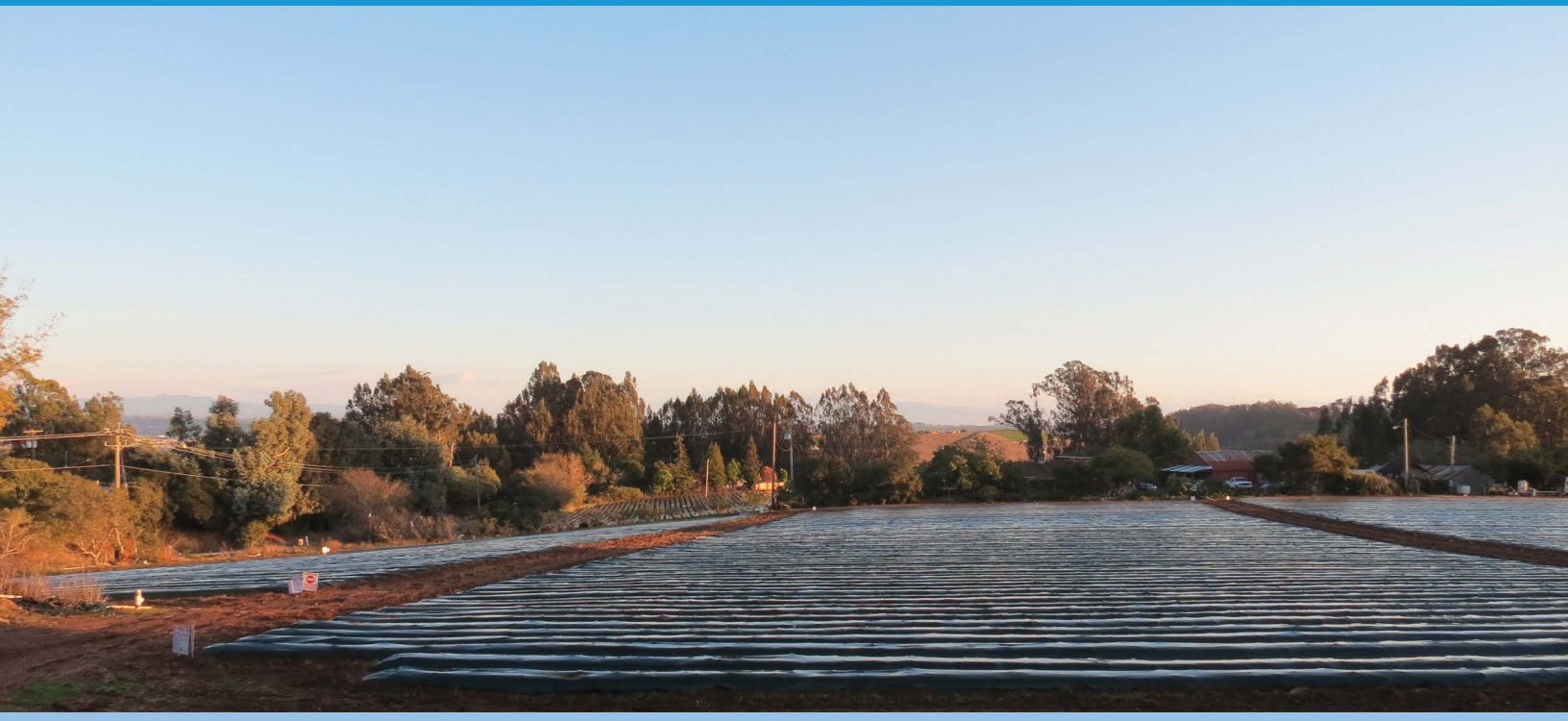
Air Monitoring in Watsonville, California

NOVEMBER 3–12, 2014



TECHNICAL REPORT

CALIFORNIANS FOR PESTICIDE REFORM
PESTICIDE ACTION NETWORK NORTH AMERICA

2015

Pesticide Action Network North America
Californians for Pesticide Reform
April 2015

Californians for Pesticide Reform (CPR) is a coalition of over 170 public interest organizations committed to protecting public health and the environment from pesticide proliferation. CPR's mission is to:

- Educate Californians about environmental health risks posed by pesticides;
- Eliminate the use of the most dangerous pesticides in California and reduce overall pesticide use;
- Promote sustainable pest control solutions for our farms, communities, forests, homes and yards; and
- Hold government agencies accountable for protecting public health and Californians right to know about pesticide use and exposure.

Pesticide Action Network North America (PAN) works to replace hazardous pesticide use with ecologically sound and socially just alternatives. For over 30 years, our international network of over 600 citizens groups in more than 90 countries has created a global pesticide reform movement with regional coordinating centers in Africa, Asia, Europe, Latin America and North America. PAN North America links the collective strengths and expertise of groups in Canada, Mexico and the U.S. with their counterparts in other countries to further sustainable agriculture, environmental protection, workers' rights, improved food security, and guaranteed human rights for all.

PAN also maintains the world's most comprehensive set of databases on pesticides available on the web. For information on pesticide toxicity, registration status, poisoning symptoms and many other factors, see www.pesticideinfo.org. For information about pesticide residues on food, see www.whatsonmyfood.org.

Support for air monitoring in Watsonville was generously provided by the Rose Foundation and by Jacobs Farm Del Cabo.

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List of Abbreviations

ARB	Air Resources Board, the California agency in charge of regulating air pollution.
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.
LOAEL	Lowest Observed Adverse Effect Level, the lowest exposure level at which there are biologically significant increases in frequency or severity of adverse effects between the treatment group and the control group from exposure to that chemical alone.
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. An 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.

Air Monitoring in Watsonville, California, November 3-12, 2014

Emily Marquez and Susan Kegley
Pesticide Action Network North America, Oakland, CA

April 2015

Executive Summary

This report presents the results of an air monitoring experiment in Watsonville, located in Santa Cruz County, California. Between November 3 and November 12, 2014, a total of 18 12-hr samples were collected at a residence in Watsonville adjacent to two chloropicrin applications on two fields that occurred on November 3 and November 5.

Of the samples collected, 61% contained chloropicrin above the limit of quantitation (LOQ) of 0.03 μg of chloropicrin per sample. The LOQ is equivalent to an air concentration of 0.21 μg/m^3 for a 24-hour sample and 0.42 μg/m^3 for a 12-hour sample. In six samples, "breakthrough" occurred from the front to the rear bed of the sample tube, meaning that chloropicrin was detected in the second "back" bed of resin in the sample tube. Breakthrough indicates that the resin in the sample tubes did not trap all of the chloropicrin that passed through the tubes, thus the results indicating concentrations of chloropicrin in the air are potentially lower than actual concentrations.

The highest concentration of chloropicrin observed for a 12-hour period was 7.9 μg/m^3 (1.2 ppb) on November 6. The time-weighted-average (TWA) concentration for the nine days sampled was 1.3 μg/m^3 (0.20 ppb). The TWA concentration calculated for the time period only while the fumigation was taking place (November 3-6) was 2.4 μg/m^3 (0.36 ppb). The top three highest 1-day TWAs were 2.3, 2.2, and 4.0 μg/m^3, all samples taken between November 3 and November 6. Results from the air monitoring are summarized in Table 2 and Figures 4 and 5.

The time-weighted-average chloropicrin concentration of 1.3 μg/m^3 does not exceed EPA's short- and intermediate-term level of concern of 1.8 μg/m^3. However, even at a distance of 350 feet from the nearest edge of the fumigated field, the TWA calculated for only the period of fumigation (November 3 to November 6) exceeds both EPA's short- and intermediate-term level of concern of 1.8 μg/m^3 and is equal to DPR's level of concern of 2.4 μg/m^3 for exposure during a season of application,[1] indicating an increased risk of adverse effects for people who spend a significant time in the vicinity of that site while fumigations are taking place. The 12-hour peak concentration of 7.9 μg/m^3 is higher than the value of 6.2 μg/m^3, determined as a level of concern for children by DPR scientists, the Scientific Review Panel, and OEHHA. DPR management did not develop a regulatory target level for 24-hour exposure.

Cancer risks calculated based on the nine days of air monitoring in this study also exceeded levels of

[1] Seasonal exposure is based on two-week TWA air concentrations.[19]

[2] 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

concern. The age-adjusted excess cancer risks were higher than the standard acceptable level of one additional cancer per million people by a factor ranging from nine (for a child exposed from birth to two years) to 39 (for an adult exposed for a lifetime 70-year residency).

Neither current nor proposed buffer zones would not have protected residents at this site from increased cancer risk, nor would the buffer zones have protected residents during the fumigation period from exposures that exceed non-cancer levels of concern. The distance of the monitoring site from the nearest field-edge of application was 350 feet, far greater than any buffer zones required by regulators. Based on the findings from this study, both current and proposed buffer zones for chloropicrin are thus inadequate for protection of human health.

The data indicate that air concentrations of chloropicrin following a tarped drip-line chloropicrin application (using a low-permeability film called "totally impermeable film," or TIF) exceeded levels of concern for both short and intermediate-term exposure and cancer risk. Most strawberry fields are fumigated annually and if similar concentrations to those observed in 2014 occur every season, people living in the area would suffer acute health effects such as respiratory distress and an unacceptably high risk of cancer as a result. In the square mile where this application was located, historical use data show 400-3,000 pounds of chloropicrin applied every year since 2010, often co-applied with 1,3-dichloropropene (Telone), also a carcinogen. Estimated exposure scenarios spanning a lifetime, 30 years, or various periods of childhood all resulted in cancer risks exceeding EPA's level of concern of one excess cancer per million people.

Further, EPA has removed all uncertainty factors for chloropicrin in its 2009 Reregistration Eligibility Decision,[46] resulting in a target margin of exposure of one. These factors take into account susceptibility of vulnerable populations, such as children. We have serious doubts that a target margin of exposure of one is sufficiently protective of all populations (see Discussion).

Comparison of the chloropicrin concentrations measured at the Watsonville site with concentrations measured by the California Air Resources Board (ARB) near fumigation sites indicate that the levels observed in this study are relatively low compared to ARB's worst-case scenarios.

Chloropicrin is used as a soil fumigant prior to planting crops. In California, over nine million pounds of chloropicrin were reported used in 2012, the latest year for which data are available. Use has increased steadily over the last several years; in 1998, only 3.0 million pounds of chloropicrin were used in the state and in 2007 and 2011, 5.5 million pounds and 7.2 million pounds were used, respectively (see Figure 1). In California, chloropicrin is used primarily as a soil pre-plant application in strawberry fields and nurseries.

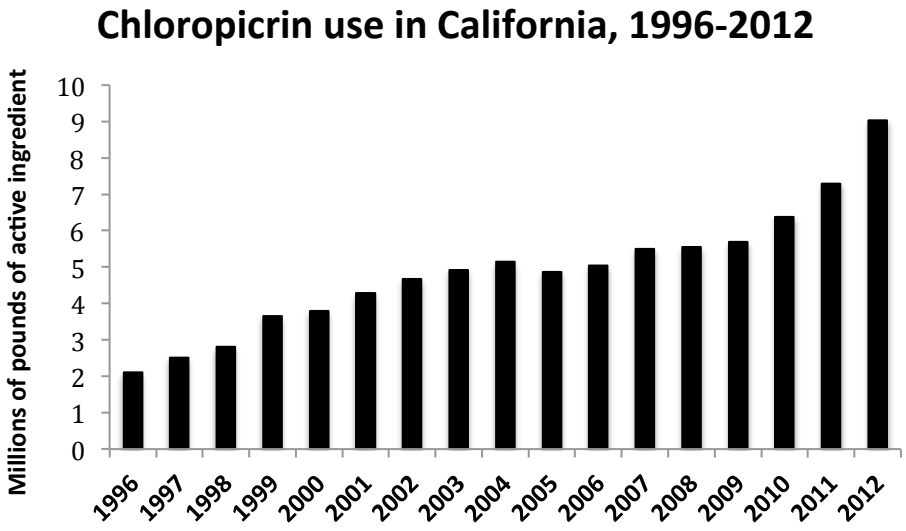


Figure 1. Chloropicrin use in agricultural applications has increased in California over the past decade. Use trends shown here include both agricultural and reportable non-agricultural applications. Production agriculture constitutes the major use category subject to reporting in California.

