Health Consultation

Ambient Air and Indoor Dust

McFARLAND STUDY AREA

McFARLAND, KERN COUNTY, CALIFORNIA

EPA FACILITY ID: CA0001118603

MARCH 22, 2006

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
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Health Consultation: A Note of Explanation

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Prepared by:

The Agency for Toxic Substances and Disease Registry
National Center for Environmental Health

Summary and Statement of Issues

The community of McFarland, California petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to evaluate potential exposure to hazardous substances in their environment in response to a childhood cancer cluster for which a causal association between health data and identified contamination could not be established [1, 2]. ATSDR completed an assessment of potential exposure to soil and municipal water in 2001 and recommended review of air quality data when available [3]. This health consultation evaluates potential exposures to ambient air quality and indoor dust in data submitted by the Environmental Protection Agency (EPA) [4]. ATSDR concluded that exposure to the reported individual contaminant levels would not be expected to result in adverse health effects.

Background

In 1995, residents of McFarland, California and the community organization Healing Our Mother Earth (HOME) petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to address health concerns about potential environmental contamination in their community. McFarland is an agricultural community located 25 miles north of Bakersfield in California's Central Valley. The town has a population of approximately 7,970 and is surrounded by crop land, pastures, and orchards. Many residents are agricultural workers. The community is concerned about exposures to hazardous substances (for example, pesticides and toxic wastes) in soil and drinking water, and about the incidence of childhood cancer. According to historical records, the McFarland area was the site of a U. S. Army Air Force basic pilot training airfield during World War II, and had extensive agricultural activity, including crop production, chemical application, storage and shipment of agricultural products [3].

The site description, history, and demographics have been previously reported in the Public Health Assessment, McFarland Study Area, released in April 4, 2001 [3]. The 2001 public health assessment evaluated contaminant levels in soil and municipal drinking water, but air data were unavailable at the time. The action plan of the 2001 assessment recommended that ATSDR evaluate air data when available. This health consultation evaluates air data and indoor dust data collected by EPA during the period from July 2001 to May 2002, and reported in 2004.

Community Health Concerns

Community health concerns were reported as an increase in the number of childhood cancer cases, the incidence of adult cancers, and low birth weights.

Methods

ATSDR evaluates contaminants by comparing the environmental media concentrations and exposure dose to levels that cause adverse effects observed in experimental studies with animals, epidemiological studies, and other sources of reported exposure such as accidents and medical treatments. In any evaluation, assumptions must be made and professional judgment used. ATSDR attempts to be transparent and presents information to assist in understanding how conclusions were reached.

ATSDR screens chemicals using chemical-specific non-cancer comparison values called Minimum Risk Levels (MRLs) which identify levels where non-cancer health effects would not be expected, even to sensitive populations (although hypersensitive populations may not be protected). If ATSDR has not developed comparison values, comparisons may be made to values

developed by other agencies. ATSDR uses a cancer risk evaluation guide (CREG) as a comparison value for cancer health effects and corresponds to an estimated cancer risk above background of one in a million (1E-06). Exceeding a comparison value does not indicate a health hazard but identifies chemicals for further evaluation. Further evaluation may include consideration of recent scientific literature, toxicological evaluation, relevance to the exposure population, risk assessment, mixtures assessment, and applies site-specific exposure values in lieu of default exposure values.

When possible, comparison values are selected to be many times below the highest exposure level where health effects have *not* been observed, called a no observed adverse effect level (NOAEL). While CVs may generally be hundreds or thousands of times below observed effect levels, the exact level depends on the degree of uncertainty. CVs are an attempt to account for identifiable uncertainty, such as individual animal variability, extrapolation from animals to humans, human variability, use of a low observed adverse effect level (LOAEL) instead of a NOAEL, incomplete database, etc. Unless a chronic comparison value is consistently exceeded and approaches a value near 1/10 of the NOAEL, it is not generally considered a public health hazard by ATSDR unless interaction with other contaminants or sensitive population exposure is suspected [5].

ATSDR evaluated results from an extensive EPA sampling and analysis project which included air data collected at two monitoring stations, one located at Browning Road School and one located at McFarland Middle School (Figure 1). Indoor dust data were collected in the same two area schools. EPA coordinated sampling with estimated agricultural pesticide application events. The number and type of contaminants for which EPA sampled was extensive and thorough. Ambient air samples were a composite of two 24-hour samples, representing a total of approximately 48-hours of sample collection, or discrete samples representing 24-hours of sampling. An exception was dioxin/furan/PCB samples which were collected over an 8-day period and two separate 11-day periods during different sampling events [4].

