Health Consultation

GILA RIVER INDIAN COMMUNITY TOXAPHENE SITE

BOUNDARY SITE
DISTRICT 6

MARICOPA COUNTY, ARIZONA

EPA FACILITY ID: AZSFN0905455

FEBRUARY 23, 2005

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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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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HEALTH CONSULTATION

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BOUNDARY SITE
DISTRICT 6, GILA RIVER INDIAN COMMUNITY

MARICOPA COUNTY, ARIZONA

EPA FACILITY ID: AZSFN0905455

Prepared by:

Gila River Indian Community
Department of Public Health
Office of Occupational Safety and Health
Under Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

Background and Statement of Issues

The GRIC Office of Occupational Safety and Health (OSH), through a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) prepared this health consultation at the request of Gila River Indian Community (GRIC) members who lived on the "Boundary site," an area south of Phoenix, Arizona. This consultation addresses concerns about possible health effects from past exposure to pesticides from previous crop dusting activities. Specifically, it evaluates whether current conditions at the site pose a threat to health, and whether the property can be re-inhabited for residential or commercial use. Data available to the OSH office is presented in the background section, followed by a discussion of the health implications, conclusions, and recommendations.

The site, also known as Border site and as Thomas-Yazzie site, is in District 6 of the GRIC, northwest of the 51st Avenue and Komatke Lane intersection, Maricopa County, Arizona. The site is within Section 20, Township 1 South, Range 2 East, of the Gila and Salt River baseline and meridian (Appendix A photographs). The Boundary site encompasses approximately 40 acres. Agricultural lands are north, northeast, and northwest, and a casino is located across 51st Avenue, which is east of the site. South and west of the site is native, undeveloped land. Geographic coordinates for the site are 33°19'31" N and 112°10'14" W.

The site is predominately undeveloped land covered by native, sparse vegetation. A former 2,357-foot, dirt runway ran northeast-southwest on the site, and is still visible. The presence of a second, shorter airstrip is suspected. A former pesticide container dumping area was located east of the longer airstrip. In the late 1970s, a single-family residence was built on the northern portion of the site. Another single-family residence was built on the southern portion of the site in 1994. Pieces of those structures, including concrete slabs, burnt support beams, and septic and utility connections, remain at the site. Several concrete-lined irrigation canals traverse the site (Appendix A). In addition, trash piles containing household refuse and two empty, rusted, 20-gallon degreaser drums were found on the premises.

According to information obtained during interviews and during reviews of historical information, several crop-dusting services operated at the property from 1959 through the early 1980s. Additionally, previous crop-dusting operators reportedly stored and mixed pesticides [toxaphene, Azodrin® (monocrotophos), methyl-parathion, Lannate® L (methomyl), DEF® 6 (S,S,S-tributyl phosphorotrithioate), Ambush® (permethrin), Bolstar 6 (sulprofos), or Furadan® (carbofuran)] on the northern portion of the property[1]. Although the site encompasses approximately 40 acres, the affected soil is limited to approximately 10.75 acres.

In the late 1970s, a family moved onto the northern portion of the Boundary site. At that time, the family consisted of two adults and three children. During the next several years, up to 14 family members lived on the premises. The family first lived in a silver trailer and later built a traditional "sandwich house" (adobe or mud house), where they slept on the dirt floor. Crop dusting sometimes occurred while the family cooked outside over an open fire and sat underneath a "watto" (i.e., an open-sided ramada or shaded patio). The children played in the unnaturally yellow soil, and bathed and swam in the yellow-colored canal water. The family also washed their clothes in the canal water. The family hauled their drinking water from the District 6 north well located approximately 0.75 miles from the site. The drinking water, which had yellow film on the surface, was stored in pails underneath the watto.

In the mid-1980s, the family living on the northern portion of the site began asking about their safety and health when they noticed strong chemical odors around their home. Family members have expressed concern that past exposure to pesticides is related to their current medical conditions. They reported having reproductive disorders (such as multiple miscarriages and sterility), kidney and liver complications, respiratory problems, severe headaches, and skin lesions and rashes.

According to 1984 records, the United States Environmental Protection Agency (EPA) conducted initial environmental sampling at the site. Organophosphorus compounds (methyl parathion, ethyl parathion, and s,s,s-tributyl phosphorotrithioate (DEF®)) and one camphene organochlorine compound (toxaphene) were found in soil on the site. Later, the area was deemed a Comprehensive Environmental Response Compensation and Liability Information System (CERCLIS) site[2]. An EPA contractor stated that most of the hazardous waste at the site was likely created by spills during mixing, loading, and handling of pesticides and herbicides before application to crops[3]. Toxaphene was detected at approximately 15,000 parts per million (ppm)1 in the site surface soil samples. EPA concluded the site constituted an immediate risk to public health and to the environment[2].

The nine family members living at the site were tested for organophosphate exposure. Blood serum cholinesterase samples were collected on July 3, 1984. The samples were drawn and analyzed for serum cholinesterase activities, which controls the proper functioning of a neurotransmitter (acetylcholine).

Organophosphates are potent cholinesterase enzyme inhibitors that interfere with the metabolism of acetylcholine, causing an accumulation of acetylcholine at neuroreceptor transmission sites. Although baseline data were not available for the patients, serum cholinesterase activities were interpreted as falling within an acceptable range of 1,900–3,800 millimoles per milliliter (mmol/mL) for five of the individuals (July 3, 1984). Four individuals had cholinesterase levels indicating they were possibly exposed to organophosphates[4]. Because the family reported direct spraying, and because low cholinesterase levels were found, exposure to organophosphates was considered a reasonable possibility. (Table 1 presents results of the tests.)

1 An example might help illustrate the part per million references. One part of 1,000,000 (10^6) is 1 ppm. Parts per million are appropriate measures for the relative abundances of rare elements in the earth's crust and concentrations of pollutants in the environment.

Table 1. Blood Serum Cholinesterase Test Results, July 3, 1984

Sex/Age (years)	*Cholinesterase Blood Test Result (mmol/mL)*	*Outside Acceptable Range?*
Male – Age 5	2,730	No
Female – Age 5	899	Yes
Male – Age 8	1,622	Yes
Female – Age 11	1,106	Yes
Female – Age 12	1,926	No
Male – Age 13	2,093	No
Female – Age 33	1,639	Yes
Female – Age 64	2,799	No
Male – Age 65	2,347	No

According to records dated August 8, 1984, EPA had the nine original family members tested again for red blood cell (RBC) and serum (plasma) cholinesterase levels. Eight other family members were also tested. Levels for three family members fell outside the acceptable range of 6,700–10,000 mmol/mL RBC, but the level was approaching the acceptable range (August 8, 1984). Serum Cholinesterase tests results were all in an acceptable range. No biological test results were found in the available data related to organochlorine exposure at the Boundary site. Because the tests lack a baseline they cannot be used to confirm exposure, but they do help support the decision to relocate the family. On August 7, 1984, again pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), EPA initiated an emergency removal action by temporarily relocating families living at this site[5].

Weston-Sper Technical Assistance Team for Emergency Response, Removal, and Prevention was contracted to conduct remedial activities at the site. After investigating the treatment options, the site was targeted for a two-phase chemical and biological treatment scheme. The goal was to reduce the concentrations of pesticides to background levels, which Weston-Sper approximated to be 12–25 ppm of toxaphene in the surrounding fields[6].

Contaminated soil was removed and disposed at an off-site disposal facility. The most contaminated soil in one zone was removed; 23 truckloads of soil were transported to U.S. Ecology facilities in Beatty, Nevada, for disposal. On-site soils were both chemically and biologically treated. The remaining on-site soil was initially treated in place (i.e., *in situ*). The Phase I chemical treatment consisted of pH adjustment (initial average of 11.8 pH units which decreased to 10.2 pH units after 69 days) using sodium hydroxide, continued moisturizing, and weekly soil turning by tractor and disc. The site was leveled, the soil neutralized, nutrients were added, and a drip irrigation system installed. In Phase II the site was covered with plastic sheeting to enhance the anaerobic biodegradation processes. To evaluate soil remediation efforts, in September 1984 additional environmental samplings were conducted on the northern portion of the site. According to the available information, the following assumptions were made:

- The applied soil was stabilized,

- The climatic conditions were dry and windy,
- The soil permeability was low,
- The depth to groundwater was greater than 100 feet, and,
- The site was generally flat, with no surrounding surface waters[7].

