Bay Area Air Quality Management District

Summary and Analysis of Cupertino Air Monitoring Results

Updated May 16, 2012

The Air District's Cupertino Air Monitoring Station began operating on September 1, 2010. The monitoring station is located at Monte Vista Park, approximately one mile east of the Lehigh Cement Plant (see Figure 1). After collecting an entire year of data from September 2010 through the end of August of 2011, Air District staff reviewed the data and developed the following summary and analysis of the results.

CRITERIA POLLUTANTS

Criteria pollutants are air contaminants for which the U.S. Environmental Protection Agency (EPA) and/or the California Air Resources Board (CARB) have adopted health-based ambient air quality standards. Ambient air quality standards adopted by EPA are National Ambient Air Quality Standards (NAAQS), and standards adopted by CARB are State Ambient Air Quality Standards. Criteria pollutants include PM_{10}, $PM_{2.5}$, ozone, carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2) and lead. Ozone, CO, SO_2, and NO_2 are gases. PM_{10} is particulate matter with a diameter less than or equal to 10 microns, and $PM_{2.5}$ is particulate matter with a diameter less than or equal to 2.5 microns. Lead is a component of particulate matter.

Table 1 summarizes Cupertino monitoring results for all criteria pollutants, provides a comparison to applicable National and State ambient air quality standards, and specifies locations with similar air quality.

GASES: Based on one year (2010 -2011) of monitoring data, Cupertino air quality levels were well below all applicable State and National Ambient Air Quality Standards for gaseous criteria pollutants including ozone, CO, SO_2, and NO_2. In general, levels of criteria pollutants were in the middle of the distribution of Bay Area air monitoring sites, with as many locations measuring levels higher as locations measuring lower than Cupertino. For ozone, levels at Cupertino were below the national standard and similar to Napa and Vallejo. NO_2 levels were similar to levels at other suburban locations, including Vallejo, Redwood City and Livermore. The same was true for SO_2 emissions with measurements similar to San Pablo and Concord. CO measurements were among the lowest in the Bay Area, with only the rural location at Bethel Island being lower.

PARTICULATE MATTER: Ambient air quality standards have been established for $PM_{2.5}$ and PM_{10}. For both $PM_{2.5}$ and PM_{10}, there is a 24-hour standard based on daily concentrations, and an annual standard based on the average of all 24-hour concentrations over a one-year period. Cupertino PM levels were among the lowest in the Bay Area, and have not exceeded the 24-hour $PM_{2.5}$ NAAQS nor the 24-hour PM_{10} NAAQS, with levels similar to Redwood City and Gilroy. The annual average $PM_{2.5}$ levels were also below the

NAAQS and the more stringent annual average State standards, with levels similar to, but lower than, Livermore.

LEAD: Cupertino lead levels were less than 1% of the State standard, less than 10% of the recently revised national standard, and less than levels in San Francisco.

Table 1. Criteria Pollutants Measured at the Cupertino Monitoring Site Compared to State and National Ambient Air Quality Standards

Pollutant	Averaging Time	State Standard	National Standard	Cupertino Concentrations	Location(s) with Similar Air Quality
Ozone	1 Hour	0.09 ppm	N/A	0.09 ppm	Napa, Vallejo
	8 Hour	0.070 ppm	0.075 ppm	0.067 ppm	
PM_{10}	24 Hour	50 $\mu g/m^3$	150 $\mu g/m^3$	27 $\mu g/m^3$	San Francisco, San Pablo, Napa
	Annual Average	20 $\mu g/m^3$	N/A	14.6 $\mu g/m^3$	
$PM_{2.5}$	24 Hour	N/A	35 $\mu g/m^3$	20 $\mu g/m^3$	Redwood City, Gilroy
	Annual Average	12 $\mu g/m^3$	15.0 $\mu g/m^3$	8.7 $\mu g/m^3$	Livermore
CO	8 Hour	9.0 ppm	9 ppm	1.0 ppm	Pittsburg, Oakland
	1 Hour	20 ppm	35 ppm	1.3 ppm	
NO_2	Annual Average	0.030 ppm	0.053 ppm	0.0087 ppm	Vallejo, Redwood City, Livermore
	1 Hour	0.18 ppm	0.100 ppm	0.043 ppm	
SO_2	Annual Average	N/A	N/A	0.0008 ppm	San Pablo, Concord
	24 Hour	0.04 ppm	N/A	0.003 ppm	
	1 Hour	0.25 ppm	0.075 ppm	0.009 ppm	
Lead	30 Day Average	1.5 $\mu g/m^3$	N/A	0.003 $\mu g/m^3$	San Francisco
	3 Month Average	N/A	0.15 $\mu g/m^3$ (Recently Revised)	0.002 $\mu g/m^3$	

Note: Cupertino concentrations listed are design values based on the form of the NAAQS recorded for the applicable 1-hr, 8-hr, 24-hr, 30 day, and 3 month averaging periods.

