FINAL REPORT

Cumulative Risk Assessment of local produce exposed to urban wildfire smoke

Produce Safety after Urban Wildfire

Citizen Science Initiative

UC Cooperative Extension Sonoma

University of California
Agriculture and Natural Resources

Bay Area Air Quality Management District

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EXECUTIVE SUMMARY

Following the 2017 Northern California fires, we analyzed 3 food production sites across Sonoma County that were impacted by wildfire smoke. Sites selected for analysis were located at varying distances from the urban burn areas in Santa Rosa. None were adjacent to burned structures. We tested washed and unwashed samples of kale leaves, collected by volunteers during the fire, as well as soil samples collected by volunteers in June of 2018.

Plant sample results support the hypothesis that there is low concern of health impacts from ingesting produce exposed to the Santa Rosa urban wildfire smoke of 2017. Our cumulative risk assessment further suggests that the cancer risk reduction due to the nutritional value of produce outweighs the maximum possible risk from ingesting wildfire-related contaminants on produce.

Soil results, however, suggest that more analysis is needed, particularly on dioxins in Santa Rosa soils. Our report provides additional information on best practices for further reducing risk and enhancing protective factors.

- Produce Summary: ***low concern***
  - Produce samples did not have any detectable Polycyclic Aromatic Hydrocarbons (PAHs), or Polychlorinated Biphenyls (PCBs).
  - Produce did not have any detectable Dioxins and Furans in 12 out of 13 samples. The sample with detectable levels was still at a concentration below California's "No Significant Risk Level" threshold for determining chemical safety under Proposition 65.
  - Produce did not have any detectable Proposition 65-regulated heavy metals in 12 out of 13 samples. One sample of unwashed produce contained nickel at levels that exceed the Prop 65 NSRL. No samples had detectable levels of lead, arsenic, mercury, or chromium.
  - Differences between washed and unwashed were not evaluated due to low sample size.

- Soil Summary: ***low concern overall, but more soil dioxin testing needed in Santa Rosa***
  - The site closest to the Santa Rosa fires had the highest levels of dioxins and furans, at levels that exceed EPA and OEHHA soil screening levels. We are unable to confirm whether these contaminants were present before the fire or are a result of the fire.
  - Heavy metal soil concentrations were below Sonoma County's post-fire clean-up goals.
  - Soil samples did not have any detectable Polychlorinated Biphenyls (PCBs).

- General Notes: ***the need for a balanced approach in assessing risk***
  - *Over long periods of time*, exposure to these chemical groups at very low levels can still contribute to health impacts, including at *levels below what our tests are able to detect*.
  - *Numerous health benefits including cancer risk reduction* have been attributed to green leafy vegetables. In this study, these benefits were found to outweigh the risk.
  - *Some individuals have higher risks* and should talk with their healthcare provider to better understand if they should take extra precautions. Individuals at higher risk may also benefit greatly from the high nutrition in green leafy vegetables and fresh produce.
  - *Best practices for reducing risk include*: wearing a respirator mask; washing produce thoroughly in running water; peeling root vegetables, testing soil regularly; containing and amending contaminated soil through sheet mulching, raised beds, and compost.
  - *Best practices that enhance protective factors* should also be pursued, such as increasing produce consumption to improve nutrition and promote resilience to chemical exposures.

Background & Methods

STUDY BACKGROUND

Urban Wildfire and Potential Contamination

The fires that spread through Northern California in October 2017 burned over 160,000 acres of wildland, suburban, urban and industrial areas, creating dangerous air quality conditions for the region that lasted long beyond the fires themselves. The wildfire smoke likely included high concentrations of toxic air contaminants.[1] Following the fires, the Food and Drug Administration wrote a letter to the California Department of Food and Agriculture and the California Department of Public Health, stating that "toxic elements, firefighting chemicals, and combustion products such as polycyclic aromatic hydrocarbons (PAHs) and dioxins are of greatest concern."[2] There are well-known human health impacts from the *inhalation* of these contaminants.[3] Additionally, plants have the potential to absorb air pollutants directly through their leaves,[4, 5, 6, 7] but little research has been done on the risk to human health from *ingesting* contaminants from smoke and ash on produce grown near a wildfire.

Impact on Local Farms and Gardens

Local farms and gardens played a significant role in food relief efforts immediately following the fires, contributing produce to shelters and kitchens. Many farmers, gardeners, and community members have been concerned about how the fire-related air pollution might impact locally-grown produce. Farmers have been unsure of the potential health impacts of the fire on themselves, their workers, and their consumers. School, community, and home gardeners have been concerned about the potential health impact on children and other vulnerable groups.

Preliminary results from UC Davis' 2018 survey of Sonoma County residents shows that a quarter of respondents (>2000) reported concerns about the safety of locally-grown produce.[8]

Citizen Science Initiative

In the weeks following the Sonoma County fires, concerned community members came together to launch the Produce Safety after Urban Wildfire Citizen Science Initiative. Sonoma County residents and members of the UC Master Gardener Program of Sonoma County collaborated to take samples from over 25 sites across the region using a sampling protocol created under advisement by University of California specialists in Environmental Health and Food Safety. Samples included washed and unwashed produce, each in triplicate, to determine if contaminants are present and whether contaminants can be easily washed off produce. Volunteers focused on leafy greens with large surface area directly exposed to air pollution: kale, collards, chard, and lettuce. In total, over 200 samples were taken and frozen for subsequent laboratory analysis.

In the months following the fire, soil contamination became a greater concern for the community. Community-led soil sampling was initiated in June 2018 using a protocol developed in collaboration with UC Berkeley graduate students. Three sites at various distances from the urban wildfire perimeter were analyzed to test for persistent chemicals in the soil.

POTENTIAL HAZARDS FROM SMOKE

The short and long-term health impacts of smoke inhalation have been well documented over the past century. Wildfire smoke dramatically increases air pollution levels, with immediate health impacts from acute exposures. A 2015 literature review of over twenty years of wildfire health research indicates that particulate matter levels may increase by up to ten times higher during wildfires, and acute exposure to wildfire smoke is associated with respiratory disease, cardiovascular disease, and mortality.[9] Preliminary results from UC Davis' survey show that cardio-respiratory issues were common during and following the fires.[10]

In the case of an urban wildfire, there is the potential for this smoke to carry toxic chemicals in the products and building materials of the built environments that burned, which has been a major theme of concern among Sonoma County residents.[11] Based on a total of 15 ash samples from 3 residential sites and 1 state park that burned in the 2017 Tubbs fire, researchers from UC Davis are currently finding that there are upwards of two thousand chemicals in residential ash that are not present in ash from the state park. However, the study's current findings do not necessarily indicate that the residential ash is more toxic compared to ash from the state park, when tested in bioassays of dioxin-like biological response and endocrine disruption.[12]

We narrowed down our evaluation of produce to the following four chemical groups, based on FDA guidance,[13] likelihood to appear in urban wildfire smoke, and likelihood of health impacts:

Polycyclic Aromatic Hydrocarbons (PAHs)[14] are a class of very small carcinogenic chemicals that are produced from the combustion of organic materials. Traffic-related air pollution is a common source. They also enter the diet through grilling, drying, and smoking foods. It is possible PAHs may have *acute* toxicity to humans, but most studies and health guidance documents have focused on long-term effects, particularly cancer, and reproductive effects on brain development and immune system.

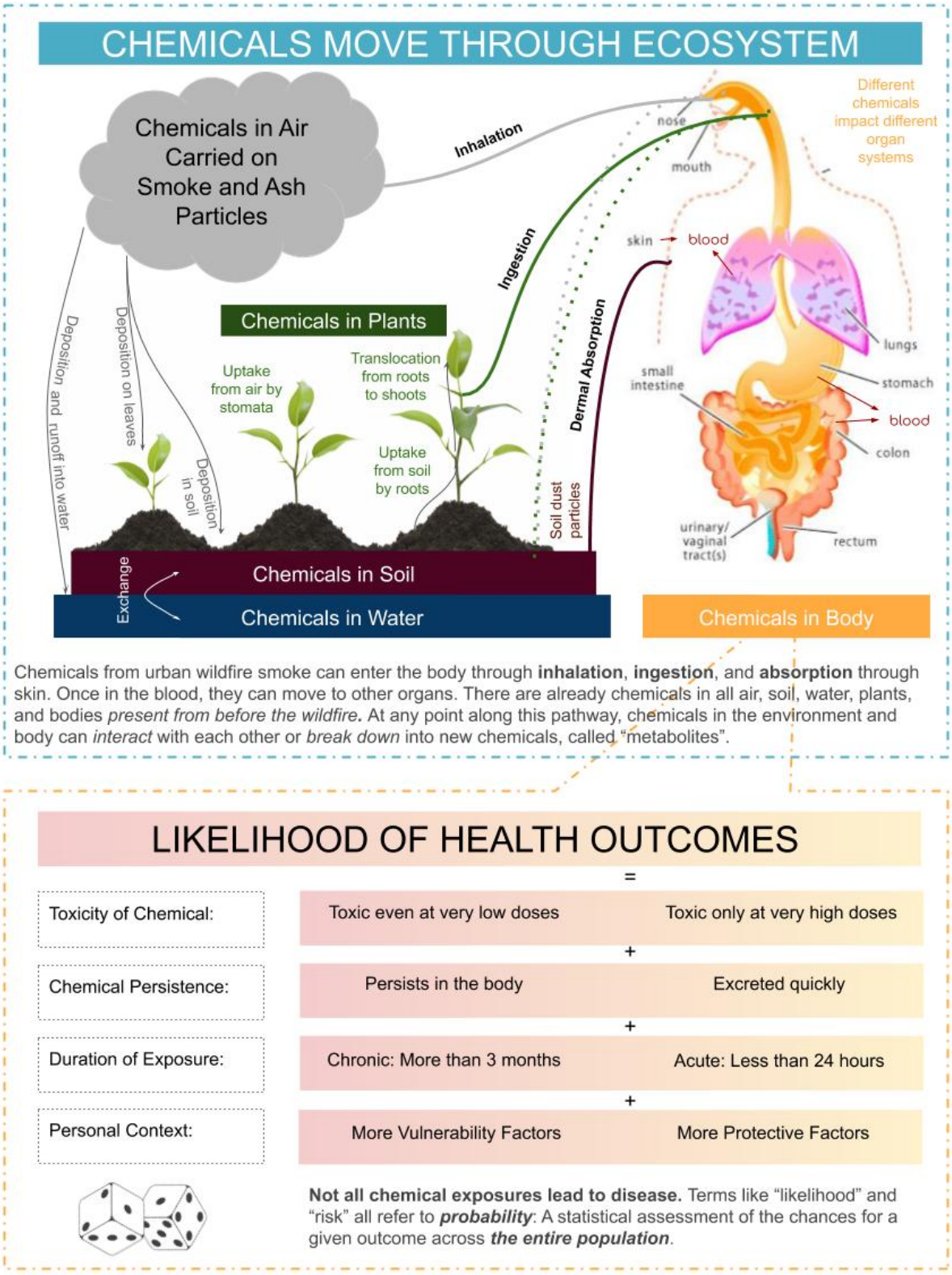
Heavy Metals [15] can exist naturally in soil but can also be emitted in toxic levels from industrial activities. During an urban fire, they could be present in smoke from burning buildings and cars. They are persistent in the environment. Some are critical nutrients, like iron for red blood cell function. Others, like lead, arsenic, and mercury are toxic to many organ systems, carcinogenic, and cause developmental effects on fetuses and children.

Polychlorinated Biphenyls (PCBs) [16] are a group of synthetic organic chemicals that persist in the environment and bioaccumulate. They were produced and widely used as coolants and lubricants in electronic devices, insulating materials, and plastic casings until 1977 when they were banned. PCBs can be released into the environment by combustion and travel long distances in air. They are associated with a wide-range of health impacts, including hormone disruption and liver cancer.

Dioxins & Furans [17] are persistent organic pollutants. They are created through the combustion of plastic products and can travel long distances through air pollution. They bind to fats and will accumulate up the food-chain, including breast milk. Toxic effects include cancer, immune toxicity, developmental, and hormonal effects. Children and breastfeeding infants are more at risk for long-term health impacts.