After Phase I samples were collected to determine the effectiveness of treatment 15, 36, and 69 days following the sodium hydroxide application. Results of sampling (Appendix B) indicated that the combination of sodium hydroxide and water had degraded ethyl parathion very quickly, probably by alkaline hydrolysis. Concentrations of ethyl parathion decreased by more than 50% in 15 days and by 76% after 69 days. Concentrations of methyl parathion decreased even more rapidly: after 15 and 69 days, reductions of 81% and 98%, respectively, were noted (Figure 1)[8]. These results are from chemical treatment only. No Phase II anaerobic treatment data were found.

Toxaphene reduction was less impressive. On the basis of the analytical data obtained during the investigation, the concentration of toxaphene remaining in the soil had been reduced by approximately 45%.

Nutrient sources and alfalfa were applied to the site to encourage biodegradation of toxaphene in the soil. A soil cap was installed near the northern home site. The soil cap, with 200 cubic yards of soil, consisted of 6–15 inches of clean fill dirt. No plan for maintenance of the soil cap was instituted. In June 1985, after 69 days, the chemical treatment was terminated[6].

EPA hired environmental consultants Roy F. Weston, Inc. (Weston-Sper), to conduct a risk assessment at the Boundary site. The risk assessment, which was completed in October 1985, concurred that either contact with or consumption of the contaminated soil could produce adverse health effects in humans. The assessment indicated a carcinogenic risk of 1 in 1 million from exposure to the toxaphene in the soil at the site. Weston-Sper estimated that it would require approximately 30 years for the toxaphene to degrade naturally to background levels. The assessment stated that residents could return to the site; however, residents needed to be advised about the implications of digging in the cap and soil, and about actions that must be taken were they to plant trees or cultivate soil. Weston also recommended that a cap monitoring program be developed to monitor the integrity of the soil cap[9].

The family that had lived on the northern portion of the land was allowed back in late 1985. Then, at the owner's request, in 1990 the entire family moved off the property. Records show that a three-member family lived on the southern portion of the site from February to May 1994. The owner of the southern portion of the site stated, however, that in August 1994 — rather than in May — EPA had ordered him to leave. The area of the site where this family lived was 660 feet south of the contaminated northern portion of the site. In July 1994, responding to the family's health concerns, the pesticide control officer of the Gila River Department of Land and Water Resources collected three soil samples from 3 feet below ground surface (bgs) at the southernmost home site. The preliminary soil samples indicated the presence of toxaphene at 41 ppm in one sample. All three samples contained dichlorodiphenyldichloroethylene (DDE) at 0.07–1 ppm. Later, the Arizona State Agricultural Laboratory confirmed and amended the results to 38 ppm toxaphene and no detection of DDE. The laboratory determined that the presence of "high levels" of toxaphene caused a false reading for DDE[10]. The family was relocated to a new, off-site location.

Under the direction of EPA and the Gila River Indian Community Department of Environmental Quality (GRIC DEQ), various environmental consultants conducted environmental samplings. A discussion of the sampling events and the results of sample analyses follow.

Environmental Contamination

On June 14, 1984, the GRIC Department of Physical Resources, Pesticide Control Office, contacted EPA's Emergency Response Section concerning the Boundary site soil-testing results. Initial sampling of soils at the site indicated the presence of toxaphene at concentrations as high as 15,845 ppm, as well as lesser concentrations of 4,4'-DDE, methyl parathion, ethyl parathion, and DEF®. Results from the September 1984 samples prompted further sampling on the northernmost portion of the site. Soils tested contained concentrations of methyl parathion at up to 8,280 ppm, ethyl parathion at up to 5,830 ppm, and DEF® at up to 91 ppm (See Table 2)[3].

Table 2. Pesticide Concentrations in Soils Collected from District 6, Boundary Site, Gila River Indian Community (GRIC), Arizona, including results from GRIC Department of Environmental Quality (DEQ) and Ecology and Environment, Inc. (E&E)

Contaminant ofConcern	*Range of Sample Concentration (ppm)*	*Soil Sample depths*	*MaximumSoil Concentration (ppm)*	*ATSDR Soil Comparison Value (CV) (ppm)*	*Residential Arizona Soil Remediation Level (SRL) (ppm)*	*Exceeds CV or SRL?*
1984						
Toxaphene	552–15,845	NA	15,845	CREG 0.6	4	Yes
DEF®	21.7– 91	NA	91	RMEG 2	Not established	Yes
Methyl Parathion	40.4–8,280	NA	8,280	EMEG 1	Not established	Yes
Ethyl Parathion	4.4–5,830	NA	5,830	0 Not established	Not established	Yes
1996						
Toxaphene	3.8–17	0.1–1 Feet bgs	17*	CREG 0.6	4	Yes
Toxaphene	2.4 – 830	0.1-1 Feet bgs	830†	CREG 0.6	4	Yes
2002 GRIC DEQ						
Toxaphene	0.48 – 710†	0.5–1 Foot bgs	710	CREG 0.6	4	Yes
Toxaphene	ND – 130*	1–1.5 Foot bgs	130	CREG 0.6	4	Yes
Chlordane	0.027 – 12†	0.5–1 Foot bgs	12	EMEG 1	3.4	Yes
4,4'-DDE	0.02 – 7.2†	0.5–1 Foot bgs	7.2	CREG 2	13	Yes

Contaminant of Concern	*Range of Sample Concentration (ppm)*	*Soil Sample depths*	*Maximum Soil Concentration (ppm)*	*ATSDR Soil Comparison Value (CV) (ppm)*	*Residential Arizona Soil Remediation Level (SRL) (ppm)*	*Exceeds CV or SRL?*
Toxaphene	ND – 130*	1–1.5 Foot bgs	130	CREG 0.6	4	Yes
4,4'-DDE	0.02 – 7.2†	0.5–1 Foot bgs	7.2	CREG 2	13	Yes
4,4'-DDD	ND – 12†	0.05–1 Foot bgs	12	CREG 3	19	Yes
2002 E&E						
Toxaphene	ND – 710	0–0.5 Foot bgs	710	CREG 0.6	4	Yes
4,4'-DDE	0.05 – 16	0–0.5 Foot bgs	16	CREG 2	19	Yes

Bgs: Below ground surface
ND: None detected
*Northernmost site
†Southernmost site
NA: None available
SRL: Soil remediation level is a clean-up standard adopted in administrative rule by the ADEQ.
SRLs are protective of public health and the environment (Arizona Administrative Code R18-7-201 through R18-7-208 of -209).
CREG - Cancer Risk Evaluation Guide for $1x10^{-6}$ excess cancer risk
REMG – Reference Dose media Evaluation Guide
EMEG – Environmental Media Evaluation Guide (ATSDR)
ppm – parts per million

According to available data, a three-member family inhabited an acre of allotted land for 3 months in 1994. The acre was on the Boundary site and approximately 660 feet from the abandoned, northernmost home site. After receiving a complaint of a strong on-site chemical smell, on July 11, 1994, the GRIC pesticide control officer performed preliminary soil testing of the southern-most portion of the site. Three soil samples were taken. The results for toxaphene were below the EPA Region 9 action level of 40 ppm. DDE was detected at 1 ppm. Because, however, high levels of toxaphene were thought to have caused a false reading for DDE, the results were later amended to "not detected"[10].