TOXIC AIR CONTAMINANTS

Table 2 summarizes toxic air contaminant monitoring results for Cupertino. Sample durations were 24-hours for either a 6-day or 12-day interval schedule. Table 2 contains the maximum concentrations for the 24-hour samples and the results for all samples averaged over a 1-year period.

The Air District estimated health risks using the ambient monitoring data and health effect values [cancer potency factors and non-cancer Reference Exposure Levels (RELs)] established by Cal/EPA's Office of Environmental Health Hazard Assessment (OEHHA). Four health risk summary tables are provided as follows: cancer risk, chronic non-cancer risk, 8-hour chronic non-cancer risk, and acute non-cancer risk. Note that each health risk summary table lists only the measured toxic air contaminant compounds for which a corresponding cancer or non-cancer health effect value has been adopted by OEHHA. Health risks were based on the following exposure pathways, where applicable, under OEHHA health risk assessment guidelines: inhalation, dermal absorption, soil ingestion, mother's milk ingestion, and homegrown produce ingestion. Non-inhalation pathway exposures were estimated based on measured pollutant concentrations and conservative default exposure assumptions established in OEHHA guidelines.

Table 3 lists the estimated cancer risk associated with lifetime exposure to the measured levels of toxic air contaminants. The estimated cancer risk includes an Age Sensitivity Factor to account for inherent increased susceptibility to carcinogens during infancy and childhood. The total cancer risk is based on the sum of the cancer risks determined for each individual compound. The compounds that contribute most significantly to cancer risk in Cupertino are diesel PM, benzene, 1,3-butadiene, carbon tetrachloride and formaldehyde. This is consistent with other monitoring sites. These pollutants are emitted primarily from mobile sources, with the exception of carbon tetrachloride. There are no known local sources of carbon tetrachloride due to the phase-out of this compound as a stratospheric ozone-depleting compound. Measured levels of carbon tetrachloride in Cupertino are consistent with global background levels observed at other monitoring sites. Diesel PM concentrations contribute approximately 70% of the total cancer risk at the Cupertino air monitoring site. Table 4 shows the concentrations of elemental carbon and Diesel PM, cancer risk due to Diesel PM and the total cancer risks for the Cupertino and several other Bay Area air monitoring sites. Total cancer risk based on the monitoring results in Cupertino is somewhat less than the risk in Benicia and significantly less than risk in San Jose, Berkeley, and San Francisco. A comparison of cancer risk at the different monitoring sites is illustrated in Figure 2.

Table 5 indicates the estimated chronic non-cancer risk represented by hazard quotient and hazard index. A hazard quotient is the ratio of the observed concentration of a particular compound to the compound's REL. RELs are concentrations at or below which no adverse non-cancer health effects are anticipated to occur in the general human population, including sensitive individuals. The hazard index is taken as the sum of the hazard quotients for each compound that affects the same target organ system (e.g., respiratory system, nervous system, etc.). A hazard index at or below one indicates that no adverse effects would be anticipated to occur. The chronic hazard index is calculated using the annual average concentration. For the