Indoor dust samples were also collected at the same area schools as short term or long term dust samples. Short term denotes samples collected and composited by vacuuming areas cleaned daily and representing a one-day accumulation of dust. All dioxin/furan/PCB samples were short term. Long term samples were collected from areas that accumulate dust for approximately six months.

ATSDR seeks to provide a realistic and relevant description of the exposures and dose-specific health information to the community of interest.

Results

Most contaminants were at or below comparison values. Other contaminants may have exceeded chronic comparison values occasionally but would not be expected to result in adverse health effects as the frequency and duration of the exposure was short-lived. Short-duration exposures did not exceed acute comparison values for non-cancer health effects and chronic non-cancer comparison values were rarely exceeded.

ATSDR identified the following chemicals as contaminants of concern for further evaluation based on toxicity and prevalence of detections in samples collected, or to provide information on the relevance of a potential exposure to the selected chemical. A summary of relevant chemicals of concern is presented in Table 1.

Contaminants Selected for Further Evaluation in Indoor Dust

Dioxins/furans/PCBs

The total toxicity equivalent quotient (TEQ) for short term dust collection did not exceed the ATSDR guidance for dioxin and dioxin-like compounds [6]. Indoor dust levels (20 pg/g maximum, 6 pg/g mean, 1.9 – 20 range) did not exceed the screening level derived by ATSDR (50 parts per trillion, or pg/g, for exposure to residential soil). In addition, guidelines are based on exposure to residential soil, not indoor dust. The exposure to dust accumulating in the school during the school day for older children is expected to be less than a young child's continuous exposure in a residential soil exposure scenario, which includes exposure to indoor dust originating from soil (does not include other potential sources of indoor dust, such as paint chips). Dust ingestion is assumed to begin around age 3 months, to peak at age 2 years, and to fall off rapidly thereafter. Soil ingestion is assumed to begin at around age 6 months, to peak at age 3 years, and then fall off slowly with age as the child presumably continues to play outdoors and perhaps participate in sports. Indoor dust exposure in schools would also be intermittent; suggesting that only a portion of the exposure would occur to the indoor dust at school during school hours and days, while residential exposures assume all exposure is to residential soil and associated indoor dust. EPA estimates 20% of the total soil/dust ingested by ages 6 – 19 comes from school dust exposure in the All-Ages Lead Model (external review draft) [7]. Therefore, exposure among school age children to indoor dust in the school would likely be less than exposure among preschool age children to residential soil and indoor dust in the home. Using a residential soil screening level is likely to be more protective in an indoor dust exposure scenario in schools.

Contaminants Selected for Further Evaluation in Ambient Air

Formaldehyde.

Formaldehyde consistently exceeded the comparison value for cancer health effects, but did not exceed the ATSDR comparison value for chronic non-cancer health effects (10 $\mu g/m^3$). The mean for formaldehyde for all sampling was 6.8 $\mu g/m^3$, which exceeds an ATSDR Cancer Risk Evaluation Guideline (CREG) of 0.08 $\mu g/m^3$. ATSDR's CREG is based on continuous lifetime exposure at 1E-06 increased cancer risk (or an increased in risk for 1 individual in a population of 1,000,000) above background and identifies a level where health effects would not be expected. The site-specific cancer risk estimate is 4E-05 (an increase in the risk to 4 individuals in a population of 100,000) for a 30 year continuous exposure, which represents a low increase in risk for cancer health effects. This level of risk would not pose a public health concern as such due to the conservative nature of the risk assessment, but identifies the contaminant for inclusion in further evaluations such as mixtures assessment.

For perspective, a study of volatile organic compounds reported nationwide annual formaldehyde levels in air to average 10 $\mu g/m^3$ outdoors (median 5 $\mu g/m^3$) with median daily concentrations ranging from 3.3 $\mu g/m^3$ (rural) to 8.0 $\mu g/m^3$ (urban) [8]. Indoor formaldehyde concentrations averaged 61 $\mu g/m^3$ (median, 52 $\mu g/m^3$). Therefore, average concentrations at McFarland are not inconsistent with nationwide averages. However, there is uncertainty in the scientific literature concerning formaldehyde and its mode(s) of action for developing cancer.