Between 1999 and 2012, chloropicrin has been the cause of over 1,000 reported poisonings in California.[61, 62] Two of the largest incidents occurred in Kern County (2003) and Monterey County (2005). The details of these and other chloropicrin-related poisonings in California are summarized in Table 11. Chloropicrin is highly irritating in low concentrations and highly acutely toxic at higher concentrations. Symptoms of acute poisoning include eye and respiratory irritation, difficulty breathing, nausea and vomiting. Chloropicrin is considered a "potent" carcinogen,[32] and causes developmental and reproductive toxicity in animal studies. 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 Watsonville 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 2014, a resident in Watsonville was growing increasingly concerned about fumigant applications in the area, with particular concern over an upcoming chloropicrin application scheduled to occur in a field located within view of the residence. The residence is located approximately 350 feet away from the nearest field-edge of one field block, and 850 feet away from the second field block. The residence has been occupied by the same family for the past five years. Among the family members are two children, ages five and nine. No past incidents of illness resulting from drift exposures have been noted by the residents. In addition to the children at the residence, 12 children ranging in age from 3-16 years old live in homes located along the first field block that was scheduled for fumigation.

In October, a PAN scientist met with the concerned resident and provided him with air monitoring equipment (a "Drift Catcher") and detailed training – including how to operate the equipment, change the sample tubes, and certification as a trained operator of the Drift Catcher. In late October 2014, the resident received notification that chloropicrin and metam potassium would be applied to the field adjacent to his residence starting on November 3rd. The resident used a Drift Catcher to monitor the air for nine days following this application, 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 field adjacent to the residence. Chloropicrin was the only fumigant monitored during this period of time due to verbal communications received by the resident that metam potassium would not be applied after all. The resident received verbal confirmation of this change from the farmer and the permit granted by the Santa Cruz County Agricultural Commissioner confirmed that metam potassium was not applied.[9]

Fumigation drift incidents have been known to cause illness in residents. Following a drift incident in 2007 with the fumigant methyl bromide, residents of a community in Sisquoc, California, reported that several children became ill with symptoms of fever, restlessness, and in some cases vomiting.[1]

Previous Drift Catcher studies in California have documented chloropicrin concentrations in the air exceeding levels of concern. Drift monitoring was done in April 2008 in Sisquoc, CA during a subsequent methyl bromide/chloropicrin application, with the results confirming chloropicrin concentrations in the air during and following application; results are detailed in a 2010 PAN technical report.[2] Community partners conducting air monitoring in 2011 for chloropicrin in Tehama County, California, have also observed effects from chloropicrin applications adjacent to their residences, reporting symptoms such as burning eyes as well as a large number of chickens that died suddenly at a residence, on a day coinciding with an adjacent fumigation. Comparisons between these data and previous Drift Catcher studies are discussed later on in this report.

Site Description and Application Details

Watsonville, California, is a city in Santa Cruz County with a population of approximately 51,000 people as of 2010. The two field blocks to be fumigated near the residence where monitoring took place were planted with strawberries following the pre-plant fumigation. Watsonville and Salinas account for about half of California's strawberry acreage, and California is the leading U.S. producer

of strawberries. Strawberries are the sixth most valuable fruit crop produced in California.[3] Due to the common practice of pre-plant soil fumigation for strawberries, they are considered a pesticide-intensive crop, ranking fourth in California for pesticide use by pounds of active ingredient per acre in 2012 (3.8 pounds of active ingredient per acre).[4] Fumigants accounted for about 87% of the pesticide active ingredients applied to strawberries in 2012.[5] Pesticide expenditures for strawberry planting in the Central Coast area have been calculated at $1,000 to $1,800 per acre for chloropicrin as of 2010.[6]

A Drift Catcher was placed in the backyard of a residence located near the intersection of Rancho Road and Buena Vista Road. The resident's family was known to spend time in the back yard, where a child's play set was located. The home was located in a residential area bordered by fields on the eastern, western, and southern edges. The site was located approximately 350 feet from the first application site and approximately 850 feet away from the second application site. In response to inquiries by the resident, the county agricultural commissioner provided as a courtesy prior notification of when the fumigation would occur.

Records obtained from the County of Santa Cruz Office of the Agricultural Commissioner show that 14 acres of this field were fumigated in two seven-acre blocks between November 3 and November 5. A note on the permit regarding the November 5th application to Block 2 indicated that the application was to end on November 6th by 11 AM. The dates that the individual blocks were treated are indicated on the map in Figure 1. The applications were scheduled to begin between 7 and 8 AM. Santa Cruz County requirements specified that fumigations were to take place using drip irrigation ("bedded drip" in this report) under "Totally Impermeable Film" (TIF). TIF is a type of low-permeability tarp that reduces emissions from a fumigated field in comparison to a standard polyethylene tarp.[7] The TIF was to remain during fumigation and to be perforated at a minimum of nine days post-application for planting preparation.[8] The fumigant product, TRICAL Inc. 0/100 Tri-Chlor EC 58266-5-11220 (94% chloropicrin and 6% other ingredients), was applied at a rate of 210 pounds per acre. The records indicate the field was treated as a preplant fumigation for strawberries.[9]

Information on the sizes of the application blocks and the approximate distances from the monitoring site to the edge of each block are provided in Table 1. The distances are estimates based on measurements made on Google Earth, with the information on field blocks obtained from the copy of the permit from the Santa Cruz County Agricultural Commissioner's Office.[9]

Table 1. Fumigation Dates and Sizes of Fumigation Blocks

Block	Date of Fumigation	Size in Acres	Approximate Distance from Site
1	November 3	7	350 ft
2	November 5-6	7	850 ft

[a] In both the permit and notification letter from the Agricultural Commissioner, the areas of blocks one and two are listed as seven acres.

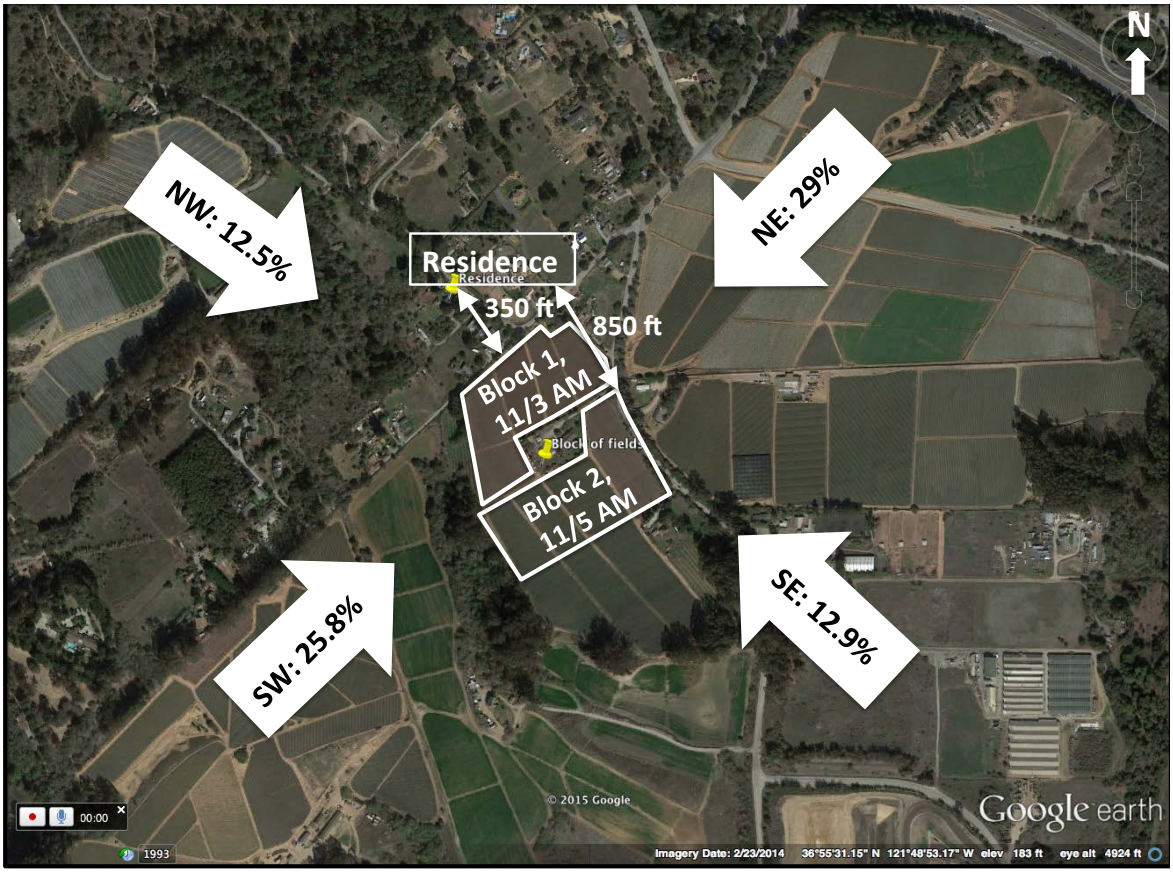


Figure 2. Approximate locations of fumigation blocks. The sketch is based on the Notice of Intent submitted to the County of Santa Cruz Office of the Agricultural Commissioner. Dates of fumigation for each block are shown. The residence was located 350 feet away from the nearest field-edge of Block 1 and 850 feet away from the nearest field-edge of Block 2. The large white arrows indicate directions and percent frequency of wind directions from the northeast (29%) and southwest (25.8%), the predominant wind directions during sampling. From the other directions, the frequencies of wind direction were as follows: east (1.61%); southeast (12.9%); south (2.82%); west (2.42%); northwest (12.5%); north (12.1%). All boundaries are approximate. Underlying map image is from Google Maps, © 2015.