Cupertino air monitoring site, the chronic hazard index is about one. Arsenic and mercury have the highest contribution to the chronic hazard index at the Cupertino site. At other Bay Area air monitoring sites, the arsenic level of detection was not nearly as sensitive as the Cupertino site, and mercury was not measured at all; for these reasons there is no Bay Area air monitoring site that is a good comparator to the Cupertino site for arsenic and mercury. However, based on a literature review, the arsenic and mercury concentrations measured at the Cupertino site appear to be within or lower than the range found for rural areas. The annual average concentration of arsenic measured at the Cupertino air monitoring site is 0.00018 $\mu g/m^3$. The range of arsenic air concentrations in remote areas is 0.001 to 0.003 $\mu g/m^3$ (ToxGuide for Arsenic As CAS# 7440-38-2, U.S. Department of Health and Human Services Public Health Service Agency for Toxic Substances and Disease Registry, October 2007, http://www.astsdr.cdc.gov/toxguides/toxguide-2.pdf). The annual average concentration of mercury measured at the Cupertino air monitoring site is 0.0024 $\mu g/m^3$. The range of mercury concentrations in rural areas is 0.001 to 0.004 $\mu g/m^3$ (Mercury Study Report to Congress, Volume III: Fate and Transport of Mercury in the Environment, EPA-452/R-97-003 December 1997, Table 3-1 Summary of Measured Mercury Concentration in Air http://www.epa.gov/mercury/report.htm).

Table 6 lists the estimated 8-hour chronic non-cancer risk. The 8-hour hazard indices are based on concentrations for the normal 8-hour exposure period for workers, and for children at schools and daycare facilities, that are repeated over an annual period. Note that 8-hour monitoring data are not available, but these concentrations were conservatively estimated by assuming that the entire 24-hour sample was collected over a single 8-hour period (i.e., 8-hour concentrations were assumed to be three times the measured 24-hour concentration). The 8-hour chronic hazard index is less than one for the Cupertino air monitoring site.

Table 7 lists the estimated acute non-cancer risk. The acute hazard indices are based on maximum concentrations for a 1-hour period. Note that 1-hour monitoring data are not available, but these concentrations were conservatively assumed to be 7.5 times the maximum 24-hour concentration (see table footnote for derivation of this adjustment factor). The acute hazard index is less than one for the Cupertino air monitoring site.

Table 2. Cupertino Maximum 24-hour Average and Annual Average Toxic Air Contaminant Ambient Air Monitoring Data			
Compound	Maximum 24-hour Average Concentration, µg/m^3	% of Samples above MDL	Annual Average Concentration, µg/m^3
Acetaldehyde	4.7E+00	100%	1.1E+00
Arsenic	7.0E-04	26%	1.8E-04
Benzene	1.1E+00	100%	4.8E-01
1,3 Butadiene	2.4E-01	5%	7.4E-02
Carbon Tetrachloride	8.2E-01	100%	6.4E-01
Chloroform	4.0E-01	93%	1.1E-01
Chromium (Total)	7.4E-03	97%	2.0E-03
Copper	2.4E-02	100%	7.7E-03
Elemental Carbon		100%	5.0E-01
Diesel PM		100%	5.2E-01
Ethylbenzene	4.3E-01	36%	1.5E-01
Ethylene Dibromide	<MDL	0%	<MDL
Ethylene Dichloride	<MDL	0%	<MDL
Formaldehyde	5.7E+00	100%	1.7E+00
Lead	5.7E-03	85%	2.1E-03
Manganese	3.5E-02	100%	7.7E-03
Mercury	5.3E-03	100%	2.4E-03
Methyl Chloroform	<MDL	0%	<MDL
Methylene Chloride	1.1E+00	43%	3.2E-01
Methyl Ethyl Ketone	2.7E+00	77%	6.2E-01
Nickel	4.3E-03	67%	1.2E-03
Perchloroethylene	1.5E-01	80%	5.6E-02
Selenium	1.7E-03	74%	5.2E-04
Toluene	3.4E+00	98%	1.0E+00
Trichloroethylene	1.6E-01	75%	5.8E-02
Vanadium	9.8E-03	92%	2.4E-03
Vinyl chloride	<MDL	0%	<MDL
M&P Xylene	1.7E+00	98%	5.7E-01
O Xylene	7.0E-01	62%	2.4E-01

Table 2 Notes:

1. MDL is the Method Detection Limit. <MDL indicates less than Method Detection Limit. When a sample is identified as <MDL, 1/2 the MDL is used to calculate the annual average concentration. If 95% or more of the sample values are <MDL, the annual average concentration is listed as <MDL.
2. Diesel PM is estimated from elemental carbon data using the MATES II factor of 1.04.
3. The annual average concentration of the following toxic air contaminants were less than their respective MDLs: Arsenic, 1,3-Butadiene, Ethylbenzene, Ethylene dibromide, Ethylene dichloride, Methyl chloroform, Methylene chloride, Trichloroethylene and Vinyl chloride.

