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JANICEK BREWER, GOVERNOR
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March 18, 2011

[redacted]

Dear [redacted]

RE: Community Garden

This letter health consultation has been completed by the Arizona Department of Health Services (ADHS), through support with funds from a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Health and Human Services, for the proposed community garden located at the confluence of Miller and Granite Creeks in Prescott, Arizona. This document has not been reviewed or cleared by ATSDR. In this report, ADHS reviewed the soil sampling results and conducted an assessment to evaluate whether people's health will be harmed if they consume fruits and/or vegetables grown in the proposed community garden.

Background and Statement of Issue

Prescott College plans to plant a community garden with all types of fruits and vegetables. The proposed garden would replace native vegetation that was established during a restoration project at a site located where Miller and Granite Creeks converge in Prescott, Arizona. Historically, a manufactured gas plant operated until approximately 50 years ago. The manufacturing activities impacted the soil at the site. Arizona Public Service (APS) currently owns the site and worked with the Arizona Department of Environmental Quality's (ADEQ) Voluntary Remediation Program (VRP) to address the contamination concerns. Groundwater monitoring wells were installed, and APS and DEQ took and analyzed water and soil samples to determine the nature and extent of the contamination. In 1999, contaminated soil was removed, treated, and restored to the site. Later, APS removed soil from about one acre of land, until the level of contaminants were either no longer detectable or below the defined target concentrations. Soil between 2-10 feet deep was removed, depending on contamination of the area. Soil from the Willow Lake area of Prescott was used as replacement fill. The removed soil was incinerated and disposed of in Maricopa Valley. In 2001 a local restoration company called "Riparia" reestablished vegetation (mainly perennial grasses and cottonwoods) on the site.

The major concern of the community is that if hazardous chemicals were not entirely removed from the site, these chemicals could be bioaccumulated in the fruits and vegetables, and subsequently impact the health of the community members who consume the fruits and vegetables harvested from the garden.

Discussion

Environmental Data

Three soil samples were collected from the site and analyzed in accordance with ADHS 8015 AZR1 method for fuel hydrocarbons, EPA 5035/8015D method for volatile fuel hydrocarbons, EPA 5035/8260B method for BTEX (benzene, ethylbenzene, toluene, and total xylene), and EPA 3545/8310 method for PAHs (polynuclear aromatic hydrocarbons). The analytical results from ADHS 8015 AZR1 method were not used to evaluate the potential health effects because this method is a general screening method. Analytical results from the ADHS 8015 AZR1 method represent the entire range of petroleum hydrocarbons in the soil samples. This method does not divide the broad chemical classes of petroleum hydrocarbons into subgroups of compounds such as alkanes, cycloalkanes and aromatics; hence the results cannot provide discrete estimates that can be used for site-specific risk assessments.

The laboratory shows that most of chemicals are at concentrations below their reporting limits. Low levels of a few polynuclear aromatic hydrocarbons (PAHs) are detected in site 2 and site 3 soil samples. The potential health effects due to exposure to these PAHs are discussed below.

Exposure Evaluation

The major ways that local residents could come into contact with the contaminants in the soil are through unintentional consumption of soil on hands or food items, and through consumption of contaminated homegrown produce.

Public Health Implication

In order to determine the risk of illness from contaminated soil, ADHS used exposure models and risk factors developed by ATSDR and U.S. Environmental Protection Agency (EPA). ATSDR or EPA's models consider only the potential intake of contaminants from incidental soil ingestion. The models do not consider exposures through homegrown produce. If the concentration of a contaminant meets or exceeds a health-based Comparison Value (CV), then it is considered for further analysis. This process is called screening. The health-based CVs are concentrations of contaminants that the current public health literature suggest are "safe" or "harmless." These comparison values are quite conservative, because they include ample safety factors that account for most sensitive populations. ADHS typically uses comparison values as follows: if a contaminant is never found at levels greater than its CV, ADHS concludes the levels of corresponding contamination are "safe" or "harmless." If, however, a contaminant is found at levels at greater than its comparison value, ADHS designates the pollutant as a *contaminant of interest* and examines potential human exposures in greater detail. Detected PAH levels in the soil were screened and no PAHs exceeded non-cancer CVs (See Table 1). Thus, people are not like to get cancer/non-cancer health effects from incidental soil ingestion at the proposed community garden area.