Methods

Sample Collection

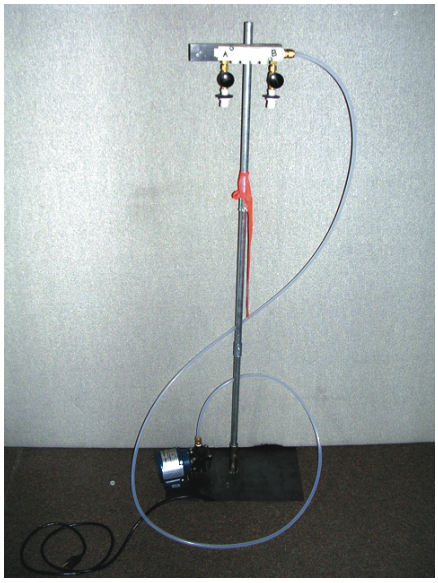
Samples were collected by pumping air through XAD-4 resin tubes at a rate of approximately 0.10 L/min. Sample tubes were obtained from SKC Inc. (#226-175, 8 x 150 mm, 400/200 mg in front/rear beds, respectively), and were generally changed every 12 hours, at approximately at 7 AM and 7 PM. This sampling 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).[10, ,11, 12]

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 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 1.5 meters in height. Flow rates were measured with a 0.05–0.5 L capacity rotameter (SKC Inc., Cat. #320-2A05). 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 PAN at -10 to 0°C by overnight express mail for analysis. At PAN, data from sample log sheets were entered into a sample log notebook and samples were stored in a -20°C freezer for six days prior to being shipped by overnight express mail to a commercial laboratory (Environmental Micro Analysis Laboratories, Inc., Woodland, California) for analysis. A chain of custody form accompanied each batch of samples during handling and transport. At the laboratory, samples were stored in a -20°C freezer prior to processing, which occurred within one month of receipt in the laboratory. The laboratory reported initial results from analysis of the front beds four weeks after sample collection, and it appeared likely that at least some of the samples were likely to have chloropicrin detected in the rear beds as well. After initial results reporting, a request to have the rear beds analyzed was made by PAN. Not more than eight weeks passed between sample collection and analysis of the rear beds in the sample tubes. Prior sample storage stability assessments conducted by the ARB indicate that chloropicrin is stable on XAD-4 resin for at least four weeks under these conditions.[10, 11, 12]

(a) 

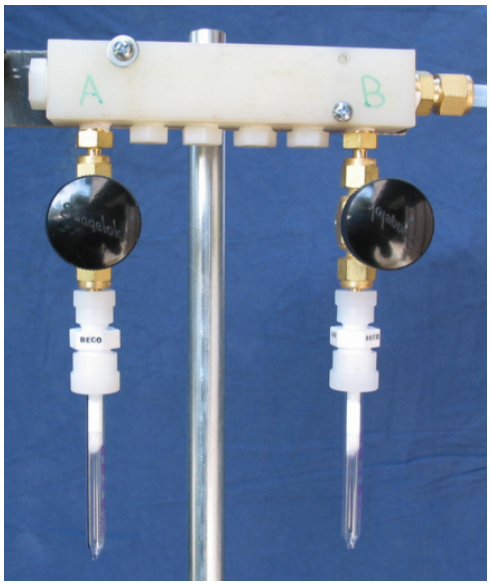
(b) 

Figure 3. (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 Laboratories, Inc. (Woodland, California) using GC with electron capture detection according to OSHA method PV 2103.[13, 14] Samples were desorbed with 3 mL of ethyl acetate rather than 1 mL as specified in the method. The laboratory reporting limit was 0.03 µg/tube, which corresponds to an air concentration of 0.21 µg/m^3 for a 24 h sample collected at 0.1 L/min.

In addition to the field samples, one trip blank sample (a negative control; please see **Quality Assurance – Quality Control** section at the end of this report for explanation of the trip blank) was sent to the lab for analysis. The lab was unaware of which samples were field samples and which were blanks. The front and rear beds of the sample tubes were analyzed separately. In six samples, pesticides were detected in the rear bed, indicating that there was breakthrough from the front to rear bed. Samples were not tested 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 Watsonville.[15] The meteorological data for the sampling period are provided in Appendix 1.

Results

A total of 18 samples (blanks excluded) were collected at the Watsonville site from November 3–12, 2014. One trip blank was made in duplicate at the end of the first 12-hour sample. No chloropicrin

was detected in the blanks nor in field samples that were collected in the latter days of sample collection. 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 less than 25%, and the average value was used to calculate the sample volume. For sample "Ogre," the difference in flow rates exceeded 25%, so the total sample volume was calculated based solely on the greater flow rate so as to over-estimate the sample air volume and thus provide a conservative estimate of the airborne pesticide concentration. The reported pesticide concentration for this sample should therefore be considered a minimum value. 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.[12] Complete results are provided in Table 2 and a plot of chloropicrin concentrations over time is presented in Figure 3.

Chloropicrin was detected in 11 (61%) of the 18 field samples from the Watsonville site (see Table 2 and Figures 3 and 4). In six samples, breakthrough occurred from the front to the rear bed, indicating that the resin in the sample tubes did not trap all of the chloropicrin that passed through the tubes, which results in measured concentrations of chloropicrin that are potentially lower than the actual concentrations in the air. Sample "Purple," collected at the site between 6:40 AM and 7 PM on November 6 – the day that fumigation of the block was completed – had the highest observed concentration of chloropicrin, at 7.9 $\mu g/m^3$. Time Weighted Average (TWA) chloropicrin concentrations were also calculated for the sampling period. No background sample was taken prior to the start of fumigation. Thus, the TWA concentrations covered the 9-day period from November 3-12, and the average was 1.3 $\mu g/m^3$. TWA concentrations covering only the 4-day period from November 3-6 had an average of 2.4 $\mu g/m^3$.

According to the permit, fumigation was scheduled to begin in Block one at 7 AM on November 3rd. The Drift Catcher operator noted that the tarps were already placed on the field in the days prior to the scheduled fumigation. As would be expected with a negative control, chloropicrin was not detected in the trip blank ("House").

Table 2: Chloropicrin Concentrations at monitoring site, Watsonville, California, November 3-12, 2014

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, b]
Shoe	11/3/14	6:40 AM	11/3/14	7:00 PM	740	0.08	2.58	D, FUM-1
Ogre	11/3/14	7:28 PM	11/4/14	7:38 AM	712	0.07	2.04	D, MV
Mom	11/4/14	7:24 AM	11/4/14	6:55 PM	691	0.06	3.71	D
Sky	11/4/14	7:00 PM	11/5/14	7:10 AM	730	0.06	0.78	D
Tree	11/5/14	7:28 AM	11/5/14	7:05 PM	697	0.07	1.44	D
Roof	11/5/14	7:10 PM	11/6/14	6:35 AM	685	0.07	0.855	D, FUM-2
Purple	11/6/14	6:40 AM	11/6/14	7:00 PM	740	0.08	7.86	D, FUM-2
Lady	11/6/14	7:15 PM	11/7/14	7:00 AM	705	0.07	0	D, MV
Cat	11/7/14	7:05 AM	11/7/14	7:15 PM	730	0.07	3.56	D
Pillow	11/7/14	7:20 PM	11/8/14	7:30 AM	730	0.07	0.479	D
Banana	11/8/14	7:40 AM	11/8/14	7:20 PM	700	0.07	0.557	D
Alpha	11/8/14	7:30 PM	11/9/14	7:15 AM	705	0.07	nondetect	
Rain	11/9/14	7:25 AM	11/9/14	7:30 PM	725	0.07	nondetect	
Laurel	11/9/14	7:37 PM	11/10/14	7:20 AM	703	0.07	nondetect	
Salt	11/10/14	7:30 AM	11/10/14	7:30 PM	720	0.08	nondetect	
Good	11/10/14	7:35 PM	11/11/14	7:19 AM	704	0.07	nondetect	
Bread	11/11/14	7:25 AM	11/11/14	7:15 PM	710	0.07	nondetect	
Joker	11/11/14	7:25 PM	11/12/14	7:20 AM	715	0.07	nondetect	
House	11/3/14	7:19 PM	-	-	-	-	nondetect	Trip Blank

[a] D = The duplicate sample was analyzed and value shown is the average of two duplicate samples; MV = minimum value (see text).
[b] FUM-1, FUM-2= fumigation of blocks 1 and 2, respectively, occurred on the dates indicated. Note that according to permit notes[9], fumigation of the 2nd block began on November 5, but any portion of the field not completed on November 5th was scheduled to end by 11 AM, November 6.

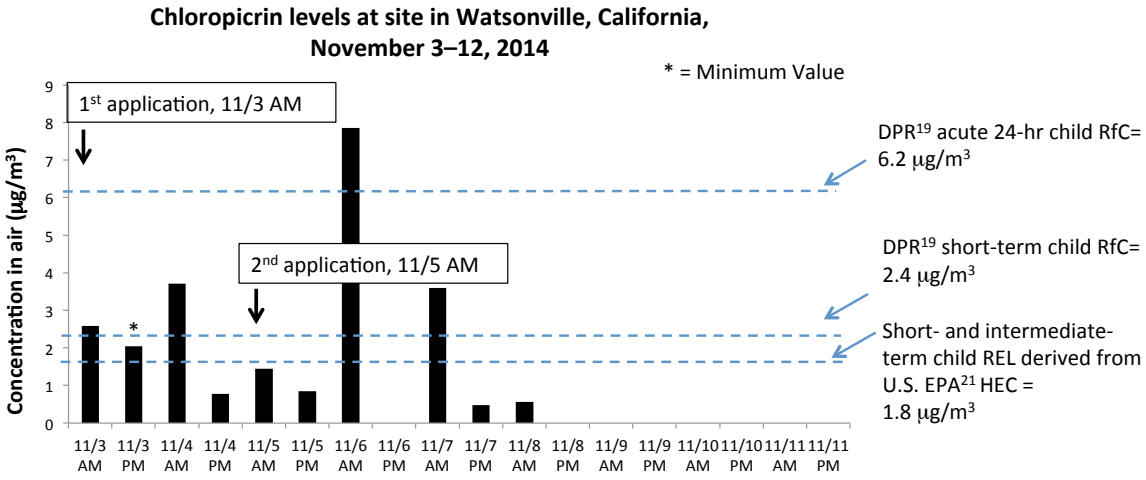


Figure 4. Chloropicrin concentrations in air at monitoring site in Watsonville, California, November 3-12. All samples taken were 12-hour samples. RELs are based on DPR toxicologists' analysis of risk.[19] RfC= reference concentration; REL= reference exposure level; HEC= human equivalent concentration.

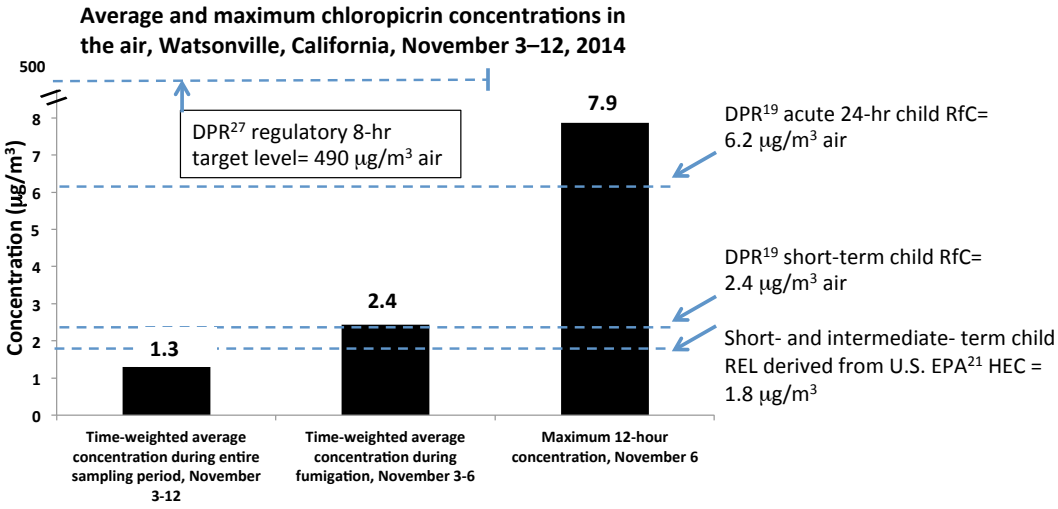


Figure 5. Average and maximum chloropicrin concentrations during the sampling period. Time-weighted average for the entire sampling period is shown (November 3-12), along with the time-weighted average calculated for only the period of time when fumigation was occurring (November 3-6). RELs are based on DPR toxicologists' analysis of risk.[19] DPR's regulatory 8-hr target level is shown above the TWAs only because it is based on an 8-hr exposure. RfC= reference concentration; REL= reference exposure level' HEC= human equivalent concentration.

Discussion

Meteorology and Timing of Fumigations

Fumigation operations on November 3 and 5 were scheduled to begin at around 7 AM. Weather station data indicate that during the monitoring, winds in Watsonville were calmer in the mornings (1-4 mph) and in the afternoon and stronger in the late afternoon (4-9 mph). The wind very rarely blew from the west during the sampling period, which would have carried the fumigant plume away from the residence. In general, winds blowing from southern directions would have carried some of the fumigant plume towards the residence, while winds blowing from the northern direction would not tend to carry the plume towards the residence.