SCHEMATIC MODEL OF HAZARD EXPOSURE AND RISKS

Figure 1: Schematic Model of Hazard Exposure and Risks



METHODS FOR DETERMINING RISK

Foliar Samples: Proposition 65

In order to determine whether levels of contaminants on produce were "safe", we compared our laboratory results to the "No Significant Risk Level" (NSRL) established by California's Office of Environmental Health Hazard Assessments (OEHHA) under Proposition 65.[18]

Proposition 65 is officially known as the "Safe Drinking Water and Toxic Enforcement Act of 1986". It was enacted as a ballot initiative to protect drinking water and to inform consumers about exposures to chemicals in consumer products shown to cause cancer, reproductive harm, and neurological impacts in products for sale in California. Under the law, businesses selling products containing these chemicals at levels that pose significant risk must inform customers with a Proposition 65 warning on the package.

"No Significant Risk Level" (NSRL)
According to the OEHHA website, Proposition 65 *"defines "no significant risk" as a level of exposure that would cause no more than 1 extra case of cancer in 100,000 people over a 70-year lifetime. So a compound can be unlabeled if a person exposed to the substance at the expected level for 70 years is estimated to have a 1 in 100,000 chance or less of getting cancer due to that exposure. The law also has similar strict cutoff levels for birth defects and reproductive harm."*[18]

Soil Samples: Soil Screening Levels

We used soil screening levels from the Environmental Protection Agency (EPA) and OEHHA.

We used the EPA's Regional Soil Screening tables, and selected the Resident Soil level with a target hazard quotient of 1.[19] We used OEHHA's California Human Health Screening Levels (CHHSLs) table and selected the Residential Scenario values.[20]

"Target Cancer Risk"
Both OEHHA and the EPA calculate their soil screening levels based on a target cancer risk of 1 extra case of cancer in a million people, making it even stricter than the Proposition 65 levels. As with Prop 65, these soil standards are calculated for cancer risk over a 70-year lifetime of exposure.

For heavy metals, we also compared our laboratory results to the Sonoma County Complex Fire Cleanup Goals set by the Sonoma County Department of Health Services Public Health Division.[21]

These methods provide an overestimate of risk

Cumulative Risk Assessment

In addition to examining each chemical in each route of exposure, our study uses a cumulative approach to examine the total set of exposures that could impact health, including an assessment of chemical mixtures, a risk-benefit analysis of ingesting psmoke-exposed roduce (as commonly recommended in the EU, including by European Food Safety Authority[22] and the European Commision-funded Benefit Risk Assessment for Food study[23]), and a literature review of social determinants of health considerations in wildfire health impacts. Our mixed-methods analysis evaluates health hazards and protective factors. Our conclusions draw from the synthesis of these traditional risk assessment and holistic methods.

Limitations

There are a number of significant limitations to our methods. These principally include: a very low sample number (3 sites and 2 preliminary sites), lack of an adequate control sample for comparison, and a risk assessment approach that assumes chronic exposures.

Due to low sample size, statistical tests of significance were not attempted so as not to assume generalizability. These results offer some initial descriptive information that could inform future studies with larger sample sizes.

The risk assessment methods that we have been using in this preliminary report ***assume a 70-year lifetime of exposure at the daily intake rate.*** Consuming local produce following an urban wildfire likely results in an acute or sub-chronic exposure due to the wildfire incident.[24] The increasing frequency of wildfires means that a person may experience recurring acute or subacute exposures from ingesting produce over their lifetime. Exposure to soil may be considered at chronic levels, as many of the chemicals in this study are persistent in the environment. However, it is unclear if recurring urban wildfire events would constitute a "chronic" exposure. ***Due to this method limitation, we suggest that results be interpreted as overestimates of risk.***

"Acute, Chronic & Subchronic"

The length of the exposure can make a significant difference in whether or not an exposure has health consequences.

Acute *= exposure for under 24 hours*
Subchronic *= repeated exposure for more than 30 days, up to 10% of the lifespan*
Chronic *= repeated exposure for more than 10% of the life span in humans (90 days to 2 years is typically used in lab animal studies)*

Finally, it is critical to note that this study examined produce and soil exposed to dispersed contaminants in *smoke* and does not address the question of contamination on food produced directly adjacent to burned structures.

Despite these significant limitations, this study provides an initial descriptive analysis of food production sites exposed to urban wildfire smoke, and gives important insights into the under-investigated field of post-wildfire food safety for further study.

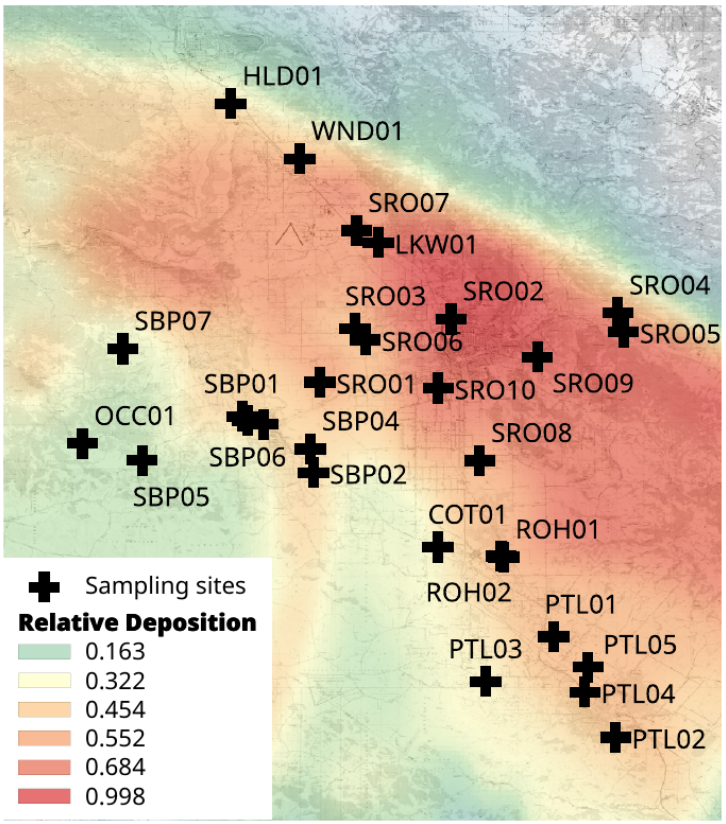
These methods provide an overestimate of risk

PRELIMINARY ANALYSIS & STUDY HYPOTHESIS

Site Selection

Our preliminary analysis tested samples from two high-priority sites that were most likely to have received deposits of toxic chemical from combustion of residential and urban structures. We created a meteorological model of particulate matter deposition from the urban burn area in Santa Rosa. The HYSPLIT model returned results in the form of geospatial polygons with discrete levels of deposition (integers). These results were post-processed using Gaussian kernel smoothing to arrive at our final model. We used this model to choose sites that were most likely to have chemicals from the smoke settle on their crops.

Figure 2: Deposition Model



Samples, Tests, and Labs

We provided two varieties of leafy greens (kale, lettuce) from the two sites to TestAmerica in Sacramento for analysis for PAHs, CAM17 metals, and dioxins and furans. We then sent another set of samples from the same two high priority sites to Enthalpy Analytics in Berkeley to help validate our first results. With this second lab, we tested for PAHs in chard samples from both sites, and we tested for dioxins using collards from one site

Based on these preliminary findings, we hypothesized that produce safety was not significantly affected by the fires and that heavy metal deposits may be mitigated by washing produce.

Preliminary results did not indicate a high degree of contamination.

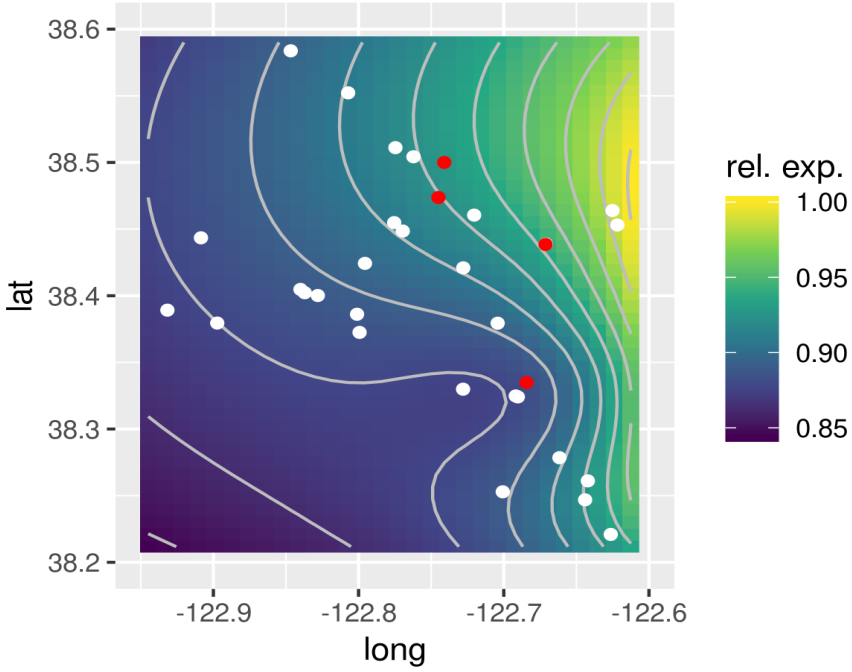
- **Polycyclic Aromatic Hydrocarbons = *inconclusive;*** Due to high method reporting limits from our laboratories
- **Heavy metals = *low concern, except for Nickel*;** No detection of lead, arsenic, or mercury. Nickel was found in 2 of 8 samples at levels exceeding Prop 65's No Significant Risk Level (NSRL). Nickel contamination appears to be mitigated by washing produce.
- **Dioxins = *some concern*;** Concentrations found above the background levels from FDA's Dioxin Monitoring Program, but at levels below NSRL.

See Produce Safety After Urban Wildfire's June 2018 Report for details on preliminary data.

HYPOTHESIS TESTING

Site Selection

Figure 3: Air Quality Data from CARB Monitors



We selected three sites for additional testing based on three variables: distance from urban burn area, ranking on meteorological deposition model used in preliminary analysis, and ranking on particulate matter levels during fire. For this third variable, we analyzed Sonoma County air quality sensor data collected during October 2017, provided by California Air Resource Board (CARB). Averages were calculated between Oct 8 and Oct 20 (capturing most peaks on sensor measurements with relatively uniform sensor coverage) from four air pollution monitors in Sonoma County. Two monitors in Sonoma County were removed from analysis due to anomalous low levels indicating possible calibration issues. Initially, the air quality data and deposition models were inversely proportional. Removing the anomalous monitors improved correlation of the two data sets. In making our decision on which sites to analyze, we used both the air quality data and the deposition model, with the assumption that the air quality data provides information on total wildfire smoke exposure at a site, whereas the deposition model provides insight into the likelihood that a site had contaminants from the urban part of the burn.

Samples, Tests, and Labs

For these three sites, we sent washed and unwashed kale samples to Enthalpy Analytical to be tested for Polychlorinated Biphenyls, Dioxins and Furans, and Heavy Metals.

We selected an additional, fourth, site to send in washed and unwashed kale samples to test for Polycyclic Aromatic Hydrocarbons. This site was selected as a high ranking site for multiple of the above site selection variables. We also selected it due to the larger sample mass available, to increase the chances of a lower detection level and thus a higher resolution of analysis for PAHs.

SUMMARY OF SAMPLES SELECTED FOR ANALYSIS

Figure 4: Samples Selected for Analysis

	Final Report Hypothesis Testing				Preliminary Sites	
Location:	Santa Rosa	Santa Rosa	Rohnert Park	Petaluma	Larkfield	Santa Rosa
Site ID:	SRO10	SRO02	ROH02	PTL04	LWK01	SRO09
Media Tested:	Plant	Plant & Soil	Plant & Soil	Plant & Soil	Plant (Soil samples available)	Plant (Soil samples available)
Tested for:	PAHs	PCBs Dioxins & Furans Heavy Metals	PCBs Dioxins & Furans Heavy Metals	PCBs Dioxins & Furans Heavy Metals	Dioxins & Furans Heavy Metals	Dioxins & Furans Heavy Metals
Average PM concentration (ug/m^3)	41.58	42.52	40.21	42.16	42.15	43.07
Peak PM concentration (ug/m^3)	187.70	186.01	195.13	193.56	174.60	197.92
Ranking* on PM 2.5 concentration	11	5	25	6	7	3
Ranking* on deposition model	4	1	12	17	5	2
Ranking* on distance from urban burn perimeter	9	1	15	24	2	7

* Rankings are based on the 25 sites sampled by volunteers during the 2017 wildfires

Laboratory Results & Risk Assessment

RESULTS: PLANT TISSUE

Polychlorinated Biphenyls (PCBs) – Plant Tissue

Interpretation: There were no PCBs detected in the plant samples from any site. However, due to the high reporting limit from standard laboratory methods, we are not able to confirm whether or not sites had PCBs at levels below our reporting limit that still exceed the Proposition 65 NSRL.

