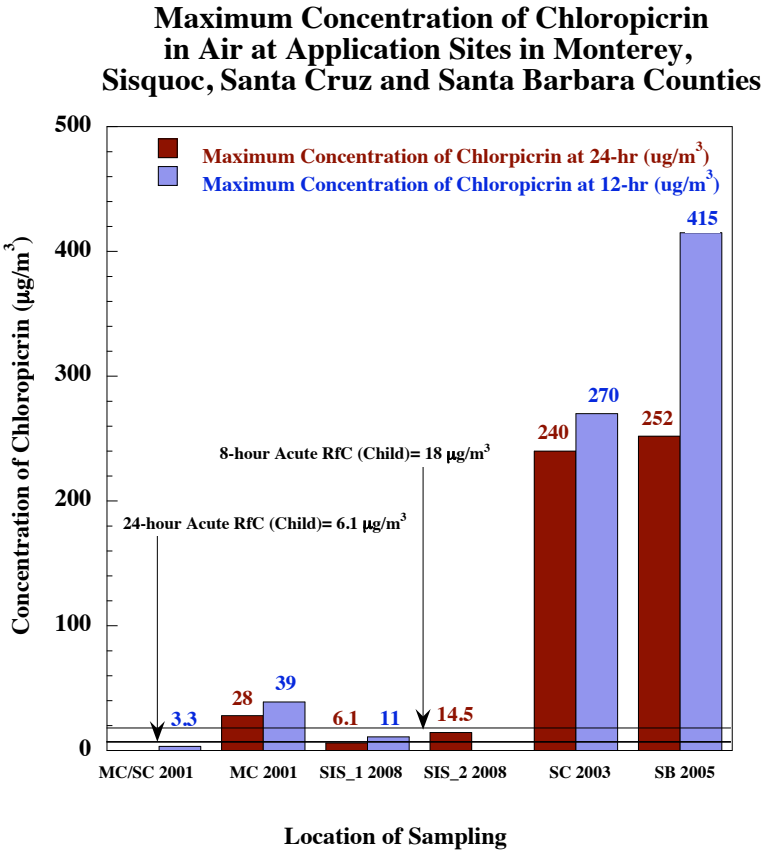

Figure 5: The maximum 12- and 24-hour concentrations of chloropicrin measured in various California counties (Monterey and Santa Cruz Counties = MC/SC, Monterey County= MC; Sisquoc, Sites 1 and 2= SIS_1 and SIS_2, respectively; Santa Cruz County = SC; Santa Barbara County= SB) during an application. See Table 10 for sampling details such as distance from the field. A 12-hr maximum concentration of chloropicrin for Site 2 in the Sisquoc, CA (SIS_2) air monitoring study was unavailable. The MC/SC 2001 sample was taken as a background sample measured prior to application and plotted here for comparison.

As described in Table 10, the details of the applications monitored by ARB differ greatly between one another (e.g. different application methods and rates, sampler placement, and field sizes), and thus it is not surprising that a wide range of chloropicrin concentrations were observed, ranging from 0.1 to 415 µg/m^3. This lack of consistency makes it difficult to draw meaningful comparisons between the Sisquoc results and those of the ARB, except to note that the concentrations measured in Sisquoc do not represent a worst-case scenario. The ARB study that most closely resembles the Sisquoc study is the one conducted in Monterey: both are tarped, shank injection applications and the application rates are similar (129 lbs/acres vs. 125 lbs/acres). While still smaller than the Sisquoc application, at 22 acres it is the largest fumigation of the three ARB studies, and as in Sisquoc, it was carried out over several days. As expected, the chloropicrin levels observed in Sisquoc are most similar to those from this study: the maximum 24-hour level in Sisquoc was 14.5 µg/m^3 versus 28

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μg/m^3 in Monterey. This 2-fold difference in maximum observed concentrations could be due to any number of factors:

- *Sampler placement:* ARB places samplers on all sides of the monitored field, in order to capture representative concentrations in the plume. Thus, ARB studies are likely to include samples from the areas with highest concentration. In our study, only two samplers were employed, both off the western edge of the field. It is possible that chloropicrin levels approaching or exceeding those observed by DPR occurred on other sides of the field.

- *Tarp type:* Tarps vary in permeability to chloropicrin, and the Sisquoc application may have employed a different type than those in the ARB studies.

- *Weather:* Differences in weather and wind patterns could have contributed to differences in results. All of the ARB studies were conducted during the fall (October-November) whereas our study was performed in the spring, when weather conditions may differ substantially. For instance, in the Santa Cruz study, the report indicated that the results may not be representative due to the occurrence of rain both before and during the monitoring period.[3]

- *Timing:* In Sisquoc, the applications took place in six blocks across 10 days, while in Monterey the three blocks were fumigated consecutively over 3 days.

- *Additional applications:* In ARB's Monterey study and others, applications of chloropicrin had recently taken place in fields adjacent to the monitored application. In fact some "blank" samples collected just prior to the monitored fumigation actually contained chloropicrin. These prior applications may have contributed to the concentrations observed in these studies. In contrast, no chloropicrin applications other than those discussed in this report were conducted near Sisquoc before or during the sampling period.

Seasonal Air Monitoring Studies for Chloropicrin

In 2001, Air Resources Board (ARB) conducted seasonal air monitoring studies for chloropicrin in Monterey,[4] Santa Cruz,[4] and Kern Counties.[5] The Monterey and Santa Cruz studies were conducted from September through November to coincide with the season when fumigants are usually applied to prepare the soil for planting strawberries. All samplers were placed on the roofs of school buildings. The four sites in Monterey and two sites in Santa Cruz were sampled over 24-hour periods, with sampling occurring randomly over the full seven-day week during the sampling period (4 sample periods/week). The range of chloropicrin measured at these two sites was <MDL to 14.3 μg/m^3 with an eight-week time-weighted average concentration of 0.41 to 2.27 μg/m^3, depending on the site.

The study in Kern County was conducted at five sites from June 30 through August 31, coinciding with the use of fumigants prior to the planting of a variety of crops in the area. Daily concentrations ranged from <MDL to 0.75 μg/m^3 with an eight-week TWA concentration of <MDL to 0.042 μg/m^3.

Figure 6 illustrates the maximum concentrations observed at these sites in comparison with those from Sisquoc. The highest level observed in the ARB ambient studies was 14.3 μg/m^3, from La Joya Elementary School site (LJE_M on Figure 6) in Monterey. The maximum concentrations observed

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in Sisquoc are slightly higher at 14.3 µg/m^3, and average concentrations at the Sisquoc sites exceed the average concentrations for the majority of sites monitored by ARB. This is in line with expectations, as the samplers in Sisquoc were located within 70 to 1,000 feet of the field being fumigated, while ARB's sites were intentionally located such that they were not in the immediate vicinity of any applications

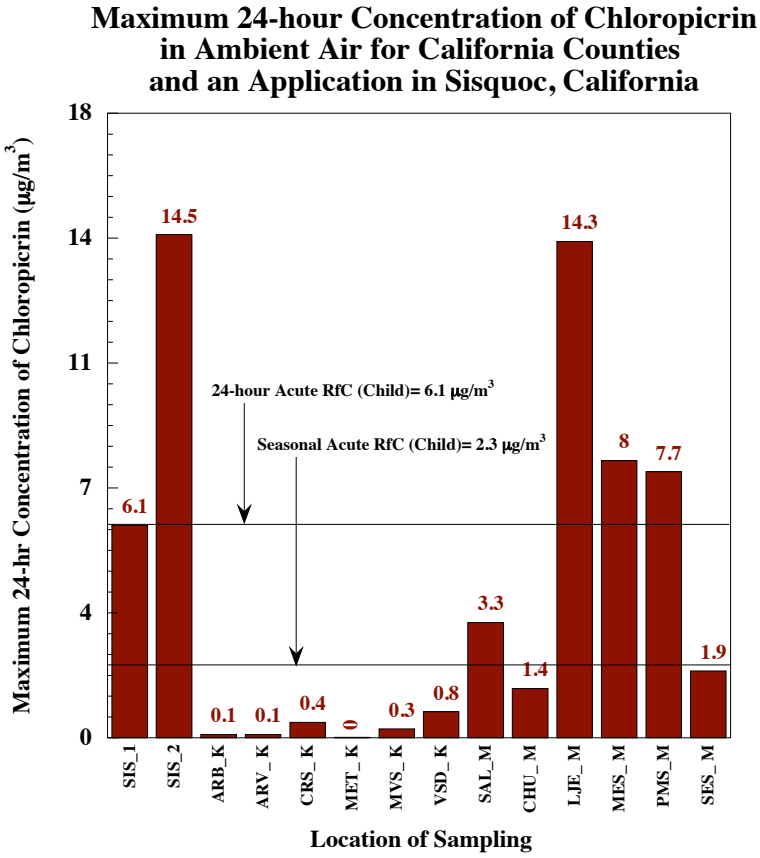

Figure 6: Maximum 24-hour concentration of chloropicrin measured in various California counties. The DPR RfC values (24-hour acute and seasonal) for a child are displayed as a basis for comparison with the measurements. The values for the application monitoring performed at SIS_1 and SIS_2 are plotted for comparison with the ambient air monitoring performed at all other sites represented here. [SIS_1= Sisquoc Site 1, SIS_2= Sisquoc Site 2, ARB_K= Ambient Air Monitoring Station in Kern Co., CRS_K= Cotton Research Station in Kern Co., MET_K= Mettler Fire Station in Kern Co., MVS_K= Mountain View School in Kern Co., VSD_K= Vineland School District-Sunset School in Kern Co., SAL_M= Ambient Monitoring Station in Monterey Co., CHU_M= Chualar School in Monterey Co., LJE_M= La Joya Elementary School in Monterey Co., MES_M= MacQuiddy Elementary School in Monterey Co., PMS_M= Pajaro Middle School in Monterey Co. and SES_M= Salsepuedes Elementary School in Monterey Co.].

