APPENDIX C

HUMAN HEALTH RISK ASSESSMENT WITH CONCEPTUAL SITE EXPOSURE MODEL

HUMAN HEALTH SCREENING ASSESSMENT
REMOVAL ACTION WORKPLAN

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1.0 INTRODUCTION

1.1 Purpose

The purpose of the human health screening assessment is to provide risk management data about the potential risks to human health and the environment associated with chemicals detected in soils at the site. This assessment will inform decisions made regarding whether further characterization, risk assessment, or remedial actions are necessary.

The methodology for identifying and assessing hazard and risk at the property follows the procedures outlined in the DTSC's 1994 (second printing June 1999) Preliminary Endangerment Assessment Guidance Manual. In addition, DTSC's Supplemental Guidance for Human Health Multimedia Risk Assessments of Hazardous Waste Sites and Permitted Facilities (DTSC, 1992), and Selecting Inorganic Constituents as Chemicals of Potential Concern at Risk Assessments at Hazardous Waste Sites (HERD, DTSC, February 1997) were consulted.

1.2 Components

The human health screening encompasses descriptions of four basic components to help to assess the potential risk present at the site: 1) exposure pathways and media of concern, 2) chemicals and exposure concentrations, 3) toxicity values, and 4) risk characterization summary. The four components are described in more detail in the following sub-sections.

1.2.1 Exposure Pathways and Media of Concern

This component of the human health screening provides a detailed description of the media at the site, which are considered impacted, potentially impacted, or which could potentially become impacted. This component also includes a description of the pathways and routes by which populations might be exposed at the site and describes the populations that are considered to be potentially exposed at the site.

1.2.2 Exposure Concentrations and Chemicals

This component of the human health screening describes the chemicals detected during site characterization, the concentrations and spatial distribution of the chemicals on the site, evaluates detected analytes as COPCs, and presents calculations or arguments for determining concentrations used as the input – or "exposure point" - concentrations (EPCs) in further COPC.

1.2.3 Toxicity Values

The Toxicity Values component describes the relevant human toxicity information available for each identified COPC, and supplies references for the source of each toxicity criterion used.

1.2.4 Risk Characterization Summary

The Risk Characterization summarizes the results of the screening exposure assessment and integrates them with the available toxicity information, presenting the significant findings, such as the risks and hazards estimated for the chemicals at the property and the conclusions regarding the human health screening.

2.0 EXPOSURE PATHWAYS AND MEDIA OF CONCERN

This section presents potential exposure pathways and receptors on the subject property using a Conceptual Site Model (CSM). A conceptual site model is a three-dimensional "picture" of site conditions that illustrate contaminant contributions, release mechanisms, exposure pathways and migration routes, and potentially exposed populations (USEPA, 1996). It describes the suspected sources of chemicals, fate and transport mechanisms that distribute the chemicals within the environment, the potentially exposed human populations, and the potentially complete exposure pathways. The conceptual site model presents a hypothesis but does not demonstrate actual exposure and/or effects on receptors. Plate C-1 presents a CSM for the potential primary exposure pathways to the human receptors at the site. Surface water tests did not yield any COPCs (see Table C-1). Moreover, surface water will not be used as a drinking or recreational water supply source. This model therefore does not incorporate water as a theoretical pathway of a site contaminant. It is reasonable, for purposes of this investigation and cleanup, for soil to be considered the only significant potential medium of concern and exposure pathway.

The default exposure pathways of potential concern for soil exposures are therefore:

- Inhalation of airborne dust
- Incidental ingestion of soil
- Dermal contact with soil.

3.0 IDENTIFICATION OF COPCs AND EXPOSURE CONCENTRATIONS

3.1 Chemicals Detected at the Property

3.1.1 Organic COPCs Detected at the Property

On the basis of the previous site sampling and analysis, organochlorine pesticide compounds DDE, DDT, endrin and methoxychlor were identified in site soils. These organic pesticides are anthropogenic and are therefore immediately categorized as COPCs. Inorganic chemical analytes detected in site samples consisted of arsenic, barium, cobalt, chromium, copper, lead, nickel, vanadium and zinc. These analyte concentrations were screened against corresponding background metals concentrations for identification of inorganic COPCs. Report Tables C-2 and C-3 present Site Data Summaries from the SSI I and the SSI II, respectively, and contain statistical elements used in the selection of inorganic COPCs.