In Figure 6 below, the frequency of wind direction is plotted, based on hourly data collected from CIMIS station 129 (Pajaro 129) for the sampling period.[15] Winds predominantly blew from the northeastern (29% of the time) or the southwestern direction (25.8% of the time). From the other directions, the percent frequency of wind direction was as follows: east (1.61%); southeast (12.9%); south (2.82%); west (2.42%); northwest (12.5%); north (12.1%). Figures A-1 and A-2 in Appendix 1 show the hourly average wind speed and direction for November 3-12, 2014.

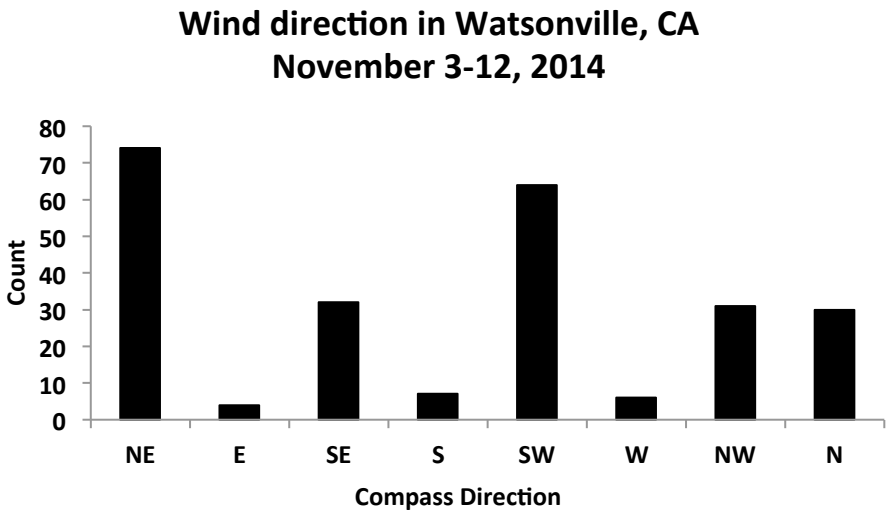


Figure 6. Plot of compass direction versus the frequency of occurrence of wind from each direction.

On November 6, the AM sample "Purple" resulted in the highest level of chloropicrin detected during the sampling period. During the period of time from approximately 6:40 AM to 7 PM on November 6, the wind predominantly blew from the southeast, southwest, and southern direction. During the hours of sample "Purple," the winds came from the southerly directions (i.e., blowing across the fumigated fields and towards the house) was 69% of the time. Winds blew from the

northeast and northwest directions (i.e., away from the fields) the remaining 30% of the time during the hours that sample "Purple" was run.

The sample following the highest 12-h sample "Purple" resulted in a nondetection ("Lady"), due to a change in wind direction. During the 12-h sample "Lady," winds blew from the northerly direction – from either the northeast (54.5% of the time) or the northwest (45.5% of the time) during that 12-h sample, i.e., towards the field and away from the residence where the Drift Catcher was located).

Generally, the highest-concentrations were measured in daytime samples. The Drift Catcher data are consistent with these observations, as the 12 hours following the morning start time of these samples included the afternoon hours, when winds generally blew from the south where the fumigated fields were located towards the location of the Drift Catcher at the residence.

Distances from Fumigated Blocks

The distances of the monitoring site at the residence from the two fumigated blocks were approximately 350 and 850 feet for blocks 1 and 2, respectively. The distance between the fumigated fields and the residence were greater than mandated buffer zones from EPA or those currently proposed by CA DPR, which for these size fields range from 30 to 40 feet only (see Table 8). Thus, current or proposed buffer zones would not have protected against the observed levels of chloropicrin in the air, which reached levels of concern based on TWA calculated for the days when fumigation was occurring. The field blocks were both about seven acres. The levels of chloropicrin measured in the air were relatively higher during the AM sample taken just after or at the time that fumigation of the second block was completed. The second block was fumigated starting on November 5, and it was noted on the permit that the application would be completed by 11 AM on November 6, at the latest. This sample ("Purple") resulted in the highest chloropicrin levels detected during the sampling period, at 7.9 $\mu g/m^3$.

The closer proximity of the first block to the Drift Catcher might lead one to predict that higher levels of chloropicrin (relative to the second, more distant fumigant application) would be detected shortly after the first fumigation on November 3. However, the maximum level of chloropicrin detected was during the second fumigation. With the fumigation of the second block, the wind may have carried drift from both fumigated blocks towards the air sampler.

Comparison to Levels of Concern

Chloropicrin is ranked by EPA as Category I (highly acutely toxic), and the state of California listed it as a Toxic Air Contaminant in April 2010.[16] 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 (fluid in the lungs) at high exposures.[17] Chloropicrin 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 a concern in terms of the potential for negative impacts on human health.

To assess whether the levels of chloropicrin observed in this study constitute a significant risk to the exposed population, we compared the measured concentrations to levels of concern developed by

EPA, CA OEHHA, and CA DPR. 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). This NOAEL is converted to a human-equivalent dose, and regulators then derive a level of concern by dividing the human-equivalent dose 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. For genotoxic chemicals, 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 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.

Non-Cancer Levels of Concern for Chloropicrin

In this study, we use levels of concern for acute and short-term exposure derived by the EPA, DPR, and the California Office of Environmental Health Hazard Assessment (OEHHA). These are:

- Reference Concentrations (RfCs) derived by DPR toxicologists in their 2010 chloropicrin risk assessment for their Toxic Air Contaminant Program[18] and subsequent 2012 Risk Characterization Document;[19]
- The actual regulatory endpoints used by DPR management;
- Reference Exposure Levels (RELs) determined by OEHHA;[20] and
- RELs derived from the Human Equivalent Concentrations (HECs) in EPA's 2009 risk assessment (see Table 3).[21]

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. 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" (MOE) 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 3. A comprehensive discussion of how to interpret air monitoring results is presented in **Appendix 2: Interpreting Air Monitoring Results.**

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

In 2009, EPA 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 included eye irritation, increased nasal nitric oxide (nNO), and differential effects on inspiratory and expiratory flow.[22] Individuals with respiratory problems or chronic illness were excluded from the study.

In US EPA's 2006 Preliminary Risk Assessment for chloropicrin,[23] the Agency used a level of concern for acute exposure of 49 $\mu g/m^3$. This was derived from a benchmark concentration ($BMCL_{10}$) of 490 $\mu g/m^3$, 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 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;[22] HED did not take this advice. The EPA 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.[24]

Table 3: Summary of Toxicity Information Used To Calculate RELs

Exposure Scenario (Timeframe)	Critical endpoint expressed as a Human Equivalent Concentration (HEC)[a] (µg/m^3)	Effects at LOAEL	Uncertainty Factors	REL or RfC[b] (µg/m^3)
U.S. EPA Acute Adult (1–24 hour)[21]	$BMCL_{10}$ = 490	Human study: 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)[21]	HEC = 54	Mouse study: Rhinitis	30 (interspecies: 10X intraspecies: 3X)	1.8 (0.26 ppb)
DPR Acute (1 hour)[16]	$BMCL_{10}$ = 296	Human study: Increased NO concentration in nasal air	10 (interspecies: 1X intraspecies: 10X)	30 (4.4 ppb)
DPR Acute (1–8 hour)[16]	Child: HEC = 1,800 Adult: HEC = 3,900	Rabbit study: 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 (24 hour)[16]	Child: HEC = 620 (92 ppb) Adult: HEC = 1,300 (190 ppb)	Rabbit study: 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)[16]	Child: HEC = 230 (35 ppb) Adult: HEC = 490 (73 ppb)	Rat study: Rhinitis in female rats	100 (interspecies: 10X intraspecies: 10X)	Child: 2.3 (0.35 ppb) Adult: 4.9 (0.73 ppb)
DPR Regulatory Target Level (8 hour)[27]	Adult & Child HC[c]=490 (73 ppb)	Human study: Eye irritation, increased nasal nitric oxide, altered breathing	1 (interspecies: 1X; intraspecies: 1X)	Adult & Child: 490 (73 ppb)
OEHHA Acute (1 hour)[33]	RD_{05} = 890[b]	5 % decrease in respiratory rate	30 (interspecies: 3X intraspecies: 10X)	29 (4.3 ppb)

[a] When calculating HECs for children, DPR uses the breathing rate of 1-year-old infant of 0.59 m^3/kg/day.
[b] 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.
[c]HC=Human Concentration, value derived from the human study.

EPA issued a Revised Risk Assessment in April of 2007.[25] This assessment removed the intraspecies uncertainty factor of 10, and a value of 490 μg/m^3 was proposed as the level of concern. The document noted that, "73 ppb [490 μg/m^3] represents a level at which upper respiratory changes and irritation (eyes, nose) would not be expected to occur." Interestingly, 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 10th and 90th percentile of the population, substantially higher than the factor of 10 they had removed.

EPA finalized the chloropicrin risk assessment in May 2009.[21] 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 NOAEL endpoint appropriate for assessing the risk of non-occupational short-term (1–30 day) and intermediate-term (1–6 months) 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 portal-of-entry effect rather than a systemic effect, EPA did not calculate different levels of concern for children and adults.[21]

California Department of Pesticide Regulation (DPR) Levels of Concern

The levels of concern developed by CA DPR toxicologists in the February 2010 risk assessment and approved by the Scientific Review Panel (SRP)[32] and California Office of Environmental Health Hazard Assessment[26] are lower (i.e. more health-protective) than those used by EPA.[18] DPR toxicologists 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 EPA described above. DPR staff 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. The seasonal/subchronic RfCs for children and adults developed by DPR staff toxicologists are 2.3 μg/m^3 and 4.9 μg/m^3. The 8- and 24-hour acute RfCs recommended by DPR toxicologists were based on a rabbit study— a different study than the human study used by EPA. Because of this difference in study choice, the DPR toxicologists recommended an RfC that was lower that EPA's RfC by a factor of 12.6.

While DPR staff toxicologists, the SRP and OEHHA all agreed on the selection of endpoints to use to develop RfCs as described above, DPR management announced a completely different regulatory target level of 490 μg/m^3 (73 ppb) for both adults and children, based on the human study that did not include any uncertainty factors.[27] This decision mirrors that of EPA, but is in conflict with the scientific recommendations for health-protective endpoints.

Consideration of the human study on which the regulatory target level is based indicates that reducing exposures to this concentration will not adequately protect against irritant effects in the exposed population. The fact that a human study was done supports the elimination of the interspecies UF, but there is no justification for the elimination of the intraspecies UF (typically a factor of 10). In fact, the variability in eye irritation thresholds among the subjects in the human study were found to vary by *greater than* a factor of 10 between study participants—see Table 4

below.[28] Note that the regulatory target level of 490 μg/m^3 (73 ppb) is twice the concentration that the 10th percentile subject responded to and the variability between the most and least sensitive subject was a factor of 42, far greater than the intraspecies factor of 10 typically used in risk assessment. In the Phase III human study, no low dose was even tested, resulting in a study that produced only a lowest observed adverse effect level (LOAEL), not a NOAEL.

Table 4: Chloropicrin Concentrations at Which Adverse Effects Were Observed

Effect	Threshold conc. 10th percentile (most sensitive subjects)	Threshold conc. 90th percentile (least sensitive subjects)	Factor by which 10th percentile differs from 90th percentile
Eye irritation	37 ppb	1,565 ppb	42
Odor	216 ppb	764 ppb	1.9

Indeed, in their review of the chloropicrin human study, members of the Human Subjects Review Board recommended that not only should the intraspecies uncertainty factor (UF) be retained, but that an additional UF be used.[29] This conclusion was based on the fact that only young healthy adults were included as study subjects. Only subjects who reported no smoking within a year, no use of recreational drugs within a year, no recent illness, and no history of chronic illness qualified to go on to screening in the laboratory. In addition, to be approved for inclusion in the study, subjects were required to have pulmonary function at or above 83% of predicted forced expiratory volume at 1 sec (FEV1) or forced vital capacity (FVC) for testing by American Thoracic Society criteria. These criteria clearly exclude people with asthma or other respiratory illness, children whose lungs are still developing, the elderly, and other vulnerable populations.