Health Effects of Chloropicrin

Short-term Effects, High Exposures

Historically, chloropicrin was used as a poisonous tear gas during World War I, inducing severe eye and respiratory system irritation, nausea and vomiting. Inhalation of high concentrations of or prolonged exposure to chloropicrin results in shortness of breath, a blue color to the skin, and weakness. Chloropicrin primarily affects the medium and small bronchi, but also injures the small

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air sacs in the lung—the alveoli—resulting in pulmonary edema, which is often the cause of death. Death can occur within a few hours of high exposure due to effects on the upper and lower airways. Even if initial symptoms are not severe, death may occur three or four days later due to respiratory infection (chemical pneumonia).[35]

Short-term Effects, Low-Level Exposures

The primary symptoms observed with short and long-term exposure to low levels of chloropicrin are eye, throat, and respiratory system irritation, lacrimation (tearing), coughing, headache, nausea and vomiting. Participants in the human study (used by US EPA and CA DPR in their risk assessments) exposed for up to one hour reported eye irritation as the most sensitive endpoint.[36, 37] This study, which was sponsored by the chloropicrin registrants, limited participation to healthy young adults; people with pre-existing respiratory conditions or illness were excluded. Nonetheless, a large variation in sensitivity among the subjects of this study was observed.

In the human study, two upper respiratory parameters, nasal nitric oxide (nNO) and air flow were measured for one-hour exposures that occurred one day at a time. These physiological changes indicated signs of nasal congestion and engorgement. Approximately 10–30% of the subjects failed to identify chloropicrin in the eyes, nose or throat at any concentration during the study, while 30–40% of the subjects could detect chloropicrin at the lowest concentration tested. The mechanism of action with respect to sensory irritation was shown to involve the direct interaction of the compound with the free trigeminal nerve endings in the respiratory mucosa, primarily affecting the medium and small bronchi.[38]

In animal studies of developmental toxicity, maternal toxicity outcomes observed included increased mortality, gasping and labored breathing, increased salivation, clear nasal discharge, red area around eyes and excessive tearing (lacrimation). Reduced body weights and food consumption, as well as red discoloration of the lungs in rabbits were also observed.

Long-term Effects

No studies are available on the effects of chronic exposure of humans to chloropicrin.[39] Animal studies indicate lowered activity levels and decreased startle response. Increased mortality was noted at higher concentrations (0.5–1 ppm). Irritation of the respiratory tract was observed, as well as increased lung and liver weights in rats. In mice, lung masses and kidney cysts were observed, as well as damage to the alveoli in the lungs and bronchiectasis (irreversible dilation of the bronchial walls).

The U.S. EPA does not consider chloropicrin to be carcinogenic by the inhalation route of exposure.[16] In contrast, DPR concluded that a genotoxic mode of action for tumor formation is likely based on increases in lung tumors in inhalation-exposed rats and in mammary tumors in orally exposed rats.[14] DPR therefore conducted a quantitative assessment of carcinogenicity for current chloropicrin use patterns.

Developmental and Reproductive Effects

While no human data are available, developmental and reproductive effects attributable to chloropicrin were seen in studies of pregnant animals, including reduced number of implantation sites, increased pre- and post-implantation losses, late-term abortions, and visceral and skeletal

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defects in fetuses. Other adverse effects reported in developmental toxicity studies were reductions in maternal body weights and food consumption, and macroscopic and microscopic lesions in the lungs of the adult.

Mechanism of Action

The mechanism of action for chloropicrin is not well understood, but current work indicates that chloropicrin reacts with thiol groups of certain proteins such as glutathione (GSH) and hemoglobin.[40] These reactions are irreversible, resulting in loss of protein function. In mutation assays, the addition of GSH alone converted chloropicrin to a mutagenic metabolite either through reductive chlorination or through the formation of a reactive intermediate GSH conjugate, such as $GSCCl_2NO_2$ or $GSCHClNO_2$. Chloropicrin has also been shown to inhibit pyruvate (PDH) and succinate dehydrogenase (SDH).[41] The PDH and SDH enzymes are possible targets for lacrimatory effects of chloropicrin because they contain thiol groups in their active sites. The data suggest that the acute toxicity of chloropicrin can be attributed to the parent compound or metabolites other than the dehalogenated metabolites. Further, chloropicrin toxicity may be associated with the inhibition of PDH and elevated oxyhemoglobin.[41]

Poisoning Incidents related to Chloropicrin

Over the course of the last several years, chloropicrin has been the cause of over 1,000 poisoning incidents reported to the California Pesticide Illness Surveillance Program.[42,43] Two of the largest chloropicrin-related incidents occurred in Kern County in 2003 and Monterey County in 2005. The details of these and other chloropicrin-related poisoning incidents in California are summarized in Table 11. In the majority of these incidents, individuals most commonly suffered respiratory distress, lacrimation, headache, nausea, and vomiting as a result of inhalation of chloropicrin. The resultant poisoning in these events was occasionally, but not always, due to improper application practices (non-adherence to buffer zone regulations) as well as effects of temperature inversions and changes in wind patterns.

It is important to note that most often poisoning incidents occur in and directly adjacent to fields where the farm workers and their families reside. Therefore, the number of poisoning incidents reported is probably under-estimated, as these communities are often reluctant to speak out regarding such occurrences. Further, follow-up with affected persons in these communities presents challenges, making it difficult to accurately document the long-term damage sustained as a result of exposure.

There are several documented accounts of more severe cases involving prolonged inhalation of chloropicrin where the affected individuals experienced shortness of breath, cyanosis, weakness and sometimes death.[44, 45, 46]

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Table 11: Summary of Poisoning Incidents in California

Location and Year	Number of People Affected	Distance from Treated Field (feet)	Concentration of Chloropicrin in Product Applied	Violations of Label Instructions?	Temperature Inversion?	Comments
Monterey County, 2005	336	-----	-----	Multiple violations	-----	-----
Kern County, 2003	172	1,320	100% chloropicrin @ 80 lbs/acre	Yes: possible failure to adequately contain chloropicrin after application.	Yes: Change in wind direction in the evening toward residences.	-----
San Joaquin County, 2003	12	100–160	34.7% chloropicrin: 61.1% 1–3 dichloropropene	No	Wind from the E and NW.	Modification of grower's permit.
San Luis Obispo County, 2002	14	140–800	19.8% chloropicrin: 80% MeBr	No	Variable wind direction and speed.	No eye irritation reported.
San Joaquin County, 2001	10	185	25% chloropicrin: 75% MeBr @ 350 lbs/acre	Yes: tears in tarp post-application and fumigation of a larger than allowed area.	Yes: wind blowing 1–4 mph from W and NW.	-----
San Luis Obispo County, 2001	12	140–800	42.6% chloropicrin: 57% MeBr @ 250 lbs/acre	-----	-----	No eye irritation reported.
Monterey County, 2000	152	160 and 250	49.5% MeBr: 41.5% chloropicrin @ 325 lbs/acre	No	Yes: temperature rose 10 deg between 8 and10 a.m. but ground temperature remained cool.	-----
San Joaquin County, 1999	6	137	42.2% chloropicrin: 56.8% MeBr @ 350 lbs/acre	Yes: buffer zone was less than the required 200 feet.	-----	Stable atmospheric conditions were partially responsible for the incident.
Monterey County, 1998	7	90	25% chloropicrin: 75% MeBr @ 275 lbs/acre	Yes: buffer zone was 17 feet not the required 30 feet.	Some wind	-----

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Location and Year	Number of People Involved	Distance from Treated Field (feet)	Concentration of Chloropicrin	Any Mistakes Made?	Temperature Inversion?	Comments
Monterey County, 1995	9	90	33% chloropicrin: 67% MeBr @ 350 lbs/acre	Multiple Violations	Some wind	Violations include insufficient buffer zone.
Ventura County, 1995	16 (underestimated)	215–875	100% chloropicrin @ 100 lbs/acre	No	Yes	Factors contributing to the incident include late afternoon application and temperature inversion
Tulare County, 1993	1	Application Worker	25% chloropicrin:75% MeBr @ 275 lbs/acre	-----	-----	-----
Merced County, 1992	6	100	33% chloropicrin: 67% MeBr @ 323 lbs/acre	No	Yes: wind speed was 5–7 mph and blowing from the NW immediately following application.	-----
San Diego, 1992	6	150	33% chloropicrin: 67% MeBr @ 436 lbs/acre	2 violations: didn't obtain a recommendation from a licensed Ag pest control advisor and for exceeding maximum label rate of 400 lbs/acre.	No	-----
Ventura County, 1992	11+	412	33% chloropicrin: 67% MeBr @ 355 lbs/acre	Multiple Violations	Yes: changes in wind speed and direction during and after application.	-----

Sources: References 42 and 43.