Figure 5 shows how Proposition 65's "No Significant Risk Level" (NSRL) compares to the "reporting limit" (RL) from our lab, which is the lowest level that our tests are able to detect.

A value is given for the lowest PCB reporting limit (min RL) and for the highest PCB reporting limit (max RL) across all sites.

Key: ND= "Non-Detect"; RL= "Reporting Limit"

"Reporting Limit"
A method reporting limit (MRL) is the lowest concentration of a chemical that a lab test would be able to detect in a sample. This is also sometimes refered to as the Detection Limit (DL), Limit of Detection (LOD), or Estimated Detection Limit (EDL) depending on the test.

Figure 5: Proposition 65 Comparisons for PCBs (ug/day)

	NSRL	Daily Intake from Levels Found at Sites ND=0	Daily Intake at Reporting Limit ND=5.6 **(min RL)**	Daily Intake at Reporting Limit ND=82 **(max RL)**
PCB Intake Rate (ug/day)	0.09	0	0.117	1.722

E.g. Concentrations above 0.09 ug/day but below 0.117 ug/day would exceed Prop 65 NSRL levels, but would not be detected by our test.

Polycyclic Aromatic Hydrocarbons (PAHs) – Plant Tissue

Interpretation: There were no PAHs detected in the plant samples from any site. However, due to the high reporting limit from standard laboratory methods, we are not able to confirm whether any sites had PAHs at levels below our reporting limit that still exceed the Proposition 65 NSRL.

Figure 6 shows how the Proposition 65's "No Significant Risk Level" (NSRL) for several different PAHs compare to the "method reporting limit" (MRL) from our lab, which is the lowest level that our tests are able to detect. The MRL listed in figure 6 is an average across all samples of washed and unwashed produce taken from all sites.

Figure 6: Proposition 65 comparisons for PAHs (ug/day)

	NSRL for Benzo(a) pyrene	Daily Intake from Levels Found at Sites ND=0	Daily Intake at Reporting Limit ND=178.33 **(average RL)**
PAH Intake Rate (ug/day)	0.06	0	0.749

For Example: Daily Intake of benzo(a)pyrene above 0.06 ug/day but below 0.749 ug/day would exceed Prop 65 NSRL levels, but would not be detected by our test.

Dioxins & Furans – Plant Tissue

Interpretation: Some dioxins or furans were detected in 10 out of 19 samples from tested sites, but all concentrations detected were below Prop 65 NSRL. Additionally, no samples had detectable levels of the higher-toxicity dioxins that are of greatest concern for public health. However, we cannot completely confirm that sites did not have dioxins & furans above the Proposition 65 NSRL due to the high detection limit from standard laboratory methods.

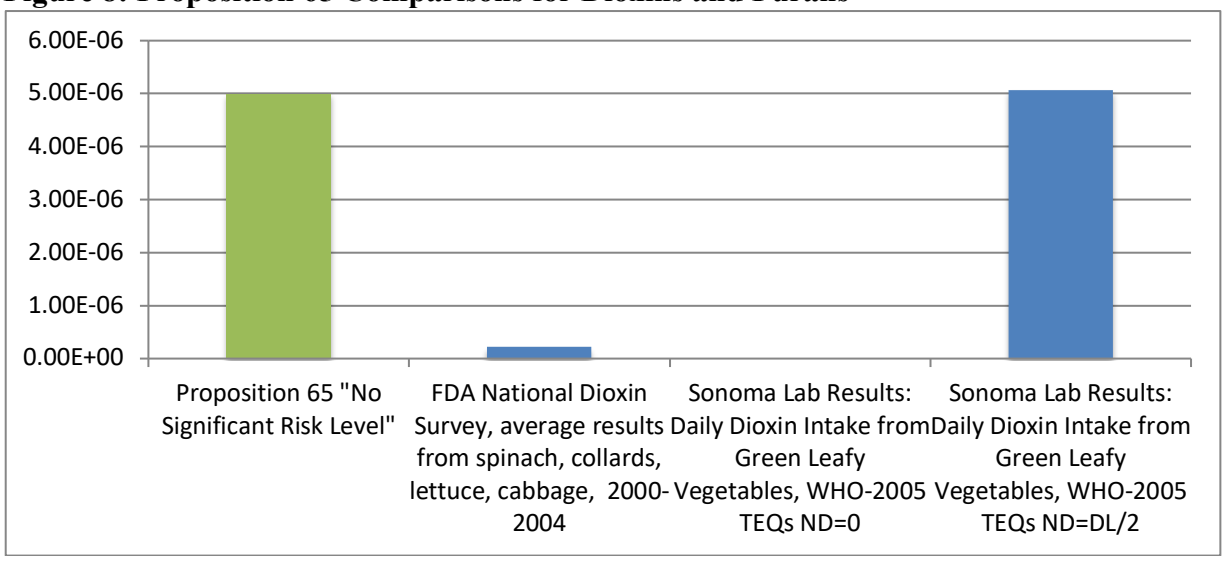
Figures 7 and 8 show how Proposition 65's "No Significant Risk Level" (NSRL) compares to the "detection limit" (DL) from our lab, which is the lowest level that our tests are able to detect. It is a common convention in scientific studies to use half of the detection limit rather than "0" for non-detect results (ND=DL/2). ND=DL/2 results are equivalent to the Prop 65 NSRL. Key: ND= "Non-Detect"; DL= "Detection Limit"

Figure 7: Proposition 65 Comparisons for Dioxins and Furans

	NSRL	Background*	Daily Intake from Levels Found at Sites ND=0	Daily Intake at Half of Reporting Limit ND=RL/2
Dioxins & Furans Intake Rate WHO-2005 TEQs (ug/day)	5×10^{-6}	2.23×10^{-7}	0	5.07×10^{-6}

* FDA National Dioxin Survey, average results from spinach, collards, lettuce, cabbage, 2000-2004

Figure 8: Proposition 65 Comparisons for Dioxins and Furans



Heavy Metals – Plant Tissue

Interpretation: Some heavy metals were detected. There were no detections of the heavy metals of greatest concern to public health, including Lead, Arsenic, Mercury, and Chromium.

Nickel was detected on one sample out of nineteen samples tested, and only on an unwashed sample. Consuming this concentration of Nickel daily would lead to consumption rates above Proposition 65's "No Significant Risk Level". However, this NSRL was established based on the toxicity of nickel refinery dust from the pyrometallurgical process, which may not accurately represent the toxicity of the nickel found in our samples. Levels found in this analysis may reflect nutritionally-beneficial nickel levels within the range of typical consumption.

Recommendations: Wash produce in running water.

Figure 9 summarizes the laboratory results of plant tissue sampled from tested sites. All sites were tested for the complete panel of CAM17 heavy metals. Figure 9 shows only the metals detected during testing. There were no detections of the heavy metals of greatest concern to public health, including Lead, Arsenic, Mercury, and Chromium.

Figure 9: Heavy Metals Concentrations ND=0 (mg/kg)

Site	SRO02		ROH2		PLT04	
Wash Condition	Washed	Unwashed	Washed	Unwashed	Washed	Unwashed
	AVG*	AVG	AVG	AVG	AVG	AVG
Barium	7.333	6.733	2.7	2.566	3.925	6.8
Copper	0.447	0.81	0.757	0.87	0.71	0.55
Molybdenum	0.657	0.747	0.49	0.477	0.697	0.657
Nickel	0	0.443	0	0	0	0
Thallium	0	0	0.817	1.09	0	0
Zinc	6.5	11.1	5	5.567	8.55	12.367

*This table shows averages of the triplicate samples taken for each site and wash condition

Heavy Metals – Plant Tissue (Page 2 of 2)

Figure 10 calculates an average nickel concentration for the one site where nickel was detected on plant tissue. Instead of using 0 for non-detections, the table shows estimated concentrations in grey. These were created by halving the detection limit for each sample (ND=DL/2).

Figure 10: Heavy Metals Concentrations ND=DL/2 (mg/kg)

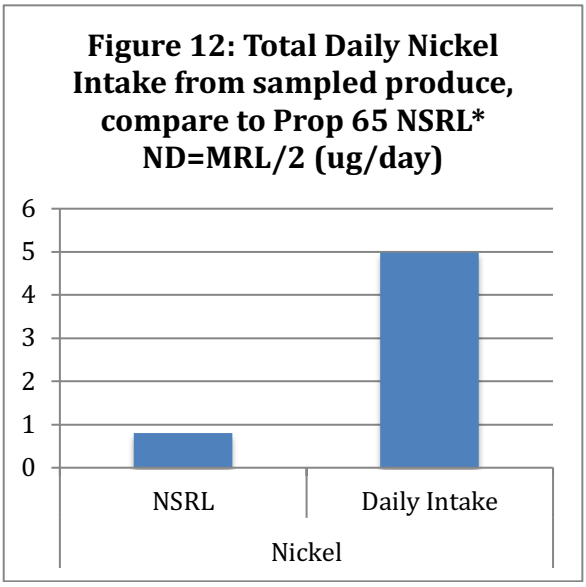
Site	SRO02						
Wash Condition	Washed			Unwashed			
Replicate	1	2	3	1	2	3	AVG
Nickel	0.16*	0.2	0.195	**0.41****	0.215	0.245	0.237

*Numbers in grey represent DL/2 **Lab measurements

Figures 11 and 12 compares the Proposition 65 "No Significant Risk Level" to the total daily Nickel intake that would occur if a person were to eat 21 grams of green leafy vegetables per day, with all produce containing **0.237 mg/kg** of Nickel.

Figure 11: Proposition 65 Comparison for Nickel

	Nickel	
	NSRL*	Daily Intake
Total Daily Nickel Intake ND=DL/2 (ug/day)	0.8	4.9875



*** NOTE REGARDING NICKEL INGESTION:**
This NSRL is based on studies of the toxicity of nickel refinery dust from the pyrometallurgical process,[25] which may not accurately represent the toxicity of the nickel found in our samples. According to the EPA's Hazard Summary, Nickel toxicity varies by compound, with soluble compounds (such as nickel acetate) being the most toxic, and the insoluble forms (such as nickel powder) being the least toxic.[26] More research is needed to determine the type of nickel compound released during the urban wildfire events, and whether they reflect a more toxic or less toxic profile.

EPA and HHS reviews of oral Nickel exposure highlight that food is the major source of nickel exposure, with average daily consumption in the range of 100 to 300 µg/d, which is 20 to 60 times higher than the concentrations detected in our study.[27, 28]

RESULTS: SOIL

Polychlorinated Biphenyls (PCBs) – Soil

Interpretation: There were no PCBs detected in the soil sample. The reporting limit from our laboratory methods are far below the EPA's PCB soil screening levels, so we can conclude that no PCBs are present above screening levels in our soil samples.

Figure 13 shows a comparison of the reporting limits from our laboratory methods with the EPA's screening levels for PCBs in soil.

Figure 13: Comparison of Reporting Limit to Screening Level (ug/Kg)

Site	EPA Screening Level	SRO02 Reporting Limit	ROH02 Reporting Limit	PTL04 Reporting Limit
Aroclor-1016	411	13	12	12
Aroclor-1221	2000	26	24	24
Aroclor-1232	1720	13	12	12
Aroclor-1242	2300	13	12	12
Aroclor-1248	2310	13	12	12
Aroclor-1254	176	13	12	12
Aroclor-1260	2400	13	12	12

Dioxins & Furans – Soil

Interpretation: At the Santa Rosa site closest to the urban burn perimeter, dioxins and furans were detected in soil at cumulative concentrations that exceed the EPA and OEHHA's Screening Levels. The Rohnert Park and Petaluma sites had detectable levels of some dioxins, but cumulative concentrations were below screening levels.