During May and August 1996, URS Greiner, Inc. (URSG) conducted further EPA-directed testing at the Boundary site. URSG collected samples from 57 on-site locations. Five samples were collected from the soil cap near the northernmost home site. The remaining soil samples were collected near the southernmost home site and along the northeastern end of the airstrip. Depths of the samples are unknown. Soil samples also were collected near the southernmost home site and along the northeastern end of the airstrip. Five samples contained chlordane at concentrations exceeding the residential soil remediation levels (R-SRL) of 0.14 ppm. Laboratory results indicate, however, that inaccuracies occurred in the field screening results. Uncertainty regarding the results arose because the enzyme immunoassay test was conducted at temperatures exceeding the recommended 60°–80°F (degrees Fahrenheit). Additionally, photometer results from prepared standards indicate a level of accuracy outside manufacturer-

specified control limits. All three standards were found to give absorbencies substantially higher than the manufacturer's stated limits. It is not clear to what extent the high ambient temperature (100°F) might have contributed to this. The manufacturer states the kits are designed for an operating range of 60° to 80°F. Because the standards fell outside the prescribed limits, URSG regards the screening results as very qualitative. In any event, organophosphate pesticides were not found in the surface soil samples[3].

Table 3 summarizes the sampling results.

Table 3. URS Greiner, Inc., Sampling Results

Area	*Contaminant*	*# of Samples*	*Detection Range*	*ATSDR Comparison Values (CREG)*
Northernmost home site	Toxaphene	5 Samples	Not detected to 17 ppm	0.6 ppm
Southernmost home site or along the airstrip	Toxaphene	16 Samples	0.7–830 ppm	0.6 ppm

CREG = Cancer Risk Evaluation Guide for $1x10^{-6}$ excess cancer risk

Ecology and Environment, Inc. (E&E) used the data to determine whether the site qualified for listing on EPA's National Priorities List (NPL). E&E used EPA's Hazardous Ranking System model and guidelines to evaluate the site. Although the site did not qualify for the NPL using the EPA model, EPA determined that toxaphene levels were high enough to recommend the site not be used for residential, commercial, or industrial activities[11].

In January and February 2002, GRIC DEQ conducted sampling which generally confirmed the 1996 data. According to the GRIC DEQ site investigation report, the field screening performed by URSG in May 1996 was conducted at ambient temperatures of approximately 100°F. The ambient temperatures were much higher than the recommended temperature range of 60° –80°F for the enzyme immunoassay test kit used. Therefore, the URSG results should be considered suspect. Any field decisions made during the 1996 investigation that were based on those results would require additional evaluation. Selected sample locations may require retesting[12]. For this reason, OSH elects to disregard other data from this sampling round and focus only on the toxaphene results[5].

Table 4 summarizes the 2002 GRIC DEQ sampling results[5]. The objective of the soil sampling and analysis was to evaluate residual concentrations of toxaphene below the cap at the northernmost site and to assess further the elevated concentrations of toxaphene on and around the southernmost site. Del Mar Analytical Laboratory, an Arizona Department of Health Services licensed laboratory in Phoenix, Arizona, analyzed the soil samples for chlorinated pesticides with EPA Test Method 8081, which uses Gas Chromatography (GC). The 13 samples from the southernmost home site were collected at 0.5–1 foot bgs. Samples from the cap at the northernmost home site were collected at 0.5–1 foot bgs or 2–2.5 feet bgs. Table 4 presents the sample results[11].

Table 4. GRIC DEQ Sampling Results

Area	*Contaminant*	*# of Samples*	*Detection Range*	*ATSDR Comparison Values*
Southernmost site	Toxaphene	12	0.48–710 ppm	CREG 0.6 ppm
Southernmost site	Chlordane	12	0.053–12 ppm	EMEG 1 ppm
Southernmost site	4,4'-DDE 4,4'-DDD	12 1	0.2–7.2 ppm 12 ppm	CREG 2 ppm CREG 3 ppm
Northernmost site at soil cap	Toxaphene	11	ND–130 ppm	CREG 0.6 ppm
Northernmost site at soil cap	Chlordane	11	0.027–10 ppm	EMEG 1 ppm
Northernmost site at soil cap	4,4'-DDE	11	0.2–5.1 ppm	CREG 2 ppm

CREG - Cancer Risk Evaluation Guide for $1x10^{-6}$ excess cancer risk
EMEG – environmental Media Evaluation Guide (ATSDR)

Highest levels were located at SS-01, SS-05, and SS-09 on the southernmost site, and at SS-49 on the northernmost site. Laboratory analysis also detected dichlorodiphenyltrichloroethane (DDT), endrin, aldehyde, dieldrin, and endosulfan II, but all concentrations were below the ATSDR comparison values. Comparison values are used to select contaminants for further exposure evaluation. If a contaminant is present in the soil at a level greater than the comparison value, the contaminant is selected for further evaluation. If the chemical is present in the soil at a level below the comparison value, no further evaluation is necessary — health effects are unlikely to occur at those levels regardless of how people come into contact with them.

In October 2002, the GRIC DEQ requested assistance from EPA Region 9's Emergency Response Section (ERS) with the assessment, removal, or remediation of toxaphene-contaminated soil at the Boundary site. The E&E Superfund Technical Assessment and Response Team (START) conducted sampling for field-testing and laboratory chemical analysis. According to the E&E February 2003 report, the team used a global positioning system to mark and identify 895 grid samples collected from October 21–25, 2002. The team used a Geoprobe™ push drill rig to collect soil samples that were analyzed for toxaphene using immunoassay technology. START also submitted 37 samples, three duplicate samples, and one performance evaluation sample to Agriculture & Priority Pollutants Laboratories, Inc. (APPL), in Fresno, California, for analysis of chlorinated pesticides using the GC-based EPA method 8018. Toxaphene concentrations were detected in surficial soil samples from most of the site locations. Table 5 presents the toxaphene results using immunoassay technology.

Table 5. Ecology and Environmental Toxaphene Sampling Results

Sampling Depth (below ground surface)	*# of Samples*	*Detection Range*	*ATSDR Comparison Value for Toxaphene (CREG)*
0–6 inch bgs	168	ND–710 ppm	0.6 ppm
6 inch–1 foot bgs	29	ND–140 ppm	0.6 ppm
1.5–2 feet bgs	54	ND–99 ppm	0.6 ppm
2.5–3 feet bgs	9	2–27 ppm	0.6 ppm
3.5–4 feet bgs	5	3–49 ppm	0.6 ppm
4–5.5 feet bgs	1	1.3 ppm	0.6 ppm

CREG - Cancer Risk Evaluation Guide for $1x10^{-6}$ excess cancer risk

Data collected during this sampling event demonstrated that most contamination is limited to near-surface soils and is consistent with properties of toxaphene — it strongly adsorbs to soil and has low water solubility. Laboratory analysis also detected 4,4'-DDE, which, like toxaphene, is persistent in soil[11]. The immunoassay technology was used for this data; the surface soil toxaphene could be overestimated because of the presence of chlordane in the surface soil, as was demonstrated in the laboratory results. The immunoassay was not specific to toxaphene, and could be chlordane rather than toxaphene sensitive (at the maximum concentrations of 12 ppm). Usually, toxaphene tends to evaporate from the top 3 inches of soil at high temperature. Also, some relatively small molecules of toxaphene are relatively soluble in water and can move to the subsurface soils.

In mid-May 2003 EPA conducted further remedial actions at the Boundary site. The remedial actions included introduction of additional chemical (monosodium phosphate) and biological agents (blood meal) to promote the removal of the chlorine molecule from the organochlorine, thereby reducing the soil contamination to a safer level. According to GRIC DEQ, as of August 27, 2003, the remediation effort had not reduced the toxaphene levels at the Boundary site.

On October 3, 2003, a second pilot study was introduced under the direction of the EPA Environmental Response Team (ERT), GRIC DEQ, START, and Lockheed Martin Services, Inc./Response Engineering and Analytical Contract (REAC). Baseline data samples were taken.

