

## **Ambient toxic trends**

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The BAAQMD and ARB have tracked selected toxic pollutants since the late 1980s. There are no ambient standards for these pollutants, but they do have known health risks either for cancer or for other acute or chronic health problems. Table 1 presents the compounds that have been measured for a significant amount of time, showing the first year sufficient data were available, and the annual percent reduction, where possible. The trend for a compound was estimated using the ratio of an estimate of the District mean based on the most recent available 5 years of data (usually 2002-06) with the earliest 5 years. BAAQMD sites were used where possible.

With some exceptions, ambient toxic compounds in the Bay Area have been reduced substantially. Reformulated fuels have reduced concentrations of benzene and 1,3-butadiene. MTBE has been eliminated from gasoline. Perchloroethylene has been reduced dramatically because of state and BAAQMD dry cleaner rules. Cleaner-burning diesel engines and cleaner diesel fuel have reduced diesel concentrations over 50%. Carbon tetrachloride is one exception, showing essentially no change. This pollutant is no longer manufactured but is long-lived in the atmosphere and ubiquitous world-wide. A second exception is chloroform, which has shown a 16% increase since the late 1980s. Formaldehyde and acetaldehyde show reductions of 14% and 8% respectively from 1996-98 to 2003-05. It is not clear that these represent statistically significant reductions.

### **Data and Methods**

Data are collected on an every 12th day schedule. For this analysis, a year was assumed to have sufficient data if there were at least 5 observations in each calendar quarter. For site/years meeting this criterion, the annual means were computed from the quarterly averages.

To determine trends, the following procedure was used. Sites were used if they had measurements in more than half the years that compound was measured at some site. Missing years were filled using a program that assumes annual mean vectors follow a multivariate normal distribution. The District average was computed for each year. If 7 or more sites were available, a trimmed mean was used – where the highest and lowest mean for that year were discarded. Otherwise, a simple arithmetic mean was used. Then regression lines were fit to the natural logs of District mean for the earliest 5 or 6 years and the most recent 5 or 6 years, and predictions for the earliest year  $p_e$ , and the most recent year,  $p_r$ , computed. The total change was estimated to be  $T = e^{p_r - p_e}$ . The annual percentage trend was estimated as  $100*[1 - t^{1/(r-e)}]$ .

The log scale was used because it affords more straightforward estimates of uncertainty. Namely a 90% confidence interval can be formed as  $100*[1 - L^{1/(r-e)}]$  to  $100*[1 - U^{1/(r-e)}]$ , where  $L = e^{p_r - p_e - w}$  and  $U = e^{p_r - p_e + w}$ , where  $w = t_{.05} * s$ , with  $t_{.05}$  = the upper 5<sup>th</sup> percentile of a t-distribution with the appropriate degrees of freedom, and  $s$  = an estimate of the standard error of  $p_r - p_e$ .

## Results

Table 1 shows the set of toxics measured, the years of measurement, trend estimates where there is sufficient data. Figure 1 shows estimates of the District means for these toxics for the first and last years of measurement.

Of the toxics for which trend estimation is indicated, substantial progress has been made in all but one case. Except for carbon tetrachloride, the reductions have been statistically significant and the majority of toxics have been reduced more than 50% since the late 1980s as has the toxic risk.

**Table 1.** Toxic compound trends 1987-2005.

Compound	Years First Measured Routinely		Sufficient Data For Trend Analysis <sup>a</sup>		Annual Percent Change (90% confidence interval) <sup>b</sup>
	BAAQMD	ARB	BAAQMD	ARB	
<i>VOCs:</i>					
1,1,1-Trichloroethane	1987	1988	+	+	11.3 (10.1, 12.4)
1,3-Butadiene	1994	1989		+	9.5 (8.2, 10.8)
Benzene	1987	1988	+	+	9.6 (8.6, 10.6)
Chloroform	1987	1988		+	
Carbon Tetrachloride	1987	1988	+	+	0.2 (-0.1, 0.5)
Ethylene Dibromide	1987	1988			
Ethylene Dichloride	1987	1988			
MTBE	1996	1998	+	+	>25
Methylene Chloride	1987	1988			5.1 (2.9, 7.2)
Perchloroethylene	1987	1987	+	+	14.9 (13.3, 16.4)
Toluene	1987	1991	+	+	8.2 (7.3, 9.1)
Trichloroethylene	1987	1988			
Vinyl Chloride	1987				
Formaldehyde		1996		+	4.1 (1.8, 6.4)
Acetaldehyde		1996		+	1.9 (0.2, 3.7)
<i>PM10:</i>					
PAHs <sup>c</sup> (toxicity-weighted)		1995-2004		+	5.4 (2.2, 8.4)
Hexavalent Chromium		1991		+	8.5 (6.2, 10.8)
<i>PM2.5:</i>					
Diesel <sup>d</sup>	1987 <sup>d</sup>		+		6.2 (4.4, 8.7)
<i>Risk<sup>e</sup></i>	1990	2005			7.0 (5.3, 9.1)

<sup>a</sup>Sufficient data above the limit of detection & enough years with non-missing data to perform trend analysis.

<sup>b</sup>Comparison of earliest 5 years with 2002-06. Regression lines fit to each 5-year period. The annual reduction from the earliest year to 2006 as predicted by the regression lines is shown in the table.

<sup>c</sup>Benzo(a)pyrene, Benzo(b)fluoranthene, Benzo(g,h,i)perylene, Benzo(k)fluoranthene, Dibenz(a,h)anthracene, Indeno(1,2,3-cd)pyrene

<sup>d</sup>Diesel estimated from Coefficient of Haze measurements and elemental carbon measurements. Annual COH means were collected over many years, but there was no observable trend until the beginning of the 1990s.

<sup>e</sup>Estimates of the 2006 risk compared with the 1990 risk.

<sup>f</sup>MTBE concentrations are now below the limit of detection making a calculation of the current mean impossible.

## Benzene

Benzene is highly carcinogenic and occurs throughout the Bay Area. Benzene also has non-cancer health impacts. Acute effects include central nervous system symptoms of nausea, tremors, drowsiness, dizziness, headache, intoxication, and unconsciousness.

Current estimates show that most of the benzene emitted in the Bay Area comes from motor vehicles, including evaporative leakage and unburned fuel exhaust. Industry-related stationary sources contribute 13 percent of the benzene statewide. The primary stationary sources of reported benzene emissions include petroleum refining, and electricity generation.

Figure 2a shows Bay Area benzene trends. In earlier years, there was substantial variation in benzene concentrations, with high levels in areas with heavy traffic. However, the reductions have been dramatic: for example, the California Air Resources Board adopted new fuel standards that by 1996 reduced the benzene content in gasoline for motor vehicles by greater than 50% and accelerated the downward trend in benzene concentrations. CARB also implemented numerous changes to regulations mandating more effective vapor recovery equipment for gasoline stations. Attrition of older motor vehicles has also reduced benzene in tailpipe emissions. In 1987, all non-background sites had mean concentrations over 1.0 ppb. By 2006, no site had mean concentrations above 0.5 ppb, and many had values below what the Fort Cronkite "background" registered in the late 1980s. Regression lines are shown in red, estimating a mean concentration of 1.8 ppb in 1987 compared with 0.26 ppb by 2006. The average Bay Area risk dropped from 144 cases/million to 24 cases/million, a reduction of over 80%.