Note: DDD was detected at 100 µg/kg in only one of 30 samples analyzed for OCPs. This value however was obtained from a composite sample. Subsequent analysis of the three individual samples comprising the composite did not yield a detection of DDD above method reporting limits in any sample. For this reason WKA chose not to include DDD as a COPC.

3.1.2 Selection of Inorganic COPCs

In previous investigations (WKA, 2006a and WKA, 2006b), CAM-17 analytes detected in shallow site soils were evaluated against background samples collected from six deep samples deemed representative of background soil conditions. This indicated that, with the exception of arsenic and lead, all detected site metals were sufficiently below the mean or maximum background concentrations to eliminate them from further consideration as COPCs. Moreover, with the exception of arsenic and lead, none of the metals detected were detected at concentrations at or above levels established as harmful to human health and the environment based on CHHSLs (DTSC, 2005).

3.2 Exposure Point Concentrations

EPCs are the representative chemical concentrations that receptors (residents) may contact through each defined exposure scenario and each exposure route (inhalation, ingestion, dermal contact) on the subject property. For purposes of this HHRA, both the maximum detected COPC concentrations, and then the 95% UCLs of the arithmetic means of the COPC concentrations are evaluated as EPCs. Given the proposed use for

this site as multifamily residential units, use of the 95% UCLs, which averages concentrations, is a reasonable approach on which to base risk management recessions. Lead is evaluated separately via the Leadspread Assessment Spreadsheet (DTSC, 1999b). Again, both the maximum and the 95% upper confidence limit (UCL) values are used to evaluate site lead.

4.0 TOXICITY ASSESSMENT

Toxicity values for many chemicals are published in the U.S. EPA on-line Integrated Risk Information System (IRIS; U.S. EPA, 2000). Additionally, the California Office of Environmental Health Hazard Assessment (OEHHA, 1994) publishes toxicity values for carcinogens. Cancer slope factors (CSFs) are chemical-specific, experimentally derived, potency values used to calculate the risk of cancer resulting from exposure to carcinogenic chemicals. A higher value implies a more potent carcinogen. Reference doses (RfDs) are experimentally derived "no-effect" values used to quantify the extent of non-carcinogenic toxic effects from exposure to chemicals. Here, a lower value implies a more potent toxicant. These criteria are generally developed by U.S. EPA risk assessment work groups and listed in U.S. EPA risk assessment guidance documents and databases. The CSFs and RfDs (Toxicity Criteria of COPCs) available for the site COPCs are presented in Table C-4.

4.1 Non Carcinogenic Toxicity

Arsenic, DDE, DDT, endrin, and methoxychlor, identified on the subject property as COPCs, have established RfDs used to evaluate non-carcinogenic adverse health effects. Arsenic has an RfD of 3×10^{-4} mg/kg-day for oral exposure, with no established inhalation exposure toxicity, the same 3×10^{-4} mg/kg-day value is therefore used as the default inhalation exposure toxicity. DDE and DDT both have an RfD of 5×10^{-4} mg/kg-day for oral and inhalation exposures. Endrin has an RfD of 3×10^{-4} mg/kg for both oral and inhalation exposures. Methoxychlor has an RfD of 5×10^{-3} mg/kg for both oral and inhalation exposures.

4.2 Carcinogenic Toxicity

Arsenic, DDE and DDT have established oral CSFs of 9.5, 0.34 and 0.34 $(mg/kg\text{-}day)^{-1}$ respectively, and inhalation CSFs of 12, 0.34 and 0.34 $(mg/kg\text{-}day)^{-1}$ respectively (OEHHA, 1994).

