Schools Air Toxics Monitoring Activity (2009)
Uses of Health Effects Information in Evaluating Sample Results

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1. Introduction

EPA has developed an initiative to implement Administrator Jackson's commitment to assess potentially elevated ambient (outdoor) concentrations of air toxics at some of our nation's schools (generally described at www.epa.gov/schoolair). This monitoring activity is described in the project monitoring plan and quality assurance project plan (available on the schools web site: http://www.epa.gov/schoolair/techinfo.html). This monitoring is targeting key hazardous air pollutants (HAPs or air toxics) at each site (i.e., those identified by either the Risk-Screening Environmental Indicators (RSEI) Model or the National Air Toxics Assessments (NATA) national-scale assessment as the local risk drivers, or by information provided by EPA, Regions and States). Key pollutants differ among the monitored schools based on what this background information indicated regarding pollutant emissions, and potential air concentrations and risk in each area. The monitoring data and other information collected for each school during this initiative will allow us to assess levels of the key pollutants[a] occurring at these sites including the potential for contributions from nearby sources.

The ambient air monitoring data collected at each site (along with other site- or source-specific information as well as information on typical regional or national levels) will be used to facilitate decisions regarding priority for and type of any follow-up actions near each school. In considering the pollutants monitored at each site and potential follow-up actions, EPA recognizes that data from ongoing monitoring programs (e.g., monitoring at the National Air Toxics Trends Sites or NATTS) indicate that levels of some pollutants are commonly higher in urban areas than elsewhere. While in some locations these pollutants (e.g., benzene and acrolein) may be associated with specific industrial facilities, they are also associated with mobile source emissions and are commonly occurring at sites near urban areas or near large roadways and other transportation facilities (http://www.epa.gov/otaq/toxics.htm). Accordingly, EPA has taken several actions to reduce mobile source emissions nationally and maintains several ongoing programs aimed at achieving reductions in ambient air concentrations of these hazardous air pollutants from mobile sources (http://www.epa.gov/otaq/toxics.htm#epamsat). In addition to these national steps that are expected to reduce concentrations on a local basis as they are implemented, there are steps that individuals and communities can take to achieve additional reductions (http://www.epa.gov/otaq/consumer.htm).

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[a] At each site, monitoring has been targeted to get information on the key HAPs about which the background information indicated a potential for concern. In analyzing air samples for these key pollutants, samples are also being analyzed for some additional pollutants that are routinely included in the analytical methods for the key pollutants. While analysis of the dataset collected for each school will focus attention on the key HAPs, results for other HAPs monitored at the school will also be reviewed consistent with the approach described in this document.

EPA/OAQPS will evaluate the full dataset for the key pollutants at each school, in light of information particular to the school and its location, in order to identify the school's priority for follow-up activities. That evaluation will include consideration of several analyses and factors. These will generally include the site-specific ambient air measurements along with other site and source-specific information, as well as, information on typical national and regional levels and long-term health risk-related exposure concentrations for monitored pollutants. Prior to the availability of a full dataset for each location, EPA will be making available on the public web site (www.epa.gov/schoolair) an interim presentation, including individual sample measurements for key pollutants. As part of this interim presentation, EPA also intends to provide, as described in section 3 below, conservatively developed health risk-related individual sample screening levels for all key pollutants. In instances where individual sample concentrations occur above these sample screening levels, the pollutant-specific health effects information will be evaluated along with information concerning the sample collection and potential sources of the monitored pollutant.[b] Additionally, for some nationally key pollutants, such as those associated with mobile sources, EPA may provide information on nationally common concentrations for reference. This document, prepared for reference by the EPA schools air toxics team, describes the various uses of health effects information in evaluating sample results, using screening or comparison levels and in further investigation of results higher than such screening or comparison levels.

2. Uses of Health Effects Information

There are several uses of health effects information for the pollutants monitored in this project. As sample results are received from the analytical laboratory[c], EPA will be reviewing the data to identify any situations where additional attention may be appropriate to confirm sample results and to assess the potential for immediate health concerns. To facilitate the identification of sample results to receive such additional attention, we have developed individual sample screening levels (described below in section 3).[d] Any sample results higher than these sample screening levels will be further considered in light of chemical-specific information on health effects (section 4), along with information concerning the sample collection and potential sources of the monitored pollutant. Lastly, the multi-faceted analysis of the full monitoring dataset at a site, which will not entail a detailed risk assessment, will include consideration of projected estimates for the long-term average concentration of the monitored air toxics in light of health-based comparison levels. Accordingly, long-term comparison levels have been developed for use in the full dataset analysis (described below in section 5).

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[b] While our main focus is on the key pollutants at each school (i.e., those pollutants for which background information indicated emissions and potential concentrations that supported the monitoring activity), we are screening sample results for other pollutants monitored using individual sample screening levels and approach described in sections 3 and 4 below.

[c] A quality assurance and quality control program is a component of the monitoring program that is followed by the sample collection team and analytical laboratory in producing sample results.

[d] At this step we are also reviewing national levels of some air toxics in recognition of their common occurrence at levels of risk-related interest nationally or in some regions. These typical ambient levels are not the focus of nor are they otherwise discussed in this document.

3. Development of Individual Sample Screening Levels

This section describes the approach employed for developing screening-level values for use in reviewing individual sample measurements. Prior to developing or identifying these screening levels, priority was given to recognition of relevant and appropriate air standards or regulations, such as in the case of the recently updated National Ambient Air Quality Standards (NAAQS) for lead. For the remaining chemicals, health risk-related sample screening levels were calculated using EPA risk assessment guidance and precedents. The objective in developing individual sample screening levels for these air toxics is to identify air concentrations to which continuous exposures over a few days would be unlikely to be associated with appreciable risk of adverse health effects. These values are intended to help EPA to identify contaminants for which closer attention may be appropriate to confirm sample results and to assess the potential for any immediate health concerns. These sample screening values are considered reasonably conservative for the presumed exposure scenario at hand, such that individual results above these concentrations should not be presumed to be cause for alarm. These sample screening levels are not intended to define clean up or action levels.

As the ambient air samples are collected over a 24-hour period, the exposure duration of interest in developing individual sample screening levels is on the order of a few days (short-term). As described below, established reference concentrations, dose-response assessments or other similar benchmarks for a time period of this length are not available for all of the monitored pollutants. In identifying surrogate exposure levels for use in lieu of short-term values, we have chosen to consider longer-term (intermediate) screening levels for which the exposure duration extends from weeks to a year. These intermediate screening levels have been developed consistent with the use of such levels in the 2005-06 monitoring conducted in association with activities in response to hurricane Katrina in the Louisiana, Mississippi, and Alabama.[e]

In summary, the individual sample screening levels presented here represent short-term (or intermediate) duration inhalation exposure estimates that are unlikely to be associated with appreciable risk of adverse health effects for continuously exposed populations (including sensitive subgroups). These levels are not designed to predict the occurrence of effects, and individual sample measurements greater than the screening levels do not imply an immediate health threat. Rather, findings of individual sample measurements above these screening levels will receive additional attention to confirm sample results and to assess the potential for any immediate health concerns (as described in section 4 below).

At each site, monitoring has been targeted to get information on a limited set of key hazardous air pollutants (HAPs or air toxics)[f]. These pollutants are the primary focus of our

[e] Intermediate duration values were selected in the Katrina recovery monitoring activity with the presumption that a one-year exposure duration would conservatively and reasonably account for the potential of burning and/or demolition activities to go on longer than the presumed initial 3-month estimate for such activities.