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Conclusions

The data collected in Sisquoc demonstrates that the levels of chloropicrin found in the air in Sisquoc following a tarped, shank injection application in April 2008 exceeded levels of concern for both short-term exposure and cancer risk. If such fumigations were to become annual events in the area, with similar concentrations to those observed in 2008 reoccurring every spring, people living in the area will suffer acute health effects such as respiratory distress and an unacceptably high risk of cancer as a result. Specifically, exposure scenarios spanning a lifetime, 30 years, and various periods of childhood all resulted in cancer risks exceeding EPA's level of concern of one excess cancer per million people.*

Co-exposure to methyl bromide also occurred, as it was applied simultaneously with chloropicrin. Methyl bromide concentrations were not determined, and thus risks associated with methyl bromide exposure could not be quantified. Additive or synergistic effects associated with co-exposure are likely.

These results are from a single fumigation; nevertheless, the data raise grave concerns about fumigant exposure generally and the failure of mitigation measures such as buffer zones to protect communities. In fact, exposures could have been much higher, for the following reasons:

- In the monitored application, chloropicrin was applied at a rate of 129 lbs/acre. Much higher application rates are allowed for chloropicrin (up to 500 lb/acre);[47] it is reasonable to assume that concentrations of chloropicrin in the air adjacent to such applications is even higher than those observed in Sisquoc.

- In 2008, some 8,304 lbs of chloropicrin where applied in the one square mile section that contains the Sisquoc community, placing it in the upper 75th percentile of California sections in which chloropicrin use was reported. While the community is on the high end of exposed communities, there are nonetheless communities within sections reporting substantially higher amounts of chloropicrin use. For example, in the strawberry growing areas around Ventura, there are residential areas where more than 20,000 lbs of chloropicrin were applied per square mile in 2008. We would expect ambient chloropicrin levels to be even higher in these areas.[48]

- The monitored application in Sisquoc appears to have been conducted in compliance with all rules and regulations. The high chloropicrin levels are not the result of a botched application or proper mitigation measures not being followed.

The data indicate overall that Sisquoc is not unusual among communities that are in areas where fumigants are used. It is not on the extreme high end of chloropicrin use for California, nor was there

* Air monitoring data provide exposure estimates and do not necessarily represent the precise exposure individuals may experience. Variables that affect an individual's exposure to airborne pesticides include the amount of time spent in areas with high concentrations of airborne pesticides, body weight and breathing rate.

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anything unusual about the application that we monitored there. Thus it is likely that hundreds of other communities across the country are experiencing chloropicrin exposures that are just as high or higher than those documented in this report. As discussed previously in this report, the U.S. EPA's newly mandated buffer zones would not have mitigated these concentrations to below levels of concern.

The California Department of Pesticide Regulation (DPR) declared chloropicrin to be a toxic air contaminant (TAC) in February 2010.[49] This conclusion is based on reasonable worst-case scenarios for bystander exposure. Chloropicrin concentrations following applications to fields, enclosed spaces, and homes were predicted using computer models derived from field studies. In general, the predicted concentrations exceeded DPR's levels of concern by several orders of magnitude.

While the concentrations observed in Sisquoc are much lower than those predicted by DPR, our results nonetheless buttress these conclusions. While DPR's conclusions are based on modeled reasonable worst-case scenarios, our data show that in a real-world, typical-case scenario, levels of concern are still exceeded, even with buffer zones.

Calculations

Air Concentrations

Pesticide concentrations in air were calculated from the analytical results obtained from the commercial lab as shown in equation (1):

$$\text{Air concentration, } \mu\text{g/m}^3 = \frac{\text{chloropicrin level in tube, } \mu\text{g}}{\text{volume of air sampled, m}^3} \qquad (1)$$

For convenience, all air concentrations reported here are expressed in units of $\mu g/m^3$. In some cases, concentrations from other studies that are quoted herein were converted from units of ppbv (parts per billion by volume, also abbreviated as ppb) according to equation (2):[15]

$$\text{Air concentration, } \mu\text{g/m}^3 = \text{air concentration, ppb} \times \frac{164.38\text{, g/mol}}{24.45\text{, L/mol}} \qquad (2)$$

Calculation of Reference Exposure Levels

In its most recent risk assessment of chloropicrin, U.S. EPA assessed inhalation exposure by the target "margin of exposure" (MOE) approach. In the first part of this approach, an appropriate toxicological endpoint is selected. Typically, the endpoint is a human equivalent concentration (HEC) or No Observed Adverse Effect Level (NOAEL) or from an animal study. This is the highest dose that did not cause observable adverse effects in the study. In the next stage, a target MOE is determined. An MOE is defined as the ratio of the NOAEL from the animal study to the human exposure dosage; a higher MOE corresponds to a greater margin between the anticipated human exposure and the level known to cause adverse effects in animals. An MOE of less than one for a scenario indicates that humans are being exposed at doses that exceed the safe dose in the test animal. A target MOE is the minimum MOE deemed acceptable for humans by the Agency. Usually the target MOE is set to at least 100. This assumes that humans are 10-fold more sensitive than the

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test animal and that there is 10-fold variability among humans (i.e. some people, e.g. infants, the elderly, or sick people, may be up to 10 times as sensitive as the average person). In setting the target MOE at 100, U.S. EPA is attempting to keep human levels of exposures to the chemical at least 100 times lower than the highest dose known to be safe in animals. In the last stage, MOEs are estimated for various human exposure scenarios. Those situations with MOEs less than the target MOE are usually considered to carry unacceptably high levels of risk and require mitigation.

To facilitate comparisons of the chloropicrin levels observed in this study with U.S. EPA's target MOE, we calculated reference exposure levels (RELs) according to equation (2). Breathing rate and body weight is not incorporated into this calculation because the short-term effects are port-of-entry effects.

$$\text{Reference Exposure Level, } \mu g/m^3 = \frac{\text{critical NOAEL, } \mu g/m^3}{UF_{intraspecies} \times UF_{interspecies} \times UF_{other}} \quad (3)$$

The REL represents the air concentration corresponding to an MOE equal to the target MOE. Air levels exceeding the REL have MOEs less than the target MOE, and represent situations with unacceptably high levels of risk. Likewise, air levels below the REL correspond to the MOEs greater than the target MOE and represent "acceptable" levels of exposure, according to the agency making the decision.

For the purpose of calculating RELs, we have used the critical toxicological endpoints and the target MOEs specified by U.S. EPA and CA DPR in their most recent chloropicrin risk assessments. As discussed in the **Discussion** section, we do not necessarily agree with EPA's choices—particularly the use of a target MOE of only 1 for acute exposure—but we have utilized their endpoints or target MOEs in our REL calculations for comparison purposed. Since the U.S. EPA expressed the critical toxicological endpoints as air concentrations adjusted for human physiology (so-called "Human Equivalent Concentrations" [HECs]), rather than as doses in units of mg/kg/day, it was not necessary to convert doses in mg/kg-day into air concentrations.

Estimation of Lifetime Cancer Risk

Lifetime cancer risk was calculated using the methods published by the California Office of Environmental Health Hazard Assessment (OEHHA).[50]
To estimate the risk of cancer from exposure to a substance over a 70-year lifetime, one must know the following:

- The **average concentration** of the substance in air during the monitoring period.
- The **exposure frequency**, or the fraction of a year in which concentrations are estimated to equal the average concentration measured during the monitoring period.
- The **average annual concentration** of the substance in air, determined from the exposure frequency and the average concentrations observed during the monitoring period.
- The **cancer potency factor, Q***, determined from toxicity studies. For chloropicrin, the DPR derived a cancer potency factor of 2.2 $(mg/kg\text{-}day)^{-1}$ calculated for the 95th percentile.[14]

Details for each calculation are shown below; see Table 6 for results.