Table 3. Cancer Risk Based on Cupertino Ambient Air Monitoring Data		
Compound	Unit Risk Values[1], $(\mu g/m^3)^{-1}$	Cancer Risk[2] (in a million)
Acetaldehyde	2.9E-06	5.4
Arsenic	1.7E-02	5.0
Benzene	2.9E-05	23.8
1,3 Butadiene	1.7E-04	21.9
Carbon Tetrachloride	4.3E-05	47.6
Chloroform	5.5E-06	1.0
Diesel PM	3.2E-04	280.1
Ethylbenzene	2.5E-06	0.7
Formaldehyde	6.1E-06	17.6
Lead	5.1E-05	0.0
Methylene Chloride	1.0E-06	0.6
Nickel	2.6E-04	0.5
Perchloroethylene	6.1E-06	0.6
Trichloroethylene	2.0E-06	0.2
	Total Cancer Risk:	**405**

Table 3 Notes:

1. All compounds were evaluated for the inhalation pathway. For Arsenic and Lead, which have multipathway impacts, the Unit Risk Values (URVs) represent the combined inhalation and noninhalation pathways (dermal, soil ingestion, mother's milk, homegrown produce ingestion); these URVs were derived using HARP and default exposure values.
2. Cancer risk is based on a residential exposure duration of 24 hours per day, 350 days per year over a 70-year lifetime and includes a cancer risk adjustment factor of 1.7 to account for the inherent greater susceptibility to carcinogens during infancy and childhood.

Table 4. Cancer Risk Based on Ambient Air Monitoring Data in the Bay Area					
Compound	Cupertino	San Jose	Berkeley	San Francisco	Benicia
Elemental Carbon	0.5	0.8	0.9	0.7	0.7
Diesel PM	0.5	0.9	0.9	0.8	0.7
Diesel PM, in a million	280	468	481	419	369
Total Cancer Risk, in a million	405	649	608	521	470

Table 4 Notes:

1. Diesel PM is estimated from elemental carbon data using the MATES II factor of 1.04.

Table 5. Chronic Non-cancer Risk Based on Cupertino Ambient Air Monitoring Data			
Compound	Chronic REL, µg/m^3	Chronic Hazard Quotient	Target Organ System
Acetaldehyde	140	0.0	Respiratory
Arsenic	0.00037	0.5	Cardiovascular, Developmental, Nervous, Respiratory, Skin
Benzene	60	0.0	Developmental, Hematologic, Nervous
1,3 Butadiene	20	0.0	Reproductive
Carbon Tetrachloride	40	0.0	Alimentary, Developmental, Nervous
Chloroform	300	0.0	Alimentary, Developmental, Kidney
Diesel PM	5	0.1	Respiratory
Ethylbenzene	2000	0.0	Alimentary, Developmental, Endocrine, Kidney
Formaldehyde	9	0.2	Respiratory
Manganese	0.09	0.1	Nervous
Mercury	0.0045	0.5	Developmental, Kidney, Nervous
Methyl Chloroform	1000	<MDL	Nervous
Methylene Chloride	400	0.0	Cardiovascular, Nervous
Nickel	0.014	0.1	Developmental, Hematologic, Respiratory
Perchloroethylene	35	0.0	Alimentary, Kidney
Selenium	20	0.0	Alimentary, Cardiovascular, Nervous
Toluene	300	0.0	Developmental, Nervous
Trichloroethylene	600	0.0	Eye, Nervous
M&P Xylene	700	0.0	Nervous, Respiratory
O Xylene	700	0.0	Nervous, Respiratory
Chronic Hazard Index:		**1.1**	**Nervous, Developmental**

Table 5 Notes:

1. A chronic inhalation hazard quotient (HQ) is the ratio of the annual average concentration to the chronic inhalation REL. A noninhalation HQ is the ratio of the estimated noninhalation dose to the oral REL. The HQ for each compound is the sum of the chemical specific inhalation HQ and non-inhalation HQ. A Hazard Index (HI) is the sum of the hazard quotients (HQ) for all compounds that affect a particular target organ system. The highest target organ specific HI is the overall HI. Arsenic, Mercury, and Nickel have noninhalation pathways; the chronic RELs for these compounds were derived from HARP and included the impacts of the noninhalation pathways.
2. Adverse health effects are not expected to occur, even for sensitive members of the population, for hazard indices less than one. An exceedance of one does not indicate that adverse effects will occur; rather, it is an indication of the erosion of the margin of safety, and that the likelihood of adverse health effects is increased.
3. Arsenic, Mercury, and Nickel have noninhalation pathways; the chronic RELs for these compounds were derived from HARP and included the impacts of the inhalation and noninhalation pathways: inhalation, dermal adsorption, soil ingestion, mother's milk ingestion and home grown produce ingestion pathways (urban area).