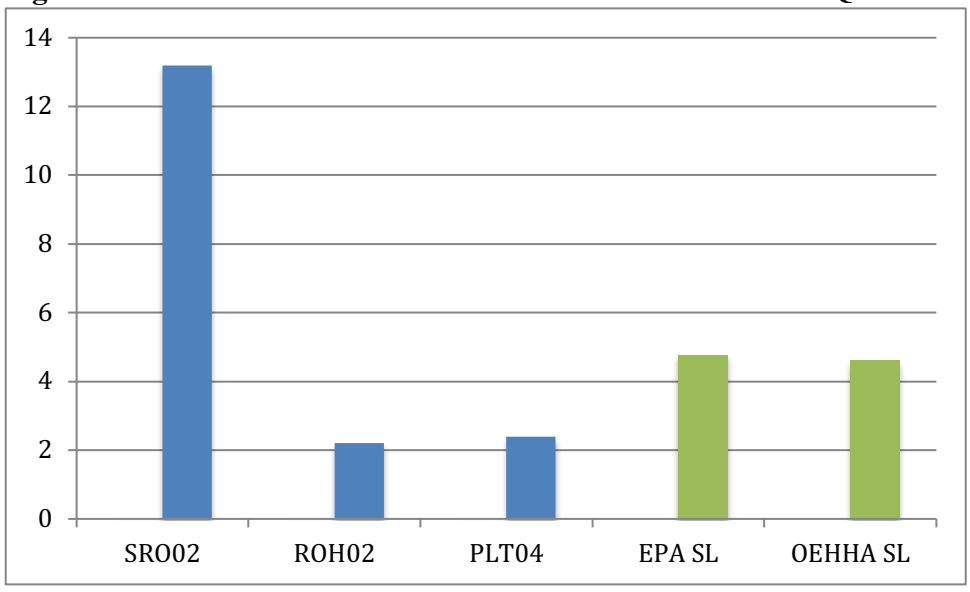
Recommendations: The main concern with soil dioxin contamination is from direct inhalation and ingestion of soil. Children are more likely to ingest soil. *Short-term*: Reduce direct contact with soil. Wash hands after working with soil. Wash produce thoroughly, and peel root vegetables. *Long-term*: Heavily amend soil with compost and mulch to dilute dioxins and build up. Use drip irrigation to reduce the up-splash of soil and dust. Re-test soil.

Figure 14 shows a ***cumulative dioxin concentration*** for the tested sites. These values are created by scaling the concentrations of each dioxin and furan detected by the relative toxicity of each dioxin or furan. This is known as the "Toxic Equivalency Factor" (TEQ). We used the TEQs proposed by the World Health Organization (WHO) in 2005. We compared this cumulative dioxin concentration to the soil dioxin screening level proposed by the EPA.
Key: ND= "Non-detect", DL= "Detection Limit", SL= "Screening Level"

Figure 14: Cumulative Dioxin Concentration WHO 2005 TEQ ND=DL/2 (pg/g)

Site	**SRO02**	**ROH02**	**PLT04**	EPA SL	OEHHA SL
Replicate	Average	Average	Average		
Soil Dioxin Concentration	13.2	2.213	2.387	4.77	4.6

Figure 15: Cumulative Dioxin Concentration WHO 2005 TEQ ND=DL/2 (pg/g)



Heavy Metals – Soil

Interpretation: All heavy metals were found at concentrations below Sonoma County Clean-Up Goals, and all metals except for arsenic were detected at levels below the EPA's Screening Levels. Arsenic was detected above EPA's Screening Level, but below background arsenic levels for Sonoma County soils.

Figure 16 compares the average heavy metals concentration from tested sites to regional background levels, federal (USA EPA) and state (CalEPA) soil screening levels, and to the Clean-Up Goals set by the Sonoma County Department of Health Services.

Figure 16: Heavy Metals comparison to standards and clean up goals ND=0 (mg/kg)

	SRO02	ROH02	PTL04	Sonoma County Complex Fire clean-up goals.			
	AVG	AVG	AVG	Background	USA EPA RSL	CAlEPA CHHSL	Clean-Up Goal
Arsenic	4.567	2.833	2.7	6.18	0.7	0.1	6.2
Barium	183.33	120	140	263.2	15000	5200	5200
Beryllium	0.49	0.577	0.303	2079	160	16	15
Cadmium	0.5233	0.26	0.43	0.184	71	1.7	1.7
Chromium	51.667	34	25	110	120000	100000	36000
Cobalt	10.667	12.67	8.5	29.2	23	660	29.2
Copper	40.667	26	18	40.35	3100	3000	3000
Lead	27.333	7.67	55.67	39.76	400	80	80
Mercury	0.17	0.088	0.062	3.19	5.1	18	5.1
Molybdenum	0.42	0.597	0.457	0.759	390	380	380
Nickel	67.333	47	16.33	102.7	1500	1600	490
Vanadium	35.333	41.667	22.667	120	390	530	390
Zinc	180	109.67	116.67	74.5	23000	23000	23000

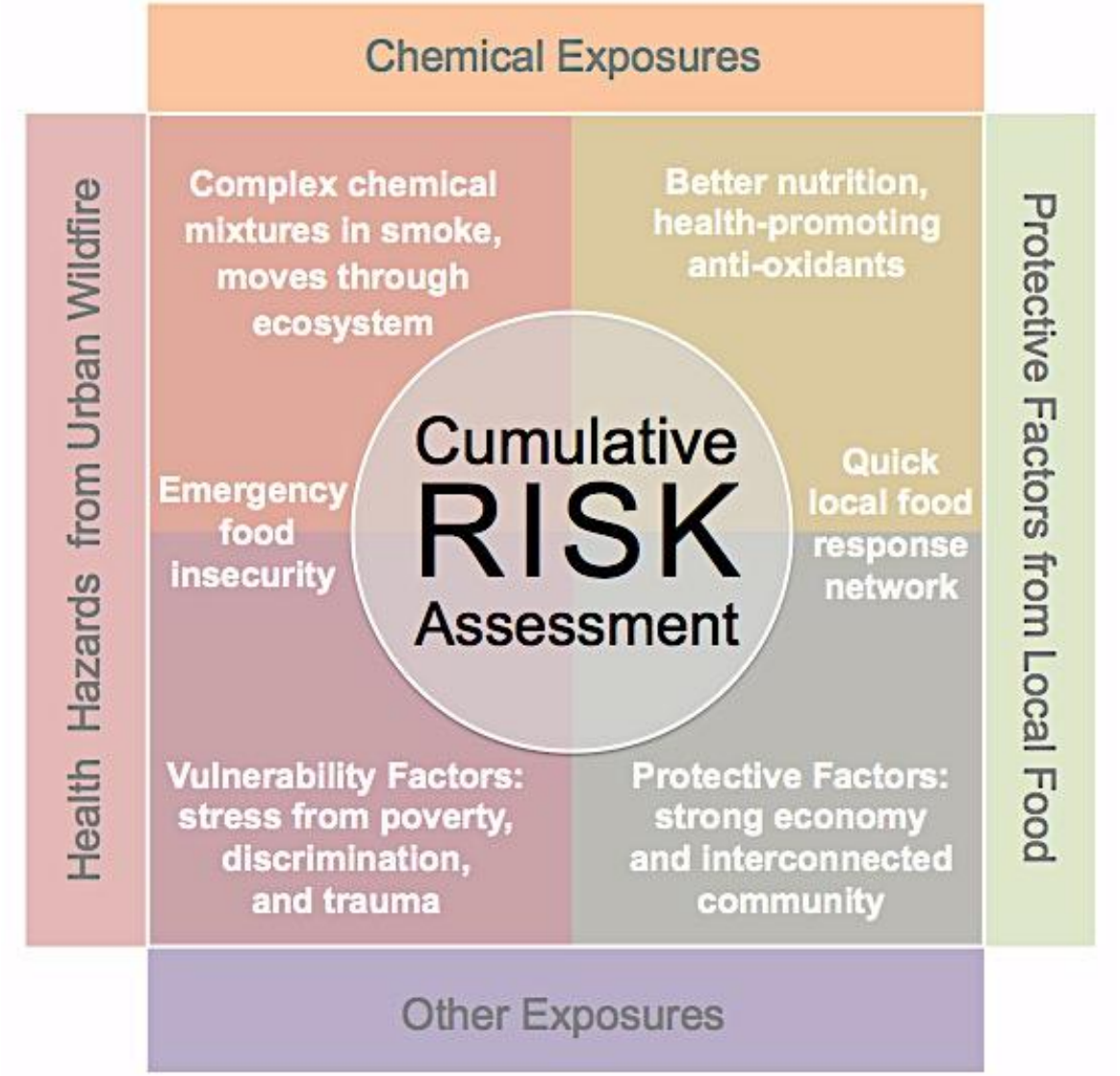
Cumulative Risk Assessment

CUMULATIVE RISK ASSESSMENT

Our cumulative assessment examines the total set of exposures that could impact health, including an assessment of chemical mixtures, a risk-benefit analysis of ingesting produce, and a literature review of social determinants of health considerations in wildfire health impacts.

We used the following model to guide our literature review. Wildfires hitting an urban area create innumerable ***health hazards*** for communities and the smoke from the fire can impact an even larger geographic area. We holistically evaluate the larger context of these health impacts, as well as the larger context of ***protective factors*** from local food, such as the health benefits of open green spaces and nutritious produce, and the socio-economic impacts of a strong local economy and interconnected community.

Figure 17: Schematic Model of the Cumulative Risk Assessment Framework used in this study



1. Evaluation of chemical mixtures from multiple media

This study examined multiple chemical groups that were likely to be present in smoke, and so an evaluation of the risk from mixtures is warranted. In 1996, the Safe Drinking Water Act required the EPA to create methods for the evaluation of mixtures of chemicals that are likely to co-occur in specific media, and since then multiple frameworks have been tested. [29, 30]

To establish cumulative risk values, we used OEHHA's Air Toxics Hotspots Exposure Assessment guidance documents.[31] As with the chemical-specific risks, this cumulative method assumes a 70-year life-time of exposure. It differs from our Proposition 65 analyses in that it calculates different risks for particular age groups, and then sums them across the 70-year life-time, which is particularly useful for behaviors such as soil ingestion, which is most likely to occur in children between 0-2 years of age.

a. Ingestion of contamination on plant tissue

We used the formulas and suggested intake levels described in the Air Toxics Hotspots Exposure Assessment Chapter 7: "Home Produced Food Exposure Assessment".

We created three scenarios in our calculations: "Maximum Possible Risk," which uses all reporting limit values for non-detected chemicals (ND=DL), "Maximum Probable Risk," which uses half the detection limit (ND=DL/2), and "Risk from Detected Levels," which examines only the risk from detected chemicals and counts all non-detects as zero (ND=0).

Figure 18: Risk from ingestion of produce in Sonoma County exposed to wildfire smoke

	MAXIMUM POSSIBLE RISK ND=DL	MAXIMUM PROBABLE RISK ND=DL/2	RISK FROM DETECTED LEVELS ND=0
Lifetime Cancer Risk*	0.00568	0.00288	1.282 E-09

*Lifetime Cancer Risk can be converted to an estimate of "Cancer cases per year" by multiplying by Sonoma County's population, divided by 70 to convert lifetime risk into an annual figure.[32] However, this should be considered a crude "upper bound" estimate. Due to the low confidence in such an estimate, the "Cancer Cases per Year" have not been calculated.

How to interpret the "Maximum Possible Risk" from produce:
If the entire population of Sonoma County ate only local produce every day for the rest of their lives, and that produce were contaminated at levels just below our ability to detect (ND=RL), it would lead to a life-time cancer risk of 0.00568.

This method provides an extremely high overestimate of risk.

Its utility is in understanding the worst-possible risk scenario given the high rate of non-detections in our analysis and the high detection limits for our PAH and PCB tests.

b. Ingestion of contamination in soil

Soil ingestion is the most common pathway of exposure to chemicals in soil. Skin absorption and inhalation of dust are secondary pathways that were not considered in this analysis. To create a risk of exposure through this media, we used the calculations described in the Air Toxics Hotspots Exposure Assessment Chapter 4: "Soil Ingestion".

We used the same three risk categories as we did for ingestion of plant tissue. There were fewer non-detects in our soil samples, eliminating the variation in risk between categories.