[f] Section 112(b) of the Clean Air Act identifies 189 hazardous air pollutants, three of which have subsequently been removed from this list. These pollutants, commonly called "air toxics" are the focus of regulatory actions involving stationary sources described by CAA section 112 and are distinguished from the six pollutants for

monitoring activities at a site and a priority for us based on our emissions, modeling and other information. Accordingly, the sample screening levels for the full set of these key pollutants are presented in the first table at the end of this section (Table 1). In analyzing air samples for these key pollutants, we will also be analyzing samples for some other pollutants (both HAPs and others) that can be economically measured at the same time. Sample screening levels for these additional HAPs are presented in Appendix A. Similar information for pollutants that are not HAPs is described in Appendix B.

Individual sample screening levels were set for the monitored HAPs, using the following prioritized approach.[g]

1. In considering a sample screening level for lead, preference was given to use of the National Ambient Air Quality Standards. A review of the NAAQS for lead was completed in October 2008. The revised primary standard[h] reflects the health effects evidence available in that review and provides increased protection for children and other at-risk populations against an array of adverse health effects, most notably including neurological effects in children, including neurocognitive and neurobehavioral effects (73 FR 66964).

2. For pollutants for which ATSDR has developed acute Minimal Risk Levels (MRLs), the individual sample screening levels were set to these MRLs. ATSDR defines each MRL as "an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure" (http://www.atsdr.cdc.gov/mrls/). Acute MRLs are considered appropriately protective comparison levels for screening of individual samples in this project because they were developed to be protective of short-term continuous exposures lasting up to 14 days (24 hours each day).

3. In looking for surrogates for chemicals for which no ATSDR acute MRL or similar duration value was available, we looked to longer- *vs* shorter-term duration exposure levels in keeping with the description that the sample screening levels be reasonably conservative for the presumed exposure scenario and that the exposure duration of interest is on the order of a few days.[i] Thus, for the remaining pollutants for which ATSDR has not established acute MRLs and for which longer-term risk-related exposure concentrations were available, surrogates for short-term screening levels were developed from longer-term risk-related exposure concentrations. The approach for these surrogates focuses on what are termed "intermediate" duration exposures, which range up to one year duration. Thus, when used in lieu of short-term values

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which criteria and national ambient air quality standards (NAAQS) are developed as described in section 108. One of the criteria pollutants, lead, is also represented, as lead compounds, on the HAP list.

[g] Although this project targets HAPs, the analytical methods employed include several non-HAPs, such that data are being generated for those pollutants as well. Although the air toxics to which EPA assigns greatest concern nationally are the HAPs, a health risk-related screening was also applied to the individual sample results for the non-HAPs. The screening levels employed for this purpose are identified in Appendix A.

[h] The NAAQS for lead is 0.15 $\mu g/m^3$ in total suspended particles, applied as an average over a 3-month period.

[i] Other short-term duration exposure levels presented in OAQPS Table 2 include the California Environmental Protection Agency's acute reference exposure levels, the exposure durations for which are 24 hours for most pollutants and somewhat shorter than that for a few.

these values for most pollutants are considered to inherently have an extra level of conservatism than would short-term values had they been available.

The approach for deriving intermediate screening levels described here is that which EPA relied upon in the Hurricane Katrina ambient air monitoring activity. Preliminary screening values were first identified separately for (a) effects for which reference concentrations have been developed (usually considered to have nonlinear or "threshold" exposure-response relationships) and, (b) if pertinent to the pollutant, linear cancer risk (built upon EPA's default carcinogen assumption, which is accepted in lieu of a conclusion to the contrary, of some cancer risk associated with any level of exposure). The final intermediate screening level is the lower of the subsequent two values (described in sections (a) and (b) below).

a. To develop a preliminary screening level for "threshold" effects for these pollutants, the ATSDR intermediate inhalation minimal risk level was used (MRL_{inter}; see http://www.atsdr.cdc.gov/mrls.html). Values for the MRL_{inter} are developed to be protective of exposures ranging from two weeks to a year.

   When the MRL_{inter} was not available for a compound, a value of ten times the chronic Reference Concentration (RfC) or other similar value[j] was used instead as an estimate of the intermediate (subchronic) comparison value. Specifically, the preliminary screening level (SL) for a threshold chemical (in the absence of a MRL_{inter}) is:

   $$\mathbf{SL_{threshold} = (10)(RfC)}$$

   The application of a factor of 10^k is commonly used by the Superfund program for making decisions regarding emergency removal actions using a time frame with an upper bound of approximately a year (USEPA, 1997) to account for the fact that chronic toxicity values (e.g., RfCs) are being applied to the less than chronic (sub-chronic) time periods of expected exposure.

b. For linear carcinogenic compounds, the linear cancer risk-based screening levels were set at concentrations for which the upper-bound lifetime probability of developing cancer would be one hundred-in-a-million ($1x10^{-4}$), if exposure occurred continuously at the concentration for one year.[l] The one-

[j] After EPA IRIS chronic RfCs (referred to as "chronic RfCs" in Tables 1-3 in this document), the next source consulted was ATSDR chronic MRLs, then California EPA chronic Reference Exposure Levels, and lastly HEAST chronic RfCs. This hierarchy for identification of chronic reference concentrations is consistent with that used in Table 1 of OAQPS toxicity values available on the EPA web site (http://www.epa.gov/ttn/atw/toxsource/summary.html). Information from that table was updated as needed.

[k] In recognition of the uncertainty of this approach for developing an intermediate duration screening level from a value derived for chronic exposures, we have deviated from this in some instances. For example, where the chronic RfC was set with human data and with a cumulative uncertainty factor equal to or less than 10), the factor of 10 was not applied and the chronic RfC was used as is. This was the case for cobalt, beryllium and selenium.

[l] The cancer risk level used here ($1x10^{-4}$) reflects the upper bound of the Agency's traditional acceptable risk range (10^{-4} to 10^{-6}), which plays a role in both the hazardous air pollutant and Superfund programs. For example, in the 1989 setting of the "benzene NESHAP", which has a precedent-setting role in the HAP program, EPA stated that an additional cancer risk (associated with air toxics emitted from a stationary source)

year exposure duration is consistent with the time frame of the intermediate MRLs. Specifically, the preliminary linear cancer risk-based screening levels were calculated as:

$$\mathbf{SL_{cancer} = (1E\text{-}04/IUR)(70)}$$

where the IUR is the inhalation unit risk factor (or slope), expressed as $(\mu g/m^3)^{-1}$. The IURs used in this exercise are those in the EPA OAQPS Toxicity Table 1 (http://www.epa.gov/ttn/atw/toxsource/summary.html).[m]

Table 1. Individual Sample Screening Levels for Key (aka "driver") HAP Analytes.