The risk from homegrown produce consumption pathway is determined by estimating the level of uptake of PAHs by produce and food consumption habits. Plants absorb PAHs from soil and readily translocate them to above-ground tissues. The amount of PAH can be transferred from soil to homegrown produce is determined by various factors such as chemical property, soil concentration, soil property, climate, and plant species. ADHS used soil-to-biota bioaccumulation equations developed by EPA (see Table 2) to estimate concentrations of PAHs in homegrown produce, and assumed soil samples consisting of 10% water. The soil-to-biota bioaccumulation uptake equations were derived based on published data and literature (US EPA 2003). The homegrown produce consumption rates for different age groups were adapted from the EPA Exposure Factors Handbook (EPA 1997). To convert the wet-weight consumption rates to dry-weight consumption rates, ADHS assumed that homegrown produce consisted 90% of water. The estimated Chronic Daily Intakes (CDIs) for various PAHs do not exceed their respective Reference Doses (RD) which is the EPA's maximum acceptable oral dose for a toxic substance (See Table 3). In

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general, CDIs less than the RfD are not likely to be associated with non-cancer health risks, and are therefore less likely to be of a health concern.

ADHS also evaluated the theoretical cancer risk from homegrown produce consumption. The carcinogenicity of certain PAHs is well established in laboratory animals. Researchers have reported increased incidences of skin, lung, bladder, liver, and stomach cancers, as well as injection-site sarcomas, in exposed lab animals. Increased incidences of lung, skin, and bladder cancer are associated with occupational exposure to PAHs in humans (ATSDR 2009). Because of the complexity of PAH mixtures, the most active compound, benzo(a)pyrene [B(a)P], is used as the indicator compound. While not all PAHs are considered carcinogenic (e.g. pyrene), the EPA has determined that benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, and indeno(1,2,3-c,d)pyrene are probable human carcinogens (ATSDR 1995).

B(a)P was used as a surrogate to assess the relative toxicity of the carcinogenic PAHs (cPAHs) classified by EPA as probable human carcinogens. To determine the toxicity of the mixture of PAHs, the concentration of each cPAH was multiplied by a Toxic Equivalency Factor (TEF) which relates its toxicity to that of B(a)P. The sum of the weighted concentrations can be used to evaluate the PAH mixture (US EPA 1993). The theoretical ingestion cancer risks for people consuming homegrown produce from site 2 and site 3 are 3.5×10^{-5} and 4.6×10^{-5}, respectively (see Table 6). That indicates that there may be a slight increase in cancer risk. The theoretical excess cancer risk is about 4 to 5 additional occurrences of cancer in a population of 100,000 due to exposure to cPAH contaminated homegrown produce[1]. The estimated excess cancer risk is within US EPA's guidance range (10^{-6} to 10^{-4}) for the protection of human health.

Conclusion

Based on the available information, the local residents are not like to get cancer/non-cancer health effects from incidental ingestion of the PAH-contaminated soil at the proposed community garden area. However, the PAHs can be transferred from soil to homegrown produce. Therefore, the local residents could uptake PAHs by consume PAH-contaminated homegrown produced foods. Based on the available information and assumptions, ADHS concludes that exposures to contaminated homegrown produce are not expected to result in non-cancerous health effects among adults and children. In addition, the estimated cancer risks were within the EPA's guidance range for the protection of human health.

Recommendation

To reduce the exposure to PAHs, ADHS recommends washing the homegrown produce properly before consumption. If you have any questions, please contact me at (602) 803-3740 or linh@azdhs.gov.