Table 6. 8-hour Chronic Non-cancer Risk Based on Cupertino Ambient Air Monitoring Data			
Compound	8-hour Chronic Inhalation REL, $\mu g/m^3$	8-hour Chronic Hazard Quotient	Target Organ System
Acetaldehyde	300	0.0	Respiratory
Arsenic	0.015	0.0	Cardiovascular, Developmental, Nervous, Respiratory, Skin
Formaldehyde	9	0.6	Respiratory
Manganese	0.17	0.1	Nervous
Mercury	0.06	0.1	Developmental, Kidney, Nervous
8-hour Chronic Hazard Index:		**0.6**	**Respiratory**

Table 6 Notes:

1. An 8-hr hazard quotient is calculated by dividing the 8-hour average concentration (e.g., for a worker or student or child at daycare that is repeated over an annual period) by the 8-hr REL. A hazard Index is the sum of the hazard quotients for all compounds that affect a particular target organ system. The greatest target organ HI is the overall HI.
2. Adverse health effects are not expected to occur, even for sensitive members of the population, for hazard indices less than one. An exceedance of one does not indicate that adverse effects will occur, rather, it is an indication of the erosion of the margin of safety and that the likelihood of adverse health effects is increased.
3. The maximum 8-hour chronic exposure was conservatively estimated based on the assumption that all the pollutants for a 24-hour sample were collected within an 8-hour period. Therefore, an adjustment factor of 3 (24 hr/8 hr) was applied to the annual average concentrations (averages of multiple 24-hr samples).

Table 7. Acute Non-cancer Risk Based on Cupertino Ambient Air Monitoring Data			
Compound	Acute Inhalation REL, µg/m^3	Acute Hazard Quotient	Target Organ System
Acetaldehyde	470	0.1	Eye, Respiratory
Arsenic	0.2	0.0	Cardiovascular, Developmental, Nervous
Benzene	1300	0.0	Developmental, Hematologic, Immune, Reproductive
Carbon Tetrachloride	1900	0.0	Alimentary Tract, Developmental, Nervous, Reproductive
Chloroform	150	0.0	Developmental, Nervous, Reproductive
Copper	100	0.00	Respiratory
Formaldehyde	55	0.8	Eye
Mercury	0.6	0.1	Developmental, Nervous
Methyl Chloroform	68000	<MDL	Nervous
Methylene Chloride	14000	0.0	Nervous
Methyl Ethyl Ketone	13000	0.0	Eye, Respiratory
Nickel	0.2	0.2	Immune, Respiratory
Perchloroethylene	20000	0.0	Eye, Nervous, Respiratory
Toluene	37000	0.0	Developmental, Eye, Nervous, Reproductive, Respiratory
Vanadium	30	0.0	Eye, Respiratory
M&P Xylene	22000	0.0	Eye, Respiratory
O Xylene	22000	0.0	Eye, Respiratory
Acute Hazard Index:		**0.9**	**sensory irritation: Eyes**

Table 7 Notes:

1. An acute hazard quotient is the value of the maximum one-hour average concentration divided by the acute REL. A hazard Index (HI) is the sum of the hazard quotients (HQ) for all compounds that affect a particular target organ system. The greatest target organ specific HI is the overall HI.
2. Adverse health effects are not expected to occur, even for sensitive members of the population, for hazard indices less than one. An exceedance of one does not indicate that adverse effects will occur, rather, it is an indication of the erosion of the margin of safety and that the likelihood of adverse health effects is increased.
3. Max. 1-hr concentrations were assumed to be 7.5 times the max. 24-hr concentration. This adjustment factor was determined by multiplying a 1-hr to 24-hr meteorological persistence factor of 1/0.4 = 2.5 ("Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised, October 1992, EPA-454/R-92-019, page 4-16), by an emission rate scalar of 3 (24 hr/8 hr), that accounts for temporal differences in emissions over the 24-hour period. This technique was used for this report to adjust concentrations based on the 24 hour monitoring data in Table 2.