Analyte	Individual Sample Screening Level[1]	Value from which Screening Level Derived
Metals[2]	ng/m^3	
Arsenic (PM_{10} focus)	150 (I)[3]	chronic REL
Chromium-hexavalent	580 (I)	IUR
Cobalt (PM_{10} focus)	100 (I)	chronic MRL
Lead[4]-TSP	The lead NAAQS is a rolling 3-month average of 150 (I) ng/m3 lead in TSP.	
Manganese (PM_{10} focus)	500 (I)	chronic RfC
Nickel (PM_{10} focus)	200 (I)	inter. MRL
VOCs/Carbonyls	µg/m^3	
Acetaldehyde	90 (I)	chronic RfC
Acrolein	7	acute MRL (0.003 ppm)
Benzene	30	acute MRL (0.009 ppm)
1,3-Butadiene	20 (I)	chronic RfC
4,4'-methylenedianiline	15 (I)	IUR
Naphthalene	30 (I)	chronic RfC
POM-PAHs[5]:	ng/m^3	
Benzo(a)pyrene	6,400 (I)	IUR
Benzo(a)anthracene	64,000 (I)	"
Benzo(b)fluoranthene	64,000 (I)	"
Benzo(k) fluoranthene	64,000 (I)	"
Chrysene	640,000 (I)	"
Dibenz(a,h)anthracene	5,800 (I)	"
Indeno(1,2,3-cd)pyrene	64,000 (I)	"
Diisocyanates	µg/m^3	
1,6-hexamethylene diisocyanate	0.2 (I)	inter. MRL
Methylenediphenyl diisocyanate	6 (I)	chronic RfC
2,4-toluene diisocyanate	0.7 (I)	chronic RfC

of 100-in-a-million should ordinarily be the upper end of the range of acceptability, with risks increasing above this benchmark becoming presumptively less acceptable under the HAP program. Additionally, in the Superfund program, acceptable exposure levels for known or suspected linear carcinogens are generally those that, using information on the relationship between dose and response, represent an excess upper-bound lifetime individual cancer risk between 10^{-4} and 10^{-6} [e.g., see 40 CFR 300.430(e)(2)(i)(A)(2)].

[m] Where the OAQPS table on web did not represent the most current information, the information was updated.

Notes for Table 1

[1] Values derived for this document, or for which units conversions were performed are assigned the same number of significant figures as those specified in the source value.

[2] Metal samples will be of 2 different types: PM_{10} and total suspended particles (TSP). With the exception of lead (for which the NAAQS was developed with explicit recognition of non-inhalation exposure pathways), metals screening levels are more suited for use with the concentration of metal in particles captured in a PM_{10} sample.

[3] The superscript (I) indicates where an intermediate duration exposure screening levels has been assigned as a surrogate in lieu of a short-term screening level.

[4] This value for lead is the level of the NAAQS, which is in terms of a 3-month average level of lead in TSP.

[5] PAHs are components of the HAP polycyclic organic matter (POM). The PAH analytes listed here are those which EPA has identified as carcinogens, and for which inhalation unit risk estimates are available (from the OAQPS Toxicity Table, per section 3.3.b above)). In assessing carcinogenicity for chemicals concluded to be carcinogenic by a mutagenic mode of action and for which early lifestage dose-response information is not available, EPA recommends use of age-dependent adjustment factors (ADAFs) which effectively increase the potency when applied to the first 16 years of age (USEPA, 2005). Dibenz[a,h] anthracene (DBA) and benzo[a]pyrene (BaP), and the other PAHs for which IURs are indexed to that of BaP are considered by EPA to act by mutagenic modes of action (Farland, 2006). In considering potential use of ADAFs in the context of the individual sample screening levels, we recognize several considerations: (1) the objective for the sample screening levels is a short-term exposure estimate (on the order of weeks), (2) application of EPA's traditional cancer risk assessment methods to such short periods is inherently uncertain, (3) the sample screening levels for these PAHs are actually surrogates for short-term levels and have been derived for a 1-year exposure, (4) the full data analysis for each school (see section 5) will consider potential risk of chronic exposure, and (5) if in the calculation of these levels, we had substituted a short-term exposure duration (e.g., 2 weeks), their values would be increased by a factor of 20, which is more than twice the value of the highest ADAF. Thus, rather than implement a cancer risk calculation specific to a 2-week duration exposure, which is not generally recommended by EPA guidance, we have retained the 1-year values and recognize that they are appropriately protective of shorter-term exposures with a potential for increased early-life susceptibility.

4. Pollutant-specific Health Effects Information

As mentioned in the previous sections, findings of sample measurements above the individual sample screening levels will be given closer attention to confirm sample results, to assess a potential for unusual circumstances at nearby sources, and to assess the potential for any immediate health concerns for the exposed population. Additionally, longer-term concentration estimates developed in the analysis of the full set of results for a school (see section 5 below) that are above long-term comparison levels (particularly in the case of noncancer-based comparison levels) will also be given close attention. In assessing the potential for any health concerns regarding the reported levels, we will be considering the health effects and toxicity information available for that pollutant and the details associated with the derivation of the particular screening or comparison level, as well as the likely exposure circumstances at the monitored location. The type of health effects and toxicity information to be considered for a particular pollutant would be expected to include the type of effects and exposure conditions (including duration, frequency and inhalation concentrations) with which they are associated, as well as the confidence level and type and magnitude of uncertainty associated with applicable reference or toxicity values.

Sources of information on toxicity and health effects that may be consulted include, but are not necessarily limited to, materials developed by:

- ATSDR, including their *Toxicological Profiles, ToxGuides™, Case Studies in Environmental Medicine, Medical Management Guidelines for Acute Chemical Exposures* and *Public Health Statements*;
- EPA, including the OAQPS Health Effects Notebook, the OAQPS Air Toxics Risk Assessment library and toxicity tables, the Integrated Risk Information System, and technical support documents for acute exposure guideline levels; and
- American Industrial Hygiene Association, the American Conference of Governmental Industrial Hygienists, the National Institute of Occupational Safety and Health and the Occupational Safety and Health Administration.

5. Development of Long-term Comparison Levels

5.1 Background and Objectives for Use of Comparison Levels

The majority of schools being monitored in this initiative were selected based on modeling analyses that indicated the potential for annual average air concentrations of some HAPs (air toxics) to be of particular concern based on approaches that are commonly used in the air toxics program for considering potential for long-term risk. For example, these analyses suggested annual average concentrations of some air toxics to be greater than long-term risk-based concentrations associated with an additional cancer risk greater than 1-in-10,000 or a hazard index on the order of or above 10. These long-term risk-based concentrations presume continuous (all-day, all-year) exposure over a lifetime. To make projections of air concentrations, the modeling analyses combined estimates of air toxics emissions from industrial, motor vehicle and other sources, with past measurements of winds, and other meteorological factors that can influence air concentrations, from a weather station in the general area. In some cases, the weather station was very close (within a few miles) but in other cases, it was much further away (e.g., up to 60 miles), which may contribute to quite different conditions being modeled than actually exist at the school. The modeling analyses are intended to be used to prioritize locations for further investigation.

The primary objective of this initiative is to investigate - through monitoring air concentrations of the key HAPs at each school over a 2-3 month period - whether the levels measured and associated longer-term concentration estimates are of a magnitude, in light of health risk-based criteria, for which follow-up activities may need to be considered. To evaluate the monitoring results consistent with this objective, we developed health risk-based air concentrations (the long-term comparison levels described below) for each monitored pollutant using established EPA methodology and practices for health risk assessment[n] and, in the case of cancer risk, consistent with the implied level of risk considered in identifying schools for monitoring. Consistent with the long-term or chronic focus of the modeling analyses based on which these schools were selected for monitoring, we intend to analyze the complete dataset of measured concentrations using routine statistical tools to derive a 95 percent confidence interval for the estimate of longer-term average concentration for each

[n] While this EPA initiative will rely on EPA methodology, practices, assessments and risk policy considerations, we recognize that individual state methods, practices and policies may differ and subsequent analyses of the monitoring data by state agencies may draw additional or varying conclusions.