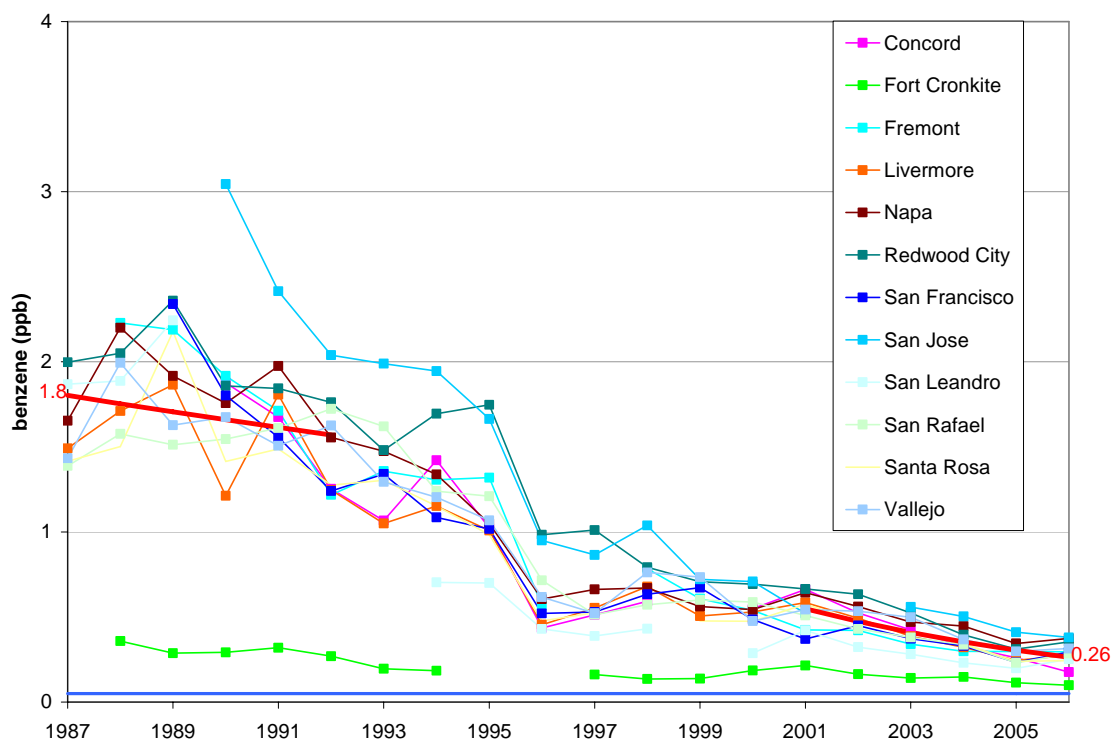


Figure 2a. Annual benzene concentrations at Bay Area sites.

## 1,3-Butadiene

1,3-butadiene is another carcinogen, with similar origins to benzene, namely mainly from gasoline evaporation and motor vehicle exhaust, with some benzene also coming from petroleum refining and electricity generation. CARB fuel standards also mandated reductions in 1,3-butadiene content in gasoline by 1996.

Long-term 1,3-butadiene measurements were only available from ARB. Figure 2b shows the trend from 1989 through 2006. Also shown are regression lines and estimated means: 0.37 ppb for 1989, and 0.07 ppb for 2006, a reduction of over 80%. The reduction in cancer risk was 131 cases/million in 1990 vs. 25 cases/million in 2006, very similar to that for benzene. This is because, although the 1,3-butadiene concentrations are lower than benzene, the cancer risk per unit concentration is correspondingly higher.

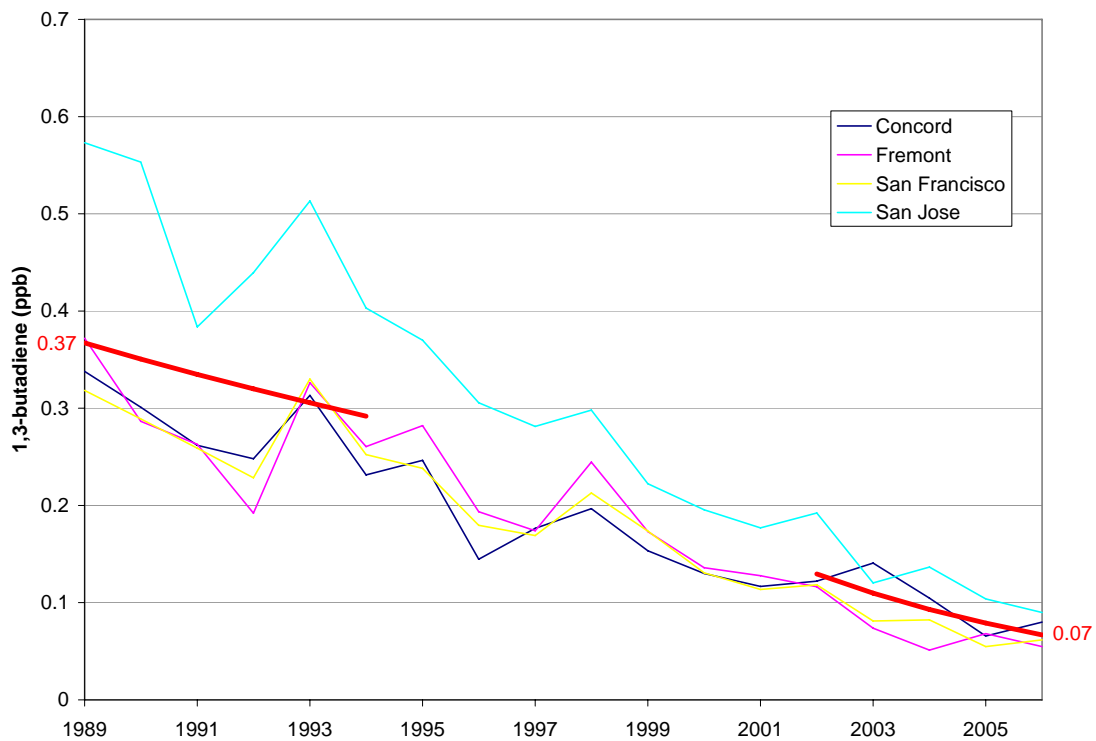


Figure 2b. Annual 1,3-butadiene at Bay Area sites.

## Carbon Tetrachloride

Virtually no carbon tetrachloride is being emitted today in the Bay Area. Concentrations represent a global background. Carbon tetrachloride was used in the early 20<sup>th</sup> century as a dry cleaning solvent and a refrigerant. However it has been found to have serious health effects, including affecting the central nervous system and degenerating the liver and kidneys. It is also a carcinogen, so it was banned in consumer products in the US in 1970, but continued to be used to manufacture Freon.

Carbon tetrachloride itself is a stratospheric ozone depleter. It was among the chemicals scheduled for phase-out by 1996 by the 1987 Montreal Protocol on Substances that Deplete the Ozone Layer. However, its atmospheric lifetime is approximately 50 years, thus much carbon tetrachloride remains.

Figure 2c shows Bay Area carbon tetrachloride concentrations from 1987 through 2006. There has literally been no trend in the concentrations over that time though it does show a modest reduction from early 1990s concentrations, perhaps reflecting global restrictions on production. Also note that Fort Cronkite concentrations are similar to those at other Bay Area sites, reflective of the fact that carbon tetrachloride in the Bay Area derives from a global background.

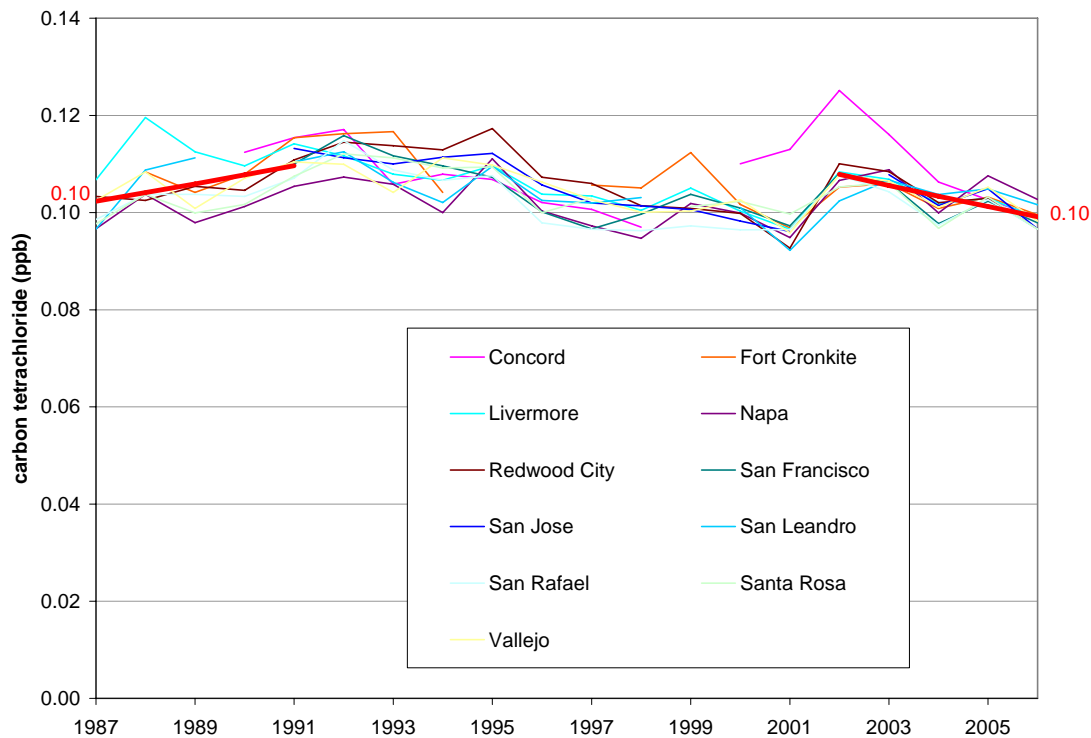


Figure 2c. Annual carbon tetrachloride concentrations at Bay Area sites.