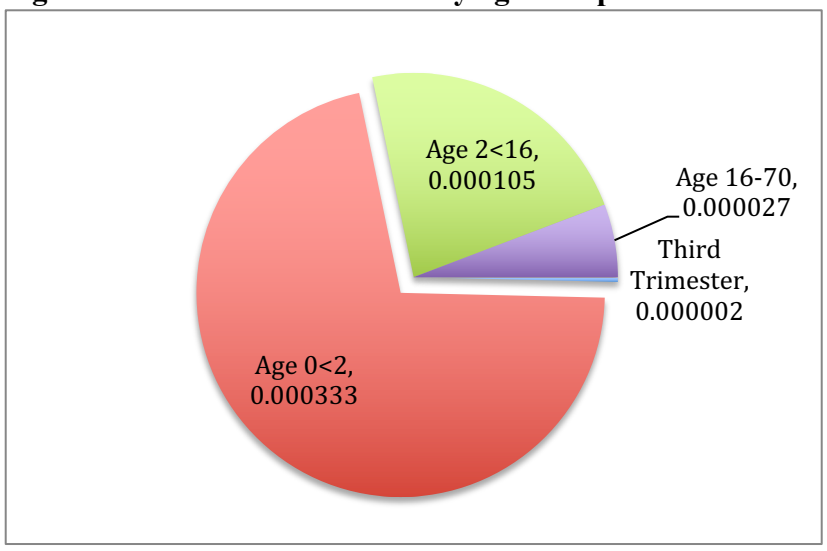
Figure 19: Risk from ingestion of soil in Sonoma County exposed to wildfire smoke

	MAXIMUM POSSIBLE ND=DL	MAXIMUM PROBABLE ND=DL/2	RISK FROM DETECTED LEVELS ND=0
Lifetime Cancer Risk*	0.000467	0.000467	0.000466

*Lifetime Cancer Risk can be converted to an estimate of "Cancer cases per year" by multiplying by Sonoma County's population, divided by 70 to convert lifetime risk into an annual figure.[33] However, this should be considered a crude "upper bound" estimate. Due to the low confidence in such an estimate, the "Cancer Cases per Year" have not been calculated.

How to interpret the "Maximum Possible Risk" from soil:
If the entire population of Sonoma County were exposed to contaminated soil every day of their life, and that soil were contaminated at levels just below our ability to detect (ND=RL), it would lead to a life-time cancer risk of 0.000467.

Figure 20: Lifetime cancer risk by age of exposure



The OEHHA Toxic Air Hot Spots method predicts that over two-thirds of this total lifetime cancer risk is attributable to exposures during 0-2 years of age.

This implies that risk reduction strategies focused on eliminating exposures for this age group would have maximum impacts on lifetime cancer risk.

This method provides an extremely high overestimate of risk.

Its utility is in understanding the worst-possible risk scenario given the high rate of non-detections in our analysis and the high detection limits for our PAH and PCB tests.

c. Combined ingestion of contamination in both produce and soil

We use an additive approach to approximate the combined health impact of ingestion of similar contaminants in produce and soil.

Figure 21: Risk from combined ingestion of produce and soil in Sonoma County

	MAXIMUM POSSIBLE RISK ND=DL	MAXIMUM PROBABLE RISK ND=DL/2	RISK FROM DETECTED LEVELS ND=0
Lifetime Cancer Risk*	0.00615	0.00335	0.000466

*Lifetime Cancer Risk can be converted to an estimate of "Cancer cases per year" by multiplying by Sonoma County's population, divided by 70 to convert lifetime risk into an annual figure.[34] However, this should be considered a crude "upper bound" estimate. Due to the low confidence in such an estimate, the "Cancer cases per year" have not been calculated.

Figure 22: Lifetime Cancer Risk from ingestion of contamination in produce and soil

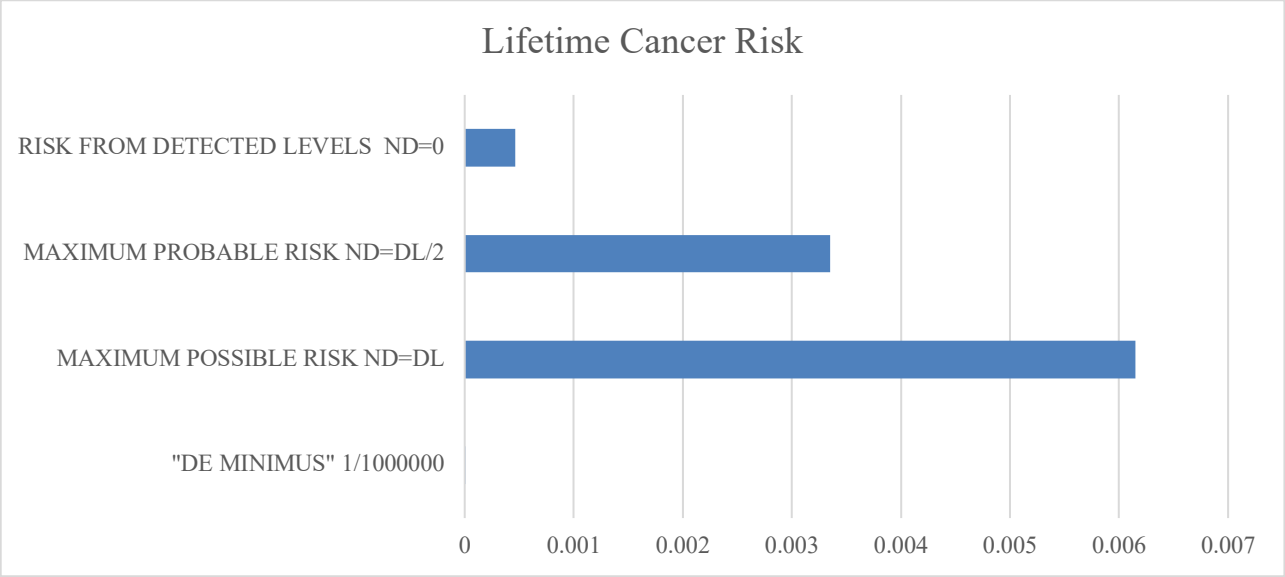


Figure 22 shows the combined risk from ingestion of contaminants in produce in soil, compared to the common "de minimus" threshold of one in a million for lifetime cancer risk. This analysis shows that the health risks from smoke contamination are not negligible. Contamination from urban wildfire smoke warrants further study to determine how closely real risks approximate these estimated maximum risk values.

This method provides an extremely high overestimate of risk.

Its utility is in understanding the worst-possible risk scenario given the high rate of non-detections in our analysis and the high detection limits for our PAH and PCB tests.

2. Risk-Benefit Analysis

The choice of whether or not to consume local produce following a wildfire event requires that consumers make other choices about what to eat instead. Risk-Benefit analyses are useful tools to weigh these various choices.

a. Risk of other chemical exposures in food system

Of the chemicals that we evaluated in this study, produce is not typically the primary route of exposure within the food system. Therefore, consumers reducing their consumption of local produce and increasing their consumption of eggs, dairy, meat, processed foods, or canned produce may increase their overall chemical exposure from food.

- Dioxins and other fat-soluble chemicals are more likely to accumulate in meat and dairy products. The FDA dioxin monitoring project showed that, compared to fruits and vegetables, dairy products likely contribute three times more dioxins to the American diet, and meats contribute nine times more.[35]
- Polycyclic Aromatic Hydrocarbons are most commonly found in food that has been processed (especially smoking or drying) and foods that are cooked at high temperatures. PAH levels in smoked meat and fish can be as high as 200 ug/kg.[36]
- Polychlorinated Biphenyl's enter the diet primarily through fish, especially sportfish caught in contaminated lakes and rivers, which can contain PCB contamination at the order of magnitude around 1mg/kg.[37]
- Heavy Metals in the food system are tracked by FDA's Total Diet Study. Meat and processed foods are typically the highest contributors to heavy metal exposure:
    - The highest dietary sources of arsenic are in fish and seafood (.99mg/kg in canned tuna, 0.5 mg/kg in frozen fish sticks, 0.424 mg/g fish sandwich, .315 mg/kg in shrimp, and .293 in salmon steaks);
    - The highest dietary sources of lead are in processed deserts (0.01mg/kg in canned fruit cocktail, 0.011mg/kg in milk chocolate candy bar, 0.016 mg/g in chocolate syrup, 0.01 mg/kg in brownies, 0.012mg/kg in canned sweet potatoes.
    - The highest dietary sources of nickel are in processed foods (2.1mg/kg in "Oat Ring Cereal", 0.947 in milk chocolate candy bar, 0.927 in chocolate syrup, 0.6mg/kg in chocolate chip cookies). Higher levels of nickel are also found in sunflower seeds (3.2mg/kg) and legumes (0.6mg/kg dried pinto beans, 0.577 in frozen lima beans, .489mg/kg in dry roasted peanuts)

Consumers switching from local produce to other produce sources may shift exposures:

- Canned produce frequently contains Bisphenol A, a chemical used in plastics that can leach into produce from can linings. BPA from canned vegetables makes up around a third to a fifth of adult BPA intake.[38]
- Close to 50% of conventional produce contain pesticide residues[39] and diet is the leading source of pesticide exposure for the general population.[40] The research on the cancer risk from pesticide residues is divided, with some risk assessments showing low risk from pesticide residues.[41] Other studies indicate negative cognitive,[42] behavioral,[43] and reproductive health impacts.[44] A recent longitudinal study of 70,000 adults shows organic food consumption is protective against several kinds of cancer.[45]

b. Cancer Risk Reduction from Produce Consumption

When considering the potential for contamination in local produce, some consumers may reduce their overall produce consumption. This is particularly true of communities receiving food from local food security projects. Knowing that green leafy vegetables are also some of the most nutritiously dense foods, we conducted a risk-benefit analysis for lifetime cancer risk, using the methods outlined in Reiss et al. (2012) "Estimation of cancer risks and benefits associated with a potential increased consumption of fruits and vegetables."

Reiss et al used results from the 2007 meta-analysis by the World Cancer Research Fund and American Institute of Cancer evaluating the available epidemiologic evidence for the relationship between various foods and cancer rates. These relative risk results compare the cancer incidence for populations with higher consumption versus lower consumption of fruits and vegetables. Using these relative risks, Reiss et al calculated the cancer risk reduction likely in the scenario that the half of the US population (155 million) with the lowest produce intake increased their daily consumption by one serving (80g) of produce per day.

We scaled the results of Reiss et al's analysis to Sonoma County's population of 500,000.

Figure 23: Annual cancer cases per year scaled to Sonoma County population

Avoided Cancer Cases in US annually due to increased produce consumption of 80g/day	Half US population	Half Sonoma County Population	Avoided Cancer Cases in Sonoma County annually due to increased produce consumption of 80g/day
21,518	155,000,000	250,000	34.7

To calculate the health risks from eating contamination on produce, we calculated a "cancer cases per year" value using the "Maximum Probable" risk calculated using contamination levels at half the detection limit (ND=DL/2)), and assuming daily ingestion rate of 80g of produce per day among half of Sonoma County's population.

Use Caution in Interpreting Results from this Method

This method requires the calculation of a "Cancer cases per year" estimate. This crude calculation should be considered an "upper bound" estimate, and not a precise measure of the number of people that will develop cancer. Due to the low confidence in such an estimate, the "Cancer cases per year" are included only for their utility in comparing to the cancer risk reductions of produce consumption found in the epidemiologic data.