HAP monitored at a given school. This projected range (most particularly the upper end of the range) will be compared to the long-term comparison levels. The analysis of the air concentrations will also include a consideration of the potential for cumulative multiple pollutant impacts in drawing conclusions regarding air toxics at each location.[o]

In drawing conclusions regarding potential follow-up activities for a particular school location EPA will evaluate the air quality information provided by the full monitoring dataset, in light of various other types of information and considerations pertinent to the air quality at each location. For the purposes of providing context, some quite general examples of key considerations and potential resulting decisions are described in Table 4 below.

In general, where the monitoring results indicate estimates of longer-term average concentrations that are above the comparison levels – i.e., above the cancer-based comparison levels or notably above the noncancer-based comparison levels - we will consider the need for follow-up actions such as:

- → Additional monitoring of air concentrations and/or meteorology in the area,
- → Evaluation of potentially contributing sources to help us confirm their emissions and identify what options (regulatory and otherwise) may be available to us to achieve emissions reductions, and
- → Evaluation of actions being taken or planned nationally, regionally or locally that may achieve emission and or exposure reductions. An example of this would be the type of ubiquitous emissions from mobile sources.

Where the monitoring data analyses do not project longer-term average concentration estimates above the comparison levels, we will further analyze the dataset to describe what it indicates in light of other criteria and information commonly used in prioritizing state, local and national air toxics program activities. State, local and national programs often develop longer term monitoring data sets in order to better characterize pollutants near particular sources. The 2-3 month dataset developed under this initiative will be helpful to those programs in setting priorities for longer term monitoring projects. The intent of this analysis is to make this 2-3 month monitoring dataset as useful as possible to state, local and national air toxics program in their longer term efforts to improve air quality nationally. To that end, this analysis will:

- → Describe the air toxics measurements in terms of potential longer-term concentrations, and, as available, compare the measurements to monitoring data from national monitoring programs.
- → Describe the meteorological data by considering conditions on sampling days as compared to those over all the days within the 2-3 month monitoring period and what conditions might be expected over the longer-term, as indicated, for example by information from a nearby weather station.

[o] As this analysis of a 2-3 month monitoring dataset is not intended to be a full risk assessment, consideration of potential multiple pollutant impacts may differ among sites. For example, in instances where no individual pollutant appears to be present above its comparison level, we will also check for the presence of multiple pollutants at levels just below their respective comparison levels (giving a higher priority to such instances).

→ Describe available information regarding activities and emissions at the nearby source(s) of interest, such as that obtained from public databases such as TRI and/or consultation with the local air pollution authority.

5.2 Long-term Comparison Levels

The risk or hazard levels considered in interpreting modeling analysis results with regard to identifying schools for monitoring under this initiative (e.g., 1-in-10,000 cancer risk and hazard index of 1) derive from aspects of the framework for regulatory decisions in the Clean Air Act (CAA) residual risk program for stationary sources of air toxics (USEPA, 1999; 70 FR 19992).[p] While the CAA identifies different criteria for different types of air sources, with technological feasibility being the key criterion for decisions on controlling air toxics from mobile sources (e.g., cars and trucks), in the risk management framework for the residual risk program, regulatory actions may generally be considered when estimated ambient concentrations from the source emissions being assessed are equal to a continuous lifetime exposure concentration associated with an estimated increase in lifetime cancer risk greater than 100-in-a-million (or 1-in-10,000). The 100-in-a-million cancer risk level is the approximate upper end of the range that is commonly described in EPA risk-based decision making (see footnote l on page 5). The lower end of the range is 1-in-a-million.

In our analysis of the air toxics measurements at each school, we recognize two areas of interest. First and most importantly, as described in section 5.1 above, we are interested in identifying circumstances for which follow-up activities need to be considered (i.e., air concentrations at or above levels considered in identifying schools for monitoring under this initiative, such as those for which long-term exposure is associated with 100-in-a million or greater cancer risk). In addressing this most important area of focus, we developed long-term health risk-related comparison levels based on cancer risk using the 100-in-a million level of additional risk (see below). This is consistent with criteria that were considered in identifying schools for this monitoring. Secondly, as we are interested in making these monitoring data as useful as possible to ongoing state, local and national air toxics programs in their air quality improvement activities, we also intend to describe findings in light of other criteria that may indicate the potential for longer-term air concentrations that while somewhat lower, may still be of a magnitude that would inform EPA, state and local decision-making on risk reduction actions for air toxics more broadly. In our analyses of the monitoring data, we intend to also provide information useful to this second area of interest, such as noting how longer-term concentration estimates relate to 10% or 1% of the cancer-based comparison level.

With regard to effects other than cancer, EPA's residual risk program for stationary sources of air toxics has not established a strict regulatory decision framework. Secondly, while EPA develops and uses estimates of cancer risk per concentration of lifetime continuous exposure to carcinogenic air toxics in assessing potential cancer risk (and in deriving cancer risk-based

[p] Section 112 of the Clean Air Act describes a two-step approach for regulating categories of stationary sources of HAPs. In the first step, EPA set technology-based standards for all categories. In the second step, EPA is required to assess the residual risk remaining in areas near these sources and where necessary set additional source standards. This second step is referred to as the residual risk program.

concentrations), we use a different type of tool for analyses that consider the potential for risk of other (noncancer) health effects. This noncancer assessment tool is the reference concentration (RfC) or comparable value. The RfC is a continuous inhalation estimate for the human population, including sensitive subgroups, that is likely to be without appreciable risk of adverse noncancer effects over a lifetime. The RfC is more informative with regard to exposures that are below it than those above it. That is, an RfC is not a direct estimator of risk but rather a reference point to gauge the potential for effects. Lifetime or chronic exposures at or below the RfC are concluded to be without significant risk of adverse effects. Lifetime exposures above the RfC, however, do not necessarily indicate a risk of adverse health effect. But with such exposures increasingly above the RfC, there is an increasing potential for risk of effects. This potential varies depending on the pollutant and information specific to that pollutant. Accordingly, we commonly use the RfC as a benchmark in considering potential concern for air toxics exposures posing risk of noncancer effects, recognizing its more direct strength in interpretations regarding exposures at or below it. For example, noncancer impacts associated with projected long-termexposures equal to or below the RfC are currently being given little regulatory attention in the residual risk program. Situations where projected long-term exposures are above the RfC may be further considered for additional regulation in light of other information, such as information specific to the pollutant and uncertainties in the assessment.

This difference in how we assess potential noncancer effects and cancer risk affects our consideration of the two areas of focus mentioned above. As we do not have an upper and lower end of an established reference risk range to draw on for noncancer effects concentrations, our analyses involving the noncancer comparison levels take this into account through recognizing that longer-term average concentration estimates that are appreciably above the noncancer comparison level (e.g., by a factor of 5-10 or more) may be more relevant to gauging significance for health concerns than estimates falling much closer to or below this comparison level. That is, for some pollutants, a situation where the noncancer comparison level is exceeded by a factor on the order of 10 or more may be more relevant to consider for the purposes of our primary objective for this study, than one where the noncancer comparison level is only just exceeded. Thus, in drawing conclusions about potential concerns associated with estimated longer-term average concentration estimates higher than the long-term comparison level, we intend to consider a variety of factors, including those specific to the site or sources involved which might influence exposures (e.g., pending source actions), as well as factors particular to the health effects information (e.g., see section 4) including: the endpoints on which the RfC is based and our confidence in the RfC, its underlying database and the principal study; whether or not the RfC represents current methods and current information for the chemical, and; the magnitude of quantitative uncertainty associated with the RfC.[q] As a result, concerns regarding potential health impacts associated with estimated longer-term average concentrations above the noncancer-based comparison level may vary in light of factors other than or in addition to the amount by which the level is exceeded.