There are two additional factors not accounted for by DPR's regulatory target level of 490 μg/m^3 (73 ppb) averaged over eight hours. First, no subject in the human study was ever exposed to chloropicrin for more than 60 minutes at a time, much less eight or 24 hours. Second, during a fumigation, the concentration of chloropicrin varies over time, with concentration peaks that are substantially above the 490 μg/m^3 (73 ppb) level. DPR's decision to eliminate the intraspecies UF for an exposure period averaged over eight hours and based on a study in which humans were exposed at most for 60 minutes will not be protective of the health of even healthy individuals, and could pose very serious risks for vulnerable populations.

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

OEHHA has 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.[30] This REL is more protective than both 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.

In OEHHA's comments[31] on DPR's Risk Characterization Document,[34] they recommended that the seasonal and chronic RfCs for children incorporate the breathing rate for infants, which would reduce those RfCs for children by 12 percent.

Comparison of Watsonville Results to Non-Cancer Levels of Concern

The results of this study indicate that acute levels of concern were exceeded at the site at least once during the ten-day sampling period. A TWA chloropicrin concentration of 1.3 μg/m^3 was calculated for the entire nine-day sampling period, which is lower than any chloropicrin reference concentrations discussed here. However, the TWA at the site for the period beginning on the day that the fumigation started and ending four days later exceeds both the 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, EPA normally takes action to reduce exposures to below levels of concern.

None of the samples exceeded the EPA, DPR, or OEHHA 1-hour or 8-hour levels of concern, but this basis of comparison is not valid, as samples in this study provided average concentrations over a 12 or 24-h sampling period. The highest observed concentration does exceed the science-based 24-h acute level of concern recommended by DPR toxicologists, the SRP, and OEHHA of 6.2 μg/m^3 for children. This is for the sample 12-h sample "Purple," taken November 6 in the AM, which was 7.9 μg/m^3. The samples taken were all approximately 12-h samples, so comparing these samples to a 24-h RfC is not necessarily appropriate. On November 6, the 12-h AM sample taken ("Purple"), resulted in the highest level of chloropicrin detected. During the hours that sample "Purple" was taken, the winds came from a southerly direction (i.e., blowing across the fumigated fields and towards the house) 69% of the time. Winds blew from the northeast and northwest directions (i.e., away from the house towards fields, so carrying the fumigant plume away from the sampler) the remaining 31% of the time during the hours that sample "Purple" was run.

Meaningful comparisons can be made to the TWA of these samples with those immediately preceding or following them. The sample following the highest 12-h sample "Purple" resulted in a nondetection ("Lady") due to a change in wind direction. During the 12-h sample "Lady," winds blew from the northerly direction (towards the field and away from the residence where the Drift Catcher was located) – from either the northeast (54.5% of the time) or the northwest (45.5% of the time) during that 12-h sample.

Four of the calculated 24-h TWAs were greater than the EPA's short- and intermediate-term REL of 1.8 μg/m^3, taken from November 3 to November 7. These 24-h TWA values ranged from 2.0-4.0 μg/m^3, and include the TWA value for samples "Purple" and "Lady" (4.0 μg/m^3). A TWA of 2.4 μg/m^3 was calculated for the period of time during the fumigation (November 3-6), which exceeded EPA's short- and intermediate-term REL of 1.8 μg/m^3 and is at the level of DPR's seasonal child RfC of 2.4 μg/m^3, indicating an unacceptably high risk of adverse effects for people who spent significant time in the vicinity of that site.

Only chloropicrin was monitored in this study, so risk estimates do not account for exposures to multiple chemicals.

Cancer Risks from Chloropicrin Exposure in Watsonville

EPA does not consider chloropicrin to be carcinogenic by inhalation exposure. The EPA 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."[25]

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 DPR toxicologists derived a cancer potency factor of 2.2 $(mg/kg\text{-}day)^{-1}$ for chloropicrin.[18] 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. The DPR staff determination of carcinogenicity and cancer potency was supported by both the SRP and OEHHA.[32, 33]

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 33.
[b] Reference 34.

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 Watsonville were found to exceed the acceptable risk of level one in one million level of concern for bystanders. Given that chloropicrin is intensively used in Santa Cruz County and given the proximity of this neighborhood to other fields that had fumigation tarps laid over them, the estimate for chloropicrin cancer risk is highly likely to be on the low end of exposures.

DPR Management Contradicts Conclusions Reached by DPR and OEHHA Scientists

DPR managers contradicted the carcinogenicity conclusion reached by DPR staff scientists, OEHHA and the SRP, stating that "the conclusion of the Toxic Air Contaminant assessment was not adequately supported and that the evidence on the carcinogenicity of chloropicrin was equivocal."[27] DPR managers also issued a memorandum reconsidering the carcinogenicity of chloropicrin[35] in an attempt to justify this decision; however, this characterization is not supported by the body of evidence from animal and in vitro studies.

Management's memo claims that the probability for carcinogenicity in the comprehensive chloropicrin risk characterization (completed in 2012) was stated with "caveats and uncertainties". However, in the executive summary in the 2012 risk assessment[19] the toxicologists are clear in their assertion that chloropicrin is carcinogenic:

> "*Although the increases in the tumors in neither study were dramatic and all the in vivo genotoxicity studies were negative, DPR made a health protective assumption that chloropicrin was carcinogenic with a genotoxic mode of action based on its electrophilic structure and the positive in vitro genotoxicity tests*."

The detailed discussion of carcinogenicity later in the risk assessment unequivocally concludes that "based on the weight of evidence it was determined that the tumor data could not be dismissed". OEHHA reiterated this point in its response to DPR's Risk Management Directive,[36] noting:

> "*OEHHA respectfully disagrees with DPR's conclusion that evidence on the carcinogenicity of chloropicrin should be viewed as equivocal. Chloropicrin has been observed to induce gene mutations and chromosomal damage. The DPR chloropicrin TAC document, the OEHHA chloropicrin findings and the SRP chloropicrin findings all state that chloropicrin is a genotoxic carcinogen and can be assigned a cancer potency factor of 2.2 (mg/kg-day)$^{-1}$. This information should be considered in the development of an RMD for chloropicrin."*

The TAC evaluation concluded that in the 78-week chloropicrin inhalation exposure study, female mice showed statistically elevated incidence of lung adenomas and carcinomas at the highest dose tested by trend analysis using the Poly-3 trend test, which better accounts for survival in the different dose groups. The assessment also pointed out that the 37% incidence of adenomas at the highest dose was clearly outside the historical control range of 0-27% reported by the supplier, and that the tumor incidence might have been higher if the study duration were the standard 104 weeks rather than 78 weeks, if dose levels were higher, and if body weights and caloric intakes were not reduced.

DPR recently had a Department statistician review the relevance of using this particular type of statistical test.[37] This critique concludes that use of the Fischer test alone without the Poly-3 test might have been more appropriate. However, DPR neglected to mention that the statistician also stated that

> *"the results from this particular study, regardless of the manner in which the data are statistically analyzed, provide some (albeit borderline) evidence that high dose female mice develop increased numbers of lung tumors."*

Chloropicrin tested positive in a total of 13 in vitro mutagenicity tests, including three tests for DNA damage, eight reverse mutation assays with Salmonella strain TA100, and two tests for clastogenicity, characterized by a DPR toxicologist as "overwhelming positive results."[38] Instead of summarizing the weight of the evidence, the DPR risk management directive focuses on the two *in vivo* tests in which chloropicrin tested negative. We note that OEHHA characterized the *in vivo* studies as quite limited, inconclusive and suffering from experimental deficiencies.[31]

The combination of the animal studies and the *in vitro* studies indicate that there is no question that there is evidence of carcinogenicity. We utilized the scientifically based cancer potency factor to assess cancer risk from exposure to chloropicrin in this study.

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 Watsonville.

For the nine-day period from November 3-12, the TWA chloropicrin concentrations were 1.34 $\mu g/m^3$, and for the purpose of these calculations, it was assumed that this represents total annual chloropicrin exposure (i.e., there is no additional exposure beyond these nine days out of the year, but that this exposure happens every year). In this area, where chloropicrin applications are common, additional exposure is likely, but not accounted for in this estimate of cancer risk. The results indicate that the lifetime cancer risk exceeds the level of concern of one excess cancer per million people by a factor of 39 (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).[18]

Table 6: Lifetime Cancer Risk Estimates for Site at Watsonville, CA

Parameter	Site
Average concentration during monitoring period (μg/m^3)	1.3
Exposure frequency as a percent of a year	2.5%
Average annual concentration (μg/m^3)	0.066
Annual exposure[a] (mg/kg-day)	1.56 x 10^{-5}
Cancer potency factor (mg/kg-day)$^{-1}$	2.2
Lifetime Cancer Risk (excess cancers per million people)[a]	39

[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. To assess this increased risk, OEHHA has devised a method for calculating cancer risks for early life exposure to carcinogens.[39, 40] The OEHHA method gives more weight 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.

We applied this method to chloropicrin exposure scenarios based on the chloropicrin air concentrations observed in this study, and calculated the resulting cancer risks for exposure during childhood. In this analysis, we utilized time-weighted-average (TWA) breathing rates for different life stages, as given in the Exposure Factors Handbook.[41] 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 are summarized in Table 7 below; see the **Calculations** section for details.

Table 7: Childhood Cancer Risk Estimates for Watsonville Site

Exposure Scenario[a]	Cancer Risk per Million at Site
Infant (birth to 2 years)	9
Juvenile (2–16 years)	16
Adult (16–70 years)	12
Lifetime (birth–70 years)[a]	39

[a] Using TWA breathing rates for different life stages. 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 one million level of concern. Even for the scenario of relatively brief exposure early in life, significant cancer risk accrues: Nine excess cancers per million for exposure during infancy (0-2 years). The excess risk calculated for juveniles (2-16 years) was 16, and for a lifetime exposure (0-70 years) was 39.

Less-Than-Lifetime Cancer Risk

The lifetime cancer risks calculated in the section above exceed 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.[39] Therefore, in this section, we calculate the cancer risks associated with the more likely scenarios of living from birth until age 9 and birth through age 30 exposed annually to the levels of chloropicrin measured at this location in Watsonville.

EPA and DPR do not typically calculate less-than-lifetime cancer risk, 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 abbreviated exposure durations, the risk ranges from 36 to 46 excess cancers per million people.

Table 8: Less Than Lifetime Cancer Risk Estimates for Watsonville Site

Exposure Scenario	Cancer Risk per Million People
9-year residency (birth to age 9)[a]	18
30-year residency (birth to age 30)[a]	23

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

Exposure Assumptions

In the above sections, we documented exceedances 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.

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-h 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.*[42] *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 µg/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.[43]

- 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.

Neither EPA Proposed Buffer Zones nor CA DPR Buffer Zone Mitigations Would Have Protected Watsonville Residents

In 2009, 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 were phased in as of 2013. Buffer zones between fumigated fields and occupied structures (e.g. homes) are one such measure that is now required to protect people who live and work around fumigated fields.[44]

The newly mandated buffer zones did not protect against the chloropicrin exposure documented in this study. The buffer zone distances are shown in Table 9, below. Approximate distances from the monitoring site to the edge of the two fumigated blocks are given as well. These buffer zones were

taken from the lookup table (Table 10 in the amended RED) for chloropicrin, 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 TRICAL 0/100 Tri-Clor EC 58266-5-11220, which is 94% chloropicrin, at a total application rate of 210 lbs/acre. Buffer zones were determined assuming an application rate of 210 lbs/acre for chloropicrin. The amended chloropicrin RED specifies that the buffer zone should be based on the component of the mixture in highest concentration, which in this case is chloropicrin.[21]

As shown in Table 9, for each block in the November 2014 Watsonville fumigation, EPA's mandated buffer zone is less than the actual distance between the edge of that fumigated block and the Drift Catcher site. In other words, the current buffer zones would not have reduced chloropicrin concentrations below levels of concern in this study. In fact, fumigations substantially closer to homes than this one are still permitted. These data indicate that EPA's new buffer zones are not adequately protective of people who live and work near fields.