Figure 1. Location of the Air District's Cupertino Air Monitoring Station

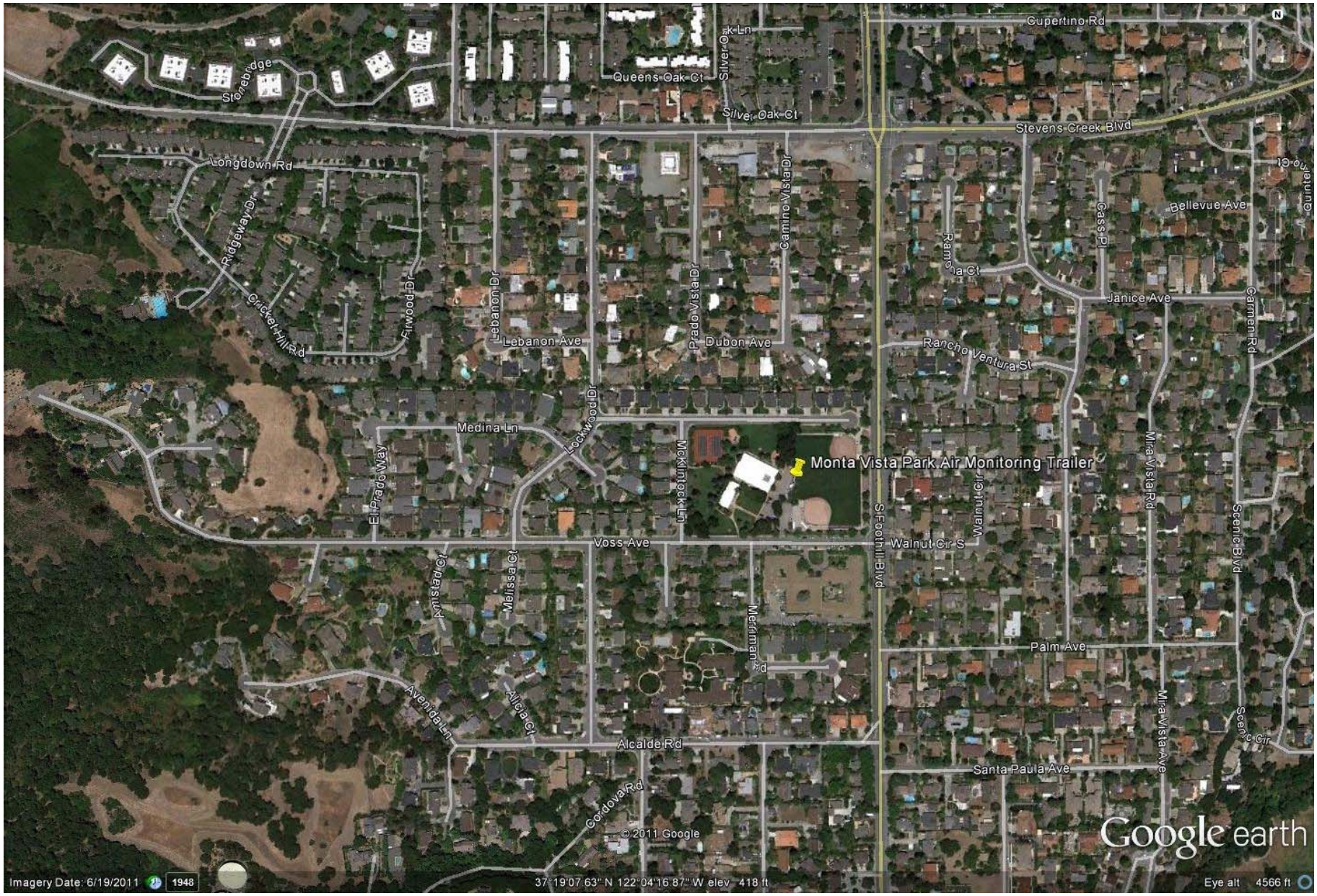

Figure 2. Cancer Risk Based on Ambient Air Monitoring Data