## Formaldehyde

Formaldehyde is a carcinogen as well as causing respiratory symptoms and eye, nose, and throat irritation. Although formaldehyde is emitted directly as a result of incomplete combustion, its major Bay Area source, like ozone, is photochemical oxidation.

Figure 2d shows Bay Area formaldehyde concentrations from 1996 to 2006. There has been a statistically significant reduction of approximately one-third.

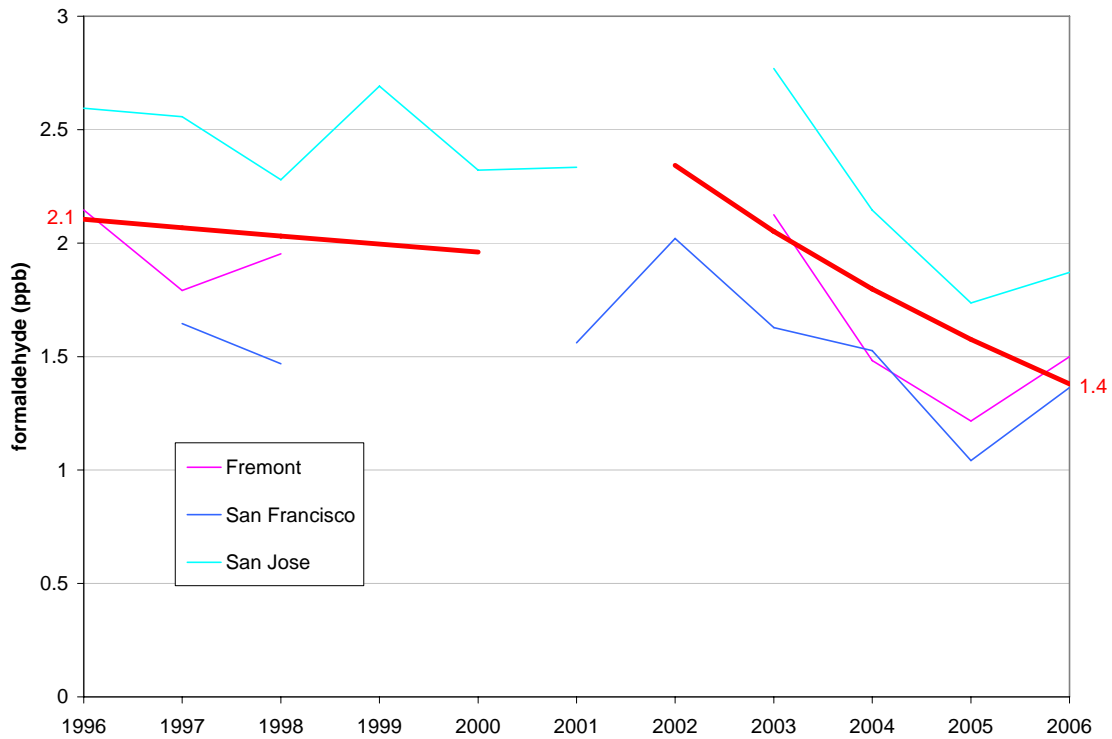


Figure 2d. Annual formaldehyde concentrations at Bay Area sites.

## Acetaldehyde

Acetaldehyde is similar to formaldehyde in that it is emitted directly, mainly from motor vehicles, but more is produced as a result of photochemical reactions in the atmosphere. Its health effects are also similar to formaldehyde, namely it is a carcinogen, and can cause irritation of the eyes, skin, and respiratory tract.

Figure 2e shows Bay Area acetaldehyde trends. There has been a modest, but statistically significant decrease in concentrations of roughly 20% from 1996 to 2006.

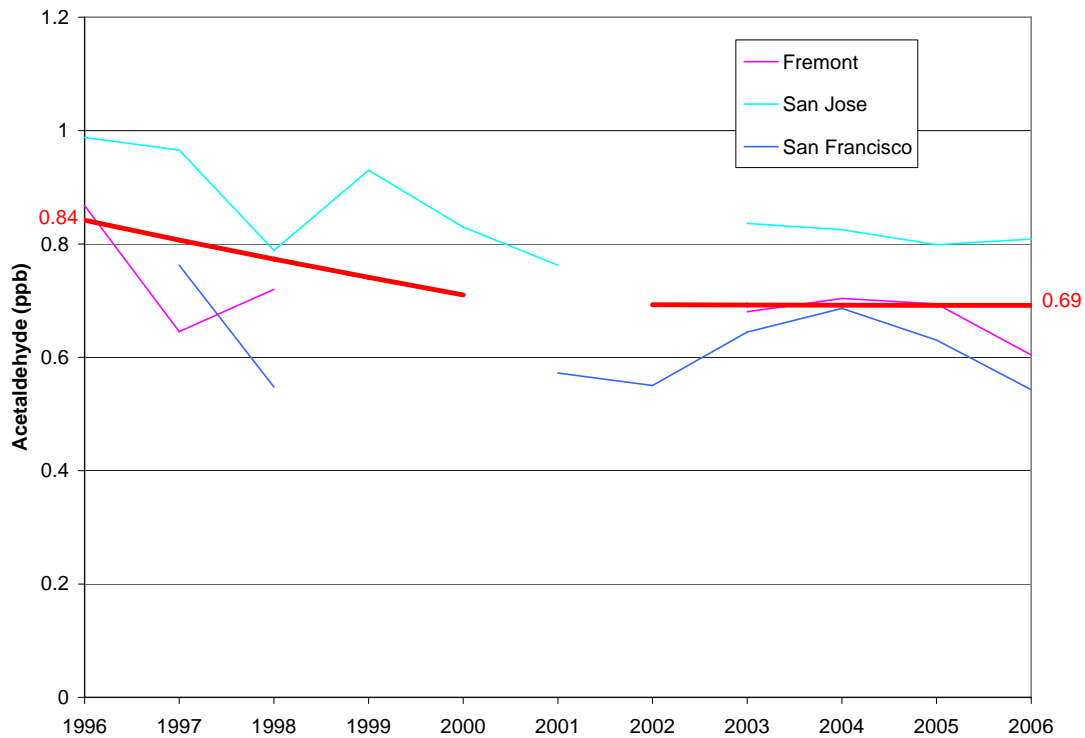


Figure 2e. Annual acetaldehyde concentrations at Bay Area sites.

## Methyl tert-butyl ether (MTBE)

Methyl tert-butyl ether, more commonly known as MTBE has been most frequently used as a gasoline additive. In the 1990s it was used to satisfy regulatory requirements for an oxygenate additive to promote more complete combustion. Unfortunately, MTBE is both an air and a water contaminant and, after considerable public outcry, its use was gradually phased out. In 2003, California was the first state to start replacing MTBE with ethanol.

Acute exposure of humans to high concentrations of MTBE can result in nausea, vomiting, dizziness, and sleepiness. Direct exposure to the skin and eyes can cause drying and irritation. It has been shown to cause cancer in tests on lab animals.

Figure 2f shows Bay Area MTBE levels since 1996. Note that concentrations have been completely below the 0.3 ppb LOD since 2004, so that the true mean concentration is unknown.