Figure 24: Cancer cases per year avoided due to increased produce consumption

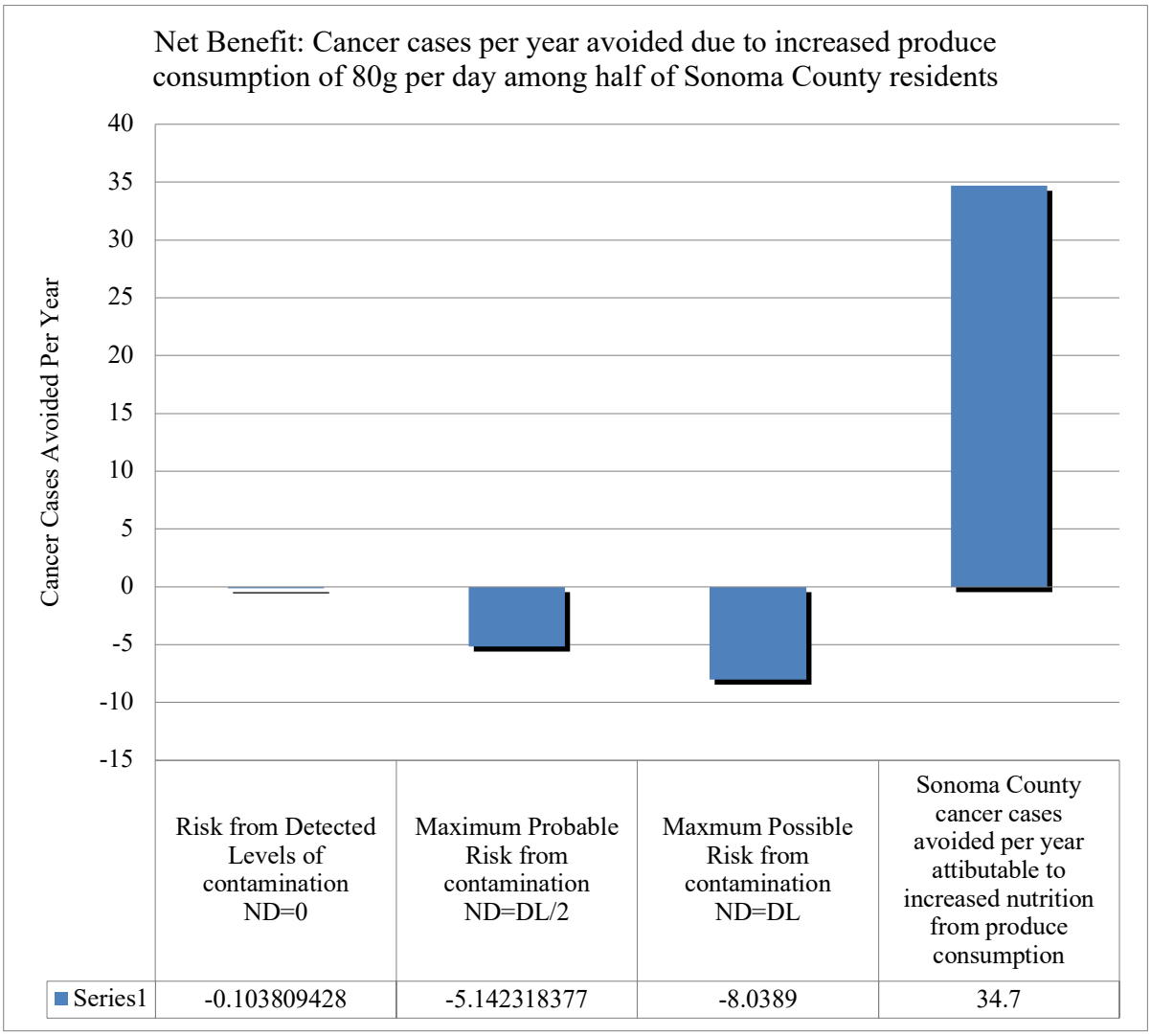


Figure 24 shows the results of our risk-benefit analysis, which yields a net benefit from the increased consumption of produce.

If half of Sonoma County's population were to eat an additional 80g of local produce per day, and if that produce were contaminated at the maximum probable levels from this study, consumption would lead to a net lifetime cancer risk reduction.

Use Caution in Interpreting Results from this Method

This method requires the calculation of a "Cancer cases per year" estimate. This crude calculation should be considered an "upper bound" estimate, and not a precise measure of the number of people that will develop cancer. Due to the low confidence in such an estimate, the "Cancer cases per year" are included only for their utility in comparing to the cancer risk reductions of produce consumption found in the epidemiologic data.

3. Social Determinants of Health: Other Health Impacts Associated with Urban Wildfire and Local Produce

a. Social Determinants of Health

Over the past several decades, public health research has increasingly expanded its focus from individual constitutional factors and lifestyle behaviors towards the larger social and economic contexts that structure disparities in health. This greater picture of the wholistic set of factors that impact the distribution of health and illness across a population gives ***perspective on the small amount of potential risk that we have shown from eating local produce exposed to wildfire smoke.***

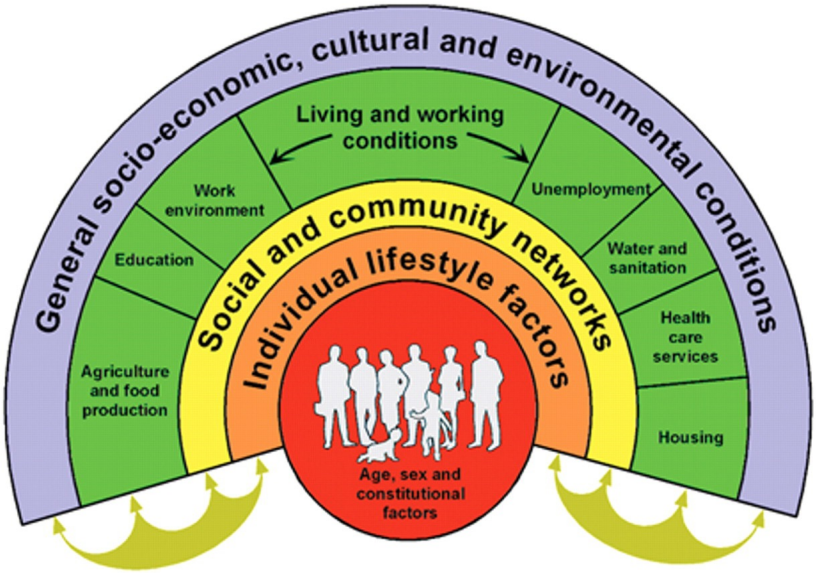


Figure 25: Social Determinants of Health, Dahlgren and Whitehead, 1991

Based on our results, we have found a low concern of health risks from the ingestion of produce and soil exposed to smoke in the 2017 urban wildfires. Furthermore, this risk represents a miniscule slice of the environmental quality and built environment conditions that also impact health. In turn, these environmental conditions are approximately only 10% of the totality of factors that shape population health, with the other major drivers of population health being access to health care (20%), health behaviors (30%), and socio-economic factors (40%).[46]

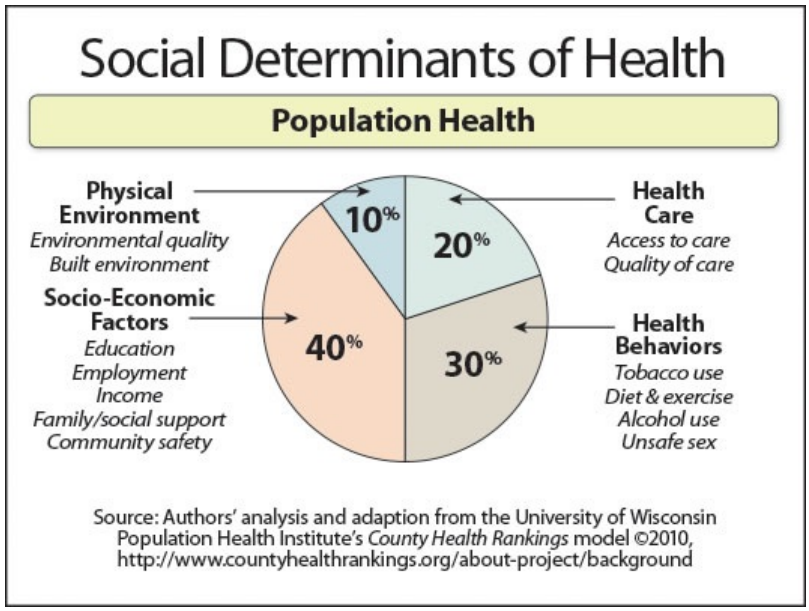


Figure 26: Population Health Institute, *County Health Rankings* model, 2010

Socio-economic factors contribute to health disparities through simultaneous and overlapping pathways. Communities marginalized by poverty, racism, and other intersectional oppressions are more likely to experience psychological stressors of marginalization[47, 48,49] and stigmatized and blighted neighborhoods,[50, 51] physical stressors including demanding physical labor, sleep deprivation and malnutrition, and chemical stressors from hazardous exposures that are more likely to be situated in low-income communities.[52, 53] It can be difficult to separate the impacts of environmental, social, and economic stressors, as communities are simultaneous exposed to multiple stressors.

One pathway that has received increasing attention over the past two decades is the biological embedding of social inequality through chronic stress. According to the National Scientific Council on the Developing Child, "toxic" stress is defined as "strong, frequent, and/or prolonged activation of the body's stress-response systems," such as the stress from chronic traumatic experiences.[54,55] According to the Adverse Childhood Experiences (ACE), a retrospective cohort study, adults with significant childhood stressors are more likely to develop CHD. The study also found a dose-response relationship, where each additional early adversity indicator increases CHD incidence by 20%.[56]

There are many competing approaches for the physiologic measurement of stress. Biomarkers that have been associated with the stress response include physiological measures like heart rate and blood pressure,[57] and hormonal responses like cortisol.[58] Adverse experiences may also be associated with biomarkers of oxidative stress,[59, 60] and telomere shortening.[61, 62, 63] which are also increasingly studied as common pathways towards negative health outcomes such as cancer and heart disease. Given the precedent from the study of chemical mixtures, these physiological measures of non-chemical stressors could be used to better understand the health impact of a mixture of chemical and social or economic stressors.

The stress from social and economic hardship has physiological impacts on the body that increase vulnerability to chemicals in the environment. For these reasons, ***an evaluation of the social and economic impacts of a strong local food system is imperative for a thorough evaluation of the relative health impact from ingestion of local produce exposed to urban wildfire smoke, especially as it relates to the most vulnerable in our community***. These social determinants of health are also reflected in Sonoma County's Recovery and Resiliency Framework, which serve as a vision and approach for how Sonoma County will recover and emerge more resilient from the October 2017 wildfires.

Sonoma County's Recovery and Resiliency Framework outlines five key strategic areas:

1. Community preparedness and infrastructure
2. Housing
3. Economy
4. Safety Net Services
5. Natural Resources

b. Health Impacts of Wildfire: Focus on Smoke Inhalation

The unknown potential health risk from the ingestion of smoke in local produce pales in comparison to the well-established health risks from inhalation of the wildfire smoke itself. In a study of the immediate health impacts of the wildfires in Alameda County in 1991, researchers conducted a retrospective review of the health records and coroner records, finding that over half of all emergency room visits in the aftermath of the fire were due to respiratory-related conditions, and that 61% were bronchospasms- irritation of the lungs due to particulates.[64] A study of the 2003 wildfires in Southern California found that exposure to wildfire smoke increases hospital admissions for cardiovascular disease.[65] Another study of the same fires found that exposure to smoke led to reduced birth weight among children born to mothers exposed to smoke, which has implications for infant development and lifelong health.[66] These cardio-respiratory impacts of acute smoke are well reported in several studies of the public health impacts of wildfire smoke and generally undisputed.[67] Other studies also point to the long-term impacts of smoke exposure among firefighters.[68, 69] Given the previously described research

on the impact of social and economic stressors on lifelong cancer risks and mortality, it is probable that social and economic factors also mediate these long-term health outcomes.

While the smoke from the wildfires impacts everyone in the region, socio-economic factors can modify the health impact of the smoke. A public health study of the cardiovascular and respiratory health impacts of wildfire smoke provides a thorough review of this issue:

> "communities with lower socio-economic status (SES) typically measured by income, education, and racial composition, have consistently been shown to be at increased risk from air pollutants but other health factors associated with low SES such as limited access to clinical care or an unhealthy diet may also play an important role in determining a community's health outcome to poor air quality… Socio-Economic Factors should be considered as modifying risk factors in air pollution studies and be evaluated in the assessment of air pollution impacts."[70]

In assessing the risks and benefits of local produce after a wildfire event, it is important to note that diet-related illnesses such as diabetes have been found to increase vulnerability to chemical exposures in air pollution.[71, 72] Chronically food insecure communities are more likely to be diagnosed with diet-related illnesses,[73] and many rely on local food security programs for free and reduced-cost produce. ***Promoting nutrition from local produce and supporting local food systems (particularly programs that serve low-income and food insecure communities) can improve community health and resilience to the cardio-respiratory impacts of a fire event.***

c. Health Impacts of Local Produce and Local Food System

A strong and connected local food system and flourishing agricultural sector help a community respond to its residents' needs for healthy food during a disaster, recover more quickly after a disaster, and provides social and economic benefits that act as protective factors for vulnerable communities. Ultimately, a robust local food system is an indicator of a resilient community.

Our local food system was an instrumental part of the emergency response during the fires, though its impact was often invisible. Following the 2017 fires, the Sonoma County Food System Alliance convened a gathering of people and organizations that were critical to the emergency food response, in order "to analyze how the emergency food response evolved during the disaster in an effort to improve the model for future disasters, to minimize the number of community members who transition from short-term emergency food assistance to long term chronic food insecurity, and to strengthen the region's food system."