The ERT returned in November 2003 to inspect the site and check on progress of the study. This pilot study consists of three treatment cells: A–*in situ*, covered; B–burrito, lined and covered; and C–no liner, covered. As of March 2004, pilot study sampling results have been reported as successful and complete[12]. The remediation team worked on modifying the mixture of chemical agent (monosodium phosphate), biological agents (blood meal), and microbes (anaerobic) from District 4 Lone Butte with the addition of starch, which further enhances the action of the blood meal. Results are pending from the third pilot study samples collected on March 10, 2004, consisting of six different mixtures. The EPA team believes it is likely that a lower concentration of blood meal can be used by fermenting starch to establish anaerobic conditions rapidly and cheaply instead of sacrificing blood meal to drive out the oxygen[12]. April 2004 results determined ratios that would in fact render the reduction of toxaphene rapidly and cheaply. EPA

has signed an action memo and deemed the Boundary site removal action time-critical. EPA mobilized and started the reduction/removal work in mid-April 2004.

Contaminants of Concern

Contaminants present in soil were matched with ATSDR soil comparison values as shown in each environmental data table. The contaminants selected for further evaluation included toxaphene, methyl parathion, ethyl parathion, S,S,S-tributyl phosphorotrithioate (tribufos, DEF®), chlordane, DDE, and DDD.

Discussion

Past Exposure

Records show former residents living at this property were exposed by accidental ingestion to pesticides through contact with soil due to crop dusting, and by intentional ingestion resulting from participation in a cultural ritual. Residents could have contacted and ingested contaminated water, and they could have inhaled contaminated air. But data for exposure through other environmental media are not available, so this health consultation focuses on soil contact. The exposure evaluation for contaminants in soil is based on the daily activities of the former residents and on past sampling results of soil at the Boundary site property.

Soil at the Boundary site has always been a medium through which pesticides could travel. Records show that an airstrip used for crop dusting was situated on the property. Chemical mixing and loading were conducted on site. Spill releases from mixing and washing out the airplanes or dumping pesticide residue on to the soil contributed to contamination, and ultimately to exposure. In this regard, we know that children played in the soil and in the water canals that traverse the site.

The sandwich mud home built on the site was made with soil from the Boundary site. Cultural practices brought women and little girls into the highest level of contact. A ritual called for them to be “one with the Earth” by eating the contaminated soil from their traditional mud home. The residents also hauled their drinking water from a community well, and stored the water in pails with no cover, but data for that drinking water are unavailable. The former residents also bathed and washed their clothes in the yellow canal water, but the canal is now empty. No data are available on what contaminant levels were in the water when it was used.

At the time the northern-most family moved onto the site, the aerial pesticide spraying was active. The family most likely inhaled sprayed pesticides and inhaled the volatilized pesticides from the ground after spraying.

Former residents of the Boundary site and other community members have stressed consideration of cultural factors in evaluating site exposures. Native vegetation is important for medicinal, ceremonial, and ritual use. The native vegetation found on — or relatively near —the Boundary site could provide an additional exposure pathway if the vegetation was eaten without washing, or if the vegetation accumulated pesticides and was then consumed[13]. It is not possible to evaluate this pathway because of a lack of data. Moreover, very little vegetation presently grows on the property.

In addition to direct contact with soil during outdoor activities, contaminated soil could have been brought inside and accumulated as indoor dust. According to records, the former residents slept on the dirt floor of the traditional sandwich mud home, which could also contribute to

exposure. Although no indoor dust data are available for the former structures by which to evaluate any such exposure, no one currently resides on the property, thus inhalation is not an imminent concern. However, because toxaphene remains in surface soil on the site, casual visitors should avoid direct soil contact to prevent tracking the contaminated soil into their homes and offices[13].

To evaluate possible health outcomes that could result from exposure, OSH assumed an adult (a man, or a woman who did not participate in soil ritual), averaging 70 kg in weight, ingested 200 mg of contaminated soil each day. Because women and young girls actually put soil in their mouths while building the traditional sandwich mud home, OSH assumed that a 70-kg woman and a 10-kg child ingested 500 mg of soil each day. This is likely an overestimate — women did not practice this ritual every day. On the other hand, because they slept, ate, and bathed outdoors, this level could in fact reflect actual exposure.

Estimated exposure doses are then compared with health guidelines, such as ATDSR's minimal risk levels (MRLs) or EPA's reference doses (RfDs). MRLs and RfDs are doses below which noncancerous adverse health effects are not expected to occur (so-called "safe" doses). They are derived from toxic effect levels obtained from human population and laboratory animal studies. In human or animal studies, the lowest-observed-adverse-effect level (LOAEL) is the lowest dose at which an adverse health effect is seen, while the no-observed-adverse-effect level (NOAEL) is the highest dose not resulting in any adverse health effects. NOAEL is a target system in a human or animal study.

LOAELS have been classified into "less serious" and "serious" effects. "Serious" effects are those that evoke failure in a biological system and can lead to morbidity or mortality (e.g., acute respiratory distress or death). "Less serious" effects are those that are not expected to cause significant dysfunction or death, or those whose significance to the organism is not entirely clear. Health effects data are discussed in terms of three exposure periods: acute (14 days or less); intermediate (15–364 days) and chronic (365 days or more)[14].

Using these scenarios OSH evaluated exposure to each contaminant of concern. A discussion of the evaluation of exposure to *organophosphate* and *organochlorine* contaminants follows. Even though chlordane, DDE, and DDD were detected in 1996 and 2002 at levels exceeding the ATSDR comparison values, these contaminates will not be discussed because no one inhabited the property during that time. If there are no receptors, there cannot be exposure.

The following information summarizes OSH's findings. To better understand how we did the analyses and, as importantly, the limitations of the information, please read the discussions of each evaluation.

- People who lived on the site were exposed to estimated doses from toxaphene found on the site, which exceeded an oral minimal risk level (MRL).
- Cholinesterase test results suggested that the some women and children were likely exposed to organophosphates at levels that affected them. The women and children had reduced cholinesterase levels. Cholinesterase inhibition can be reversed when an individual is removed from the exposure of organophosphates.
- Given ATSDR's estimated exposure dose, people exposed to toxaphene and ethyl parathion are not likely to develop cancer.

- There is no evidence that methyl parathion causes birth defects in humans or affects the ability of humans to produce children.
- Oral exposure studies were limited for DEF®. Studies were not found that provided information on possible health effects at the levels found at the Boundary site.

Organochlorines

Toxaphene

Toxaphene is the trade name for an organochlorine pesticide that is a mixture of a least 670 chlorinated camphenes. Toxaphene was first introduced in 1947 and, after the ban on DDT, was probably the most heavily used pesticide on cotton and other crops in the United States during the 1970s. Toxaphene was banned for most uses in 1982 and banned completely in 1990. Toxaphene has a relatively long half-life of 1–14 years in soils. Toxaphene can enter the human body through eating contaminated food or soil, through the skin after direct contact with contaminated substances, and through the lungs after breathing its vapors and particulate. Once toxaphene enters the human body, it rapidly spreads to all organs. Toxaphene is quickly broken down in the human body, and it is excreted in urine and feces. Nearly all (approximately 90%) of the toxaphene is eliminated from the human body within 24–36 hours[14].

OSH calculated estimated exposure doses for the stated exposure scenarios. OSH then compared the estimated doses with health guidelines. These estimated doses are based on the highest levels found, which represents a worst-case scenario. Actual exposures, especially for some people, were probably less. Estimated exposures exceeded ATSDR's intermediate oral MRL of 0.001 milligrams per kilogram a day (mg/kg/day) and the acute oral MRL of 0.005 mg/kg/day.

A child who ingested levels of toxaphene found on site in 1984 (15845 ppm) had an estimated dose of 0.79 mg/kg/day; the estimated exposure dose of a man and woman who did not participate in the soil ritual was 0.0045 mg/kg/day. Because women and young girls actually put soil in their mouths while building the traditional mud home, OSH assumed that a 70-kg woman could conceivably ingest 500 mg of soil each day. Again, this is likely an overestimate because women did not practice this act every day. Still, for those women their dose would average more than men of the same size or more than women who did not participate in the ritual. An estimated dose for a woman who participated in the soil ingestion ritual was 0.113 mg/kg/day.