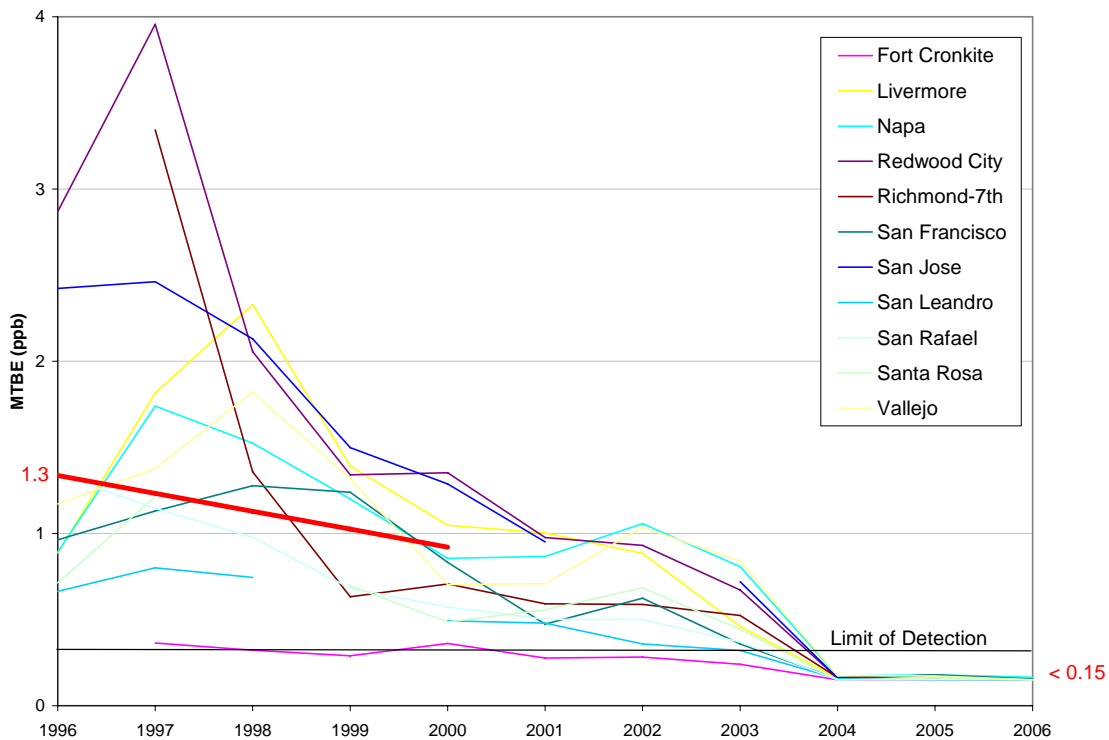


Figure 2f. Annual MTBE concentrations at Bay Area sites.

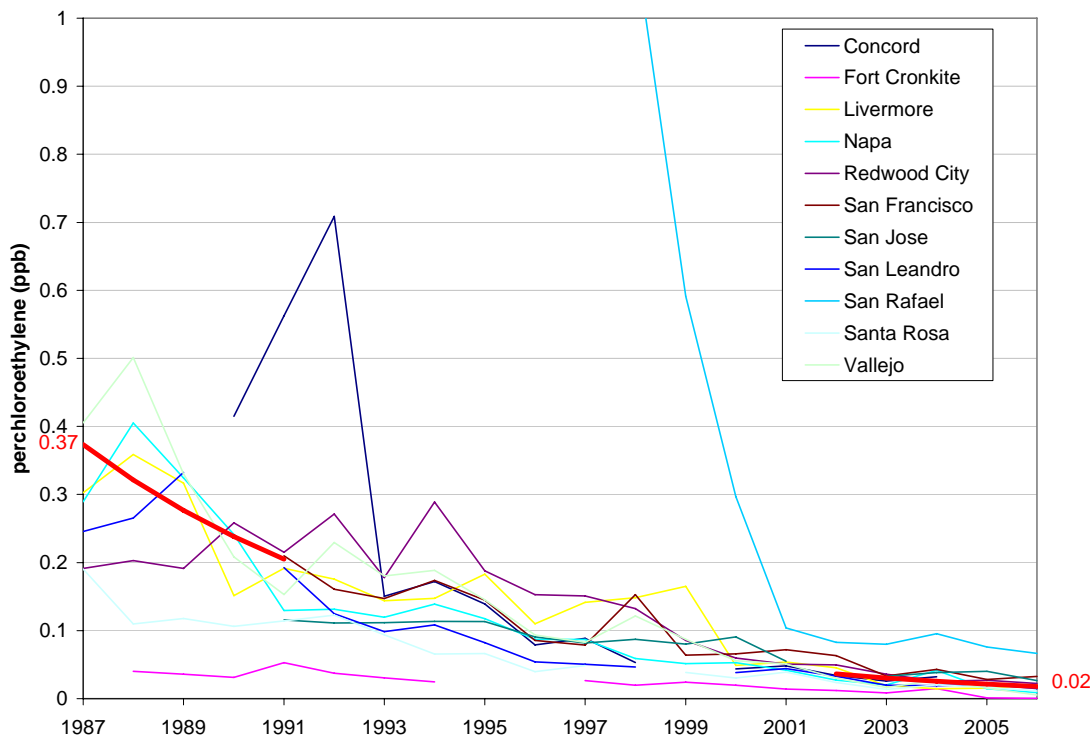


## Perchloroethylene

Perchloroethylene is most commonly used as a solvent. In the BAAQMD 2002 emissions inventory, dry cleaners produced 95% of Bay Area perchloroethylene point source emissions.

Perchloroethylene is a health hazard in several respects. It is a central nervous system depressant that can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in speaking and walking, unconsciousness, and death. Exposure to high concentrations may cause menstrual problems and spontaneous abortions. Workers have shown signs of liver toxicity and kidney dysfunction. It is considered a probable carcinogen by the US EPA, and the state of California has listed it as a carcinogen under the Toxic Air Contaminant Identification Program and for Prop 65. CARB adopted an ATCM in 1993 to reduce emissions from perchloroethylene dry cleaning operations and prohibited its use for automotive brake cleaning and degreasing products in 2001. In 1994, the BAAQMD instituted the nation's most stringent perchloroethylene rule, requiring secondary control machines and vapor barrier rooms.

Figure 2g shows a dramatic reduction in perc concentrations,<sup>1</sup> from an average of 0.37 ppb to 0.02 ppb.



**Figure 2g. Annual perchloroethylene concentrations at Bay Area sites. Not shown were San Rafael values were > 1 ppb for 1987-1997.**

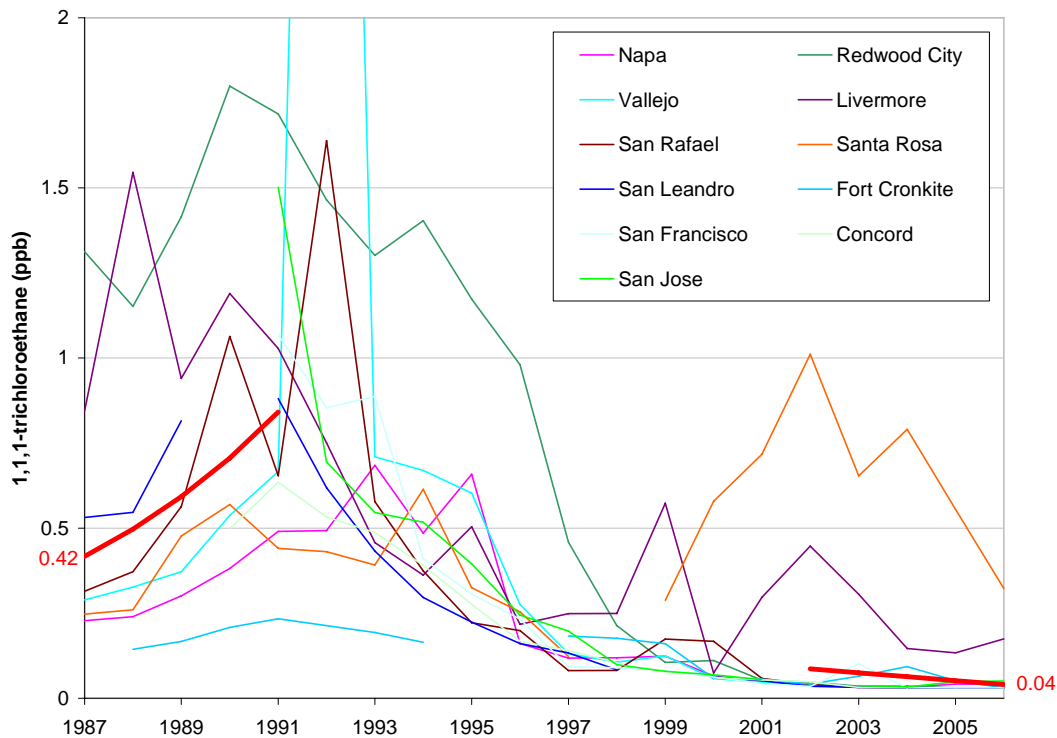
<sup>1</sup> The Figure shows only perc means  $\leq 1$  ppb from actual measurements, whereas the regression curves include means using sites with filled-in missing values. In the earliest years, Concord was predicted to have elevated perc levels, which pushed up the trimmed mean.