Sincerely,

Hsin-I Lin, ScD, MSPH
Toxicologist

[1] There is a background incidence of cancer in the general population due to everyday exposure to common materials. Nearly half of all men and one-third of all women in US population will develop cancer at some point in their life (American Cancer society 2008).

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Reference

1. Agency for Toxic Substances and Disease Registry (ATSDR). 1995. Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs). Atlanta, GA: US Department of Health Services, Public Health Service

2. Agency for Toxic Substances and Disease Registry (ATSDR). 2009. Case Studies in Environmental Medicine (CSEM). Toxicity of Polycyclic Aromatic Hydrocarbons (PAHs). Available at: http://www.atsdr.cdc.gov/csem/pah/index.html.

3. U.S. Environmental Protection Agency (U.S. EPA). 1997. Exposure Factors Handbook (Final Report). Washington, DC, EPA/600/P-95/002F a-c.

4. U.S. Environmental Protection Agency (US EPA). 2003. Guidance for Developing Ecological Soil Screening Levels. US EPA, Office of Solid Waste and Emergency Response, Washington, DC. OSWER Directive 92857-55.

5. U.S. Environmental Protection Agency (US EPA). 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons

6. U.S. Environmental Protection Agency (US EPA). Integrated Risk Information System (IRIS) 2011. http://www.epa.gov/iris Accessed March 2011.

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Table 1. Summary of Detected PAH Results and their respective screening levels

PAH	Concentration (mg/kg)		Screening Level (mg/kg)	Source	Toxic Equivalent Factor	EPA Cancer Class
	Site 2	Site 3				
Fluoranthene	0.065	0.130	2,300	SRL		D
Phenanthrene	0.044	0.075	2,300*			D
Benzo(a)anthracene	0.019	0.032	0.69	SRL	0.1	B2
Benzo(a)pyrene	0.040	0.054	0.069	SRL	1	B2
Benzo(b)fluoranthene	0.035	0.072	0.69	SRL	0.1	B2
Benzo(g,h,i)perylene	0.041	0.040	2300*			D
Benzo(k)fluoranthene	0.017	0.036	6.9	SRL	0.01	B2
Chrysene	0.024	0.057	68	SRL	0.001	B2
Indeno(1,2,3-cd)pyrene	0.046	0.049	0.69	SRL	0.1	B2
Pyrene	0.077	0.130	2,300	SRL		D
Total B(a)P Equivalent	0.050	0.069	0.069			

* Pyrene SRL value was used as a surrogate
SRL: Arizona Soil Remediation Level
B2: Probable human carcinogen (inadequate human, sufficient animal studies)
D: Not classified as to health carcinogenicity
Total B(a)P Equivalent: sum of all carcinogenic polycyclic aromatic hydrocarbons (cPAH) toxic equivalent (TEQ); all cPAH are added using the TEQ approach to obtain Total B(a)P Equivalent

Table 2. soil-to-biota bioaccumulation equations used to predict the amount of PAH bioaccumulated in homegrown produce (EPA 2003)

Chemical	**Equation**
Fluoranthene	$C_p = 0.5 \times C_s$
Phenanthrene	$\ln(C_p) = 0.6203 \times \ln(C_s) - 0.1665$
Benzo(a)anthracene	$\ln(C_p) = 0.5944 \times \ln(C_s) - 2.7078$
Benzo(a)pyrene	$\ln(C_p) = 0.9750 \times \ln(C_s) - 2.0615$
Benzo(b)fluoranthene	$C_p = 0.31 \times C_s$
Benzo(g,h,i)perylene	$\ln(C_p) = 1.1829 \times \ln(C_s) - 0.9313$
Benzo(k)fluoranthene	$\ln(C_p) = 0.8595 \times \ln(C_s) - 2.1579$
Chrysene	$\ln(C_p) = 0.5944 \times \ln(C_s) - 2.7078$
Indeno(1,2,3-cd)pyrene	$C_p = 0.11 \times C_s$
Pyrene	$C_p = 0.72 \times C_s$