The inhalation slope factor (1.3E-05/μg/m^3) used to develop the cancer screening value was derived from experimental animal studies observing cancer in the nasopharyngeal region [9]. The prevalence of toxicological information indicates that formaldehyde is very reactive and acts at the site of inhalation exposure, resulting in cancers of the lung and nasopharyngeal regions [10]. Most epidemiological studies are generally consistent with results of these animal studies. In contrast, some epidemiological studies have reported an association between formaldehyde and leukemia in certain occupations (morticians, embalmers, and certain laboratory workers), although co-exposures may also have occurred to other chemicals [11,12]. The increased risk may be associated with increasing peak, average levels, and duration of exposure, but not with cumulative exposure []. These results may be confounded by exposures to other chemicals and the lack of an identifiable mode of action, but introduce some uncertainty in the evaluation based on experimental results in animals. Multiple inhalation bioassays have not induced leukemia in animals [13].

Some scientists think effects of formaldehyde at sites other than the upper respiratory tract are unlikely. Formaldehyde is a naturally-occurring biological compound that is present in all tissues, cells, and bodily fluids. Cells have the metabolic capacity to deal with environmental levels of formaldehyde, although these pathways are saturable at high levels of formaldehyde [14]. Experiments involving inhalation exposures have not statistically increased blood levels; which does not support cancer generation at distant sites [15,16]. Thus far, a biologically plausible mechanism of action to account for the development of leukemia from exposure to formaldehyde has not been demonstrated. Inconsistent results from studies suggest that further research is needed before definite conclusions can be drawn.

Arsenic

The ATSDR CREG (0.0002 μg/m^3) was consistently exceeded by arsenic. The arsenic mean concentration was 0.0012 μg/m^3, but the mean is skewed by a few high values. Based on unit risk of 0.0043/μg/m^3, the estimated risk for developing cancer from a continuous 30-year exposure to this concentration is 2E-06 (increased risk to 2 individuals in 1,000,000), a slight increase in cancer risk above background [17]. Inhalation comparison values for non-cancer endpoints have not been developed by ATSDR but the California reference exposure level (REL) for acute inhalation is 0.19 μg/m^3, and the chronic inhalation REL is 0.03 /μg/m^3. RELs are concentrations at or below which no adverse health effects are anticipated for a specified exposure duration and are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety [18].

Arsenic has been suggested as a developmental toxicant at least at levels also causing maternal toxicity. Epidemiological evidence indicates an association in the Hispanic population with ambient air levels greater than 0.10 μg/m^3, and suggesting possible genetic polymorphisms affecting folate metabolism [19]. Arsenic levels reported in McFarland were at least 100 times less than levels potentially causing adverse birth outcomes.

Chromium

Total chromium was reported, but hexavalent chromium is the chromium species of greatest toxicological interest because of its greater hazard. Following usual convention to estimate toxicity from total chromium levels in the absence of specific hexavalent chromium sources, it is assumed that 1/6 of the total chromium is hexavalent chromium. Continuous exposure to the

mean concentration (0.00065 μg/m^3) attributable to hexavalent chromium exceeds the ATSDR CREG for hexavalent chromium (0.00008 μg/m^3), resulting in a 30-year estimated increase in cancer risk of 4E-06 above background (4 individuals in 1,000,000), based on a unit risk of 0.012/μg/m^3 [20]. The ATSDR chronic non-cancer comparison value (0.005 μg/m^3) was not exceeded.

Manganese

The ATSDR non-cancer comparison value (0.04 μg/m^3) for lifetime chronic inhalation exposure to manganese was exceeded in 7 (range, 0.0009 – 0.63) of the 76 samples. Exposure to this frequency is considered rare and not a cause for public health concern for chronic (long-term) exposures. Adverse effects from acute (short-term) inhalation exposure to manganese have not been reported in the scientific literature and health effects have not been reported in humans for chronic inhalation exposures less than 27 μg/m^3 [21]. Manganese has not been classified as to its carcinogenicity because of inadequate scientific information.

Benzene

Benzene (mean of detected values, 0.58 μg/m^3) slightly but consistently exceeded the ATSDR CREG (0.1 μg/m^3) in all detections (66 detections/76 samples).The resulting 30-year estimated cancer risk for continuous exposure ranges from 2E-06 to 6E-07 (2 in 1,000,000 to 6 in 10,000,000), based on unit risks of 2.2E-06 to 7.8E-06/μg/m^3 [22]. EPA non-cancer comparison values were not exceeded (EPA RfC, 30 μg/m^3).