[q] For example, derivation of the RfCs for acrolein and benzene included division of the exposure concentrations associated with the critical effect by cumulative uncertainty factors of 1000 and 300, respectively. For these pollutants, levels at which health effects have been observed are more than 10-fold higher than the RfCs.

In summary, depending on the information available for each pollutant, two types of long-term comparison levels have been derived for comparison to the projected range of longer-term average ambient concentration of each key pollutant at each school (Table 2).[r]

- The *cancer-based comparison level* is the estimated continuous (24 hours a day, daily), lifetime exposure concentration associated with an estimated 100-in-a-million lifetime cancer risk[s].
- The *noncancer-based comparison level* is the EPA Reference Concentration (RfC) or a comparable value[t], which is the estimated continuous (24 hours-per-day daily) exposure concentration considered likely to be without adverse effects over a lifetime.

In developing or identifying these comparison levels, we have given priority to use of relevant and appropriate air standards and EPA risk assessment guidance and precedents. These levels are based upon the most recent assessments of potential toxicity of the monitored toxic air pollutants by EPA, and, as needed, the U.S. Agency for Toxic Substances and Disease Registry, and the California EPA. These agencies recognize the need to account for potential differences in sensitivity or susceptibility of different groups (e.g., asthmatics) or lifestages/ages (e.g., young children or the elderly) to a particular pollutant's effects so that the resulting comparison levels are relevant for these potentially sensitive groups as well as the broader population. Further, the comparison levels are based on exposure to the pollutants all day, every day over a lifetime.[u]

5.3 Interpretation of Monitoring Results in Light of Comparison Levels

The report for each school that describes the analysis of the full monitoring dataset collected at each school (the *school-specific report*), will include discussion of all of the information collected as part of this initiative, including the meteorological measurements, information regarding nearby sources as well as the chemical concentration measurements. As mentioned above, analysis of the chemical concentration measurements will include consideration of what the full monitoring dataset indicates with regard to potential longer-term levels of the monitored pollutant. In doing this we will calculate the mean concentration for each pollutant during the monitoring period along with the 95% confidence limits on the mean which we will then use as an estimate of longer-term concentration in

[r] Comparison levels will be developed consistent with the method described here for use in considering results for other HAPs monitored at each school.

[s] This is derived by dividing 10^{-4} by the inhalation unit risk (IUR) value for a pollutant. IURs were taken from OAQPS toxicity table 2 (http://www.epa.gov/ttn/atw/toxsource/summary.html). In the case of the chemicals listed here as PAHs, which EPA considers to be carcinogenic by a mutagenic mode of action, either by explicit finding or by indirect finding due to the recognition of BaP as an index for IUR derivation, an additional step is taken as recommended by EPA guidance. Derivation of the cancer-based comparison levels for these pollutants included division by a factor of approximately 1.6 reflecting the application of age-dependent adjustment factors which effectively increase the potency when applied to the first 16 years of age (USEPA, 2005).

[t] Where EPA Reference Concentrations are unavailable, ATSDR chronic Minimal Risk Levels or California EPA chronic Reference Exposure levels are substituted in that order, consistent with the hierarchy implemented in the OAQPS toxicity table 2 (http://www.epa.gov/ttn/atw/toxsource/summary.html). The term "RfC" is used here to indicate any of these values (which are similarly derived and defined).

[u] This exposure assumption is explicit in its impact on derivation of the cancer-based comparison levels, such that an alternate assumption such as exposure limited to only childhood years would have resulted in higher (less protective) comparison levels.

considering the implications of the dataset with regard to longer-term levels. The paragraphs below provide a general description of how we intend to consider these summary statistics that describe the measurements at a school in light of the comparison levels described in the sections above.

Cancer-based Comparison Levels: Air toxics for which the upper 95% confidence limit on the mean concentration falls above the cancer-based comparison level will be fully discussed in the school-specific report and may be considered a high priority for potential follow-up activities in light of the full set of information available for that site. Air toxics for which the upper 95% confidence limit falls below 1% of the comparison level (i.e., those for which longer-term average concentration estimates are below the corresponding 1-in-1-million cancer risk based concentration) are generally considered a low priority for follow-up activity. Situations where the summary statistics for a pollutant are below the cancer-based comparison level but above 1% of that level will be fully discussed in the school-specific report.

Noncancer-based Comparison Levels: Air toxics for which the upper 95% confidence limit on the mean concentration is near or below the noncancer-based comparison level (i.e., those for which longer-term average concentration estimates are below a long-term health-related reference concentration) are generally of low concern and will generally be considered a low priority for follow-up activity. Pollutants for which the 95% confidence limits extend appreciably above the noncancer-based comparison level will be fully discussed in the school-specific report and may be considered a priority for follow-up activity, if indicated in light of the full set of information available for the pollutant[v] and the site. Interpretation in the case of lead entails additional specific considerations related to the use of the national ambient air quality standard as the comparison level.

Multi-pollutant Cumulative Risk: The school-specific report will also consider and discuss the potential for multiple monitored pollutants (e.g., in addition to the key pollutant(s)) to be present at levels that might contribute to a potential for cumulative risk, particularly in situations where multiple pollutants are present at longer-term levels just below their respective comparison levels.

[v] Such information includes that described in section 4 above.

Table 2. Long-term Comparison Levels for Key (*aka* "driver") Pollutants.

Analyte	Cancer-based Comparison Level[1]	source	Noncancer-based Comparison Level[1]	source	2004-07 NATTS[2] Arithmetic Mean (Median) [Maximum]
Metals[3]	ng/m^3		ng/m^3		PM_{10} Metals ng/m^3
Arsenic (PM_{10} focus)	23	IUR	15	REL	1.1 (0.69) [48]
Chromium-hexavalent	8.3	IUR	100	RfC	0.05 (0.03) [3]
Cobalt (PM_{10} focus)	-		100	MRL	0.32 (0.17) [20]
Lead[4] -TSP	-		150[3]	NAAQS	5.6 (3.5) [751]
Manganese (PM_{10} focus)	-		50	RfC	11 (4.8) [410]
Nickel (PM_{10} focus)	420[5]	IUR	90	MRL	2.6 (1.8) [110]
VOCs/Carbonyls	µg/m^3		µg/m^3		µg/m^3
Acetaldehyde	45	IUR	9	RfC	1.9 (1.4) [93]
Acrolein[6]			0.02[6]	RfC[6]	0.63 (0.44) [7.3]
Benzene	13	IUR	30	RfC	1.1 (0.86)[10]
1,3-Butadiene	3.3	IUR	2	RfC	0.15 (0.09) [16]
4,4'-methylenedianiline	20	Cal-IUR	0.22	REL	--
Naphthalene	3	Cal-IUR	2.9	RfC	
PAHs[7]:	ng/m^3				
Benzo(a)pyrene	57	IUR			
Benzo(a)anthracene	570	IUR			
Benzo(b)fluoranthene	570	IUR			
Benzo(k) fluoranthene	570	IUR			
Chrysene	5,700	IUR			
Dibenz(a,h)anthracene	52	IUR			
Indeno(1,2,3-cd)pyrene	570	IUR			
Diisocyanates	µg/m^3		µg/m^3		--
1,6-hexamethylene diisocyanate			0.01	RfC	--
Methylenediphenyl diisocyanate			0.6	RfC	--
2,4-toluene diisocyanate	9.1	Cal-IUR	0.07	RfC	--

Table Notes

[1]Values derived for this document, or for which units conversions were performed are shown here with the same number of significant figures as those specified in the source value.