Both non-carcinogenic toxic effects (Hazard index) and theoretical upper-bound incremental lifetime cancer risks are evaluated for the identified COPCs in the following section of this report.

5.0 RISK CHARACTERIZATION

In this step of the HHRA, the estimated rate at which a person incidentally takes in a chemical is compared with information about the toxicity of that chemical to estimate the potential risks to human health posed by exposure to the chemical. This section presents the risk characterization methods used in this assessment.

5.1 Quantification of Exposure

The PEA method follows the standard approach used by both U.S. EPA and Cal/EPA for assessing theoretical risks to human health. PEA (DTSC, 1999) equations for calculating risks to default residential receptors from exposure to soil were used in this assessment. PEA equations combine the assumptions of exposure with COPC toxicity information. COPCs identified are non-volatile compounds. The PEA's estimation equation for air concentrations for non-VOCs is therefore used.

Soil

The PEA equation for hazards and risks associated with soil is as follows:

$$Hazard_{soil} = ((C_s / RfD_o) \times (1.28 \times 10^{-5})) + ((C_s / RfD_o) \times (1.28 \times 10^{-4}) \times ABS)$$

$$Risk_{soil} = (C_s \times SF_o \times (1.57 \times 10^{-6})) + (C_s \times SF_o \times (1.87 \times 10^{-5}) \times ABS)$$

where:

C_s	=	Soil concentration (mg/kg)
RfD_o	=	Oral reference dose (mg/kg-day)
1.28×10^{-5}	=	Non-cancer incidental soil ingestion exposure factor (day^{-1})
1.28×10^{-4}	=	Non-cancer dermal contact exposure factor (day^{-1})
ABS	=	Dermal absorption fraction (unitless)
SF_o	=	Oral cancer slope factor $(mg/kg\text{-}day)^{-1}$
1.57×10^{-6}	=	Carcinogenic incidental soil ingestion exposure factor (day^{-1})
1.87×10^{-5}	=	Carcinogenic soil dermal contact exposure factor (day^{-1})

Air

The PEA equation for hazards and risks associated with air is as follows:

$$Hazard_{air} = (C_a / RfD_i) \times 0.639$$
$$Risk_{air} = (C_a \times SF_i \times 0.149)$$

where:

C_a	=	air concentration (mg/m^3)
RfD_i	=	Inhalation reference dose (mg/kg-day)
SFi	=	Inhalation slope factor (mg/kg-day)

C_a, the air concentration term for non-volatile compounds is defined as:

$$C_{air} = (C_s \times 5 \times 10^{-8} kg / m^3)$$

where:

5×10^{-8}	=	National ambient air quality standard for dust (kg/m^3)

5.2 Methods for Assessing Non-Cancer Health Effects

The PEA approach generates a non-cancer hazard quotient for each pathway of exposure for a default residential child. If a person's average exposure is less than the RfD (that is, if the hazard quotient is less than one), the chemical is considered unlikely to pose a significant non-carcinogenic health hazard to individuals under the given exposure conditions. Unlike carcinogenic risk estimates, a hazard quotient is not expressed as a probability. Therefore, while both cancer and non-cancer risk characterizations indicate a relative potential for adverse effects to occur from exposure to a chemical, a non-cancer adverse health effect's estimate is not directly comparable with a cancer risk estimate.

The hazard quotients for each pathway are summed to determine whether exposure to a combination of pathways poses a health concern. This sum of the hazard quotients is known as a hazard index.

$$Hazard\ Index = \sum Hazard\ Quotients$$

5.3 Methods for Assessing Cancer Risks

In the risk characterization, carcinogenic risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of a chemical exposure. Because cancer risks are averaged over a person's lifetime, longer-term exposure to a carcinogen will result in higher risks than shorter-term exposure to the same carcinogen, if all other exposure assumptions are constant. The PEA approach generates a cancer risk estimate for each pathway of exposure for the default age-adjusted resident.