Methyl Chloride (chloromethane)

Levels of methyl chloride (maximum, 8 μg/m^3) were below the ATSDR Minimum Risk Level (MRL) of 103 μg/m^3. Methyl chloride is not currently classifiable as to its carcinogenicity.

Methylene Chloride

Two samples (maximum, 24 μg/m^3) of 76 collected exceeded the ATSDR CREG (3 μg/m^3). Exposure to this frequency is considered rare and not of public health concern for cancer risk. The ATSDR non-cancer comparison value was not exceeded (1059 μg/m^3).

Methyl Bromide (bromomethane)

One sample (13 μg/m^3) of 76 samples collected exceeded a non-cancer comparison value (chronic inhalation reference media evaluation guide, 5μg/m^3). Exposure to this frequency would be a rare event and not of public health concern. Methyl bromide is currently not classified as a carcinogen.

Carbon Tetrachloride

Carbon tetrachloride did not exceed the ATSDR chronic non-cancer comparison value (192 μg/m^3). Carbon tetrachloride (mean of exceedances 0.91 μg/m^3) exceeded the ATSDR CREG (0.07 μg/m^3) in about one-third of the samples. The quantitation limit (1.3 μg/m^3) also exceeded the CREG. Numerical values below the quantitation limit represent the approximate concentration in the sample. The resulting cancer risk estimate for a continuous 30-year exposure is 6E-06 (6 individuals in 1,000,000), based on exposure to the mean of detections exceeding the CREG and an inhalation unit risk of 0.000015/μg/m^3 [23].

Methyl isothiocyanate

Methyl isothiocyanate can be produced in moist soil from the application of the pesticides dazomet [24] and metam-sodium [25]. Maximum value (4 detections/76 samples, 0.96 $\mu g/m^3$ maximum) did not exceed California's reference exposure level (REL) for acute (1.6 $\mu g/m^3$) or subchronic (35 $\mu g/m^3$) exposures [26].

Particulate Matter 2.5

PM 2.5 (14 $\mu g/m^3$, mean) did not exceed EPA's National Ambient Air Quality Standards annual average value of 15 $\mu g/m^3$; nor was the 24-hour comparison value (65 $\mu g/m^3$) exceeded by the maximum 24-hour value of 42 $\mu g/m^3$. The annual standards are designed to protect public health, including the health of sensitive populations such as asthmatics, children, and the elderly [27].

Other Contaminants of Interest

Contaminants Without Comparison Values

Some detected chemicals do not have inhalation comparison values and the contribution to toxicity may be unknown or not indicated at these levels, considering the scarcity of toxicological reports. These contaminants included:

1,1,2-trichloro-1,2,2-trifluorethane (CFC113)

(11 detections/76 samples, 3.4 $\mu g/m^3$ maximum) CFC 113 is a type of Freon that has not been reported to be toxic to humans by inhalation exposures at levels as high as 500,000 $\mu g/m^3$ [28].

Methyl benzamide

(2 detections/78samples) Benzamide is formed when mepronil, a fungicide, and other benzanilides are exposed to sunlight [29]. Specific toxicological comparison values were not located but significant effects would not be indicated by the low exposure frequency and concentration (0.0021 $\mu g/m^3$, maximum).

Endosulfan II

(12 detections/78samples, maximum 0.0023 $\mu g/m^3$) Endosulfan I and Endosulfan II are isomers of Endosulfan. Endosulfan is not classifiable as to its carcinogenicity. Inhalation studies are rare and insufficient for development of inhalation comparison values. One substantive experimental animal study identified a no observed adverse effect level (NOAEL) of 1000 $\mu g/m^3$ in rats exposed subchronically to Endosulfan (21 exposures in 29 days) and a low observed adverse effect level (LOAEL) of 2000 $\mu g/m^3$ [30].

Phenanthrene and *acenaphthylene*

Phenanthrene (79 detections/79 samples) and acenaphthylene (73 detections/78 samples) are polycyclic aromatic hydrocarbons (PAHs) for which comparison values have not been developed. Where plausible, chemicals without comparison values are evaluated by comparing to a similar chemical with a known level of toxicity that is the highest for that class of chemical. For example, polycyclic aromatic hydrocarbons (PAHs) without comparison values are often compared to benzo(a)pyrene for cancer effects and pyrene for non-cancer effects. This is the type of evaluation conducted for the PAHs phenanthrene and acenaphthylene. The toxic equivalence factor (TEF) for both is 0.001 [31]. TEF compares the relative potency of other PAHs to

benzo(a)pyrene, for which the TEF =1. Since the maximum concentrations of phenanthrene and acenaphthylene did not exceed 0.008 μg/m^3, exposure would pose no apparent health hazard when compared to benzo(a)pyrene (TEF=1 and 1E-06 risk level = 0.0022 μg/m^3) . Neither phenanthrene nor acenaphthylene is classifiable by EPA as to its carcinogenicity due to insufficient scientific information.