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Estimation of Average Air Concentrations during the Application Period

The time-weighted average concentration of chloropicrin measured in this study was 1.44 μg/m^3 at Site 1 and 2.40 μg/m^3 at Site 2 for the period from April 4 to April 22.

Estimation of Exposure Frequency

The length of the application season (and hence exposure frequency) for chloropicrin in the Sisquoc vicinity of California is not precisely known. In these cancer risk calculations we have assumed that exposure to chloropicrin is limited to just the portion of the year in which we observed it: April 4–22 (19 days, 5.2% of the year). This assumption may underestimate the actual duration of exposure, and therefore cancer risk, since chloropicrin may be used on other fields in the area.

Estimation of Average Annual Air Concentration and Exposure

Average annual air concentrations were calculated by multiplying the average air concentration during the monitoring period by the exposure frequency, according to equation (4).

$$\text{Avg. annual conc. } (\mu\text{g/m}^3) = (\text{Avg. conc. during monitoring period}) \times (\text{Exposure frequency}) \quad (4)$$

Annual exposure was calculated by multiplying the average annual air concentration by the adult breathing rate of 0.28 m^3/kg-day, according to equation (5). This calculation assumes the annual average air concentrations remain at the same level from year to year.

$$\text{Annual exposure, mg/kg - day} = (\text{Avg. annual conc., } \mu\text{g/m}^3) \times (10^{-3} \text{ mg/}\mu\text{g}) \times (0.28 \text{ m}^3\text{/kg - day}) \quad (5)$$

Determination of Lifetime Cancer Risks

To obtain the lifetime (70-year) cancer risk, the average annual exposures in mg/kg-day were multiplied by the potency factor (Q*) in (mg/kg/day)$^{-1}$, according to equation (6).

$$\text{Lifetime cancer risk} = \left(\text{Annual exposure (mg/kg - day)}\right) \times \left(Q_1^* \text{ (mg/kg - day)}^{-1}\right) \quad (6)$$

The lifetime cancer risk is defined as the estimated number of cancer cases per million people. Lifetime cancer risks exceeding one in one million represent risks of concern, therefore for convenience the values given in Table 6 has been multiplied by 1×10^6.

Determination of Age-Adjusted Cancer Risks

OEHHA has devised a method for calculating cancer risks that accounts for differences in cancer susceptibility between life stages.[25] The life stages considered are postnatal (birth to 2 years), juvenile (2 to 16 years, prior to the reproductive years), and adult, from 16 years onward. The postnatal and juvenile life stages are considered to be early life stages. The methodology OEHHA uses to estimate age-adjusted cancer risks is based on rodent studies performed on a series of carcinogens using two experimental approaches: multi-life stage studies in which exposure occurs in at least two groups during different life stages, and single life stage exposure experiments. These experiments provided the basis for the development of age sensitivity factors (ASF), which account for both the inherent sensitivity of developing animals as well as the time available since exposure to develop cancer.

The cancer risk accrued in year$_i$ is calculated according to equation (7),

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$$\text{Risk accrued in year}_i = Q_1^* \times \text{ASF} \times \text{DOSE}_i \quad (7)$$

where Q_1^* is the cancer potency factor, ASF is the age sensitivity factor, and $DOSE_i$ is the annual exposure in year *i* calculated according to equation (9) with EF = AT = 1 year. The total cancer risk associated with an exposure scenario is the sum of the risks accrued each year for the duration of the exposure, as shown in equation (8):

$$\text{Cancer risk} = \sum_{i=y_1}^{y_1+ED-1} Q_1^* \times \text{ASF} \times \text{DOSE}_i \quad (8)$$

where y_1 is the year of age when exposure commenced, and ED is the exposure duration in years. For example, to calculate the cancer risk associated with 3 years of exposure beginning at age 6, one would calculate the yearly risk accrued for years 6, 7, and 8, using the appropriate ASF and BR for each year, and then sum these risk values to arrive at the total cancer risk associated with the 3-year exposure. Values for the ASF and BR for each life stage are given in Table 12, below.

Table 12: Age-Specific Factors and Breathing Rates

Life stage	Age range (years)	Duration (years)	Age Sensitivity Factor, ASF, 50th percentile value	TWA Breathing Rate, BR (m^3/kg-day)
Postnatal	0 to < 2	2	10	0.49
Juvenile	2 to < 16	14	3	0.38
Adult	16 through 70	54	1	0.24

To determine the cancer risk for a particular multiyear exposure window, the accrued risk values for the corresponding years are summed according equation (8). For example, the lifetime cancer risk associated with exposure to chloropicrin at Site 2 is 3.87×10^{-5} (postnatal) + 3.75×10^{-5} (juvenile) + 3.02×10^{-5} (adult)—91 excess cancers per million people.

Estimation of Less-Than Lifetime Cancer Risks

OEHHA has devised methodology for calculating cancer risks resulting for shorter than lifetime exposures,[50] which we apply here to two scenarios: exposure to the levels of chloropicrin observed in Sisquoc from birth to age 9 and from birth to age 30. These scenarios were chosen because 9 and 30 years are the figures OEHHA recommends for the "central tendency and high end estimates," respectively, of residency time.[51]

This methodology relies on the use of a cancer potency factor derived from chronic animal studies. Since short-term high-dose exposures are not necessarily equivalent to chronic low-dose exposures (even if they result in identical lifetime doses), this methodology increases the uncertainty associated with the calculated cancer risk. Therefore, OEHHA does not support the use of this methodology for risk calculations of less than 9 years. Furthermore, these calculations are breathing rate dependent, therefore the 9-year exposure scenario developed here applies specifically to period of birth to age 9.

The OEHHA methodology provides an estimate of dose based on annual exposure for less-than lifetime exposures according to equation (9):[50]

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$$\text{Dose} = \frac{C_{air} \times BR \times ED \times A \times EF \times 10^{-6}}{AT} \quad (9)$$

where:

DOSE = Annual daily exposure (mg/kg-day)

C_{air} = Average daily air concentration of contaminant (μg/m^3)

BR = Average daily breathing rate (L/kg-day)

A= Inhalation absorption factor

EF = Exposure frequency, days/year

ED = Exposure duration, in years

10^{-6} = Conversion factor for μg/m^3 to mg/L

AT = Averaging time

In this calculation, C_{air} is the time the time-weighted average concentration of chloropicrin measured in this study: 1.44 μg/m^3 for Site 1 and 2.40 μg/m^3 at Site 2, and exposure frequency, EF, is 19 days/year, or 5.2%. The BR used in the calculation of age-adjusted and less-than-lifetime cancer risk calculations is the TWA breathing rate, calculated using the Exposure Factors Handbook.[26] The inhalation absorption factor, A, is equal to one based on the assumption that the human lung absorbs chloropicrin from the air as efficiently as the rat lung. Finally, the exposure duration, ED, is the length of time for the specific exposure scenario and AT is the averaging time or the period over which exposure is averaged, in years. For carcinogenic effects, the averaging time is 70 years.

Cancer risk is then calculated by multiplying the calculated annual daily dose by the cancer potency factor, Q_1*. This is analogous to the calculation of lifetime cancer risk with Equation (6). See Table 8 for the results of the calculation for 9- and 30-year exposure periods. For convenience, the cancer risk values have been multiplied by 1×10^6 to show risk per million people.

Quality Assurance–Quality Control

Operator Training

All Drift Catcher Operators participated in a hands-on training workshop on the operation of the Drift Catcher at which time they were provided with a Drift Catcher Users' Manual. They were then tested on their knowledge of the procedures and practices by a PANNA scientist. Participants were certified if they could successfully demonstrate:

(1) Mastery of the technical set-up and operation of the Drift Catcher
(2) Correct use of Sample Log Sheets and Chain of Custody Forms
(3) Ability to troubleshoot and solve common operational problems
(4) Knowledge of the scientific method

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Sample Labels

Sample labels were affixed directly to the sorbent tubes and to the corresponding sample log sheets prior to the start of sampling. The following information was contained on the labels: Sample ID, project name, and project date.

Sample Check-In

On arrival at PANNA's office, samples were checked into a Sample Log Database organized by project and sampling dates. Sampling dates and times, extraction dates, analysis dates, analytical methods and sample results were all logged in the database. Appendix 6 shows a screen shot of the main Sample Log Database page.

Leak Check

All monitoring equipment was fully leak-checked prior to use by attaching the tubing-manifold combination to a pump generating a positive airflow and testing for leaks at each connection point with a soap solution.

Flow Calibration

Rotameters used in the field to determine flow rates were calibrated using a Bios Defender Dry-Cal primary standard flow meter(medium and low range), factory calibrated by the manufacturer to ± 1% against a high precision Dry-Cal standard on 5/4/07 and 10/14/06 respectively. All rotameters used in this experiment deviated less than 5% (the rated accuracy for these rotameters) from the mass flow meter readings.