## 1,1,1-Trichloroethane (Methyl chloroform)

Methyl chloroform is widely used as an industrial solvent and degreaser, as a dry cleaning agent, as a component of aerosols formulations, and as a coolant and lubricant in metal cutting oils. In the 2002 emissions inventory, the largest Bay Area point source was the American Brass and Iron Foundry in Oakland. Methyl chloroform is considered a stratospheric ozone depleter and its use is being phased out.

Methyl chloroform is a central nervous system depressant, and is mildly irritating to the eyes and respiratory system in humans. Acute inhalation exposure in humans may cause hypotension, mild hepatic effects, dizziness, nausea, vomiting, diarrhea, and respiratory arrest. It is not considered a carcinogen by US EPA.

Figure 2h shows 1,1,1-trichloroethane concentrations at Bay Area sites for 1987 through 2006. There has been a large decrease in that period, from 0.42 ppb to 0.04 ppb, a reduction of approximately 90%, although there are individual sites exhibiting considerably higher concentrations.



**Figure 2h. Annual 1,1,1-trichloroethane concentrations at Bay Area sites. Not shown were Vallejo values were > 2 ppb for 1992.**

## Toluene

Gasoline evaporation and tailpipe emissions are the major sources of ambient toluene in the Bay Area. Toluene occurs naturally as a component of crude oil and is produced in petroleum refining operations. It is used in household aerosols, nail polish, paints and paint thinners, lacquers, rust inhibitors, adhesives, and solvent based cleaning agents. Toluene is also used in printing operations, leather tanning, and chemical processes.

In the 2002 emissions inventory, the main point sources were the Chevron Products Company, auto body shops, and landfills.

"Dysfunction of the central nervous system and narcosis are the major effects of acute exposure to toluene. Irritation of the skin, eye, and respiratory tract can also result." (OEHHA 1999)

Figure 2i shows annual toluene concentrations at Bay Area sites between from 1987 through 2006. During that time toluene concentrations dropped from about 3.7 ppb to 0.7 ppb, a reduction of about 80%. The figure shows that in 1987, all non-background sites had toluene levels exceeding 2.5 ppb. By 2006, toluene levels were below 1.5 ppb at all sites.

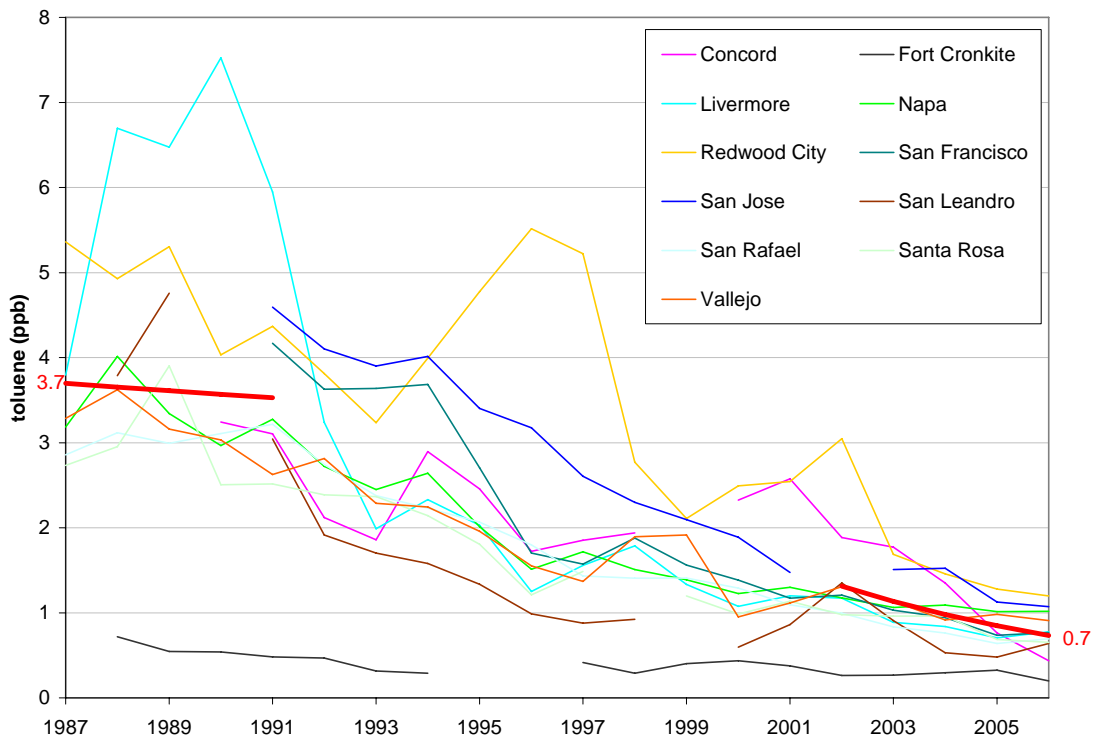


Figure 2i. Annual toluene concentrations at Bay Area sites.

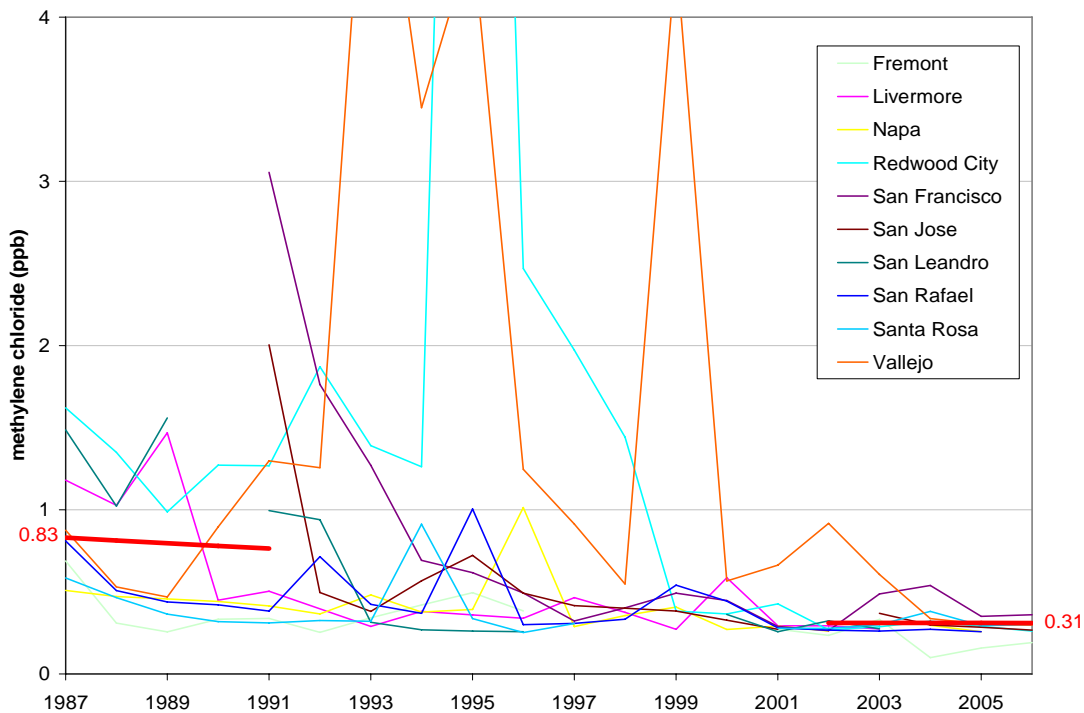
## Methylene chloride

"In California, paint removers account for the largest use of methylene chloride, which is the primary ingredient in paint stripping formulations used for industrial, commercial, military, and domestic applications. Because methylene chloride is also a constituent in many consumer products, including aerosol paints and automotive products, short-term indoor concentrations may be several orders of magnitude higher than the ambient concentrations. Many manufacturers of consumer products are voluntarily phasing-out their use of methylene chloride." CARB (2006)

In the Bay Area, the largest point sources are sewage treatment plants, furniture refinishers and landfills.

"Methylene chloride vapor is irritating to the eyes, respiratory tract, and skin. It is also a central nervous system depressant including decreased visual and auditory functions and may cause headache, nausea, and vomiting. At high exposures, methylene chloride can cause pulmonary edema, cardiac arrhythmias, and loss of consciousness. Chronic exposure can lead to bone marrow, hepatic, and renal toxicity." (CARB 1997)  
Methylene chloride is listed as a probable carcinogen by US EPA, and is on the Prop 65 list of cancer-causing compounds.

Figure 2j shows that, although there were a few exceptionally high annual methylene chloride concentrations, the Bay Area-wide concentrations have declined from 0.83 ppb in 1987 to 0.31 ppb in 2006, decline of 60%.