Mixtures Assessment

The interactions of a chemical mixture may involve synergism, antagonism, inhibition, and promotion. By convention, ATSDR assumes additivity in the absence of specific mixtures information and considers qualitatively whether potential interactions are likely to be greater or less than additive. Scientific information on mixtures is limited.

For inhalation exposure to the mixture of benzene, toluene, ethylbenzene, and xylene (BTEX), the hazard index approach for non-cancer neurological effects was used and did not indicate that interactive effects would be likely. In addition, physiologically-based pharmacokinetic modeling results predict that metabolic interactions are negligible at concentrations < 20 ppm of each component [32]. Therefore, greater or less than additive non-cancer effects at these concentrations are unlikely for this mixture. ATSDR evaluation guidance for possible hematotoxic and carcinogenic hazards from exposure to BTEX is best approached by evaluation of benzene as a single component, resulting in a risk estimate ranging from 2E-06 to 6E-07 for a continuous, 30-year exposure.

ATSDR's summation of the estimates of cancer risk for the above contaminants (5E-05; 5 individuals in 100,000) was less than 1E-04 (1 individual in 10,000), which is ATSDR's selected level of concern for an exposure to mixtures [33].

Sensitive Populations (Child Health Considerations)

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults; this means they breathe dust, soil, and vapors close to the ground. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

When ATSDR conducts a public health assessment, it considers any factors that may result in a higher level of exposure or exposure to potentially sensitive population, such as children, infants, or the fetus. Inhalation exposures to pregnant women are of particular concern because of the distribution of volatile chemicals by the blood to the placenta and fetus compared to ingestion exposures where first-pass metabolism may reduce chemical levels before distribution to the placenta and fetus. Direct maternal exposure leading to indirect fetal exposure is a subject area with little concrete scientific information at low doses, but ATSDR considers it prudent to be

cautious where these exposures are possible. Research is needed to fill the many gaps in this area.

At McFarland, detected contaminants have not been identified as representing a unique threat to children or the fetus at the concentrations observed. ATSDR surveyed contaminants for fetotoxic potential and found no public health concern at these contaminant concentrations. However, potential exposures may have occurred during times when sampling was not being conducted or to unknown contaminants. The extensive sampling and analyses conducted at McFarland reduces this uncertainty but cannot eliminate the possibility. Exposures may also occur through residential use of contaminants or occupational exposure and subsequent take-home of contamination.

Relevant to community concerns in the McFarland area, exposure to residential or occupational pesticides has been associated with childhood leukemia in some epidemiological studies [34,35]. Take-home exposure pathways have been identified as an exposure of concern for children [36,37,38, 39]. Recent epidemiological studies have not identified an association between exposure to agricultural pesticides and childhood cancers [40,41]. Newborn children have low levels of paraoxanase-1, an enzyme which detoxifies organophosphate pesticides [42]. Individual differences in genetic makeup may also influence how one reacts to an exposure [43]. When the fetus is exposed is perhaps as important as to how much, as critical windows of development are vulnerable while exposure outside the window may not result in effects [44,45]. While research indicates increased exposure to children living near agricultural areas or whose parents work in agricultural areas, childhood cancers have not been associated with such exposure in California.

Discussion

Adverse health effects would not be expected from exposure to individual contaminants if sampling is representative of actual exposure concentrations. Considering the comprehensive sampling and analysis effort, confidence in the representativeness of the data is high. What science cannot predict with confidence is the effect that interactions between multiple chemicals may have on health. Because chemicals with similar modes of action were present in low concentrations, significant interaction would not be expected, but the uncertainty remains.

The effect of contaminants whose detection limits were above comparison values represents additional uncertainty, but it is also unlikely that these non-detections would make a significant contribution, as exceeding a comparison value does not indicate that health effects would be likely and the comparison value would have to be significantly exceeded before the contaminant would be of public health concern.