C_p: chemical concentration in the plant (mg/kg, dry weight)
C_s: chemical concentration in the soil (mg/kg, dry weight)

Table 3. Estimated chronic daily intakes (CDI) from homegrown produce consumption

Chemical	Exposure Group	Site 2 Soil	Site 3 Soil	RfD (mg/kg/day)
Fluoranthene	Child (1 – <2 yr)	0.000039	0.000079	0.04
	Child (2 – <6 yr)	0.000018	0.000037	
	Adult (6 – <19 yr)	0.000013	0.000026	
	Adult (19 – <31 yr)	0.000010	0.000019	
Phenanthrene	Child (1 – <2 yr)	0.000163	0.000227	0.03*
	Child (2 – <6 yr)	0.000076	0.000106	
	Adult (6 – <19 yr)	0.000053	0.000074	
	Adult (19 – <31 yr)	0.000040	0.000056	

Chemical	Exposure Group	Site 2 Soil	Site 3 Soil	RfD (mg/kg/day)
Benzo(g,h,i)perylene	Child (1 – <2 yr)	0.000012	0.000012	0.03*
	Child (2 – <6 yr)	0.000006	0.000006	
	Adult (6 – <19 yr)	0.000004	0.000004	
	Adult (19 – <31 yr)	0.000003	0.000003	
Pyrene	Child (1 – <2 yr)	0.000067	0.000112	0.03
	Child (2 – <6 yr)	0.000031	0.000053	
	Adult (6 – <19 yr)	0.000022	0.000036	
	Adult (19 – <31 yr)	0.000016	0.000028	

*Pyrene RfD value was used as a surrogate
RfD: Reference Dose, which is the EPA's maximum acceptable oral dose for a toxic substance

Table 4 Formula and parameters used to calculate chronic daily intake (CDI) from homegrown produce ingestion (mg/kg/day)

$$CDI = \frac{Conc \times IR \times EF \times ED}{AT}$$

Parameter		Unit	Child (1 – <2 yr)	Child (2 – <6 yr)	Adult (6 – <19 yr)	Adult (19 – <31 yr)
Conc	Chemical concentration in homegrown produce	mg/kg	Chemical-specific			
IR	Homegrown produce Ingestion rate	kg/kg/day	0.001394	0.000653	0.000452	0.00342
EF	Exposure frequency	day/year	350	350	350	350
ED	Exposure duration	year	1	4	13	12
AT	Averaging time	days	365	1460	4745	4380

Table 5. Formula and parameters used to calculate Cancer Risk from homegrown produce ingestion. *i*: age-group *i*

$$Cancer\ Risk_i = Conc \times \frac{IR_i \times EF_i \times ED_i}{AT} \times SF \times ADAF_i \times TEF$$

$$Total\ Cancer\ Risk = \sum Cancer\ Risk_i$$

Parameter		Unit	Child (1 – <2 yr)	Child (2 – <6 yr)	Adult (6 – <19 yr)	Adult (19 – <31 yr)
Conc	Chemical concentration in homegrown produce	mg/kg	Chemical-specific			
IR_i	Homegrown produce Ingestion rate	kg/kg/day	0.001394	0.000653	0.000452	0.00342
EF_i	Exposure frequency	day/year	350	350	350	350
ED_i	Exposure duration	year	1	4	13	12
AT	Averaging time	days	25,550	25,550	25,550	25,550
SF	Slope Factor for BaP	$(mg/kg/day)^{-1}$	7.3	7.3	7.3	7.3
$ADAF_i$	Age-dependent adjust factor	–	10	3	1	1
TEF	Toxic Equivalent Factor	–	Chemical-specific			

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Table 6. Estimated Cancer Risk from homegrown produce consumption