The Food System Alliance released a report following the gathering, describing how farmers, distributors, chefs, and emergency food providers leveraged existing and new community-based connections to provide a quick local response in order to feed thousands of evacuees and first responders. The "spontaneous outpouring" of food from local farmers who provided a "surge of local produce," and local chefs who "stepped up" to capitalize on their pre-existing food business relationships and use excess food to "get meals out" to the community were highlights of what functioned well during the disaster. The report concludes with suggestions for learning from the emergency food system that emerged during the fire in order to strengthening the local food system to better prepare for future disasters, and to ensure ongoing food security for the region.[74]

Beyond moments of disaster, a strong and connected local food system provides other health impacts, social impacts, and economic impacts that benefit communities and increase resilience, according to a literature review on urban agriculture conducted by the University of California Agriculture and Natural Resources division.[75]

One of the most direct connections to resilience from local food systems is the increase in *Community food security*- a term commonly used to describe "a condition in which all community residents obtain a safe, culturally acceptable, nutritionally adequate diet through a sustainable food system that maximizes community self-reliance, social justice, and democratic decision-making."[76]

University of California Agriculture and Natural Resources Division report on the Benefits of Urban Agriculture:

S*ocial impacts* include the creation of safe places, community development, the building of social capital, and cross-generational and cultural integration.

Health impacts include enhanced food access and food security, increased fruit and vegetable consumption, and general well-being through improved mental health and physical activity.

Economic impacts of urban agriculture include job creation, training and business incubation, market expansion for farmers, economic savings on food for low-income consumers, savings for municipal agencies, and increased home values.

Urban agriculture is the growing of food beyond that which is strictly for home consumption or educational purposes--which includes the production, distribution and marketing of food and other products within the cores of metropolitan areas and at their edges.[66]

Beyond providing meals, the local food response gave people a sense of community connectedness and support, which is critical for modulating the toxic impacts of stressful circumstances.[77,78] Gardens hosted spaces for community members to come together, share resources, access donations, provide emotional support, offer legal consultations, clinical health and wellness support, and more.

Using the social determinants of health approach, it is clear that supporting strong local food system is critical for community health and resilience. A quantitative assessment of this dimension of health would be difficult to operationalize, and methodological challenges still remain in merging data and methods from environmental and social sciences.[79, 80] However, the County Health Rankings empirically-based model of the social determinants of health (reviewed in 3a of this report) suggests that these factors may be as important, if not more important, to health outcomes as small levels of environmental contamination, and thus warrant consideration in risk management decisions.

CONCLUSIONS

Produce sample results support the hypothesis that there is a low concern for health impacts from eating local produce exposed to the urban wildfire smoke in Sonoma County in the fall of 2017. Our cumulative analysis further suggests that eating trace contaminants on produce does not provide a significant chemical exposure during an urban wildfire event, and the potential cancer risk may be outweighed by the cancer risk reduction from the nutritional value of eating produce.

Regarding soils, results show low heavy metal concentrations, non-detectable PCBs, and generally low soil dioxin & furan levels across the region, indicating low concern for health impacts from contact with soil in local gardens near urban burn area. However, ***more analysis is needed, particularly on dioxin in Santa Rosa soils***. Direct ingestion of soil is the main route of concern for soil contamination, and OEHHA Air Toxics Hot Spots models predict that over two-thirds of this total lifetime cancer risk is attributable to exposures during 0-2 years of age.

Food safety of produce following an urban wildfire event is under-investigated, and much is unknown. Despite the dearth of information, by using a cumulative risk approach informed by the social determinants of health, we conclude that the nutritional, social, and economic benefits of promoting a strong local food system outweigh the potential risks. We conclude that when considering the impact on vulnerable communities, it is important to consider both the additional health risk from exposure to chemicals in the environment including produce, as well as the protective factors that the nutrition of local produce and a strong local food system can provide, particularly for communities for which local food assistance programs are one of their primary sources of produce.

Further research is needed to continue to investigate this hypothesis. More research is needed on the long-term impacts of trace contaminants on ***other local foods such as eggs and meat where fat-soluble chemicals can bioaccumulate***. It is also critical to note that this study examined produce and soil exposed to dispersed contaminants in smoke, and does not address the question of contamination on ***food produced directly adjacent to burned structures.*** Future studies would benefit greatly from a cumulative risk approach that takes into consideration the larger context of chemical contamination in the food system, and the potential impacts on the most vulnerable in the community, to best inform consumers making difficult and emotional choices about their food following a wildfire disaster.

Finally, we conclude that the community-driven work to build a local food system, a strong economy, and community resilience is a critical part of improving our region's health on an ongoing basis, and also a critical part of community resilience after a wildfire disaster. Wildfire preparedness should take into account not only how to reduce risk, but also how to enhance protective factors for the most vulnerable in our communities.

DURING A WILDFIRE

SAFETY TIPS FOR LOCAL FOOD GROWERS

PROTECT YOUR LUNGS

Inhaling smoke has very severe health risks

WASH PRODUCE WITH RUNNING WATER

Remove outer leaves or peel if you see ash

NUTRITION BUILDS RESILIENCE

Low risk of harm from produce exposed to smoke

TAKE EXTRA PRECAUTIONS

Children – Elders – People with Respiratory and Heart Disease

Produce Safety After Urban Wildfire

UC Cooperative Extension of Sonoma County

ucanr.edu/produceafterfire

SUGGESTED BEST PRACTICES FOR LOCAL FOOD GROWERS

Protect Your Lungs

During a wildfire, the number one thing you can do to protect your health is to avoid inhaling the smoke. Protect your lungs by staying indoors whenever possible and wearing a respirator mask when outdoors. An N95 respirator is the minimum protection recommended, while a P100 will provide additional protection from petroleum-based chemicals and smaller particles.

Wash Your Produce

Thoroughly wash produce under running water before storing, cooking and eating. Remove older, outer leaves of lettuce or leafy greens before eating. Peel root vegetables before eating.

Wash Your Hands & Clothes

Wash your hands with soap and water after working in a farm or garden that may have contamination. Reduce bringing soil contaminants into your home by removing boots and changing clothes immediately when returning home. Wash gardening clothes immediately.

Test Your Soil

After a wildfire, you can check for soil contamination by collecting soil samples from your garden and sending them to a lab. Ask for a heavy metals panel that includes lead, arsenic, chromium, and mercury. Heavy metals tests typically cost under one hundred dollars per sample and could be considered a barometer for other kinds of contamination. If heavy metals are not detected, it is probably that other contaminants are also at low levels or undetectable. Research on site history is important as contaminants detected in post-fire soil testing may have been present from before the fire. See ANR's guides on Soil Contamination in Urban Agriculture for more information.

Contain Your Soil

Sheet mulching and building raised beds can keep kids from ingesting soil, prevent soil from getting kicked up in dust, and create a barrier between soil and produce. Mulch soils beds with straw and use sub-surface irrigation (such as inline or drip-irrigation) to prevent the up-splashing of soil particles onto the under-sides of leaves or other plants' parts. A landscape fabric or weed cloth can also be used to create a barrier between soil and produce.

Amend Your Soil

Add compost and "clean" soil to your beds to dilute contaminants. Some research suggests that adding compost increases soil microorganisms and fungi that break down organic chemicals (like dioxins) in soil, and would be appropriate for low-level contamination,[81, 82, 83] while compost high in phosphorous can help bind lead in soil.[84, 85] Wear a face mask when turning soils, and reduce dust by turning soil when it is wet.

Eat Fresh Produce

Increasing produce consumption, particularly green leafy vegetables, promotes healthy nutrition and resilience to chemical exposures.

REFERENCES

[1] Lemieux, Paul M. "Emissions of Organic Air Toxics from Open Burning." *Washington, DC, United States Environmental Protection Agency* 62 (2002).
[2] Mayne, Susan and Steven Solomon. Letter from the FDA Center for Food Safety and Applied Nutrition and Center for Veterinary Medicine to California Department of Food & Agriculture and California Department of Public Health. December 19, 2017. Email.
[3] US Environmental Protection Agency, "How Smoke From Fires can Affect Your Health". EPA (2017). Accessed June 6, 2018. <https://airnow.gov/index.cfm?action=smoke.index>
[4] Uzu, Gaëlle, et al. "Foliar lead uptake by lettuce exposed to atmospheric fallouts." *Environmental Science & Technology*44.3 (2010): 1036-1042.
[5] Kipopoulou, A. M., E. Manoli, and C. Samara. "Bioconcentration of polycyclic aromatic hydrocarbons in vegetables grown in an industrial area." *Environmental pollution* 106.3 (1999): 369-380.
[6] Schreck, Eva, et al. "Metal and metalloid foliar uptake by various plant species exposed to atmospheric industrial fallout: mechanisms involved for lead." *Science of the Total Environment* 427 (2012): 253-262.
[7] Wennrich L, Popp P, Zeibig M. Polycyclic Aromatic Hydrocarbon Burden in Fruit and Vegetable Species Cultivated in Allotments in an Industrial Area. Int J Environ Anal Chem . (2002);82(10):667-690. doi:10.1080/0306731021000075401.
[8] Hertz-Picciotto, Irva. Personal Correspondence. February 20, 2019. Email.
[9] Liu, J. C., Pereira, G., Uhl, S. A., Bravo, M. A., & Bell, M. L. (2015). A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environmental research*, *136*, 120-132.
[10] Hertz-Picciotto, Irva. Personal Correspondence. February 20, 2019. Email.
[11] "New Research on Wildfires in California." Environmental Health Scienes Center, University of California, Davis. Accessed Februrary 2, 2019. <https://environmentalhealth.ucdavis.edu/california-wildfire-research>
[12] Young, Thomas. Personal Correspondence. February 22, 2019. Email.
[13] Mayne, Susan and Steven Solomon. Letter from the FDA Center for Food Safety and Applied Nutrition and Center for Veterinary Medicine to California Department of Food & Agriculture and California Department of Public Health. December 19, 2017. Email.
[14] US Environmental Protection Agency, "Toxicological Review of Benzo[a]pyrene: Executive Summary". EPA (2017). Accessed June 6, 2018. <https://cfpub.epa.gov/ncea/iris/iris documents/documents/subst/0136 summary.pdf>
[15] World Health Organization. "Health risks of heavy metals from long-range transboundary air pollution." *Geneve: WHO* (2007). Accessed June 6, 2018. <http://apps.who.int/iris/bitstream/handle/10665/107872/E91044.pdf>
[16] Agency for Toxic Substances and Disease Registry (ATSDR), "Health Statement for PCBs". Accessed January 2019 < https://www.atsdr.cdc.gov/phs/phs.asp?id=139&tid=26>
[17] World Health Organization. "Exposure to dioxins and dioxin-like substances: a major public health concern." *Geneve: WHO* (2010). Accessed June 6, 2018. <http://www.who.int/ipcs/features/dioxins.pdf>
[18] "Current Proposition 65 No Significant Risk Levels (NSRLs) Maximum Allowable Dose Levels (MADLs)." Office of Environmental Health and Hazard Assessment. Accessed May 2018. <https://oehha.ca.gov/proposition-65/general-info/current-proposition-65-no-significant-risk-levels-nsrls-maximum >
[19] "Regional Screening Levels (RSLs) – Generic Tables; Updated November 2018." Environmetnal Protection Agency. Accessed December 2018. <https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables>
[20] "California Human Health Screening Levels (CHHSLs)." Office of Environmental Health and Hazard Assessment. Accessed December 2019. <https://oehha.ca.gov/risk-assessment/california-human-health-screening-levels-chhsls>
[21] Robinson, Barbie, and Ellen Bauer. "Sonoma County Complex Fires Health Screening Level Guidance, Cleanup Goals and Background Data Sets." Sonoma County Department of Health Services, Public Health Division. February 28, 2018.
[22] EFSA Scientific Committee. "Guidance on human health risk-benefit assessment of foods." *EFSA Journal* 8.7 (2010): 1673. Accessed 4/25/2019. https://www.efsa.europa.eu/en/efsajournal/pub/1673
[23] Hoekstra, Jeljer, et al. "BRAFO tiered approach for benefit–risk assessment of foods." *Food and Chemical Toxicology* 50 (2012): S684-S698.
[24] US Environmental Protection Agency, "Integrated Risk Information System Glossary." EPA (2011). Accesed June 6, 2018 <https://iaspub.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?details=&vocabName=IRIS%20Glossary>
[25] Office of Environmental Health Hazard Assessment. Proposition 65 No Significant Risk Levels (NSRLs) for Carcinogens and Maximum Allowable Dose Levels (MADLs) for Chemicals Causing Reproductive Toxicity. March 2019. Accessed 4/24/2019. https://oehha.ca.gov/media/downloads/proposition-65//safeharborlist032519.pdf
[26] U.S. Environmental Protection Agency. Nickel Compounds. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. January 2000. Accessed 4/24/2019. https://www.epa.gov/sites/production/files/2016-09/documents/nickle-compounds.pdf
[27] U.S. Environmental Protection Agency. Health Assessment Document for Nickel. EPA/600/8-83/012F. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1986.
[28] Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Nickel (Update). Public Health Service, U.S. Department of Health and Human Services, Altanta, GA. 1997.
[29] Sexton K. Cumulative Risk Assessment: An Overview of Methodological Approaches for Evaluating Combined Health Effects from Exposure to Multiple Environmental Stressors. *Int J Environ Res Public Health*. 2012;9(2):370-390. doi:10.3390/ijerph9020370.
[30] Pressman JG, Richardson SD, Speth TF, et al. Concentration, Chlorination, and Chemical Analysis of Drinking Water for Disinfection Byproduct Mixtures Health Effects Research: U.S. EPA's Four Lab Study. *Environ Sci Technol*. 2010;44(19):7184-7192. doi:10.1021/es9039314.
[31] "Notice of adoption of technical support document for exposure assessment and stochastic analysis Aug 2012." Office of Enironmetnal health and Hazard Assessment. Accessed December 2018. <https://oehha.ca.gov/air/crnr/notice-adoption-technical-support-document-exposure-assessment-and-stochastic-analysis-aug>
[32] Reiss et al
[33] Reiss et al
[34] Reiss et al
[35] " 2001-2004 PCDD/PCDF Exposure Estimates; Based on PCDD/PCDF Concentrations assuming ND=0". Food and Drug Administration. Acccessed January 2019 < https://wayback.archive-it.org/7993/20170406021806/https://www.fda.gov/Food/FoodborneIllnessContaminants/ChemicalContaminants/ucm077498.htm>
[36] https://ec.europa.eu/food/sites/food/files/safety/docs/sci-com_scf_out154_en.pdf
[37] Agency for Toxic Substances and Disease Registry (ATSDR), "Health Statement for PCBs". Accessed January 2019 < https://www.atsdr.cdc.gov/phs/phs.asp?id=139&tid=26>
[38] Lorber, Matthew, et al. "Exposure assessment of adult intake of bisphenol A (BPA) with emphasis on canned food dietary exposures." *Environment international* 77 (2015): 55-62.
[39] US Food and Drug Administration. "Pesticide residue monitoring program fiscal year 2015 pesticide report." (2017).
[40] Riederer, Anne M., et al. "Diet and nondiet predictors of urinary 3-phenoxybenzoic acid in NHANES 1999–2002." *Environmental health perspectives* 116.8 (2008): 1015-1022.
[41] Reiss et al
[42] Engel, Stephanie M., et al. "Prenatal exposure to organophosphates, paraoxonase 1, and cognitive development in childhood." *Environmental health perspectives*119.8 (2011): 1182-1188.
[43] Quirós-Alcalá, Lesliam, Suril Mehta, and Brenda Eskenazi. "Pyrethroid pesticide exposure and parental report of learning disability and attention deficit/hyperactivity disorder in US children: NHANES 1999–2002." *Environmental health perspectives* 122.12 (2014): 1336-1342