Buffer zones proposed by California DPR in its Chloropicrin Mitigation Measures released for public comment on January 6, 2015 (Table 13 of the document) would require a zone of 40 feet based on the buffer zone set for a block of 10 acres and based on an application rate of 200 lbs/acre – a larger application block and slightly lower application rate than the application monitored in the current study. As the fumigated blocks were located at 350-850 feet away from the residence where monitoring took place, this buffer zone distance would not have protected children in residence at the Watsonville site from exposure to concerning levels of chloropicrin. The minimum buffer zone set by California DPR for using TIF is 25 feet, which would have also alito protect against the exposures determined in the current sampling project. The buffer zone distances were determined from proposed buffer zone credits for TIF. The TIF buffer zone credit is 60%, which is specified by EPA on labels. California DPR evaluated data from TIF not available to EPA at the time that they assigned label buffer zone credits, and concluded that the data support a greater credit than 60%. California DPR will follow the 60% buffer zone credit for TIF specified EPA on labels.[45]

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

Block	Buffer zone based on proposed CA DPR chloropicrin buffer zone mitigations[a] (ft)	Buffer zone based on chloropicrin EPA RED[b] (ft)	Approximate distance of Block 1 border from monitoring site (ft)	Approximate distance of Block 2 Border from Site 2 (ft)
1	40	30	350	850
2	40	30	350	850

[a] Based on Table 13 of the California DPR Chloropicrin Mitigation Measures,1/6/15, page 17.[45] 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 14 of the EPA Amended Chloropicrin RED at page 56.[46] Block sizes and application rate were rounded up to closest value listed in the table. No buffer zone credits were applied.

EPA has removed all uncertainty factors for chloropicrin, based on studies done with human subjects. We have serous doubts that a reference dose based on exposure of young, healthy adults is sufficiently protective of all populations. 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*."[46]

This air concentration of 0.073 ppm or 73 ppb is equivalent to 490 $\mu g/m^3$.

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, and peer reviewers of the human study recommended even higher uncertainty factors to protect susceptible populations. 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 10^{th} and 90^{th} 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 one 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. EPA itself 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]."[21]

Comparison of Watsonville 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.[47] 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. In 2014, ARB began monitoring for chloropicrin at three sites located in Oxnard, Santa Maria, and Watsonville from August to October.[48]

Application Site Monitoring Studies for Chloropicrin

The three chloropicrin applications monitored by ARB took place between 2001 and 2005 in Monterey,[49] Santa Cruz,[10] and Santa Barbara counties.[50] The details of these studies are summarized in Table 10, and Figure 7 shows the maximum 12- and 24-hour chloropicrin concentrations measured in these studies along side the Watsonville data. Three additional studies not conducted by ARB (Rotondaro, 2004[51] and two PAN studies[2, 53]) are also reported in Table 10.

Table 10: Chloropicrin Application Monitoring

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, ARB, 2001	Shank tarped bed; 50:50 chloropicrin: MeBr @ 125 lbs/acre each	22	870	2–39	49
Santa Cruz County, ARB, 2003	Shallow shank tarped bed; 50:50 chloropicrin: MeBr @ 150 lbs/acre each	4.8	160	0.084–270	10
Location in CA, Rotondaro, 2004[a]	Drip tarped bed, 99.1% chloropicrin @ 156 lbs/acre	4.5	50	54-349	51
Santa Barbara County, ARB, 2005	Drip tarped bed; 94% chloropicrin @ 200 lbs/acre	8.2	60	0.3–415	50
Santa Barbara County, PAN, 2008[b]	Shank tarped bed; 57% MeBr, 43% chloropicrin @300 lbs/acre	42 total	35 (Site 1; 94 (Site 2)	0.54-14.5	2
Tehama County, PAN, 2012[c]	Tarped ground; 67% MeBr, 32% chloropicrin @351 lbs/acre	5	60	0.62-26.8	53

[a]These data were unpublished, but details were reported in references 51, 52, 61. See "Reference" column above. The other studies in CA counties were conducted by PAN where noted or by the CA Air Resources Board.
[b]PAN study in Sisquoc, California,[2] fumigation of the 42 acres occurred in 6 blocks between April 4-April 14, 2008. The sizes of the fumigated blocks consisted of 1, 5, 6, or 12 acres.
[c]PAN study in Tehama, California,[53] monitoring was done in the yards of two residences, which were directly adjacent to the field being fumigated.

In the Rotondaro 2004 monitoring of a drip tarped bed application described above in Table 10, sampling intervals were four hours per sample during the first 48 hours, and 12 hours per sample for an additional 8-10 days (10-12 days total). The highest concentration from the field drip irrigation was 349 $\mu g/m^3$ (51.9 ppb), measured 4-8 hours following the application.[61]

The highest concentrations adjacent to a fumigation were observed in Santa Barbara County in 2005 at 252 $\mu g/m^3$ (24-h) and 415 $\mu g/m^3$ (12-h). The maximum concentrations observed in the Monterey County study, 28 $\mu g/m^3$ (24-h) and 39 $\mu g/m^3$ (12-h) were the lowest of the three studies. The peak concentration observed in Watsonville was lower: 7.9 $\mu g/m^3$ for a 12-h measurement.

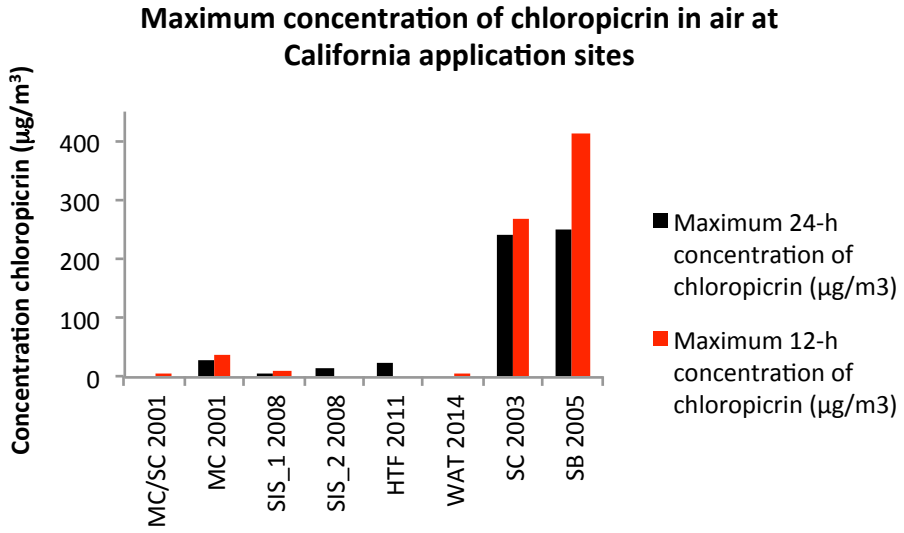


Figure 7. The maximum 12 and 24-h concentrations of chloropicrin measured in various California counties [Monterey and Santa Cruz Counties = MC/SC, Monterey County= MC; Sisquoc, Sites 1 and 2= SIS_1 and SIS_2, respectively; HTF=Healthy Tehama Farms,[53] Tehama County; WAT=Watsonville (current study); 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 Watsonville results and those of other studies, except to note that the concentrations measured in Watsonville do not represent a worst-case scenario. Based on mode of application, the study in Table 10 that most closely resembles the Watsonville study is the one conducted by Rotondaro: both are bedded drip tarp applications, though application rates are different (210 lbs/acre in Watsonville vs. 156 lbs/acre) and sampling schemes were different (all Watsonville samples were 12-hr vs. 4-12 hours by Rotondaro). While smaller than the Watsonville application, at 4.5 acres the Rotondaro study is comparable to the size of the two field blocks (7 acres each).

The chloropicrin levels observed in Watsonville are more comparable to those observed in Sisquoc, in a previously conducted study from PAN.[2] The maximum 12-hour level in Sisquoc was 11 µg/m^3 versus 7.9 µg/m^3 in Watsonville. The differences in maximum observed concentrations could be due to any number of factors:

- *Sampler placement:* The Watsonville study only had one sampler employed, located in proximity to the north side of the field. In the PAN Sisquoc study, 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. In

contrast, 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 of the field with highest concentration.

- *Tarp type:* Tarps vary in permeability to chloropicrin. The Watsonville application used TIF, which is a low-permeability tarp expected to reduce emissions in comparison to standard polyethylene tarps.[7] The Sisquoc application may have employed a different type tarp than those employed in the ARB studies.

- *Weather:* Differences in weather and wind patterns can contribute to differences in results. All of the ARB studies were conducted during the fall (October-November) as was the current study. For instance, in ARB's Santa Cruz study, the report indicated that the results might not have been representative due to the occurrence of rain both before and during the monitoring period.[10]

- *Timing:* In Watsonville, the applications took place in two blocks over four days, while in Sisquoc, six blocks were fumigated over 11 days and in CA ARB's study in Monterey, three blocks were fumigated consecutively over three 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. No background sample was taken prior to the beginning of fumigation in the current study. At the time of the writing of this report, it is not known whether other applications in proximity to the monitoring site at Watsonville were taking place during the sampling period. Towards the end of the sampling period in Watsonville, several nondetections were made (i.e., the last seven samples taken, between November 8 and November 12), suggesting that other applications of chloropicrin close enough to drift were not occurring during the latter part of sampling.

Seasonal Air Monitoring Studies for Chloropicrin

In 2001, Air Resources Board (ARB) conducted seasonal air monitoring studies for chloropicrin in Monterey,[11] Santa Cruz,[11] and Kern Counties.[12] 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 $\mu g/m^3$ with an eight-week time-weighted average concentration of 0.41 to 2.27 $\mu g/m^3$, 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 $\mu g/m^3$ with an eight-week TWA concentration of <MDL to 0.042 $\mu g/m^3$.

Figure 8 illustrates the maximum concentrations observed at these sites in comparison with those from Watsonville. The highest level observed in the ARB ambient studies was 14.3 μg/m^3, from La Joya Elementary School site (LJE_M in Figure 6) in Monterey. The maximum concentration observed in Watsonville is comparable to or exceeds some of the levels detected in various ARB studies. Levels detected in the PAN studies indicated on the left of the graph (WAT, HTF, SIS_1 and SIS-2 in Figure 6) exceed the concentrations for the majority of sites monitored by ARB. This is in line with expectations, as the samplers in these three PAN studies had samplers located within 60 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.