[2] NATTS concentrations, presented to provide a reference for ambient levels of these pollutants, have been rounded to 2 significant figures.

[3]Metal samples will be of 2 different types: PM_{10} and total suspended particles (TSP). With the exception of lead (for which the NAAQS was developed with explicit recognition of non-inhalation exposure pathways), metals comparison levels may be more suited for use with the concentration of metal in particles captured in a PM_{10} sample. While a Pb-PM_{10} average above the Pb comparison level is a finding of similar (or greater) interest than that for Pb-TSP, long-term averages for other metals in TSP that fall above a comparison level primarily indicate the need to consider results for that metal in PM_{10} on a case-by-case basis.

[4]The value here is the level of the NAAQS, which is in terms of a 3-month average level of lead in TSP.

[5] The value here is based on the IRIS IUR for nickel refinery dust.

[6] The comparison level shown for acrolein is the EPA RfC which is set below a level associated with health effects. The RfC is set as a factor of 1000 below an exposure concentration associated with sensitive nasal effects in laboratory animals. Since the EPA RfC was derived, the California EPA has derived a chronic REL

based on more recently available information on acrolein and its effects. The Cal-EPA REL, which is 0.35 ug/m^3, is somewhat more than a factor of 100 below the level associated with effects in the more recently available study.

[7]The PAHs listed here are those identified as carcinogens, and for which inhalation unit risk estimates are available (from OAQPS Toxicity Table per section 3.3.b above)). The cancer-based comparison levels for these pollutants reflect EPA recommended application of age-dependent adjustment factors to reflect a potential for increased susceptibility to these substances in the first 16 years of life.

Table 4. General examples of key considerations and potential follow-up conclusions.

- If a key source of air pollution near a particular school is known to have temporarily ceased its operations during a significant portion of the monitoring period (e.g., due to temporary closure, periodic shutdowns, reduced operational capacity, etc.), EPA may not be able to draw conclusions from the initial 60-90 day dataset regarding the potential for "usual" air toxics levels to pose a potential for chronic health impacts and may wish to consider additional monitoring if and when the key source or sources restart operations
- If the windrose during the sampling days shows significantly less windflow in the source-to-school direction as compared to what is seen across the full monitoring period or what might be expected over the longer term, EPA may choose to continue monitoring even if measurements suggest impacts are low
- Frequent rainfall throughout the monitoring period may cause EPA to decide to continue monitoring even if measurements suggest impacts are low
- If the concentrations of the key pollutants are all low, but the monitoring captures significant concentrations of an unexpected pollutant, EPA may choose to continue monitoring for that pollutant
- If the entire estimated long-term concentration range for each key pollutant falls below the respective long-term comparison levels and the wind measurements indicate inclusion of sampling dates with winds in the direction of primary sources and the source activity has been consistent with usual conditions, EPA may conclude that there is low potential for significant risk of chronic health impacts associated with air toxics at that school and recommend that we cease monitoring there.
- For pollutants where the measured concentrations are of a magnitude that may indicate some potential for chronic health concerns but where the measurements correspond to typical historic levels nationwide (e.g., this may be the case for acrolein and perhaps some others), EPA will give particular consideration to the school-specific data in light of the data for situations nationally, any associated national activities that are underway and information about other activities to address the pollutant sources. Such information will inform conclusions regarding next steps at the school. Based on this consideration, EPA may, for example, focus follow-up activities on a range of areas potentially including making the public aware of the available information, working with state and local partners to consider steps that may be available to us to reduce the pollutants locally, and/or examining how national activities underway may reduce the pollutants in the future.
- For pollutants where measured levels are indicative of some potential for chronic impacts (for continuous long-term exposure scenarios), but where requirements are in place to reduce key source emissions within next year or so (e.g., consent order), EPA may indicate that while this school's air has levels that might be of potential concern over a long term, source requirements are expected to preclude the occurrence of these levels over the long-term, and may conclude to revisit monitoring on some altered longer-term schedule.
- If the entire estimated long-term concentration range for any key pollutant falls above the long-term comparison levels (and concentrations are not typical nationally and we have no information indicating activities in place to reduce concentrations in future), EPA may conclude that the potential for chronic health impacts of concern associated with that pollutant at that school indicate a need to pursue (in concert with the State or local air agency) options for emission reductions of that pollutant at the key source(s), while also continuing monitoring to more accurately characterize long-term concentrations and track the progress of emission reduction efforts.

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APPENDIX A. Individual Sample Screening Levels for Additional (non-key) HAP Analytes. The methodology and important considerations for their interpretation aredescribed in section 3 above and the footnotes following the table.

Additional HAP Analytes	Individual Sample Screening Level[1]	Value from which Screening Level Derived
Metals[2]	ng/m^3	
Antimony[7] (PM_{10} focus)	2,000 (I) 3	chronic RfC
Beryllium (PM_{10} focus)	20 (I)	chronic RfC
Cadmium (PM_{10} focus)	30	acute MRL
Mercury (elemental)[8]	3,000 (I)	chronic RfC
Selenium (PM_{10} focus)	20,000 (I)	chronic REL
VOCs/Carbonyls	µg/m^3	
Acetonitrile	600 (I)	chronic RfC
Acrylonitrile	200	acute MRL
Benzyl chloride	140 (I)	IUR
Bromoform	6,400 (I)	IUR
Carbon disulfide	7000 (I)	chronic RfC
Carbon Tetrachloride	200 (I)	inter. MRL
Chlorobenzene	10,000 (I)	chronic REL
Chloroform	500	acute MRL
Chloroprene	70(I)	chronic RfC
1,4-Dichlorobenzene	10000	acute MRL
1,3-Dichloropropene	40 (I)	inter. MRL
Ethyl acrylate	7000 [9]	[9]
Ethyl benzene	40,000	acute MRL
Chloroethane (ethyl chloride)	40,000	acute MRL
Ethylene dibromide	12(I)	IUR
Ethylene dichloride	270 (I)	IUR
Ethylidene dichloride (1,1-dichloroethane	4400 (I)	HEAST chronic RfC
Formaldehyde	50	acute MRL
Hexachlorobutadiene	320 (I)	IUR
Bromomethane (methyl bromide)	200	acute MRL
Chloromethane (methyl chloride)	1000	acute MRL
1,1,1-Trichloroethane (methyl chloroform)	10,000	acute MRL
Methyl isobutyl ketone	30,000(I)	chronic RfC
Methyl methacrylate	7000 (I)	chronic RfC
Methyl tert-butyl ether	7,000	acute MRL
Methylene Chloride	2000	acute MRL
Propionaldehyde	80 (I)	chronic RfC
	µg/m^3	
Propylene dichloride (1,2-dichloropropane)	200	acute MRL
Styrene	9,000	acute MRL
1,1,2,2-Tetrachloroethane	120 (I)	IUR

Additional HAP Analytes	Individual Sample Screening Level[1]	Value from which Screening Level Derived
Tetrachloroethene	1,400	acute MRL
Toluene	4,000	acute MRL
1,2,4-Trichlorobenzene	2,000 (I)	chronic RfC
1,1,2-Trichloroethane	440 (I)	IUR
Trichloroethylene	10,000	acute MRL
Vinyl Chloride	1,000	acute. MRL
Vinylidene chloride (1,1-dichloroethylene)	80 (I)	inter. MRL
Xylene	9,000	acute MRL

Notes for Appendix A

[1] Values derived for this document, or for which units conversions were performed are assigned the same number of significant figures as those specified in the source value.