Exposure to levels similar to those found at the site has been associated with noncancer health effects in animal studies. Detected levels of toxaphene were as high as 15,845 ppm at the Boundary site in 1984. No animal studies at the exact level of child dose (0.79 mg/kg/day) were found in the chronic exposure studies listings regarding the 1984 estimated dose.

The only doses close to the estimated dose were found in an intermediate exposure developmental rat study. The estimated child dose exceeded the LOAEL of rat developmental effects and a NOAEL dog study in which hepatic, renal, and endocrine system effects were noted. The child dose (0.79 mg/kg/day) was 4 times above the LOAEL of 0.2 mg/kg/day in this dog study. This does not mean the child dose was safe. The important point of this study was that the child dose was approximately 3 times below the LOAEL (2 mg/kg/day) that caused health effects in rats. Studies in animals show that low levels of toxaphene may remain in fat for days[14]. The former residents lived on the Boundary site 7 years or more, and to date there is no available data on testing for toxaphene exposure on any of them.

Toxaphene primarily targets the nervous system. Individuals with latent or clinical neurological diseases, such as epilepsy or behavioral disorders, could be at higher risk. The sensitive populations also include infants with developing nervous systems.

For example, studies in animals show that chronic exposure (1–2 years) to toxaphene can damage the liver, kidneys, adrenal glands, immunological systems, and neurological systems at high-dose levels (at or above 12.9 mg/kg/day), and at low-dose levels (0.05 mg/kg/day) toxaphene can cause behavior effects in early development[14].

Animal studies suggest that developmental toxicity is associated with toxaphene through effects observed in adults. In comparison to adults, these effects will pose higher risks to children. Because their immune systems do not reach maturity until 10–12 years of age, infants and children are especially susceptible to immunosuppression. Animal studies suggest that immature animals cannot detoxify a toxaphene mixture as efficiently as they can the single components of the mixture[15]. Embryos, fetuses, and neonates up to age 2–3 months could also be at increased risk of adverse effects because their enzyme detoxification systems are immature.

Toxaphene can also cause cancer in laboratory animals. Female and male rats fed up to 56 mg/kg/day for 80 weeks had an increase in thyroid adenomas, and, in female rats alone, follicular-cell carcinoma. The occurrence of tumors was however at a relatively low incidence and was inconclusive. A liver cancer effect level (CEL) was seen in male mice that had oral exposures of 12.9 mg/kg/day and in female mice of 25.7 mg/kg/day for 80 weeks. EPA has classified toxaphene as a probable human carcinogen (Group B2) as a result of oral exposure studies in animals[14]. No conclusive human epidemiological studies are available for toxaphene. Oral administration of toxaphene resulted in an increased incidence of hepatocellular (liver) carcinomas and neoplastic nodules in mice, and thyroid tumors in rats[15].

Toxaphene has recently been observed to have estrogenic effects on human breast cancer estrogen-sensitive cells. Xenoestrogens have been hypothesized to have a role in human breast cancer. In addition to potential carcinogenic effects, toxaphene may also cause disruption of the endocrine system due to its estrogenic activity[15].

Individuals who may be at greater risk from toxaphene are those with diseases of the renal, nervous, cardiac, adrenal, and respiratory systems. Individuals using certain medications are also at potential risk through the induction of hepatic microsomal enzymes by toxaphene. Hepatic microsomal enzymes affect the metabolism of some drugs and alcohol. This was observed in a man using warfarin as an anticoagulant while he used toxaphene as an insecticide. The effectiveness of the drug was reduced through an increase in metabolism arising from toxaphene's induction of microsomal enzymes[14].

Blood and urine tests can confirm that a person has been exposed recently to toxaphene, but these tests cannot yet predict the kind or severity of any health effects that might occur. No studies demonstrate a reliable correlation between blood levels and levels of exposure, especially an exposure that occurred 26 years ago.

Organophosphates

Ethyl parathion (or parathion)

OSH estimated the exposure doses for a child exposed to ethyl parathion at the maximum level found in 1984. The child's estimated dose was 0.29 mg/kg/day. For a woman who participated in the soil ingestion ritual, the estimated exposure was 0.042 mg/kg/day, while for other adults the

estimated dose was 0.017 mg/kg/day. Because no comparison values have been established for ethyl parathion, further evaluation is warranted.

Exposure to the levels found at the site has been associated with noncancer health effects. In 1984 detected levels of ethyl parathion were as high as 5,830 ppm at the Boundary site. Study results indicate that ingestion of parathion poses acute and chronic risks to birds and mammals. Studies also show that wildlife incident data link bird and mammal mortality to parathion use. In one study, parathion was fed to dogs at 1 ppm (avg. 0.021 mg/kg/day), 2 ppm (0.047 mg/kg/day), and 5 ppm (0.117 mg/kg/day) for 24 weeks. At 1 ppm, (avg. 0.021 mg/kg/day) a minimal, but significant, reduction in plasma cholinesterase occurred. At higher dosages [2ppm (0.047 mg/kg/day), and 5 ppm (0.117 mg/kg/day)] plasma and cholinesterase activity was reduced by 60%–70%[16]. In another study, dogs were given 15% parathion wettable powder in gelatin capsules 6 days/week for 90 days. At 2 mg/kg/day, dogs lived for 3 weeks but exhibited signs of toxicity. At 1 mg/kg/day, animals survived but were nervous. This is approximately the same level that a child would experience if that child had daily exposure to the maximum amount found on the site. It is, however, unlikely that a child would have experienced that level of exposure; still, actual doses could have been high. Animals became irritable during early stages of treatment. Later, their behavior seemed normal. No gross pathology was evident, but histopathologic examination after termination of the experiment revealed degenerative changes in the liver[16].

Male dogs (six per group) were given oral doses of 0.50 mg/kg, 5 days/week, for 6 weeks. Erythrocyte and plasma cholinesterase activities were 42% and 15% of normal, respectively, after 6 weeks of exposure[17].

Epidemiological evidence indicates that children are more sensitive to parathion than are adults through dermal exposure. Children 7 and 9 years of age died from bathing in a tub in a house that had been sprayed several days earlier with 10% parathion intended for ornamental plants in a greenhouse. Such evidence, however, did not prove that children are more susceptible to parathion poisoning.

In another incident, a group of city children found a coarse cloth sack in a trash pile, stuffed it with rags, suspended it from a tree by a rope, and used it as a swing. Their bare arms, legs, and faces rubbed against the rough fabric as they clung to it while swinging back and forth. The swing was used briefly on the day it was made, and it was used almost constantly from 10:00 a.m. to 5:30 p.m. the next day. Between 8:00 p.m. and 9:00 p.m. of the first full day of swinging, the children became sick and were taken to different hospitals. Three of them, a 10-year-old and a 13-year-old boy and 10-year-old girl, survived, but two, a 5-year-old boy and 9-year-old girl, died. Parathion was found in the bag and in the clothing of the dead children. The ethyl parathion metabolite, p-nitrophenol, was identified in the only sample of urine available. Red cell cholinesterase activity was also greatly reduced[18].

No deaths were reported in connection with Boundary site exposures; however, cholinesterase tests conducted suggest that children were likely exposed at levels that could have affected them. Children living on the northern-most site played in the contaminated soil and canals outside of their homes.

EPA has classified ethyl parathion as a possible human carcinogen (i.e., no human, limited animal studies—Group C). This classification is based on the increased adrenal cortical tumors that developed in male and female Osborne-Mendel rats, and on possible trends for thyroid

follicular adenomas and pancreatic islet cell carcinomas in male rats in the same study. No mutagenicity was seen in any study[19].