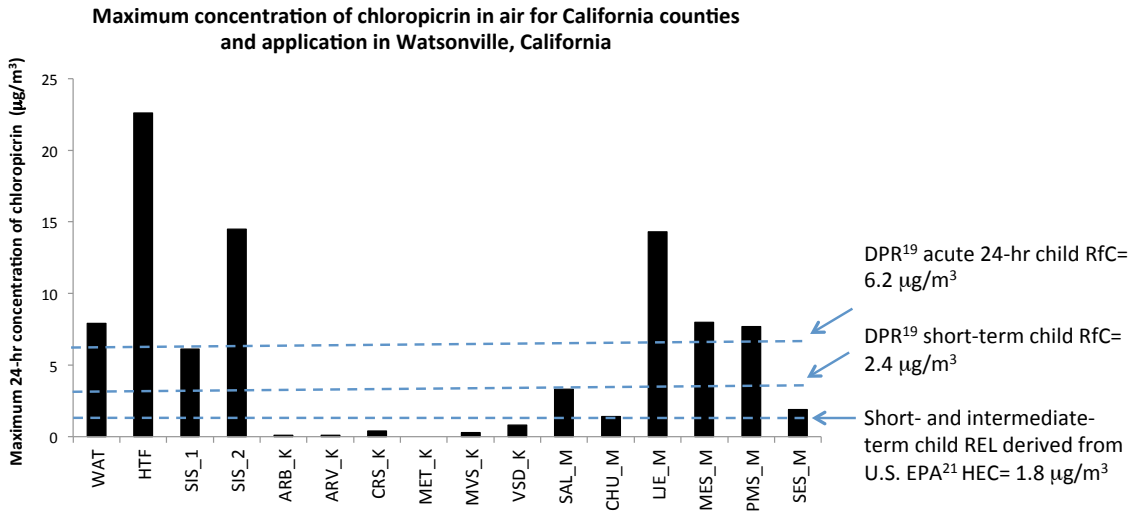


Figure 8. Maximum 24-hour concentration of chloropicrin measured in various California counties. The DPR toxicologists' RfC values (24-hour acute, short-term, and seasonal) for a child are displayed as a basis for comparison with the measurements.[19] DPR management's regulatory target level of 490 μg/m^3 (73 ppb) is not shown here. The values for the application monitoring at HTF, SIS_1, SIS_2, and WAT are plotted for comparison with the ambient air monitoring performed at all other sites represented here. [WAT=Watsonville, HTF= Healthy Tehama Farms in Tehama Co.,[53] 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

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).[54]

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.[55, 56] 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.[57]

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.[58] 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).

EPA does not consider chloropicrin to be carcinogenic by the inhalation route of exposure.[21] 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.[18] 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

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 research indicates that chloropicrin reacts with thiol groups of certain proteins such as glutathione (GSH) and hemoglobin.[59] 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).[60] 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.[60]

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.[61,62] 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.

From 1999 to 2012, at least 1,641 workers and community members have been poisoned by drifting fumigants in California.[63,64] A 2011 study of reported drift incidents and acute illnesses caused by drift from 1998-2006 in 11 states including California examined 643 drift events and 2,945 acute illness cases. In the same study, fumigants accounted for only 8% of drift events, but were the cause of 45% of the acute illness cases. Of those drift events that included information on violations of pesticide regulation, 74% had one or more violations; however, not all of the violations may have directly contributed to drift exposure. Violations contributing to the drift exposure were identified in 52% of the cases. In five agriculture-intensive California counties examined in the same study, the two groups with the highest overall incidence were agricultural workers and residents.[65]

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 harm 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.[66, 67, 68]

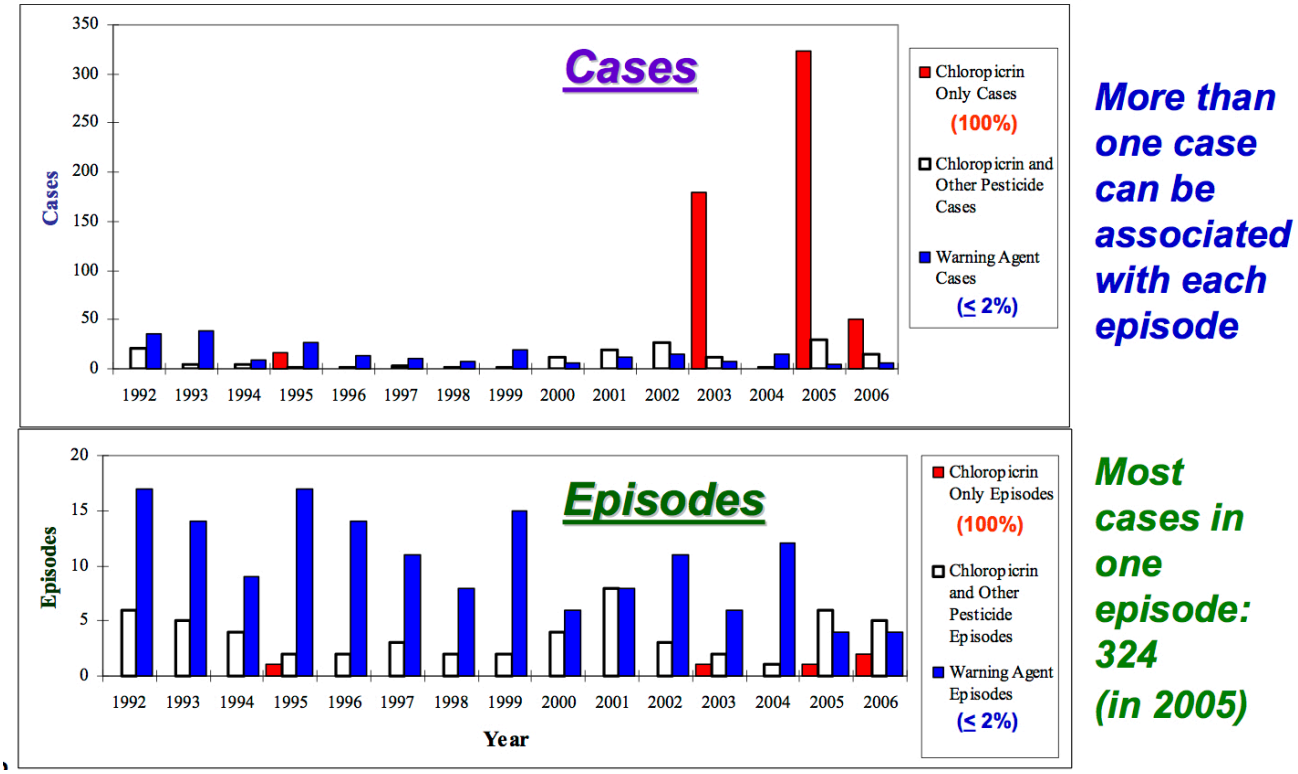


Figure 9. Illnesses Associated with Chloropicrin, 1992-2006. Percentage refers to the product used in fumigation, i.e., chloropicrin only, chloropicrin mixed with other pesticides, and episodes where chloropicrin was used as a warning agent. An "episode" can have multiple "cases," or reports made. Source: California Department of Pesticide Regulation presentation "Public Exposure to Chloropicrin in California," *www.arb.ca.gov/srp/**chloropicrin**(A).pdf*.

Table 11: Summary of Poisoning Incidents in California Involving Chloropicrin

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, 2012	52	920-2,240	Chloropicrin, 1,3-Dichloropropene (Telone)	No	No	Cited for failing to take all workers for medical treatment.
Santa Barbara County, 2012	42	50	Chloropicrin	Yes: Failure to notify workers, who entered field 3 hours early.	No	Pre-plant treatment for strawberry field.
Monterey County, 2011	39	115	Chloropicrin, Methyl bromide	Yes: Multiple, including failure to notify farmworkers.	Yes, slight wind blowing towards crew.	Tears in tarp post-application.
Monterey County, 2010	26	----	Chloropicrin, Methyl bromide	Yes: Inadequate posting and failure to notify.	No	Farmworkers fell ill.
Santa Barbara County, 2008	25	30	Chloropicrin, 1,3-Dichloropropene (Telone)	No	No	Farmworkers fell ill, adjacent to previously fumigated preplant lettuce field.
San Bernadino County, 2006	26	32-265	99% chloropicrin	No	No	Mainly workers from nearby busineses complained of symptoms.
Monterey County, 2007	At least 62	300	Chloropicrin, Methyl bromide	PCO applied 3 more acres than allowed by permit. Buffer zones lay within field.		Canvassers in neighborhood interviewed 62 people, more were likely affected.
Monterey County, 2005	336	-----	-----	Multiple violations	-----	-----

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
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	-----

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 61 and 62.

Conclusions

The data collected in Watsonville demonstrates that the levels of chloropicrin found in the air in Watsonville in the time period following a TIF, drip-line application in November 2014 exceeded levels of concern for both short and intermediate-term exposure and cancer risk. If such fumigations were to become annual events in the area, with similar concentrations to those observed in 2014 reoccurring every fall, 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, ranging from 9 to 39 excess cancers per million people.[2]

In the case where mixtures of fumigants are applied, co-exposure to another fumigant could occur. The original letter informing the Watsonville resident of the pending application included a request to apply metam potassium shortly after the chloropicrin application. However, the grower indicated that metam potassium would not be used in the application, nor was metam potassium monitored for, due to limited resources for air monitoring. Additive or synergistic effects associated with co-exposure are possible.

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

- In the monitored application, chloropicrin was applied at a rate of 210 lbs/acre. Much higher application rates are allowed for chloropicrin (for the product used in this application, up to 300 pounds of active ingredient per acre, but other chloropicrin products can legally be applied at even higher rates);[69] it is reasonable to assume that concentrations of chloropicrin in the air adjacent to such applications would be even higher than those observed in Watsonville.

- In 2012, 429,317 lbs of chloropicrin where applied in Santa Cruz County, 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 in other areas 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 higher in these areas.[70]

[2] 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.

- The monitored application in Watsonville appeared to have been conducted in compliance with all rules and regulations. The chloropicrin levels are not the result of an illegal application or failure to follow proper mitigation measures.

Overall, the data indicate that Watsonville 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 anything unusual about the application monitored there. Thus it is likely that hundreds of other communities across the state are experiencing chloropicrin exposures that are as high or higher than those documented in this report. As discussed previously in this report, both buffer zones proposed by California DPR and EPA's recently 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.[71] 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 Watsonville are much lower than those predicted by DPR, our results nonetheless support these conclusions. While DPR's conclusions are based on modeled reasonable worst-case scenarios, our data indicate that in real-world scenarios, levels of concern are still exceeded during the period of fumigation, even well outside of 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):[20]

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

Calculation of Reference Exposure Levels

In its most recent risk assessment of chloropicrin, 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) 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. 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 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, 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 EPA's target MOE, we calculated reference exposure levels (RELs) according to equation (3). Breathing rate and body weight are 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 a 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 EPA and CA DPR in their most recent chloropicrin risk assessments. As outlined 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 purposes. Since the 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).[72] 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.[18]

Details for each calculation are shown below; see Table 7 for results.

Estimation of Average Air Concentrations during the Application Period

The time-weighted average concentration of chloropicrin measured in this study during the entire sampling period was 1.34 $\mu g/m^3$, from November 3-12. The time-weighted average concentration of chloropicrin measured in this study was 2.44 $\mu g/m^3$ for the period from November 3 to November 6.

Estimation of Exposure Frequency

The length of the application season (and hence exposure frequency) for chloropicrin in the Watsonville 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: November 3-12 (9 days, 2.5% 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).

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

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

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

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).

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

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 -have 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.[40] 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),

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

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 three years of exposure beginning at age six, 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 to 70	54	1	0.24

For example, the lifetime cancer risk associated with exposure to chloropicrin at the site is 1.03 x 10^{-5} (postnatal, 0-2 years) + 1.66 x 10^{-5} (juvenile, 2-16 years) + 1.33 x 10^{-5} (adult, 16-70 years) – which is 39 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,[72] which we apply here to two scenarios: exposure to the levels of chloropicrin observed in Watsonville from birth to age nine and from birth to age 30. These scenarios were chosen because nine and 30 years are the figures OEHHA recommends for the central tendency and high-end estimates, respectively, of residency time.[73]

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 nine years. Furthermore, these calculations are breathing rate dependent, therefore the nine-year exposure scenario developed here applies specifically to period of birth to age nine.

The OEHHA methodology provides an estimate of dose based on annual exposure for less-than lifetime exposures according to equation (9):[72]

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

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: 2.7 μg/m^3 for, and exposure frequency, EF, is 9 days/year, or 2.5%. 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.[41] 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 7 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

A Drift Catcher Operator participates in a hands-on training workshop on the operation of the Drift Catcher at which time a Drift Catcher Users' Manual is provided. Operators are then tested on their knowledge of the procedures and practices by a PAN scientist. Participants are certified if they can 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

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 the PAN office, samples were logged into a sample log notebook kept in the PAN offices.