Trip Blanks

Three pairs of trip blank tubes were prepared over the course or the sampling period. These tubes were stored and transported with the samples from that location, and one from each pair was processed and analyzed as part of the batch on arrival in the lab. No pesticide residues were detected in the trip blanks. These are shown is Table 2 (samples "Badger," "Petal," and "Banana.")

Spiked Samples

Two spiked samples were prepared by PANNA and sent to the commercial lab for analysis along with the field samples and trip blanks. The results are shown in Table 13, below. The lab was unaware that these samples were spikes rather than field samples. The average recovery was 88%, and the results of the field sample analysis were not corrected for this recovery.

Table 13: Spike Results

Sample ID	Fortification (µg)	Recovery (µg)	Recovery (%)
Manta	3.00	2.39	79.7
Lunes	1.00	0.97	97.0

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Appendix 1: Meteorological Data

The CIMIS weather station data[10] indicate that during the monitoring, winds in Sisquoc generally blew strongly (7–12 mph) from the west in the afternoon/early evening (noon–8 pm) and gently (1–3 mph) from the east late at night and early in the morning (11 pm-8 am). Figures A-1 through A-4 show the hourly average wind speed and direction for April 2-8, April 9-15, April 16-22, and April 23–30, 2008. In Figure A-3 we note the prolonged period of winds from the west on April 18–20 coinciding with the last few days of the application period. The Drift Catcher data are consistent with this observation, with chloropicrin not detected at the sampling sites west of the field on the day immediately following the last application day. On the previous day with easterly winds, the concentrations of chloropicrin were significantly higher. Thus, on the evenings of April 18 and 19, the westerly winds (Figure A-5) considerably reduced the concentration of chloropicrin in the air near the residences (See map on p. 9). This occurrence of prolonged wind from the west for two days toward the end of the chloropicrin application likely protected the community from additional exposure.

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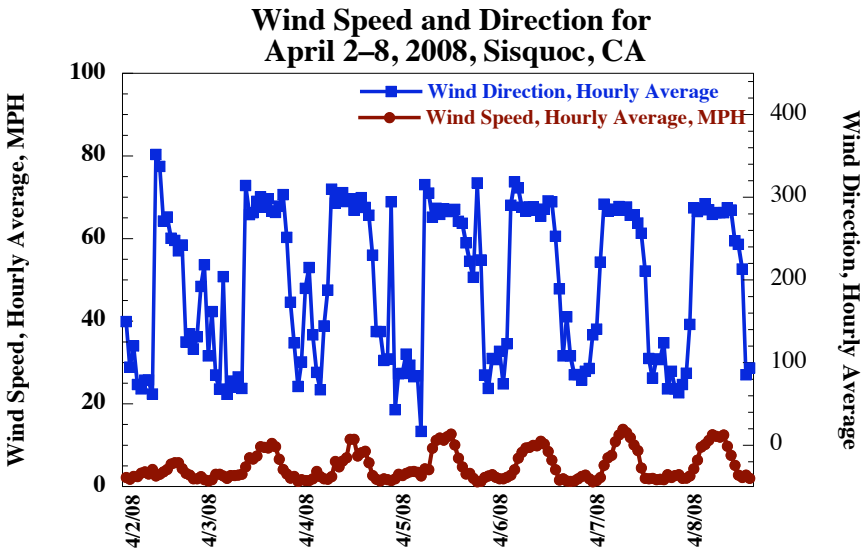

Figure A-1: Wind speed and direction for April 2–8, 2008

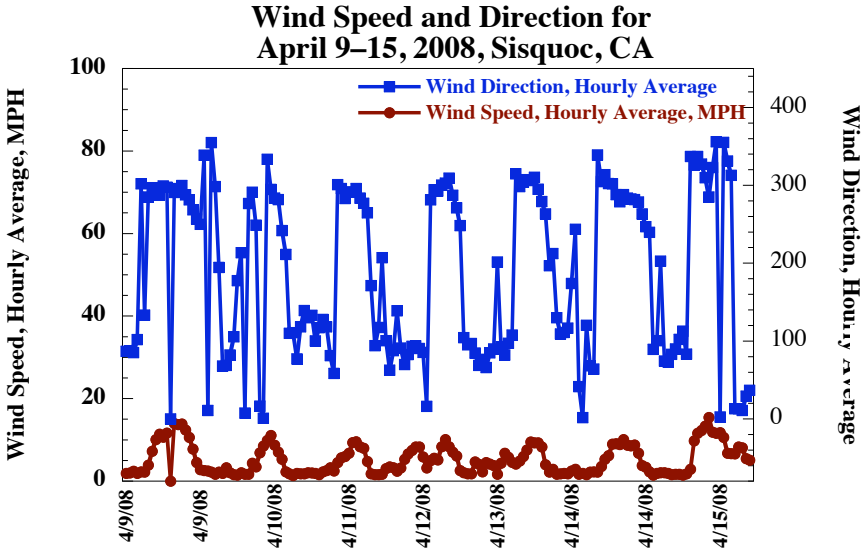

Figure A-2: Wind speed and direction for April 9–15, 2008.

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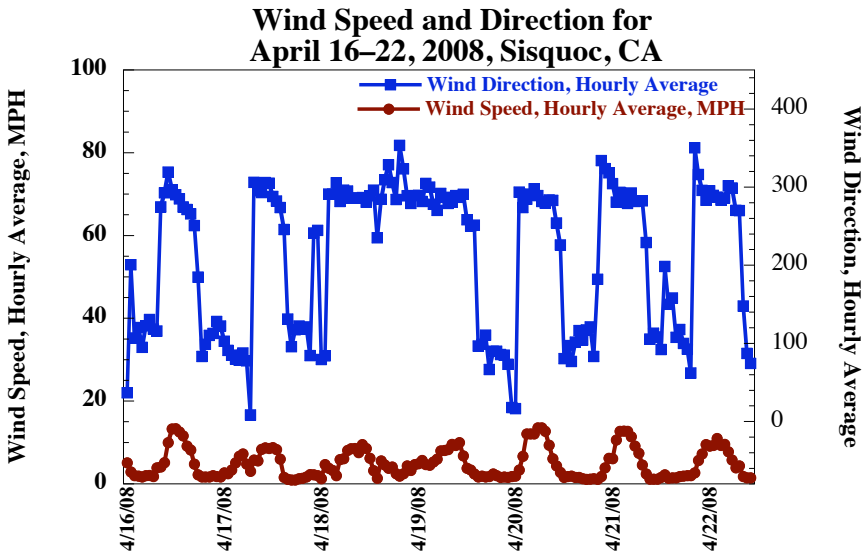

Figure A-3: Wind speed and direction for April 16–22, 2008.