Methyl parathion

OSH calculated estimated exposure doses for the stated exposure scenarios. OSH then compared the estimated doses with health guidelines. Methyl parathion exceeded ATSDR's chronic oral MRL of 0.0003 mg/kg/day, ATSDR's intermediate oral MRL of 0.0007 mg/kg/day, and EPA's chronic oral RfD of 0.00025 mg/kg/day. A child who ingested levels of methyl parathion found on the Boundary site in 1984 had an estimated dose of 0.41 mg/kg/day. A woman who participated in the soil ingestion ritual had an estimated dose of 0.059 mg/kg/day, and an adult's estimated exposure dose was 0.024 mg/kg/day.

In 1984 levels of methyl parathion as high as 8,250 ppm were detected in samples from the Boundary site. Exposures to methyl parathion for an adult were below those associated with health effects in available studies. Exposure doses of 0.22 mg/kg/day and higher have been associated with noncancer health effects associated with ingestion, according to the ATSDR toxicological profile for methyl parathion. A chronic duration oral MRL of 0.0003 mg/kg/day was derived for methyl parathion, using the observation of reduced mean hematocrit and erythrocyte counts in rats fed methyl parathion in the diet for 2 years. Significantly decreased mean hematocrit and erythrocyte counts were observed at 24 months in males who consumed either 0.25 mg/kg/day or 2.5 mg/kg/day for 24 months; no effect in males was observed at 0.025 mg/kg/day. Significantly decreased mean hemoglobin, hematocrit, and erythrocyte counts were seen at 6–24 months in female rats that ingested 2.5 mg/kg/day, with no effect at 0.025–0.25 mg/kg/day. In the same study, chlolinesterase activities decreased in plasma, erythrocyte, and the brain. Abnormal gait, tremor, and peripheral neuropathy were observed in the rats that consumed 2.5 mg/kg/day of methyl parathion, but not in rats consuming the lower doses at 0.25mg/kg/day[20].

Methyl parathion estimated doses in a woman who participated in the soil ingestion ritual exceed the chronic MRL of 0.0003 mg/kg/day by 200 times, but are approximately 4 times below the LOAEL of 0.22 mg/kg/day for neurological effects in animal studies. As result of their exposure, women who lived at the Boundary site and were exposed to methyl parathion could possibly develop non-cancer health effects.

Estimated doses for a child (0.41 mg/kg/day) were above those associated with possible adverse health effects, based on animal studies such as decreased brain and plasma cholinesterase activity, and mean hematocrit, erythrocyte count. EPA has determined that methyl parathion is "not classifiable as to human carcinogencity—Group D." No reports of cancer in humans associated with exposure to methyl parathion by any route have been found. The available data in experimental animals are negative[20].

S,S,S-Tributyl Phosphorotrithioate (DEF®, tribufos)

OSH estimated exposure doses of this substance for the stated exposure scenarios. OSH then compared the estimated doses with health guidelines. Estimated exposures to DEF® did exceed EPA's chronic oral RfD of 0.00003 mg/kg/day. A child who ingested levels of DEF® found on the Boundary site in 1984 had an estimated dose of 0.0046 mg/kg/day; for a woman who participated in the soil ingestion ritual, the estimated dose was 0.0007 mg/kg/day. An adult's estimated exposure dose was 0.0003 mg/kg/day. In 1984 levels of DEF® found at the Boundary

site were as high as 91 ppm . This level was found 4 years after aerial applications stopped. Therefore, levels could have been higher between 1977 and the early 1980s at the Boundary site, which is when the former residents inhabited the property.

DEF is registered as a cotton defoliant. N-Butyl mercaptan (nBM) is a volatile degradation product of DEF that has a strong, skunk-like odor. The odor threshold for DEF® in humans is approximately 0.01–0.1 parts per billion (ppb), based on inhalation studies[21]. DEF® is a designated toxic air contaminant in the state of California, pursuant to Food and Agricultural Code (FAC) section 14023, and to the stringent evaluation of the California Department of Pesticide Regulations (CDPR). Also, after evaluation in 2000, CDPR requires a 7-day restricted entry interval to fields treated with DEF®; that is, farm workers must wait 7 days before they are allowed to enter a site that has been sprayed [22]. Because the compound is highly volatile, most DEF® studies focus on inhalation effects. Nevertheless, for the reasons previously stated, this health consultation will continue to focus on exposure to contaminants in soil.

Higher-than-estimated exposure doses (0.0046 mg/kg/day, 0.0007 mg/kg/day, and 0.0003 mg/kg/day, respectively, for a child, a woman participating in ritual, and an adult) found at the Boundary site have been associated with noncancer health effects. Oral studies conducted in hens used levels higher than an exposure dose of 0.5 mg/kg/day to determine health effects; these are above the estimated dose levels identified at the Boundary site for DEF®. A neurotoxicity study reported a NOAEL of 0.1 mg/kg/day in hens in which mild ataxia (i.e., clumsiness or loss of coordination) was observed at 0.5 mg/kg/day when DEF® was administered in capsules for 90 days. The ataxia with oral exposure could have been caused by the parent compound DEF® or its metabolite, nBM, which can be formed in the gastrointestinal tract of hens from the hydrolysis of DEF®[23]. Another oral administration study concluded that single doses of DEF® ranging between 250 mg/kg and 1,000 mg/kg are capable of producing delayed neurotoxicity among hens, according to studies by M.B. Abou-Donia in 1979[24]. In another study a mouse that was fed for 8 weeks (NOAEL of 40 mg/kg/day and a LOAEL of 140 mg/kg/day) was found to have decreased brain chlolinesterase levels (74%)[23].

EPA has not classified DEF® as a possible human carcinogen. The National Toxicology Program (NTP) has classified DEF® as Group 3–not classified cancer class.

OSH did not find studies to give a solid representation of health effects of the levels found at the Boundary site. Most likely, the estimated doses found at the Boundary site did not pose a risk of developing adverse health effects. Still, maximum sample results were found 4–5 years after aerial applications stopped. Therefore, exposures could conceivably have been higher. EPA's *Tribufos Summary* concludes that higher levels of tribufos than found at the Boundary site can cause cholinesterase inhibition in humans. That means the compound can over-stimulate the nervous system, causing nausea, dizziness, and confusion. At high exposures, it can cause respiratory paralysis and death. Tribufos also exhibits irreversible visual system toxicity in rats[25]. Information on how humans might be affected at levels found at the Boundary site is not available.

Child Health Considerations

ATSDR and OHS recognize that infants and children can be more sensitive than are adults to environmental exposure in communities faced with contamination of their water, soil, air, or food. Children are smaller than adults are, and therefore could receive a higher dose of chemical exposure relative to their body weight. Children's developing systems also can be more

vulnerable to the toxic effects of a chemical. For example, children are far more likely to engage in activities that involve "getting dirty." Playing in the dirt, combined with frequent hand-to-mouth activity, provides toddlers and children with an increased chance of exposure to soil contaminants through ingestion and skin contact. Some children engaging in behavior known as "pica" are more likely to ingest soil and other nonfood items. For young girls who lived on the site, this was particularly true if they participated in the ritual associated with building their home. The fetus is also considered when evaluating possible health effects. A pregnant mother could possibly pass on effects of exposure to the fetus and subsequently, the fetus could be harmed. Because of these issues, ATSDR uses health guidelines that are protective for children, and considers children a special population that can be more sensitive than are adults to chemical exposures.

OSH has estimated that from 1977 to 1990 three children living on the Boundary site were exposed to several contaminants (except for 1 year from latter 1984 to 1985 when remediation was taking place). Several relatives (with children) also were reported to have lived on the property from 3 months to well over 2 years. Children's estimated exposure doses and possible health effects were discussed in detail in previous sections.