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.

Trip Blanks

One pair of trip blank tubes was prepared over the course of the sampling period as a negative control. 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 blank. This is shown in Table 2 (sample "House").

Appendix 1: Meteorological Data

The CIMIS weather station data indicate that during the monitoring, winds in Watsonville 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-2 show the hourly average wind speed and direction for November 3-12, 2014.

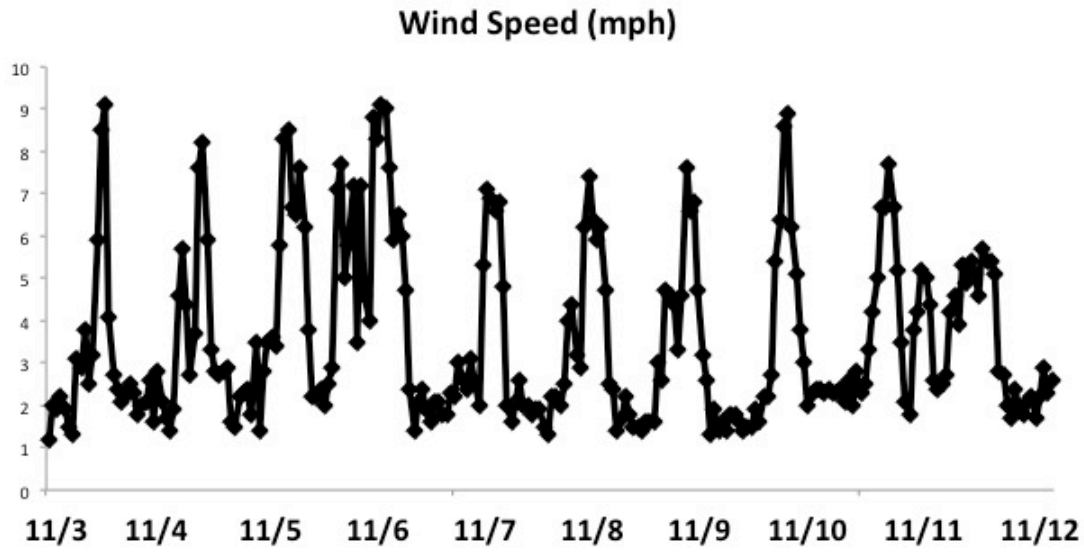


Figure A-1: Wind speed for Watsonville, CA, November 3-12, 2014.

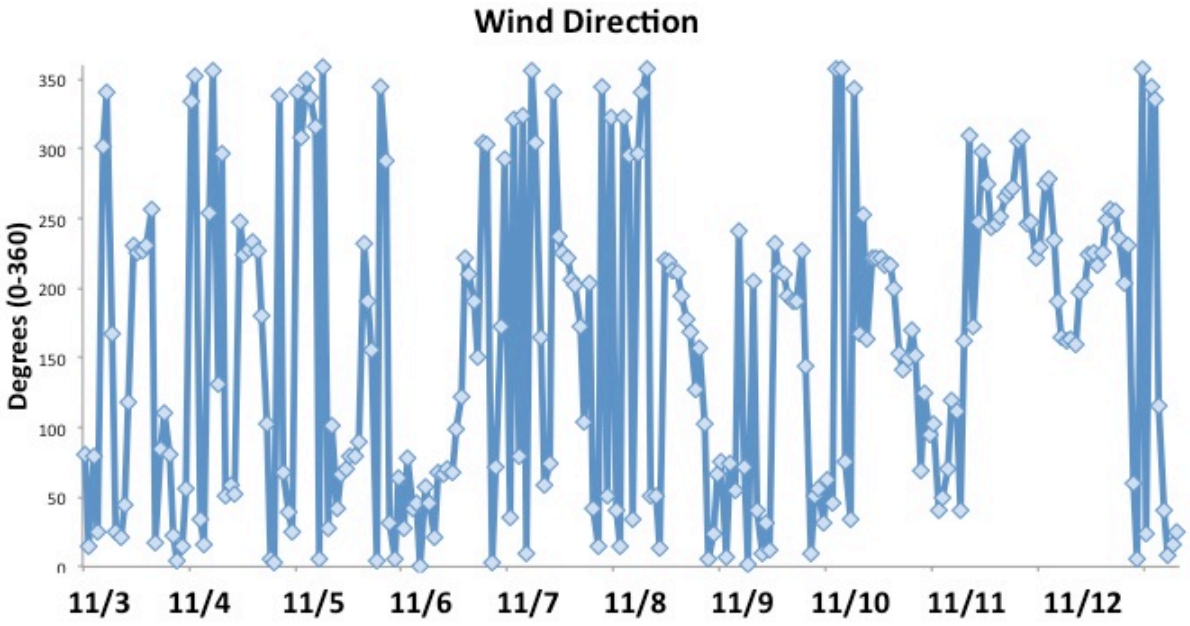


Figure A-2: Wind direction for Watsonville, CA, November 3-12, 2014.

Appendix 2: Interpreting Air Monitoring Results

Interpreting air monitoring results requires understanding of how regulatory authorities like the 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 increase, 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;[74] 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), 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, 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.[75]

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 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 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 EPA uses in its dietary assessments as a Level of Concern (LOC). To minimize exposure risk, 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, EPA would take action to reduce risk. Unfortunately, there are regulatory gaps for inhalation exposure—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 EPA in its toxicological analysis.[76] Additionally, as in this study or in past studies, 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.

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.[41] 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. PAN's Drift Catcher provides 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.

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.[23] 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 EPA in 2009.[46] Chloropicrin was listed by DPR as a Toxic Air Contaminant in February 2010.[71] It is not registered for use in Europe[77] or 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.[23] 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,[78] more chloropicrin is now being used in fumigations, with products now containing concentrations of chloropicrin ranging from 2% to 99%. Figure A-3 shows the increasing use of both 1,3-dichloropropene (Telone) and chloropicrin over the last several 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.[79] The counties with the highest use are Monterey, Ventura, Santa Barbara and Santa Cruz counties. Most fumigations occur during the months of September and October, for preplant applications. California's Department of Public Health conducted a 2014 survey of pesticide use near schools in 15 agricultural counties. Chloropicrin ranked as the top pesticide active ingredient in terms of pounds applied within ¼ mile of schools. In the same report, chloropicrin was also identified as a priority pesticide for assessment and monitoring. [80]

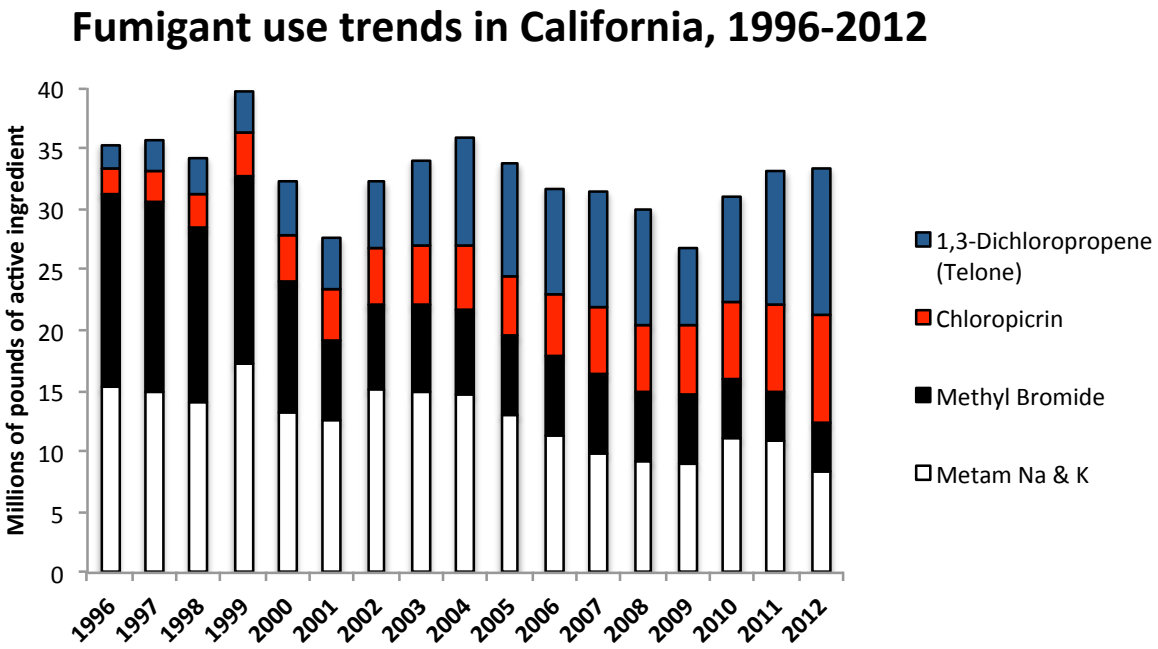


Figure A-3. Use of fumigants over time has remained relatively constant over the last several years in California, but the mix of different fumigants has changed substantially over the period, with increasing use of Telone and chloropicrin. Use includes both agricultural and reportable non-agricultural applications. Production agriculture constitutes the major category of use subject to reporting in California.

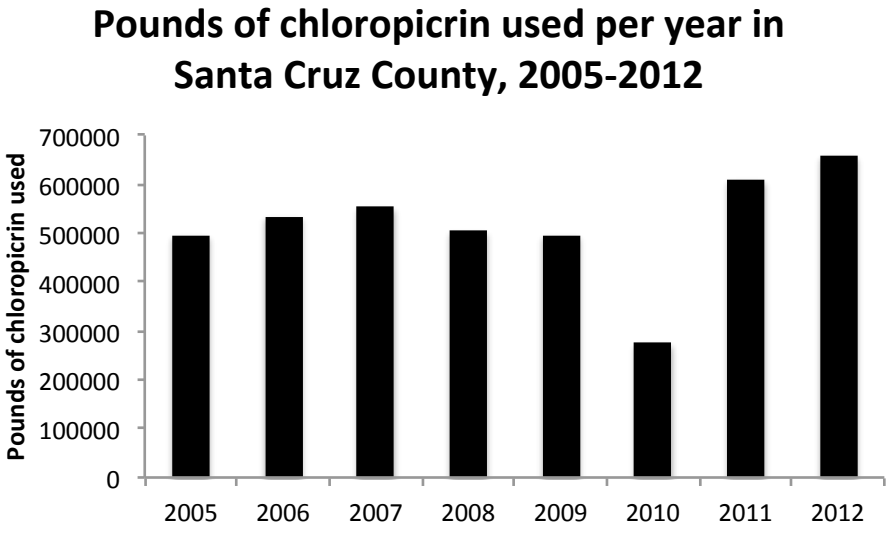


Figure A-4. Chloropicrin use trends in Santa Cruz County, California. Data are from California DPR's pesticide use reporting.

In 2004, EPA indicated that 5–9 million pounds of chloropicrin were used per year, making it the 18th most commonly used pesticide nationwide.[81] In 2007, EPA reported that 9-11 million pounds of chloropicrin were used, making it the 9th most commonly used pesticide in the country.[82] Use patterns are changing quickly, and in 2012, over nine million pounds of chloropicrin were used in California alone. 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.

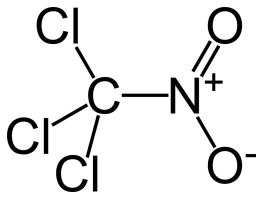


Table A-1: Physical Properties of Chloropicrin

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 21.

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: _____

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

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				

Pesticide Action Network, 49 Powell Street, Suite 500, San Francisco, CA 94102, (415) 981-1771

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