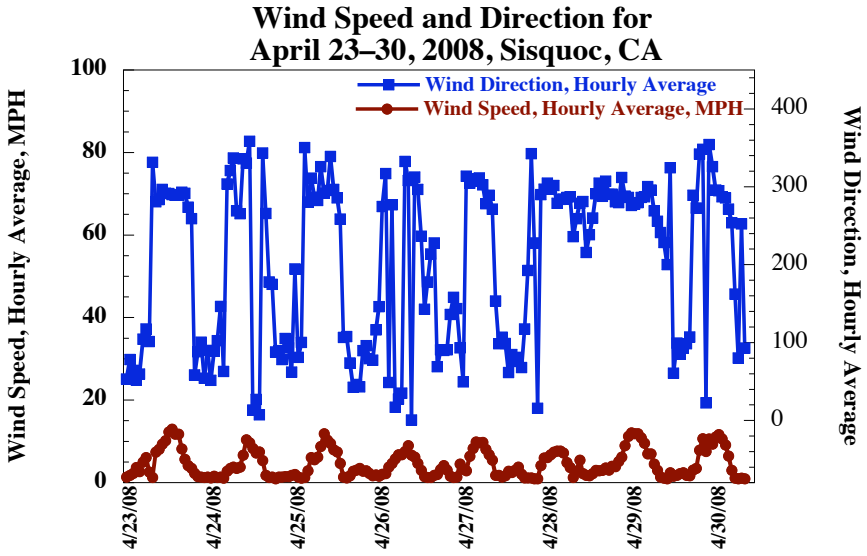

Figure A-4: Wind speed and direction for April 23–30, 2008

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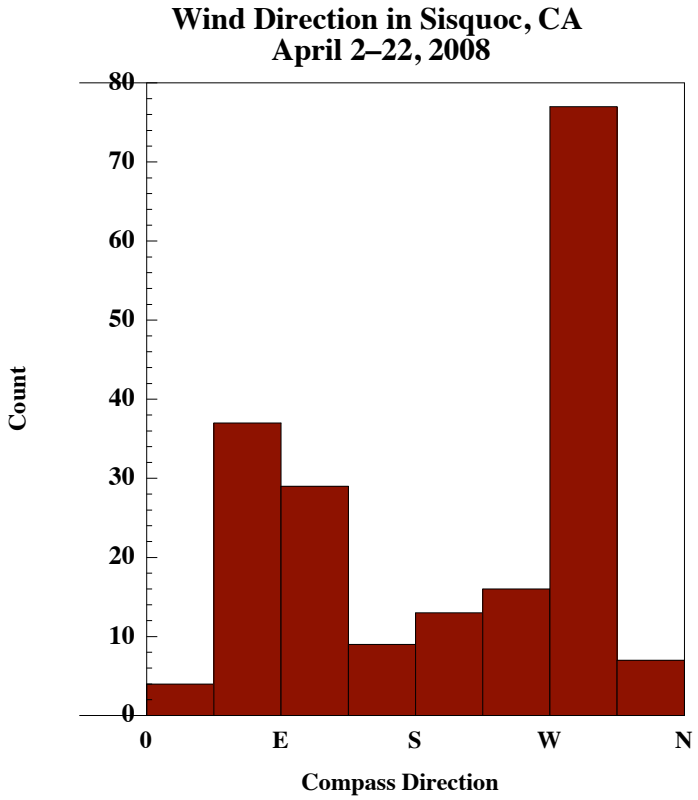

Figure A-5: Plot of compass direction versus the frequency of occurrence of wind from each direction.

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Appendix 2: Interpreting Air Monitoring Results

Interpreting air monitoring results requires understanding of how regulatory authorities like the U.S. EPA assess the toxicity of pesticides. In this section we answer the following questions.

How Are "Safe" Levels of Pesticides in Air Determined?
Are RELs and RfCs Air Quality Standards?
Are Levels Below the Level of Concern "Safe"?
What Do Air Monitoring Results Tell Us About Exposure?

How Are "Safe" Levels of Pesticides in Air Determined?

It is generally assumed that humans can be exposed to tiny amounts of most chemicals without suffering ill effects. As doses increases, usually both the severity and incidence of adverse effects increase, hence the adage: "the dose makes the poison." (In recent years, this assumption has been challenged for a class of toxicants known as endocrine disruptors;[52] nonetheless, this idea forms the basis of modern risk assessment.) Thus, rather than trying to prevent any and all exposures to chemicals of concern, regulators instead try to limit exposure to levels that are so small that the risk of harm is negligible.

Risk assessors use a variety of closely related techniques to quantify the risk posed by exposure to chemicals. These techniques go by various names but almost always involve identifying the largest dose that does not cause observable harm to animals in controlled experiments (the "No Observed Adverse Effects Level," or NOAEL), and then extrapolating from this dose to an acceptable dose in humans that is anticipated to be without harm. This extrapolation often takes into account physiological differences between the test animal and humans such as body weight, breathing rate, absorption, and metabolism.

The NOAEL usually comes from an experiment that uses only a few dozen animals (usually rats, mice, or rabbits) that are nearly genetically identical. Therefore, the extrapolation also includes factors to account for the inherent uncertainty that arises when extrapolating to a human dose that is supposed to be without risk for all members of an exceedingly large and diverse population. An interspecies factor of 10 is generally used to account for the fact that laboratory animals and humans are different and an intraspecies factor of 10 is used to account for variability among different people. The acceptable human dose calculated with these uncertainty factors is thus often several orders of magnitude smaller than the animal NOAEL that it is based on.

In assessing the risk of dietary exposure to pesticides, U.S. EPA uses oral dosing studies to establish a "Reference Dose" (RfD) following the procedure described above. The Agency defines a RfD as:

> an estimate, with uncertainty spanning perhaps an order of magnitude, of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects of a lifetime.[53]

An RfD should not, therefore, be considered a threshold level above which adverse effects are guaranteed or even expected. Rather, it should be understood as a level of concern, above which the risk of adverse effects is unacceptably high (although perhaps still quite small in absolute terms), and

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below which the risk is acceptably small. The agency uses RfDs to determine worker protection rules, mitigations for exposures the general public might experience, and acceptable limits for the maximum amount of pesticide residue permissible in food items. With these regulations, the Agency tries to limit human exposure to an amount less than the RfD.

For a constant dose, the incidence and severity of adverse effects generally increase as the duration of exposure increases. In other words, a dose that does not cause acute toxicity after a single exposure may cause chronic toxicity if exposure occurs repeatedly. For this reason, different RfDs are often calculated for acute and chronic exposure, and for 1-hour and 24-hour exposure, etc.

Reference doses are defined specifically for dietary exposure, but similar levels of concern can be derived for inhalation exposure using analogous methods: usually starting with a NOAEL from an animal study and then applying uncertainty factors to extrapolate to an acceptable human dose. The conversion from an acceptable dose (in units of mg of chemical per kg bodyweight per day) to a level of concern (in units of mg or ng of chemical per a certain volume of air) is complicated by variations in breathing rates among human beings. For example, infants and children have proportionately higher breathing rates than adults, so if an infant and an adult are exposed to the same airborne concentration of a toxicant for the same period of time, the infant will receive a larger dose (measured in mg of pesticide per kg of body weight) than the adult. Similarly, breathing rates vary with physical activity, so, for example, a person exercising in contaminated air would receive a greater dose than a person napping in the same environment for the same length of time. Since the resulting levels of concern are air concentrations rather than doses these are called Reference *Concentrations* or Reference *Exposure Levels,* rather reference *doses*.

In this air monitoring study, we compare concentrations of pesticides measured in air for acute and short term RfCs and RELs calculated by DPR and OEHHA. We also derive a REL from U.S. EPA data as described in the **Calculations** section of this report.

Are RELs and RfCs Air Quality Standards?

No. A REL or RfC is not an enforceable standard like a water quality standard or a worker protection standard. They are analogous to a RfD, a dose that the U.S. EPA uses in its dietary assessments as a Level of Concern (LOC). To minimize exposure risk, U.S. EPA typically takes action to reduce dietary exposures of the 99.9th percentile person to below the LOC. This means that if even one-tenth of one percent of the people were exposed to a pesticide in their diet at this level, U.S. EPA would take action to reduce risk. Unfortunately, there are regulatory gaps for inhalation exposure—U.S. EPA does not currently assess bystander inhalation exposures for most pesticides but rather assumes that inhalation is not a significant contributor to total exposure.

Are Levels Below the Level of Concern "Safe"?

Concentrations below the REL do not necessarily indicate that the air is "safe" to breathe. In particular, a number of recent studies evaluating people's capacity to metabolize toxic substances show that the variability among different people can be substantially greater than the variability assumed by U.S. EPA in its toxicological analysis.[54] Additionally, as in this study, people are often exposed to multiple pesticides simultaneously, or are taking prescription or non-prescription drugs, or are exposed to other chemicals, thus reducing their capacity to detoxify the pesticides to which they are exposed.

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What Do Air Monitoring Results Tell Us About Exposure?

Air monitoring data provide exposure estimates that may or may not represent worst-case exposure scenarios, and do not represent the precise exposure individuals may experience. Variables that affect an individual's exposure to airborne pesticides include the amount of time spent in areas with high concentrations of airborne pesticides, body weight and breathing rate.

The breathing rates used to derive the levels of concern in this study (see the **Calculations** section) represent the breathing rates of individuals *averaged over the course of 24 hours*. An individual's breathing rate will vary substantially over the course of 24 hours. For example, the typical breathing rate of a 10-year old child during resting activity (e.g. sleeping, reading or watching television) is 0.4 m^3/hr, while during moderate activity (e.g. climbing stairs) it is 2.0 m^3/hr, and during heavy activity (e.g. playing sports) it is almost ten times greater at 3.9 m^3/hr.[26] The breathing rate of a child at play during recess or exercising during a gym class is best approximated by the moderate or heavy activity breathing rate. Thus, children are outside and maximally exposed to air contaminants precisely when their breathing rates are expected to be the highest. The RELs used in this report are calculated using lower than moderate breathing rates—the daily averages—and assuming 24-hour exposure.

For most pesticides, only a limited number of monitoring studies are available for comparison, and most of the available studies only provide results for applications conducted according to label instructions and for exposure estimates to a single pesticide. The Drift Catcher project is providing additional monitoring data for comparison, and as we gather more data, a clearer picture of pesticide levels in the air near homes, schools, parks and workplaces will emerge.

Notwithstanding that available monitoring data are not comprehensive, the data indicate that many people are routinely exposed to levels of airborne pesticides that exceed both acute and sub-chronic levels of concern.