Site 2 Soil

Chemical	Cancer Risk for Each Age Interval				Total Risk
	1 – <2 yr	2 – <6 yr	6 – <19 yr	19 – <31 yr	
Benzo(a)anthracene	8.4×10^{-7}	4.7×10^{-7}	1.1×10^{-6}	2.5×10^{-7}	2.6×10^{-6}
Benzo(a)pyrene	8.4×10^{-6}	4.7×10^{-6}	1.1×10^{-5}	2.5×10^{-6}	2.6×10^{-5}
Benzo(b)fluoranthene	1.4×10^{-6}	7.8×10^{-7}	1.8×10^{-6}	4.1×10^{-7}	4.3×10^{-6}
Benzo(k)fluoranthene	4.2×10^{-8}	2.4×10^{-8}	5.3×10^{-8}	1.2×10^{-8}	1.3×10^{-7}
Chrysene	2.8×10^{-9}	1.6×10^{-9}	3.5×10^{-9}	8.2×10^{-10}	8.7×10^{-9}
Indo(123-cd)pyrene	7.0×10^{-7}	3.9×10^{-7}	8.8×10^{-7}	2.1×10^{-7}	2.2×10^{-6}
Total Cancer Risk from the Mixture					3.5×10^{-5}

Site 3 Soil

Chemical	Cancer Risk for Each Age Interval				Total Risk
	1 – <2 yr	2 – <6 yr	6 – <19 yr	19 – <31 yr	
Benzo(a)anthracene	1.3×10^{-6}	7.1×10^{-7}	1.6×10^{-6}	3.7×10^{-7}	3.9×10^{-6}
Benzo(a)pyrene	9.8×10^{-6}	5.5×10^{-6}	1.2×10^{-5}	2.9×10^{-6}	3.0×10^{-5}
Benzo(b)fluoranthene	2.8×10^{-6}	1.6×10^{-6}	3.5×10^{-6}	8.2×10^{-7}	8.7×10^{-6}
Benzo(k)fluoranthene	9.8×10^{-8}	5.5×10^{-8}	1.2×10^{-7}	2.9×10^{-8}	3.0×10^{-7}
Chrysene	5.6×10^{-9}	3.1×10^{-9}	7.0×10^{-9}	1.6×10^{-9}	1.7×10^{-8}
Indo(123-cd)pyrene	7.0×10^{-7}	3.9×10^{-7}	8.8×10^{-7}	2.1×10^{-7}	2.2×10^{-6}
Total Cancer Risk from the Mixture					4.6×10^{-5}

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Appendix A: Qualitative Descriptors for Excess Lifetime Cancer Risk

Cancer Risk	Qualitative Descriptor
Equal to or less than one per million (Cancer Risk $\leq 10^{-6}$)	Very Low
Greater than one per million to less than one per ten thousand (10^{-6} < Cancer Risk $\leq 10^{-5}$)	Low
Greater than one per ten thousand to less than one per thousand (10^{-5} < Cancer Risk $\leq 10^{-4}$)	Moderate
Greater than one per thousand to less than one per ten (10^{-4} < Cancer Risk < 10^{-1})	High
Equal to or greater than one per ten (Cancer Risk $\geq 10^{-1}$)	Very High

An estimated increased excess lifetime cancer risk is not a specific estimate of expected cancers. Rather, it is a plausible upper-bound estimate of the probability that a person may develop cancer sometime in his or her lifetime following exposure to that contaminant.

There is insufficient knowledge of cancer mechanisms to decide if there exists a level of exposure to a cancer-causing agent below which there is no risk of getting cancer, namely, a threshold level. Therefore, every exposure, no matter how low, to a cancer-causing compound is assumed to be associated with some increased risk. As the dose of a carcinogen decreases, the chance of developing cancer decreases, but each exposure is accompanied by some increased risk.

There is general consensus among the scientific and regulatory communities on what level of estimated excess cancer risk is acceptable. The EPA considers an acceptable cancer risk range from 10^{-6} to10^{-4}.

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