Although epidemiological data may not prove causation, some studies have identified an association between household pesticide exposure and childhood cancers. Recent studies in California have not identified an association between agricultural pesticides and childhood cancers. Since 1990, statewide commercial agricultural pesticide (full use) has been tracked by California's Department of Pesticide Regulation [46]. Pesticide use reporting before 1990 may not have been complete for all pesticide uses and use may have been higher in the more distant past than the more recent past.

<u>What we know</u>

Individual chemicals were detected at low levels in ambient air or indoor dust. The levels of individual chemicals in ambient air or indoor dust reported in the study are not of public health concern for exposures to individual chemicals and mixtures for which sufficient toxicological data exists. Exposure levels were far below levels reported to cause health effects and below levels believed to protect sensitive populations. There is a low increase in the estimate of cancer risk above background for some chemicals, but the upper-bound cancer risk estimates indicate a low probability of cancer health effects.

These conclusions are based on reported levels and are assumed to be representative of exposure. Sampling was planned to capture the most relevant periods of potential exposure to agricultural pesticide applications. EPA sampled for over 180 contaminants in ambient air including pesticides, metals, semi-volatile and volatile organics. Indoor dust samples were also collected from the schools and analyzed. Sampling, analysis, and data presentation were transparent and comprehensive.

What we do not know

While every attempt is made to include all contaminants of potential concern, there may be contaminants that have not been considered. Sampling is designed to be representative of environmental conditions over time, but may not capture all conditions. Some chemicals do not have comparison values because of inadequate scientific information. Scientific information on the joint toxic actions of chemical mixtures is very limited. Scientific knowledge of the effects of indirect fetal exposures is limited.

Conclusions

Exposure to levels of individual contaminants reported in ambient air and indoor dust represent *no apparent health hazard* for those contaminants with sufficient toxicological information to establish relevant comparison values.

Conclusions regarding the potential hazard from exposures to individual contaminants with no comparison value are made with much less confidence. Such exposures are unlikely to be a health hazard considering indications from limited data, limited exposure, and low level of contamination.

Conclusions regarding exposures to mixtures of contaminants are also made with less confidence but no health hazard is apparent for those mixtures having toxicological information. Significant interaction among other contaminants is unlikely considering the low levels of contamination and limited exposure.

Recommendations

None.

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Table 1: Contaminants of Concern from Initial Screening, Ambient Air and Indoor Dust, McFarland, California

Contaminant/ media	*Detections>CV/ Samples*	*Concentration[a] µg/m³ air or pg/g dust*	*Comparison Value*	*Estimated Cancer Risk*
Dioxins/furans/PCBs in indoor dust	0/9	1.9-20 range 6. mean	50 pg/g ATSDR[b]	NA[c]
Formaldehyde in ambient air	68/73	ND[d]-20 range 6.8 mean	0.08 µg/m³ CREG[e]	4E-05
Arsenic in ambient air	64/76	0.00025-0.011 range 0.0012 mean	0.0002 µg/m³ CREG	2E-06
Chromium in ambient air	57/76	ND-0.014 range 0.00065 mean	0.00008 µg/m³ CREG	4E-06
Manganese in ambient air	7/76	0.0009-0.63 range	0.04 µg/m³ EMEG[f]	NA
Benzene in ambient air	66/76	ND-1.3 range 0.59 mean	0.1 µg/m³ CREG	2E-06
Methylene Chloride in ambient air	2/76	24. maximum	3. µg/m³ CREG	NA
Methyl Bromide in ambient air	1/76	13.	5. µg/m³ RMEG[g]	NA
Carbon Tetrachloride in ambient air	24/76	ND-1.8 0.95 mean	0.07 µg/m³ CREG	6E-06
Particulate Matter (2.5 µm) in ambient air	0/73	4.1- 42. range 14. mean	15. average 65. 24-hour max	NA

a µg/m³ (micrograms per cubic meter) air; pg/g (picograms per gram) dust.

b ATSDR Updated Policy Guideline for Dioxins and Dioxin-like Compounds in Residential Soil, March 25, 2005.

c NA. Not Available or Not Applicable.

d ND. Not Detected.

e CREG. Cancer Risk Evaluation Guideline. Levels based on continuous lifetime exposure at 1E-06 risk.

f EMEG. Environmental Media Exposure Guide. Calculated media concentrations from predicted exposure at ATSDR Minimum Risk Levels (MRLs).

g RMEG. Reference Media Exposure Guide. Calculated media concentrations from predicted exposure at EPA Reference Concentration (RfC).

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