[2] Metal samples will be of 2 different types: PM_{10} and total suspended particles (TSP). With the exception of lead (for which the NAAQS was developed with explicit recognition of non-inhalation exposure pathways), metals screening levels may be more suited for use with the concentration of metal in particles captured in a PM_{10} sample. While a Pb-PM_{10} sample above the Pb screening level is a finding of similar (or greater) interest than that for Pb-TSP, results for other metals in TSP that fall above a screening level may primarily indicate the need to consider results for that metal in PM_{10} on a case-by-case basis.

[3]The superscript (I) indicates where an intermediate duration exposure screening levels has been assigned as a surrogate in lieu of a short-term screening level.

[4]This value for lead is the level of the NAAQS, which is in terms of a 3-month average level of lead in TSP.

[5]As neither an ATSDR intermediate MRL or a chronic value from the OAQPS table is available for total nickel, the screening level provided here is that for nickel refinery dust.

[6]PAHs are components of the HAP polycyclic organic matter (POM). The PAH analytes listed here are those which EPA has identified as carcinogens, and for which inhalation unit risk estimates are available (from the OAQPS Toxicity Table, per section 3.3.b above)). In assessing carcinogenicity for chemicals concluded to be carcinogenic by a mutagenic mode of action and for which early lifestage dose-response information is not available, EPA recommends use of age-dependent adjustment factors (ADAFs) which effectively increase the potency when applied to the first 16 years of age (USEPA, 2005). Dibenz[a,h] anthracene (DBA) and benzo[a]pyrene (BaP), and the other PAHs for which IURs are indexed to that of BaP are considered by EPA to act by mutagenic modes of action (Farland, 2006). In considering potential use of ADAFs in the context of the individual sample screening levels, we recognize several considerations: (1) the objective for the sample screening levels is a short-term exposure estimate (on the order of weeks), (2) application of EPA's traditional cancer risk assessment methods to such short periods is inherently uncertain, (3) the sample screening levels for these PAHs are actually surrogates for short-term levels and have been derived for a 1-year exposure, (4) the full data analysis for each school (see section 5) will consider potential risk of chronic exposure, and (5) if in the calculation of these levels, we had substituted a short-term exposure duration (e.g., 2 weeks), their values would be increased by a factor of 20, which is more than twice the value of the highest ADAF. Thus, rather than implement a cancer risk calculation specific to a 2-week duration exposure, which is not generally recommended by EPA guidance, we have retained the 1-year values and recognize that they are appropriately protective of shorter-term exposures with a potential for increased early-life susceptibility.

[7]As neither an ATSDR intermediate MRL nor a chronic assessment from the OAQPS toxicity table is available for total antimony, the screening level provided here, and limited to use in this project, is that for antimony trioxide.

[8]As neither an ATSDR intermediate MRL nor a chronic assessment from the OAQPS toxicity table (per Section 3.3) is available for particulate mercury, the screening level provided here is that for elemental mercury, which is expected to be lower than that which would be pertinent to particulate mercury (if one existed).

[9]For ethyl acrylate, ATSDR has not established any MRLs of any exposure duration and OAQPS has identified no chronic dose-response values (http://www.epa.gov/ttn/atw/toxsource/summary.html). The value provided as a surrogate sample screening level is the screening level for methyl methacrylate, which is of the same chemical class

and which is lower than that indicated by other available health effects information. In considering additional health effects information for this pollutant, emergency planning guideline levels (Acute Exposure Guideline Levels and Emergency Response Planning Guidelines), developed for various levels of severity and durations of exposure up to 8 hours, and their basis were reviewed. Additionally, chronic exposure values developed by the Occupational Safety and Health Administration and the American Conference of Governmental Industrial Hygienists (ACGIH) were reviewed, while recognizing that these values were developed for use in workplace environments and are not intended for ambient, environmental exposures of the general population. For example, the ACGIH threshold limit values (TLVs), while developed for use in considering time-weighted average worker exposures over an 8-hour shift, are based on recognition of repeated workday exposures in the context of 40-hour work weeks throughout a worker's career. The ACGIH TLV of 5 ppm (20 mg/m^3) is based on minimizing irritant effects of chronic exposures to ethyl acrylate, including such effects to the respiratory tract, skin, eyes, and gastrointestinal tract (as documented in *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices, ACGIH)*. As might be expected given the long-term exposure aspect of its basis, the ACGIH TLV is the lowest among the values considered here. The surrogate screening level shown in the table above, however, is lower than the ACGIH TLV.

Appendix B. Individual Sample Screening Levels for non-HAPs

As mentioned in section 1 above, the ambient air monitoring methods being used in this project for HAPs of interest also produce measurements for several non-HAPs. Some of these chemicals are well studied and individual sample screening levels (as described in section 2 above) have been developed for them.[23] These are presented in Table B-1 below.

Table B-1. Analytes that are not CAA Section 112 Hazardous Air Pollutants for which data support development of sample screening levels

Additional (non-HAP) Analytes	CAS #	Individual Sample Screening Level (ug/m^3)[1]	Value from which Screening Level Derived	2003-2007 Monitoring Data in AQS, Maximum (median) ($\mu g/m^3$)
Acetone	67-64-1	62,000	acute MRL	2600 (4×10^{-3})
Cis-1,2-dichloroethylene	156-59-2	800	value for trans isomer	0.52 (ND)
Trans-1,2-dichloroethylene	156-60-5		acute MRL	0.53 (ND)
Methyl ethyl ketone	78-93-3	50,000 (I)	chronic RfC	16 (7×10^{-4})
Propylene	115-07-1	30,000 (I)	chronic REL	3.2 (8×10^{-4})
m-dichlorobenzene (1,3-DCB)	541-73-1	10,000**	Val for para- isomer (Appendix B)	6.6 x 10^{-1} (ND)

For the remaining non-HAP analytes, the available chemical-specific information (including that recognized in section 3 above) and sample screening levels for structurally similar chemicals in Tables 1 or Appendix A (see Table B-2 below) will be considered in reviewing individual sample results.

[23] In the case of 1,3-dichlorobenzene, no chemical-specific information useful to assigning a screening level was identified. So the sample screening level for the para-isomer (1,4-DCB), presented in Appendix A above was assigned (see Table A-1).

Table B2. Pollutants for which data limit development of sample screening levels* (e.g., no EPA, ATSDR or Cal-EPA acute or chronic assessments).