**Figure 2j. Annual toluene concentrations at Bay Area sites. Values > 4 ppb at Vallejo and Redwood City not shown.**

## Polycyclic aromatic hydrocarbons (PAHs)

PAHs are a set of hydrocarbons formed of multiple benzene rings. Several PAHs have been shown to be carcinogenic, the best-studied of which is Benzo(a)pyrene. Although Bay Area PAHs are emitted in petroleum refining, the vast majority derive from fossil fuel and wood combustion.

The set of PAHs that were measured in the Bay Area also included Benzo(b)fluoranthene, Benzo(k)fluoranthene, Benzo(ghi)perylene, Indeno(1,2,3-cd)pyrene, and Dibenz(a,h)anthracene. Because both the sources and the health effects of the PAHs are similar, the PAHs have been summed in this analysis, weighted by their relative cancer risk.

Figure 2k shows the toxicity-weighted PAH concentrations from 1995 through 2004. (ARB has since discontinued routine PAHs measurements.) PAH concentrations have been reduced from an average of  $0.15 \text{ ng/m}^3$  to  $0.09 \text{ ng/m}^3$ , a reduction of about 40%.

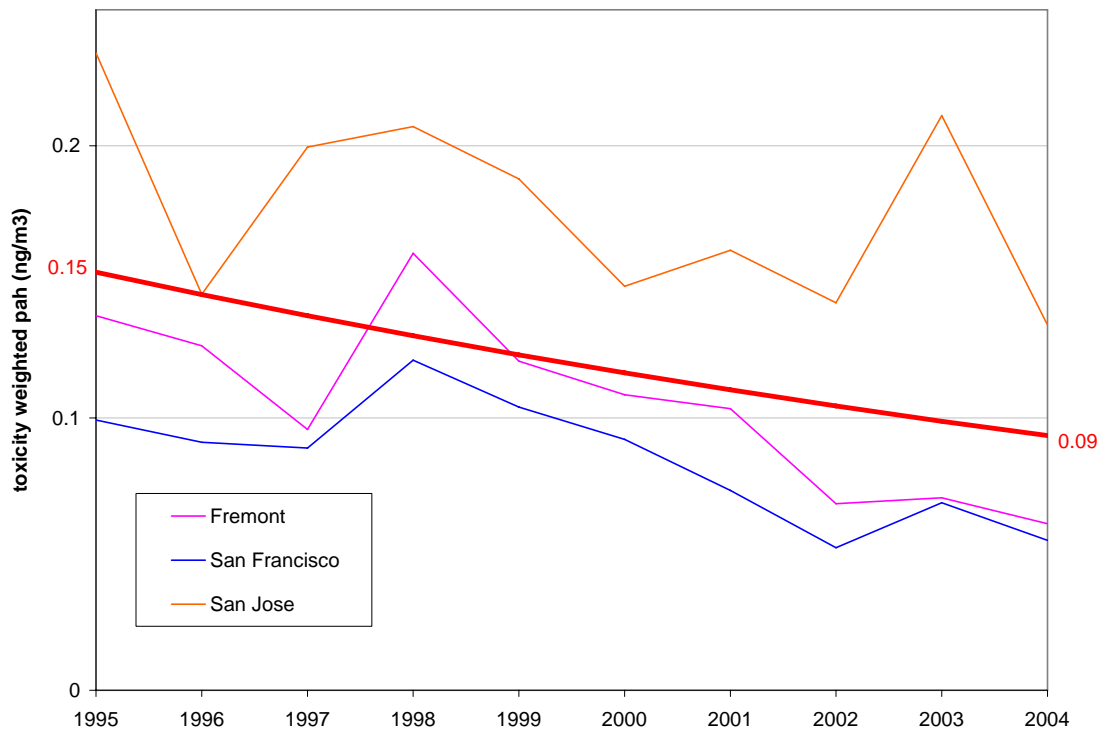


Figure 2k. Annual toxicity-weighted PAH concentrations at Bay Area sites.

## Hexavalent Chromium [Chromium (VI)]

The major source of hexavalent chromium in the Bay Area is motor vehicles. Chromium is a trace element in most crude oils, and may oxidize to hexavalent chromium during fuel combustion. Refinery hexavalent chromium emissions constitute about half of all Bay Area point source emissions. CARB adopted several ATCMs to reduce emissions of hexavalent chromium from plating operations (1988), cooling towers (1989), motor vehicle coating (2001), and thermal spraying (2005).

Chromium (VI) is among the larger sources of cancer risk in the Bay Area. Non-cancer effects of chromium (VI) exposure are renal toxicity, gastrointestinal hemorrhage, and intravascular hemolysis.

Figure 21 shows Bay Area chromium (VI) trends. Concentrations were reduced from an average of  $0.27 \text{ ng/m}^3$  in 1991 to  $0.07 \text{ ng/m}^3$  in 2006, a reduction of nearly 75%.

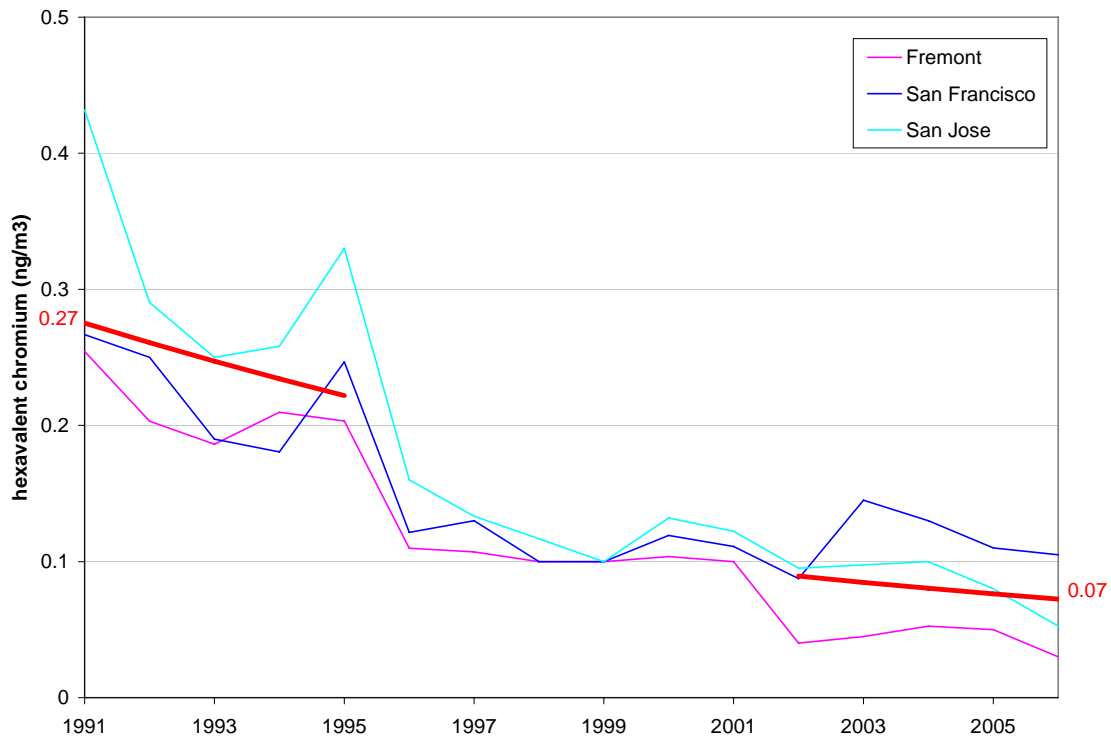


Figure 21. Annual hexavalent chromium concentrations at Bay Area sites.