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Appendix 3: Chloropicrin

Chloropicrin Use and History

Chloropicrin was first used as an insecticide in 1917 and as a soil fumigant in 1920. It was registered in the U.S. in 1975.[18] It is used as a general biocide, for control of bacteria, fungi, nematodes, insects, and weeds. As a fumigant pesticide, application of chloropicrin can sterilize the soil prior to planting of multiple agricultural crops including tobacco, potatoes, strawberries, and peppers. Other applications for this compound include treatment of tree replant sites, empty grain bins, nurseries, and as a warning agent in structural fumigations.

Chloropicrin is labeled as toxicity category I, Danger and is a Restricted Use Pesticide (RUP). It was reregistered by U.S. EPA in 2009.[31] Chloropicrin was listed by DPR as a Toxic Air Contaminant in February 2010.[49] It is not registered for use in Europe[55] and Canada.

Chloropicrin is a broad-spectrum fumigant that is usually used in combination with other fumigants, such as methyl bromide and 1,3-dichloropropene, for both increased potency and as a warning agent.[18] Chloropicrin is used as a warning agent because it has a low odor threshold and causes sensory irritation at low concentrations, unlike the fumigants (methyl bromide and sulfuryl fluoride, for example) with which it is often combined. With the 2005 phase-out of methyl bromide (with the exception of Critical Use Exemptions) mandated by the Montreal Protocol,[56] more chloropicrin is being used in fumigations, with products now containing concentrations of chloropicrin ranging from 2% to 99%. Figure A-6 shows the increasing use of chloropicrin over the last five years, as methyl bromide production and use have been curtailed by the Montreal Protocol.

Chloropicrin is used in large volumes in California on strawberries, as a soil pre-plant fumigant for unspecified crops and at outdoor nurseries.[57] The counties with the highest use are Monterey, Ventura, Santa Barbara and Santa Cruz counties. Figure A-7 below shows the typical use pattern for chloropicrin in Santa Barbara County, with most fumigations occurring during the months of September and October.

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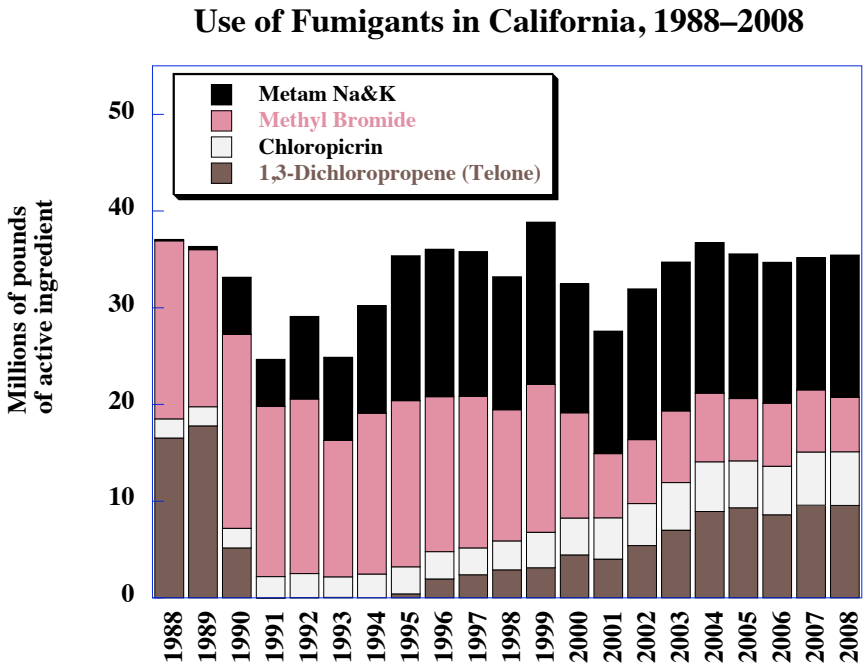

Figure A-6: Use of fumigants over time has remained relatively constant over the last twenty years in California, but the mix of different fumigants has changed substantially over the period.

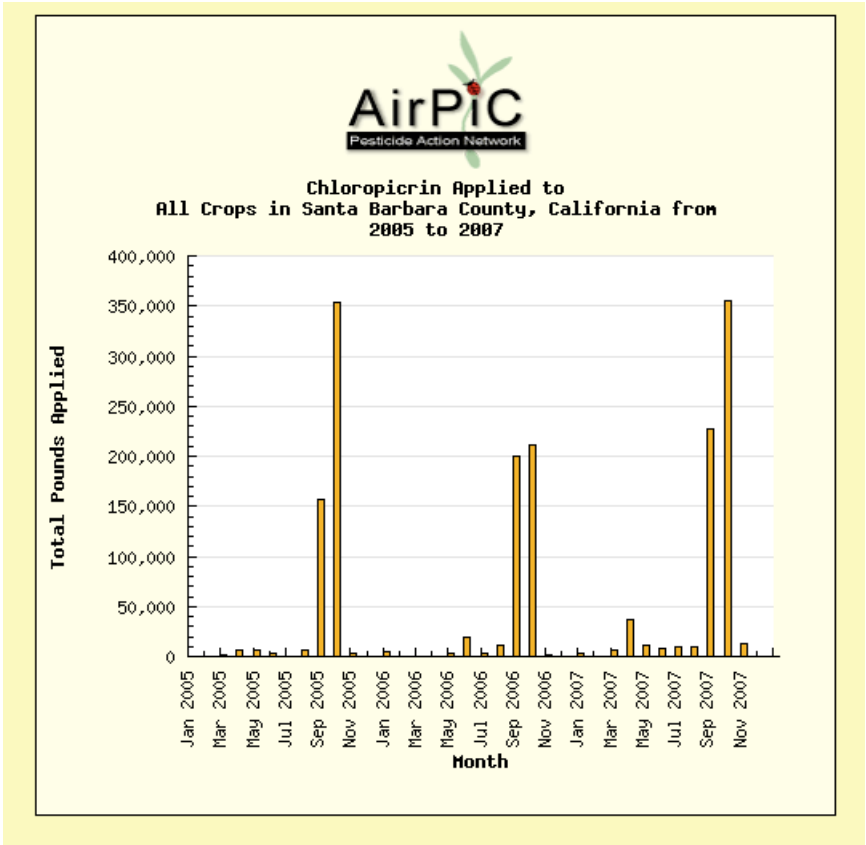

Figure A-7: Pounds of chloropicrin applied in Santa Barbara County from 2005 to 2007.

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In 2004, US EPA indicated that 5–9 million pounds of chloropicrin were used per year, making it the 18th most commonly used pesticide.[58] Use patterns are changing quickly however, and in 2007, 5.5 million pounds of chloropicrin were used in California alone. No recent data are available for nationwide use. A partial list of manufacturers includes Niklor Chemical Company, Ashta Chemicals, Angus Chemical Co., Trinity Manufacturing, Great Lakes Chemical Corporation, Soil Chemical Corporation Products, TRICAL, and Dow Agrosciences LLC. For the 99% pure agent, chloropicrin is sold under a product label of Metapicrin® or Chlor-O-Pic®.

Physical Properties of Chloropicrin

Chloropicrin (trichloronitromethane) is a colorless oily liquid at room temperature with a strongly irritating sharp odor. With a vapor pressure of 24 mm Hg at 25 °C, chloropicrin is highly volatile and can readily drift from areas where it has been applied. The chemical structure of chloropicrin is shown below, and the physical properties of chloropicrin are summarized in Table A-1 below.

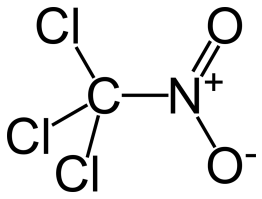

Table A-1: Physical Properties of Chloropicrin

Property or Identifier	Chloropicrin
CAS Number	76-06-2
Chemical Formula	CCl_3NO_2
Molecular Weight (g/mol)	164.4
Melting Point (°C)	-64
Water Solubility (mg/L)	2,000 @ 25°C
Vapor Pressure (mm Hg)	23.2 @ 25 °C
Henry's Law Constant (atm-m^3/mol)	2.51 x 10^{-3} @ 25 °C
Avg. Hydrolysis Half-life	31.1 hours
Avg. Aerobic Soil Half-life	0.374-5.13 days
Avg. Anaerobic Soil Half-life	1.3 hours

Data source: Reference 16.

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Appendix 4: Sample Log Sheet

Drift Catcher Sample Log Sheet

STARTING THE SAMPLE

Project: _____ Location: _____

YOU NEED: A Drift Catcher, a sample bag with pre-labeled tubes, caps, and labels, a tube cracker, a rotameter, two light shields, orange flag material, a compass, and a wind meter.

❒ **1. LABELS:** Make sure the labels included in the sample bag MATCH the labels on the pre-labeled tubes. If they match, affix the labels to this log sheet under Steps 4 & 11.

❒ **2. TUBES:** Break the tips of the glass sample tubes and insert them into the manifold.

❒ **3. PUMP:** Plug in the pump and note the EXACT TIME using the clock on the compass.