Additional (non-HAP) Analytes	CAS #	Available information regarding levels of interest (ug/m^3)[1]	Structurally similar chemicals	2003-2007 Monitoring Data in AQS, Maximum (median) ($\mu g/m^3$)
Acetylene	74-86-2	Lowest concentration at which effects reported is 10^8 $\mu g/m^3$. CNS depression is most sensitive effect reported – it was used as anesthetic in 1920s. Use stopped due to explosive characteristics. The NIOSH REL, as a ceiling, is 2500 ppm, 3 x 10^6 $\mu g/m^3$ (based on concern regarding phosphine which occurs in industrial grade acetylene, 2500ppm).	There are no straight-chain Alkyne HAPs. The most structurally similar alkyne, propyne (74-99-7), has ACGIH TLV-TWA of 1000 ppm. 1000 ppm acetylene=$1x10^6 \mu g/m^3$.	6.2×10^{-2} * ($\sim 3 \times 10^{-3}$)
Bromochloromethane	74-97-5	Lowest exposures in animal studies has been 370 ppm in dog and rat (subchronic [6mo] exposure) – some increased liver weight in rat. The ACGIH TLV-TWA is 200 ppm (10^6 $\mu g/m^3$). This was developed based on consideration of animal exposure data and concerns regarding CNS and liver effects.	The sample screening level for a structurally similar HAP, chloroform, is 0.1 ppm. In $\mu g/m3$, 0.1 ppm is: $500\ \mu g/m^3$ bromochloromethane, $700\ \mu g/m^3$ bromodichloromethane, $900\ \mu g/m^3$ dibromochloromethane	$1.1x10^{-1}$ * (ND)
Bromodichloromethane	75-27-4	Lowest exposures indicative of effects in animal studies has been 10 ppm in mice (prechronic [3 week] exposure) – some kidney effects which were not seen in exposures at this concentration lasting 13 weeks. The next lower concentration studied (3 ppm or $2x10^4 \mu g/m^3$) did not show effects in mice at 3 weeks or at 13 weeks of exposure (NTP).		$1.3x10^{-1}$ (ND)
Dibromochloromethane	124-48-1	No inhalation studies identified (HSDB).		$1.7x10^{-1}$ (ND)

Additional (non-HAP) Analytes	CAS #	Available information regarding levels of interest (ug/m^3)[1]	Structurally similar chemicals	2003-2007 Monitoring Data in AQS, Maximum (median) (µg/m^3)
Dichlorodifluoromethane (Freon 12)	75-71-8	Lowest concentration at which effects reported is 800 ppm (4 x 10^6 µg/m^3) in guinea pig (subchronic [90day continuous] exposure) – liver pathological changes not seen in dog, monkey, rat or rabbit exposures. The ACGIH TLV-TWA is 1000 ppm (5 x 10^6 µg/m^3). Sweden has an occupational value of 500 ppm (2 x 10^6 µg/m^3).	Among these four Freons, the lowest health-related concentration identified is 2 x 10^6 µg/m^3	7 (2.6x10^{-3})
Dichlorotetrafluoroethane (Freon 114)	76-14-2	Lowest concentration at which effects reported is 25,000 ppm (µg/m^3) in dog (acute exposure) – cardiac arrhythymia. No significant clinical, hematologic, histopathologic changes in rats and rabbits reported from repeated (2 hr/day; 5 day/wk) chronic (8-9 mos) exposure to 10,000 ppm. The ACGIH TLV-TWA is 1000 ppm (7 x 10^6 µg/m^3)		8.3 (1x10^{-4})
Trichlorotrifluoroethane (Freon 113)	76-13-1	Short term exposure to fluorocarbon 113 can occur by inhalation, ingestion, and skin absorption. Inhalation and ingestion of fluorocarbon 113 will result in drowsiness, narcosis, central nervous system depression and significant impairment of manual dexterity and vigilance. Irritation of the eyes, nose, throat, and skin can also occur. Breathing concentrations that are greater than 2000 ppm may cause irregular heartbeat or heart arrest. Concentrations that exceed 2000 ppm are considered as immediately dangerous to life and health (NIOSH). 1000 ppm (7.6 x 10^6 µg/m^3). A similar purpose standard in Sweden and Germany is 500 ppm (4 x 10^6 µg/m^3).		0.05 (9 x 10^{-4})
Trichlorofluoromethane (Freon 11)	75-69-4	The ACGIH ceiling and the OSHA PEL is 1000ppm (5.6 x 10^6 µg/m^3) based on a subchronic animal test NOAEL.		31 (1 x 10^{-3})

Additional (non-HAP) Analytes	CAS #	Available information regarding levels of interest (ug/m^3)[1]	Structurally similar chemicals	2003-2007 Monitoring Data in AQS, Maximum (median) (μg/m^3)
Ethyl tert-butyl ether	637-92-3	The ACGIH TLV-TWA is 5 ppm (20,000 μg/m^3).	The sample screening level for a structurally similar HAP, MTBE, is 2 ppm. In μg/m3, 2 ppm is: 8000 μg/m^3 ETBE or TAME	0.4 (ND)
Tert-amyl methyl ether	994-05-8			0.5 (ND)
n-octane	111-65-9	The NIOSH REL is 75 ppm (350,000 μg/m^3), on the basis of weight given to neurotoxic effects associated with hexane exposures. The ACGIH TLV-TWA is 300 ppm (1,400,000 μg/m^3), established on the basis of comparison of the acute response to other paraffinic hydrocarbons.	The sample screening level for a structurally similar HAP, hexane, is 2 ppm. In μg/m3, 2 ppm is: 9000 μg/m^3 octane	4 (1 x 10^{-4})
1,2,4-trimethylbenzene	95-63-6	The ACGIH TLV-TWA is 25 ppm (100,000 μg/m^3) The 8 hr AEGL-1 is 45 ppm.	The sample screening level for a structurally similar HAP, xylene, is 2 ppm. In μg/m3, 2 ppm is: 10,000 μg/m^3 1,2,4- or 1,3-5-trimethylbenzene	5.7 (3 x 10^{-4})
1,3,5-trimethylbenzene	108-67-8			1.9 (8 x 10^{-5})
Aldehydes				
Benzaldehyde	100-52-7	The ACGIH WEEL TWA is 2 ppm .	The sample screening level for a structurally similar HAP, formaldehyde, is 0.04 ppm. In μg/m^3, 0.04 ppm is: 200 μg/m^3 benzaldehyde, 2,5-dimethylbenzaldehyde, hexaldehyde, isovaleraldehyde, or the	8.2 (1 x 10^{-4})
Butyr- and Isobutyraldehyde	123-72-8 78-84-2	The ACGIH WEEL TWA is 25 ppm (74 mg/m^3)		25 (2 x 10^{-4})
Crotonaldehyde	123-73-9	The ACGIH TLV-TWA Ceiling is 0.3 ppm (0.86 mg/m3) to protect the skin. The NIOSH REL-TWA is 2 ppm (6000 μg/m^3). The ACGIH TLV-TWA is 2 ppm to protect against the respiratory irritancy effects. The AEGL-1 (8hr)=0.2 ppm.		0.8 (1.5 x 10^{-4})
2,5-Dimethylbenzaldehyde	5779-94-2			

Additional (non-HAP) Analytes	CAS #	Available information regarding levels of interest (ug/m^3)[1]	Structurally similar chemicals	2003-2007 Monitoring Data in AQS, Maximum (median) (μg/m^3)
Hexaldehyde (hexanal)	66-25-1		tolualdehydes 100 μg/m^3 butyraldehydes or valeraldehyde	5.8 (1.3x10^{-4})
Isovaleraldehyde (3-methylbutanal)	590-86-3			1 (ND)
o-, m- and p-tolualdehyde	529-20-4, 620-23-5, 104-87-0			0.4(1.5x10^{-3})
Valeraldehyde (pentanal)	110-62-3	The ACGIH TLV-TWA is 50 ppm (176 mg/m^3). The NIOSH REL is 50 ppm.		1.3 (1x10^{-4})