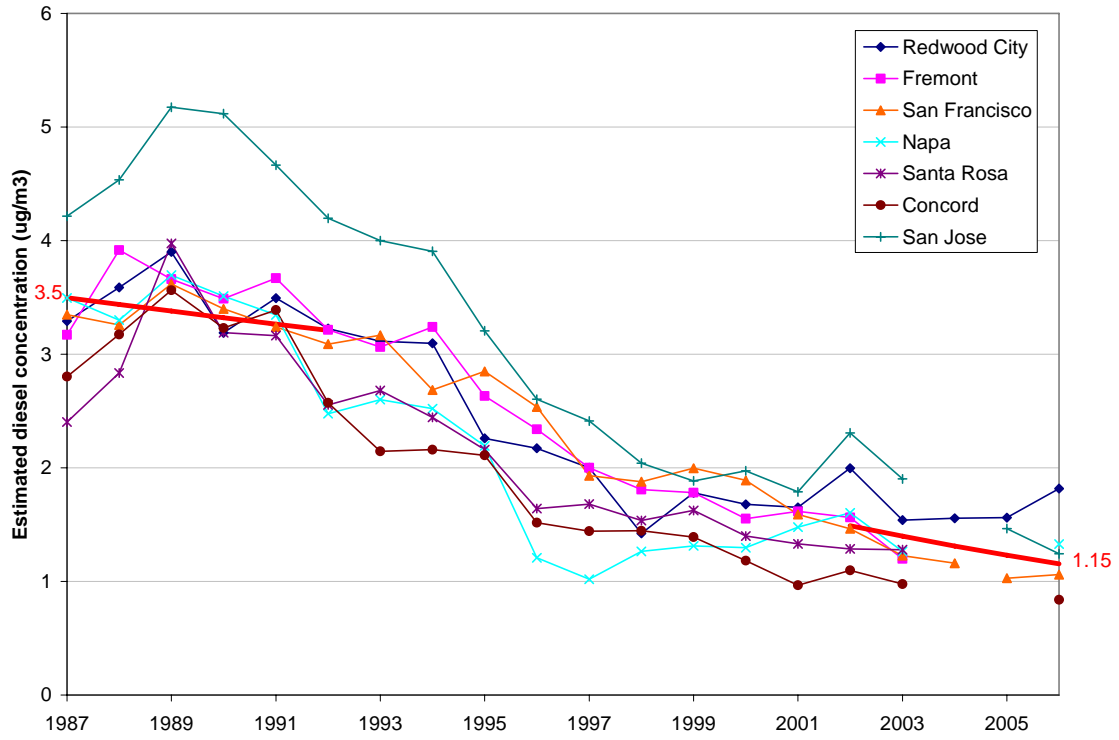
## **Diesel exhaust particulates**

Unlike the other toxics in this report, diesel exhaust is not a simple compound (or class of compounds, as are PAHs). It is a complex chemical mixture of gases, semivolatile compounds and particles. A number of diesel components are known carcinogens, including arsenic, benzene, nickel, 1,3-butadiene, benzo(a)pyrene, and formaldehyde. Yet, epidemiological evidence from workers with long exposure to diesel exhaust, as well as animal studies, suggested that the overall carcinogenicity of diesel was much larger than that computed from adding the risks from individual compounds. In other words, it is likely that there is synergy among the components of diesel exhaust that greatly magnifies its carcinogenicity. Based on our current knowledge, it is the largest source of ambient carcinogenicity in the air of the Bay Area.

Diesel fuel is burned in a wide range of sources, but the principal sources in the Bay Area are medium and heavy-duty trucks, and construction equipment.

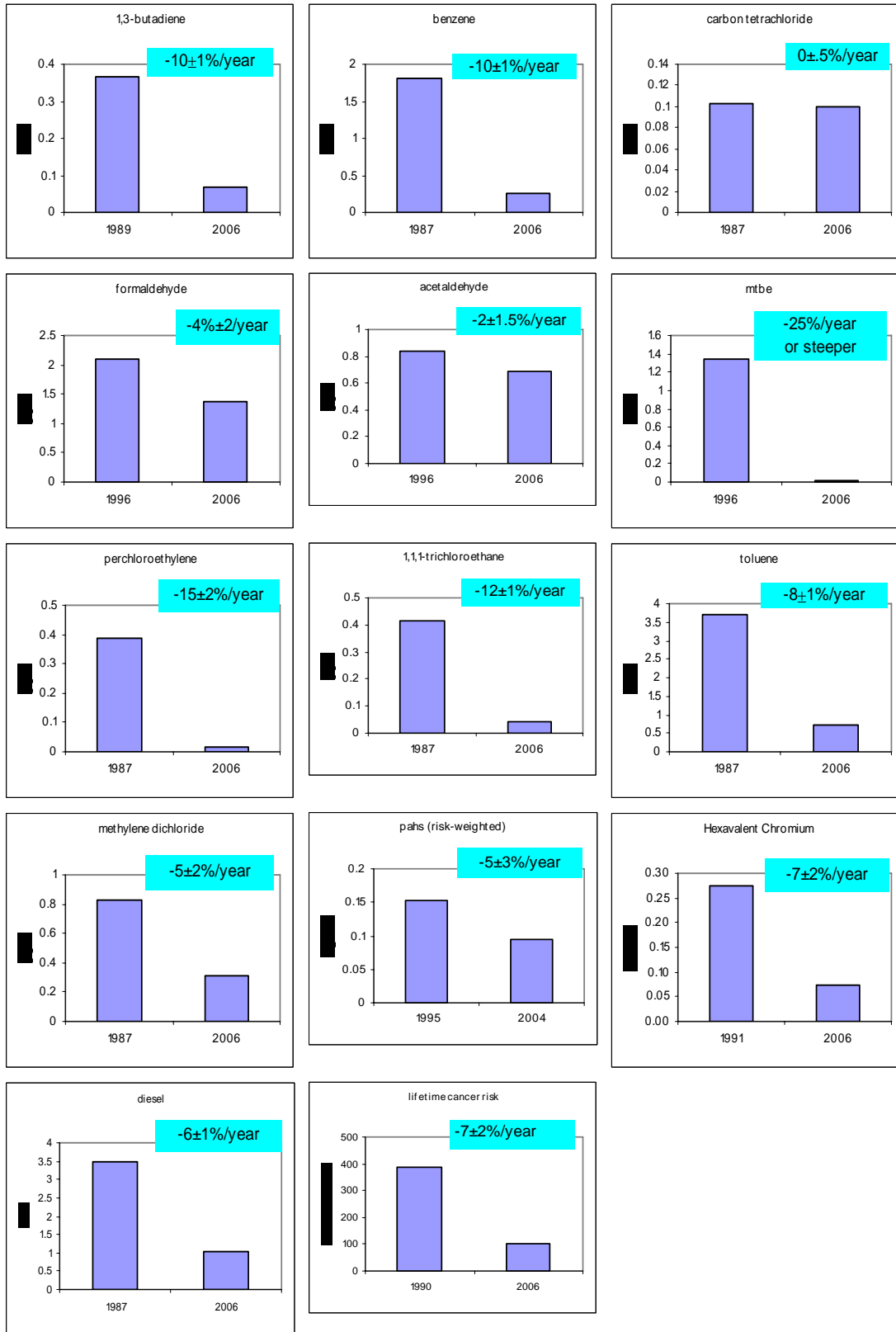
Another contrast with other compounds discussed here is that diesel exhaust cannot be measured directly. A substantial fraction of diesel exhaust is elemental carbon (EC) aka "soot", whereas it is not a large fraction for other major Bay Area PM<sub>2.5</sub> sources such as wood smoke and gasoline exhaust. Thus, EC is a surrogate for diesel exhaust. EC was measured at a few sites during special studies, but has only been measured routinely in the Bay Area since mid-2004. Previously, another measurement, coefficient of haze (COH) was made at a number of Bay Area sites.

Figure 2m shows diesel trends estimated from COH and EC. COH values were used through 2003 at all sites except San Francisco, 2004, and Redwood City, 2006. EC measurements were used for 2005 and 2006, otherwise. Estimated Bay Area average diesel exhaust dropped from 3.5 µg/m<sup>3</sup> in 1987 to 1.15 µg/m<sup>3</sup> in 2006.