Limitations of Toxicological Evaluations

Incomplete data is a problem often encountered during the public health evaluation process. Of the many thousands of commonly used chemicals, relatively few have been thoroughly evaluated for toxicity. Some information is missing for most chemicals. Information on the noncarcinogenic adverse health effects of a particular chemical might be available, but not information as to its potential to cause cancer. Information regarding the toxicity of a chemical for short exposures at high concentrations — such as what could occur in the workplace — might be easily found, but information regarding toxicity at low concentrations for long periods might be scarce to nonexistent. In these situations, researchers cannot thoroughly evaluate the health implications of exposures. Limitations on data availability are provided in the toxicological discussion for each chemical of concern.

Conclusions

1. Former residents living at this property have been exposed to pesticides from crop dusting and in soil through accidental—and sometimes intentional—ingestion, skin contact, and dust inhalation. The levels of exposure to some of the pesticides were at levels associated with noncancer health effects. Biological tests conducted on residents suggest that exposure occurred at levels that could have affected some residents, although because of a lack of baseline data, the data are inconclusive. *Past exposure posed a public health hazard for the residents.*
2. Currently, no one lives on the property. That means potential exposure is limited to those who trespass on the property or occasionally walk on the dirt and carry the dust to the home or office. Therefore, *the site currently poses no apparent public health hazard.* That said, the property is still contaminated with toxaphene and 4,4'-DDE as stated in 2002 E&E sampling report.

Future Use

The 2003 remedial efforts to date had not successfully reduced toxaphene levels to allow the property to be used for commercial or residential purposes, although in the first part of 2004 the

pilot study was successful and was completed. In mid-April 2004, EPA ERT began remediation efforts to bring toxaphene contamination levels from 600 ppm to 17 ppm, which is the Arizona non-residential soil remediation level for industrial/commercial use. For residential use, 4 ppm is the maximum level of toxaphene allowed in Arizona. Although, 4 ppm would be above the ATSDR soil comparison values for toxaphene, it is below levels associated with health effects for most exposure scenarios. Also, this process will reduce the remaining 4,4'-DDE. Total treatment time will take between 3–8 months. OSH will evaluate post-remediation samples to determine whether the property is safe for any type of use. The GRIC Council will make the final decision about how the land is used.

Recommendations

- Restrict access to contaminated areas of the site.
- Provide post-remediation data to OSH.
- Discuss the results of the HC with the former boundary site residents and provide them with other information as requested.

Public Health Action Plan

OSH has developed a public health action plan to ensure that recommendations are implemented and are meaningful for the affected families. The public health action plan is described in the following table.

Table 6: Public Health Actions to be Implemented

Public Health Action	*Who Will Implement the Action*	*Time Frame for Implementation*	*Desired Outcome When Implemented*	*Public Health Impact*
Restrict access to contaminated areas of the site	EPA	Completed	People will avoid contaminated areas	Prevent exposure to pesticides at levels that could harm health and prevent disruption of the remedial process
Provide post-remediation data to OSH	EPA	When data are quality assured	Provide families with better information on safe use of property	Prevent exposure to pesticides at levels that could harm health
Provide this health consultation and other information to the affected families	OSH	Information sharing is ongoing; the document will be shared immediately upon completion	Answer as many health questions as possible	Affected families have the information they need to discuss health effects with their health care providers and can make decisions as to whether they can safely use their property

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Certification

This Health Consultation for the Boundary Site was prepared by the Gila River Indian Community Department of Public Health under cooperative agreement with the Agency for Toxic Substances and Disease Registry. It is in accordance with approved methodology and procedures existing at the time the exposure investigation report was begun.

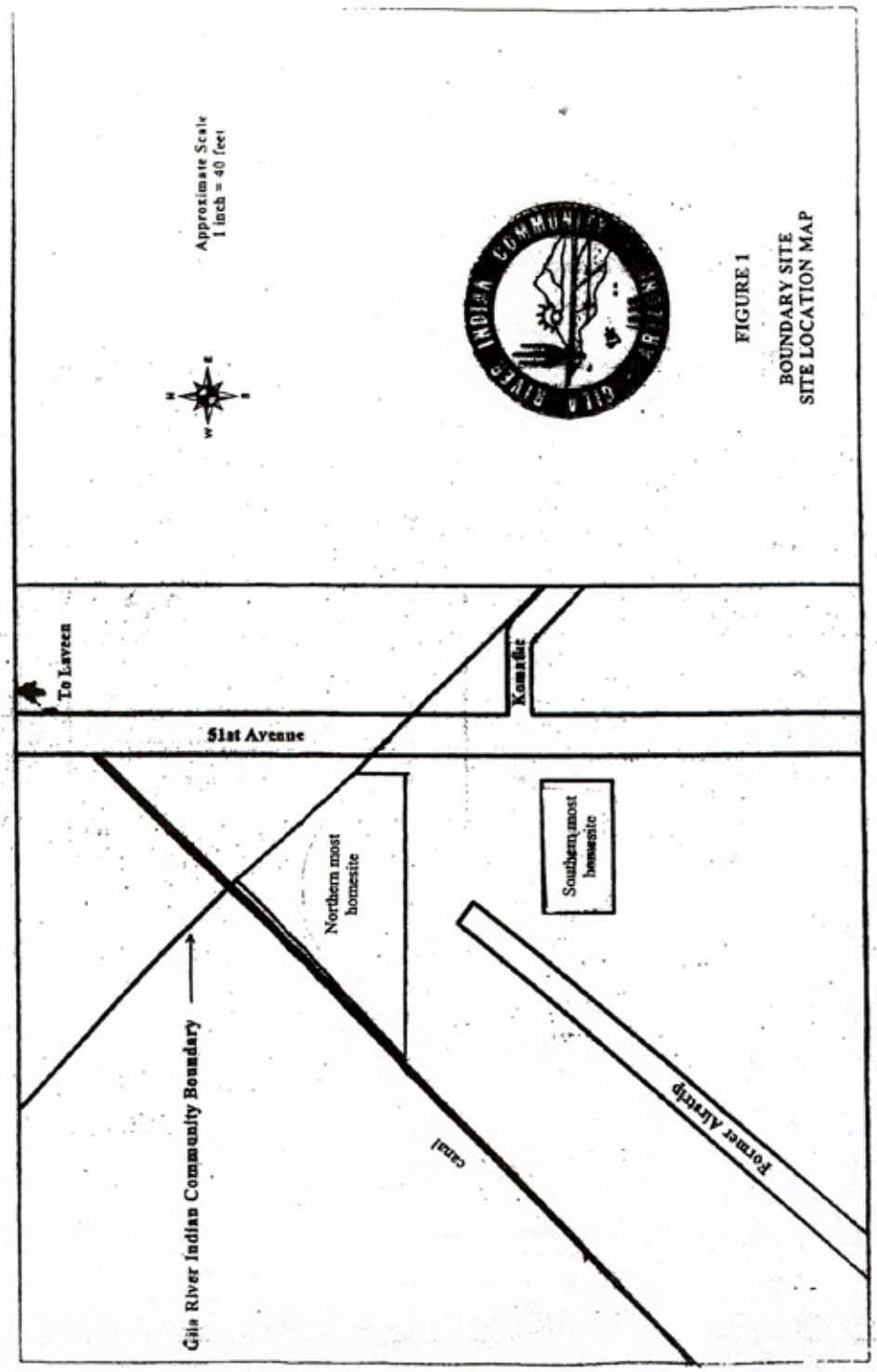
Technical Project Officer
CAT, SSAB, DHAC, ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this health consultation and concurs with the findings.