[44] Bretveld, Reini W., et al. "Pesticide exposure: the hormonal function of the female reproductive system disrupted?." *Reproductive Biology and Endocrinology* 4.1 (2006): 30.
[45] Baudry, Julia, et al. "Association of frequency of organic food consumption with cancer risk: findings from the NutriNet-Santé prospective cohort study." *JAMA internal medicine* 178.12 (2018): 1597-1606.
[46] Population Health Institute, *County Health Rankings* model, 2010
[47] Krieger, N., Rowley, D. L., Herman, A. A., & Avery, B. (1993). Racism, sexism, and social class: implications for studies of health, disease, and well-being. *American Journal of Preventive Medicine*.
[48] Krieger, N., Smith, K., Naishadham, D., Hartman, C., & Barbeau, E. M. (2005). Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health. *Social science & medicine, 61*(7), 1576-1596.
[49] Meyer, I. H. (2003). Prejudice, social stress, and mental health in lesbian, gay, and bisexual populations: conceptual issues and research evidence. *Psychological Bulletin, 129*(5), 674.
[50] Atari, D. O., Luginaah, I., & Baxter, J. (2011). "This is the mess that we are living in": residents everyday life experiences of living in a stigmatized community. *GeoJournal, 76*(5), 483-500.
[51] Sandel, M., & Wright, R. (2006). When home is where the stress is: expanding the dimensions of housing that influence asthma morbidity. *Archives of disease in childhood, 91*(11), 942-948.
[52] Morello-Frosch, R., Zuk, M., Jerrett, M., Shamasunder, B., & Kyle, A. D. (2011). Understanding the cumulative impacts of inequalities in environmental health: implications for policy. *Health Aff (Millwood), 30*(5), 879-887. doi: 10.1377/hlthaff.2011.0153
[53] O'Neill, M. S., Jerrett, M., Kawachi, I., Levy, J. I., Cohen, A. J., Gouveia, N., . . . Schwartz, J. (2003). Health, wealth, and air pollution: advancing theory and methods. *Environmental health perspectives, 111*(16), 1861.
[54] Shonkoff JP, Garner AS, Health TC on PA of C and F, et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. *Pediatrics*. 2012;129(1):e232-e246. doi:10.1542/peds.2011-2663.
[55] Shonkoff JP, Boyce W, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA*. 2009;301(21):2252-2259. doi:10.1001/jama.2009.754.
[56] Dong M, Giles WH, Felitti VJ, et al. Insights Into Causal Pathways for Ischemic Heart Disease Adverse Childhood Experiences Study. *Circulation*. 2004;110(13):1761-1766. doi:10.1161/01.CIR.0000143074.54995.7F.
[57] Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and Age Patterns of Allostatic Load Scores Among Blacks and Whites in the United States. *Am J Public Health*. 2006;96(5):826-833. doi:10.2105/AJPH.2004.060749.
[58] Ising H, Lange-Asschenfeldt H, Moriske HJ, Born J, Eilts M. Low frequency noise and stress : Bronchitis and cortisol in children exposed chronically to traffic noise and exhaust fumes. *Noise Health*. 2004;6(23):21.
[59] Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychol Bull*. 2011;137(6):959-997. doi:10.1037/a0024768.
[60] Liu J, Mori A. Stress, Aging, and Brain Oxidative Damage. *Neurochem Res*. 1999;24(11):1479-1497. doi:10.1023/A:1022597010078.
[61] Tyrka AR, Price LH, Kao H-T, Porton B, Marsella SA, Carpenter LL. Childhood Maltreatment and Telomere Shortening: Preliminary Support for an Effect of Early Stress on Cellular Aging. *Biol Psychiatry*. 2010;67(6):531-534. doi:10.1016/j.biopsych.2009.08.014.
[62] Epel ES. Telomeres in a Life-Span Perspective A New "Psychobiomarker"? *Curr Dir Psychol Sci*. 2009;18(1):6-10. doi:10.1111/j.1467-8721.2009.01596.x.
[63] Shalev I, Moffitt TE, Sugden K, et al. Exposure to violence during childhood is associated with telomere erosion from 5 to 10 years of age: a longitudinal study. *Mol Psychiatry*. 2013;18(5):576-581. doi:10.1038/mp.2012.32.
[64] Shusterman, Dennis, Jerold Z. Kaplan, and Carla Canabarro. "Immediate health effects of an urban wildfire." *Western journal of medicine* 158.2 (1993): 133.
[65] Delfino, R.J.; Brummel, S.; Wu, J.; Stern, H.; Ostro, B.; Lipsett, M.; Winer, A.; Street, D.H.; Zhang, L.; Tjoa, T.; *et al*. The relationship of respiratory and cardiovascular hospital admissions to the Southern California wildfires of 2003. *Occup. Environ. Med.* **2009**, *66*, 189–197.
[66] Holstius, David M., et al. "Birth weight following pregnancy during the 2003 Southern California wildfires." Environmental health perspectives 120.9 (2012): 1340-1345.
[67] Youssouf, Hassani, et al. "Non-accidental health impacts of wildfire smoke." *International journal of environmental research and public health* 11.11 (2014): 11772-11804.
[68] LeMasters, Grace K., et al. "Cancer risk among firefighters: a review and meta-analysis of 32 studies." *Journal of occupational and environmental medicine* 48.11 (2006): 1189-1202.
[69] Navarro, Kathleen M., et al. "Wildland firefighter smoke exposure and risk of lung cancer and cardiovascular disease mortality." *Environmental Research* (2019).
[70] Rappold, Ana G., et al. "Cardio-respiratory outcomes associated with exposure to wildfire smoke are modified by measures of community health." *Environmental Health* 11.1 (2012): 71.
[71] Schneider, Alexandra, et al. "Association of cardiac and vascular changes with ambient PM 2.5 in diabetic individuals." *Particle and fibre toxicology* 7.1 (2010): 14.
[72] O'neill, Marie S., et al. "Diabetes enhances vulnerability to particulate air pollution–associated impairment in vascular reactivity and endothelial function." *Circulation* 111.22 (2005): 2913-2920.
[73] Seligman, Hilary K., et al. "Food insecurity is associated with diabetes mellitus: results from the National Health Examination and Nutrition Examination Survey (NHANES) 1999–2002." *Journal of general internal medicine* 22.7 (2007): 1018-1023.
[74] Borgeson, Phina et al.. "Sonoma County Emergency Food Response Gathering – Summary of Findings and Recommendations, August 2018." Sonoma County Food System Alliance. Accessed 3/20/2019. <https://sonomacofsa.files.wordpress.com/2018/11/efr-report-final-2018-8-14.pdf>
[75] Golden, Sheila. **"**Urban Agriculture Impacts: Social, Health, and Economic: A Literature Review." University of California, Agriculture and Natural Resources. 2013. Accessed online 6/20/2019. https://ucanr.edu/sites/UrbanAg/files/251671.pdf
[76] Hamm, Michael W., and Anne C. Bellows. "Community food security and nutrition educators." *Journal of Nutrition Education and Behavior* 35.1 (2003): 37-43.
[77] Franke, Hillary. "Toxic stress: effects, prevention and treatment." *Children* 1.3 (2014): 390-402.
[78] Weissbecker, Inka, et al. "Psychological and physiological correlates of stress in children exposed to disaster: Current research and recommendations for intervention." *Children Youth and Environments* 18.1 (2008): 30-70.
[79] Clougherty JE, Kubzansky LD. A framework for examining social stress and susceptibility to air pollution in respiratory health. *Ciênc Saúde Coletiva*. 2010;15(4):2059-2074. doi:10.1590/S1413-81232010000400020.
[80] Sexton K, Linder SH. Cumulative Risk Assessment for Combined Health Effects From Chemical and Nonchemical Stressors. *Am J Public Health*. 2011;101(S1):S81-S88. doi:10.2105/AJPH.2011.300118.
[81] Chen, Wei-Yu, et al. "Bioremediation of polychlorinated-p-dioxins/dibenzofurans contaminated soil using simulated compost-amended landfill reactors under hypoxic conditions." *Journal of hazardous materials* 312 (2016): 159-168.
[82] Hiraishi, Akira. "Biodiversity of dioxin-degrading microorganisms and potential utilization in bioremediation." *Microbes and Environments* 18.3 (2003): 105-125.
[83] Tachibana, Sanro, Yukinori Kiyota, and Michifusa Koga. "Bioremediation of dioxin-contaminated soil by fungi screened from nature." *Pakistan Journal of Biological Sciences* 10.3 (2007): 486-491.
[84] Hettiarachchi, G. M., G. M. Pierzynski, and M. D. Ransom. "In situ stabilization of soil lead using phosphorus." *Journal of Environmental Quality* 30.4 (2001): 1214-1221.
[85] Zwonitzer, John C., Gary M. Pierzynski, and Ganga M. Hettiarachchi. "Effects of phosphorus additions on lead, cadmium, and zinc bioavailabilities in a metal-contaminated soil." *Water, Air, and Soil Pollution* 143.1-4 (2003): 193-209.

Authorship

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Project Goals

Goal 1:

Address community concerns regarding the impact of air pollution generated by the wildfires on local produce

Goal 2:

Build a body of knowledge about the impact of air pollution on produce, a critical and emerging public health topic that has little research data available

Goal 3:

Increase the air pollution and environmental health knowledge of communities engaged in local food and promote awareness of air pollution mitigation strategies

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WEBSITE

Reports and resources for community and researchers

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The Pollination Project

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