**Figure 2m. Estimated diesel concentration trends, 1987-2006. Diesel concentrations for 1987-2003 estimated using COH, also SF for 2004, and Redwood City for 2004-06. Diesel concentrations at SJ 2005-06, Concord 2006, and SF 2005-06 estimated from EC measurements.**



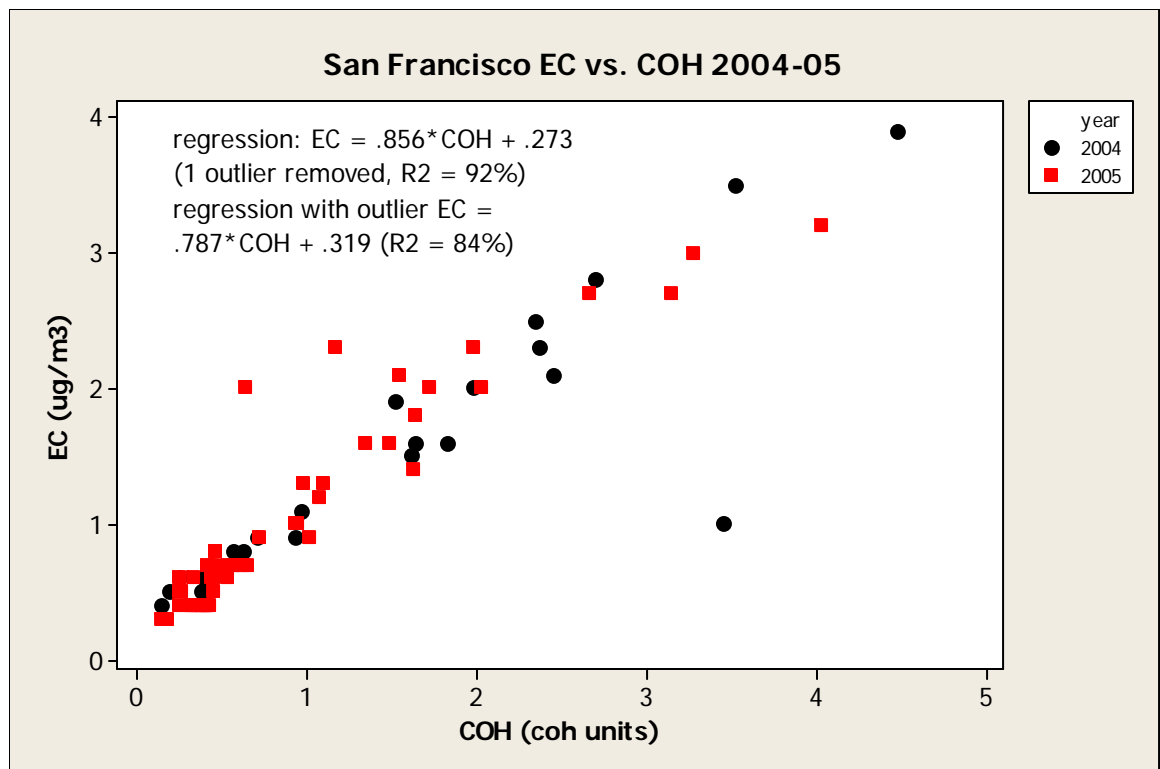


**Figure 1. Trends in individual toxics. Bay Area-average concentrations estimated for first and last year with data from sufficient sites. Annual compounded reductions also shown.**

## Appendix – Estimating diesel emissions and trend

As previously mentioned, ambient diesel measurements do not exist, but a surrogate, elemental carbon (EC), has been measured at several Bay Area sites. The conversion factor from  $\mu\text{g}/\text{m}^3$  EC to  $\mu\text{g}/\text{m}^3$  diesel is 1.04, so that the concentration of  $\text{PM}_{2.5}$  from diesel is approximately the EC concentration.<sup>2</sup> Unfortunately, routine measurement of EC has only recently begun; previous measurements were made only as part of special studies.

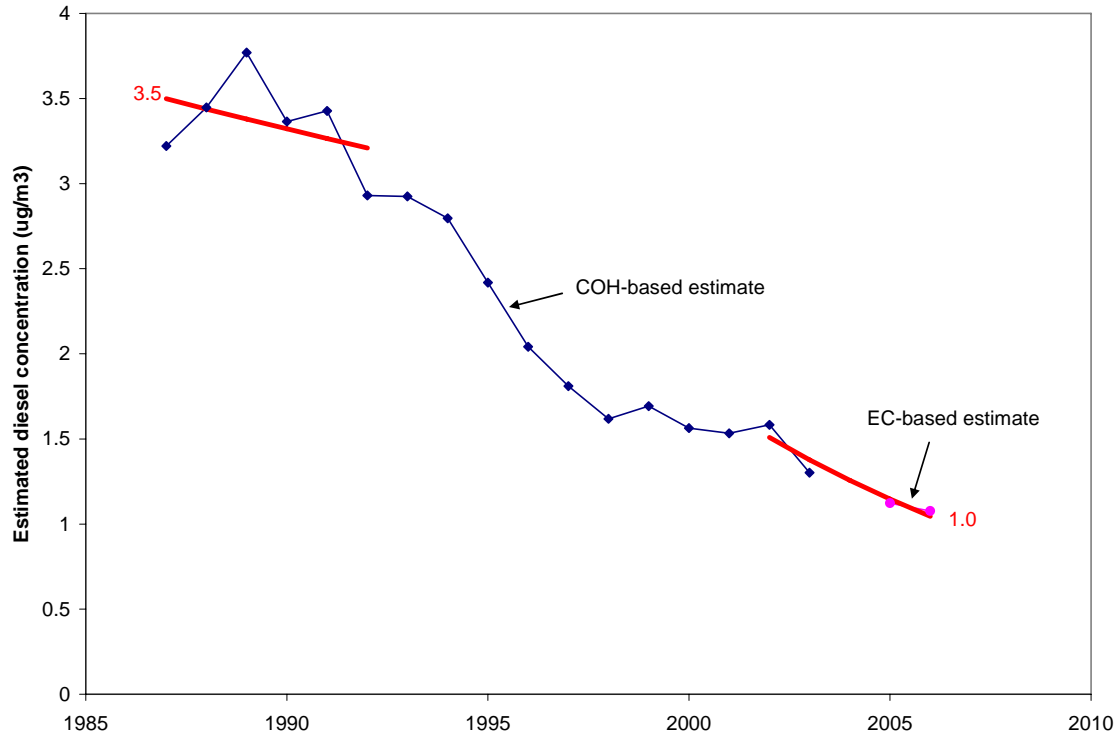
Using the limited matched data (for San Francisco in 2004 and 2005), an equation for estimating EC from COH was developed.<sup>3</sup>



Also shown in Figure 3 are log-scale regressions for 1987-1992 and 2002-2003, 2005-06. The regression predictions are  $3.5 \mu\text{g}/\text{m}^3$  for 1987 and  $1.0 \mu\text{g}/\text{m}^3$  for 2006, a reduction of 70%.

<sup>2</sup> The conversion factor comes from a South Coast study. There are other sources of EC, including gasoline exhaust and wood burning. Thus, if these are significant sources in an area, assuming a 1-to-1 ratio may cause diesel to be overestimated.

<sup>3</sup>  $\text{EC} = .856 \cdot \text{COH} + .273$ , adjusted  $R^2 = 92$ .



**Figure 3. Estimated diesel trend. The values for 1987-2003 are based on the trimmed COH mean of seven sites, using the formula  $\text{diesel} = .854 \cdot \text{COH} + .275$ . The values for 2005 and 2006 are based on mean EC at 5 sites, with the assumption  $\text{diesel} = \text{EC}$ .**

There is considerable uncertainty in the trend estimate, although more in the absolute level than the relative change. The estimate of diesel involves several levels of estimation – the conversion of EC to diesel, the conversion of COH to EC, and the uncertainty related to the measurements themselves from measurement error, atmospheric variation, and choice of sampling location. Quantifying the effect of these uncertainties on trend is complex. The formula for standard error of the prediction from a regression captures the last source of error but not the other two. The regression error is about 10%. Somewhat arbitrarily I've assigned a 10% error to trend uncertainty caused by the COH/EC conversion, and a 15% error caused by the EC/diesel conversion. Combining the root mean square of these errors yields a total error of 21%.

### Estimated Risk Trend

Unit cancer risk values were obtained from OEHHA/ARB 2002. Lifetime cancer risks were estimated based on reductions from 1990 and 2006, using multi-site means from those years except for PAHs, where the 1990 and 2006 means were extrapolated from the estimates for 1995 and 2004; MTBE, where the 1990 mean was assumed to be zero; acetaldehyde and formaldehyde where the 1990 mean was extrapolated from the estimates for 1996 and 2006. The estimated risks were 1,310 in a million for 1990 and 420 in a million for 2006, a reduction of approximately two-thirds.

It should be noted that there is considerable uncertainty in these risks. First, because of detection limitations, several monitored carcinogens are not included, such as hexavalent chromium, vinyl chloride, ethylene dibromide, and ethylene dichloride. However, even using upper bounds for the measurements of these compounds, the additional risks are small relative to the total risk and would not influence the reduction estimate substantially.

A larger source of uncertainty is the diesel risk, which constitutes over 70% of the total risk. There is considerable uncertainty in the absolute concentrations, and hence, in its contribution to each risk estimate. There is also uncertainty in its estimated trend, since the quantities measured to estimate it, EC and COH, are influenced by other sources. Another large source of uncertainty is that there may be other sources where, as with diesel, the total risk is much higher than the sum of the risks from individual components. Possibilities include wood smoke and gasoline exhaust.

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