Team Leader-Cooperative Agreement Program
CAT, SSAB, DHAC, ATSDR

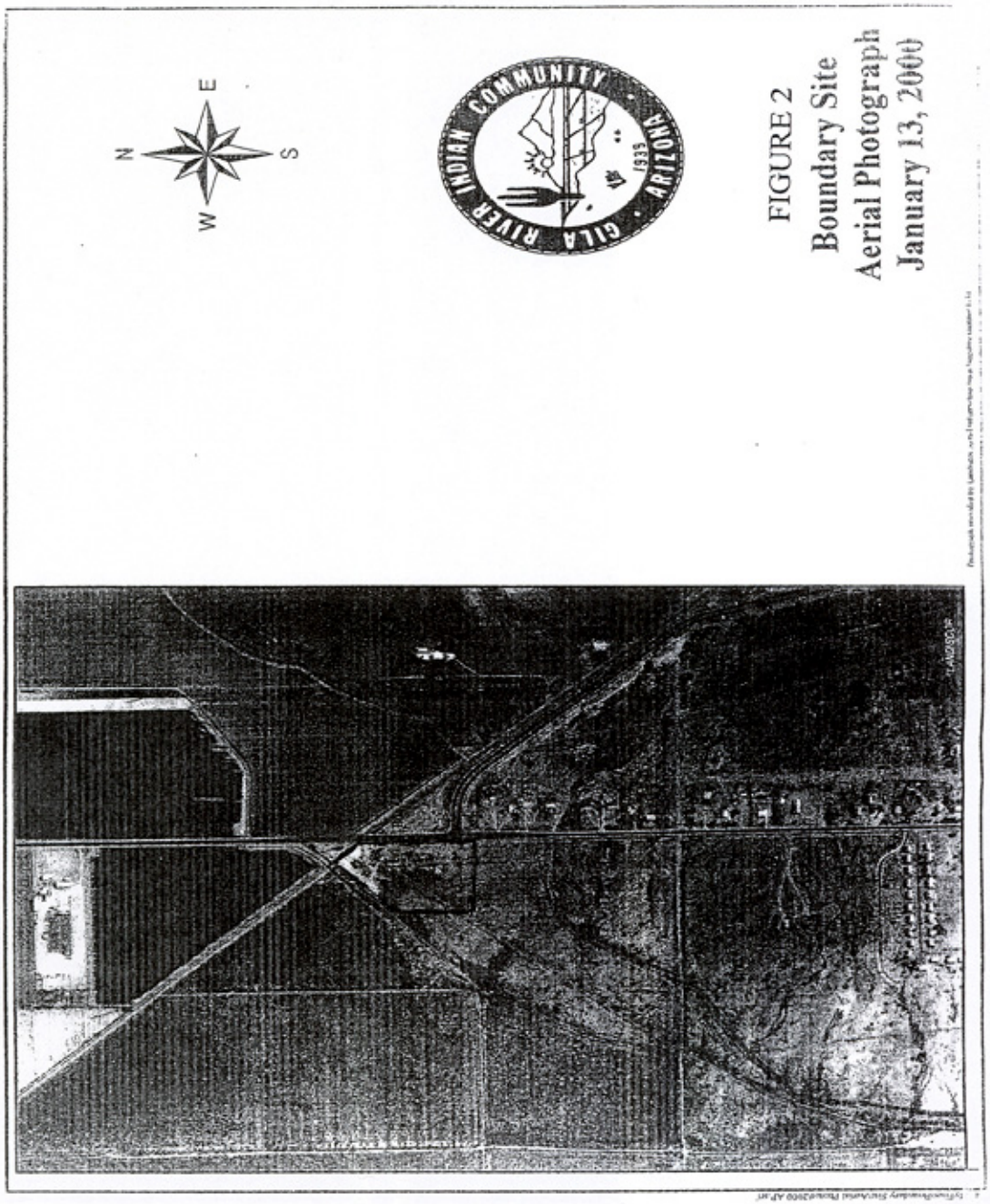
APPENDIX A: Site Map and Site Photos

Figure 1 - Site Map



APPENDIX A: Site Map and Site Photos

Figure 2 - Aerial Photo of Boundary site



APPENDIX A: Site Map and Site Photos

Figure 3 - Boundary Site Northern-most Homesite

Figure 4 - Debris scattered west of Boundary site

APPENDIX A: Site Map and Site Photos

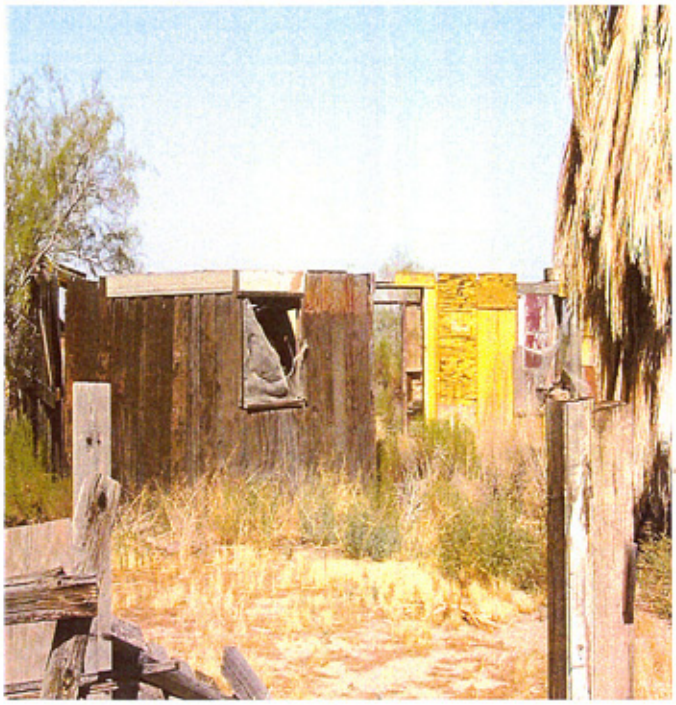


Figure 5 –Northern-most home from a southern perspective

Figure 6 - Soil Cap West of Northern-most Home site

APPENDIX B

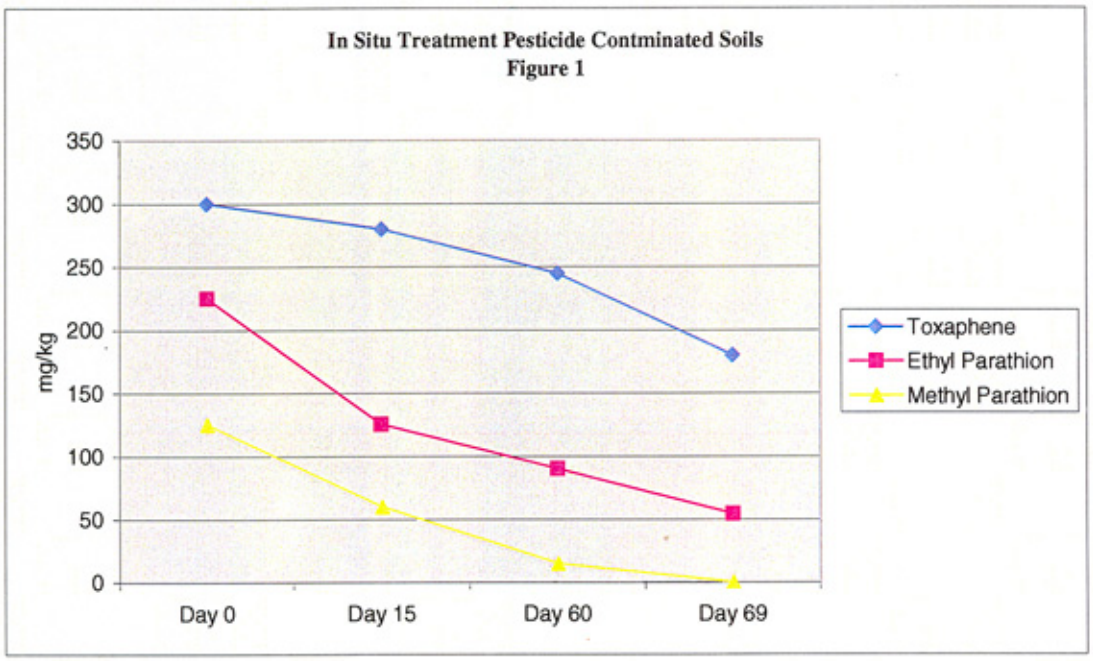



Figure 1 - Organophophates Reduction Results