Today's Date		Exact Pump START Time	**AM or PM?**

❒ **4. ROTAMETER:** Use the rotameter to measure the flow rate for each tube.

	Tube Name	Starting Flow Rate	
Tube A	[stick label here]	**L/min**	**NOTE: Adjust the flow rates so that they are equal to each other!**
Tube B	[stick label here]	**L/min**	

❒ **5. LIGHT SHIELDS:** Attach both light shields.

❒ **6. COMPASS & ORANGE FLAG:** Use these to find the direction of the wind.

Which direction is the wind blowing FROM?	N NE E SE S SW W NW calm

❒ **7. WIND METER:** Face the wind meter into the wind for 2 minutes.

What is the wind speed?	maximum: mph	average: mph
What is the temperature? **(Remember to wave wind meter back and forth!)**		° F

❒ **8. YOUR SENSES:** Use your own senses to answer the following questions.

What is the weather like?	foggy sunny mix of sun and clouds cloudy rainy humid other:
Do you smell anything?	sweet rotten eggs perfume skunk none other:

There is space for other observations and notes at the bottom of the other side of this page.

Name: _____ Initials: _____

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STOPPING THE SAMPLE (cont'd from other side)

❒ **9. PUMP:** Is the pump running? ❒ Yes ❒ No (If not, skip to Step #13)

❒ **10. LIGHT SHIELDS:** Remove both light shields.

❒ **11. ROTAMETER:** Use the rotameter to measure the flow rate for each tube.

	Tube Name	Ending Flow Rate	
Tube A	[stick label here]	L/min	**DO NOT adjust the flow rates. Just measure them.**
Tube B	[stick label here]	L/min	

❒ **12. PUMP:** Unplug the pump and note the EXACT TIME, using the clock on the compass.

Today's Date		Exact Pump STOP Time	**AM or PM?**

❒ **13. TUBES:** Remove the sample tubes, cap them, place them in the sample bag.

❒ **14. COMPASS & ORANGE FLAG:** Use these to find the direction of the wind.

Which direction is the wind blowing FROM?	N NE E SE S SW W NW calm

❒ **15. WIND METER:** Face the wind meter into the wind for 2 minutes.

What is the wind speed?	maximum: mph	average: mph
What is the temperature? **(Remember to wave wind meter back and forth!)**		°F

❒ **16. YOUR SENSES:** Use your own senses to answer the following questions.

What is the weather like?	sunny mix of sun and clouds cloudy rainy humid other:
Do you smell anything?	sweet rotten eggs perfume skunk none other:

❒ **17. TRIP BLANK:** If this is the first sample of your sample run in this location, prepare a Trip Blank sample (follow instructions on Trip Blank form).

Name: _____ Initials: _____

OBSERVATIONS AND NOTES

Please record observations or notes below (known pesticide applications nearby, equipment failure, nearby activities that could interfere with the sample, etc.)

Date	Time	Observation/Note

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Appendix 5: Freezer Log and Chain of Custody Form

Chain of Custody Form and Freezer Log

This form is used to keep track of where all your samples are and who has been responsible for them at all times.

Name: _____ Phone Number: _____

Project Name: _____

Sample Site (Include full address): _____

_____

Date Sampling Started: _____ Date Sampling Finished: _____

Freezer Log

Sample Name	Sample Placed in Freezer on Date	Time (am/pm)	Notes or Comments	Initials
Ejemplo – A	6/8/05	8:18 pm	This is an example entry.	JD

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Chain of Custody Form

This section tracks who has control of the batch of samples as they are being transported and how they are handled.

When you receive the samples,
- Make sure all samples are accounted for.
- Record the time and date and put your initials in the **Received by** column.
- If you are unpacking samples from a shipping box, note the temperature of the ice packs.

When samples are passed from one person to another, you should record the method of storage (freezer, cooler, dry ice, etc). If you change the method of storage (i.e. from a freezer to a cooler) please also record this along with the date and time of change, even though the samples are still in your custody.

Date Sent	Time Sent	Sent by (Initials)	Storage Before Transfer	Storage During Transfer	Storage After Transfer	Date Received	Time Received	Received by (Initials)	Temperature upon arrival (Circle one)*
6/9/05	2:43 pm	JD	Freezer	Cooler	Freezer	6/10/05	9:08 am	SK	(1) - 2 - 3 - 4
									1 - 2 - 3 - 4
									1 - 2 - 3 - 4
									1 - 2 - 3 - 4
									1 - 2 - 3 - 4

*note the shipping container temperature by choosing the ice pack description that best describes the condition of the ice packs.
1: Fully frozen; 2: Partially frozen; 3: Not frozen but still cold; 4: Room Temperature

Names and signatures of sample handlers:

Each person who handles the samples will need to sign off on this form. Your signature and initials are your verification that the samples were handled as indicated on the form.

	Name (Please print)	Phone Number	Signature	Initials
Example	Juan Diego	(234) 567-8901	Juan Diego	JD
1				
2				
3				
4				

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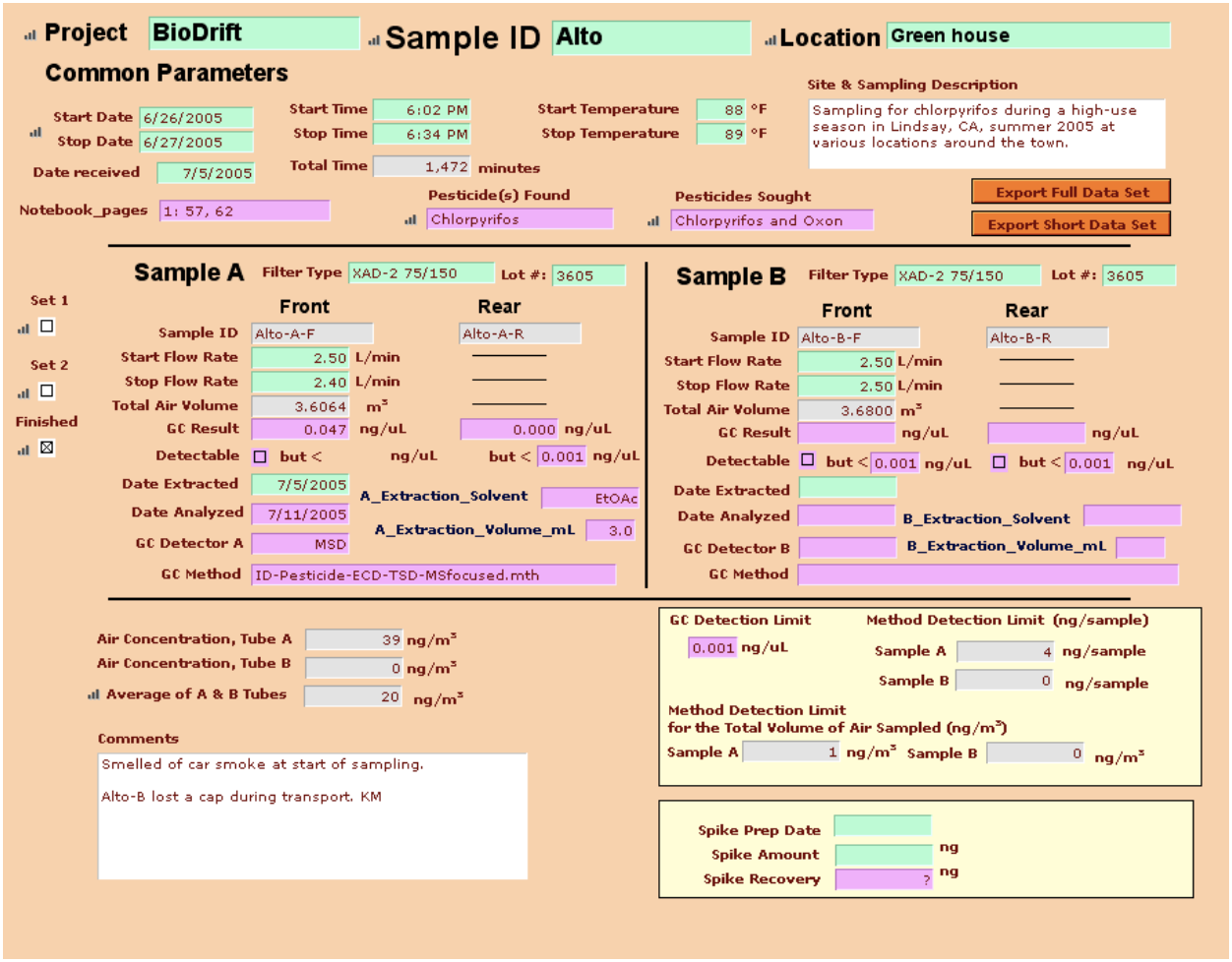
Appendix 6: Sample Log Database Screen Shot



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