Theoretical risk associated with low levels of exposure in humans is assumed directly related to an observed cancer incidence associated with high levels of exposure in animals. According to U.S. EPA (1989), this approach is appropriate for theoretical upper-bound incremental lifetime cancer risks of less than 1×10^{-2}. The following equation was used to calculate chemical-specific, pathway-specific, and total risks:

$$\textit{Total Carcinogenic Risk} = \Sigma \textit{Individual Chemical and Pathway Specific Risks}$$

This assessment assumes that cancer risks from various exposure pathways are additive. Thus, the result of the assessment is a high-end estimate of the total carcinogenic risk. High-end carcinogenic risk estimates are compared to the range of 10^{-6} to 10^{-4} used as the acceptable risk by the USEPA for Superfund Cleanup Sites. A risk level of 1×10^{-6} represents the "Bright Line" probability goal of one in one million that an individual could develop cancer from exposure to the potential carcinogen under a *defined set of exposure assumptions*. (Exposure parameters used in this risk assessment are summarized in Table C-5). If the estimated risk falls below this risk value ($<10^{-6}$) the chemical is generally considered unlikely to pose a significant carcinogenic health risk to individuals under the given exposure conditions.

5.4 Human Health Screening Results

Table C-6 presents a summary of the hazard/risk calculations using the maximum detected COPC concentrations as EPCs. Based on the use of the PEA methodology and using the highest identified organic COPC concentrations and the highest detected arsenic concentration as EPCs, the non-cancer hazard index for a residential child receptor on the subject property results in a Hazard Index number of **4.0**, and a calculated theoretical upper-bound incremental lifetime cancer risk of **1.4×10^{-3}**.

Table C-7a presents a summary of the hazard/risk calculations using the 95 % UCL of the arithmetic means of the detected COPC concentrations as EPCs. Using this approach, the non-cancer hazard index for a residential child receptor on the subject property results in a Hazard Index number of **1.4**, and a calculated theoretical upper-bound incremental lifetime cancer risk of $\mathbf{4.8 \times 10^{-4}}$. Table C-7b provides the supporting statistical analysis for Table C-7a.

Lead

An evaluation of lead in soil from Mitigation Area 1 indicated six of the ten samples exceeding the Residential CHHSL for lead (150 mg/mg). Table C-8 shows the results of the Leadspread calculation using the maximum site lead concentration on the site (300 mg/kg). The resulting blood lead concentration for a child at the 99th percentile (15.8 µg/dL) clearly exceeds the blood lead level value of 10 µg/dL used by the DTSC. However, as shown on Table C-9a, using the more representative lead concentration value of 132 mg/kg, based on the 95% UCL of the arithmetic mean of the total site lead concentration values, yields a value of 9.5 µg/dL child blood lead at the 99th percentile. This value falls acceptably within the DTSC's screening value for child blood lead. Table C-9b provides the supporting statistical analysis for Table C-9a.

5.5 Uncertainty Analysis

A health risk assessment is not intended to estimate actual health risks to a person or population in conjunction with exposure to chemicals in the environment. Estimating actual risks is unlikely because of the multitude and variability of factors potentially affecting the exposed or potentially exposed populations. This is especially true of the PEA process, which is designed only to give risk managers enough screening-level information to decide whether additional site characterization, a detailed risk assessment, mitigation, or no further action is required. Therefore, risk assessment is a means of estimating the probability that an adverse health effect (for example, cancer) will occur in a person or population at some point in the future. Risk estimates are not likely to underestimate real risk due to the numerous conservative assumptions used in the process.

Risk estimates are calculated by combining site data, assumptions about the potential exposures to impacted media, and toxicity data. As with any type of risk-based analysis, uncertainties exist because of the assumptions used throughout the process. These

assumptions are conservative, meaning that they are more likely to over-predict rather than under-predict the risks associated with a site.

The selection of exposure pathways is based on the potential for actual exposure. Based on the potential exposure pathways evaluated with ultraconservative exposure assumptions, it is evident that existing concentrations of COPCs on the subject property within certain identifiable areas could potentially result in adverse health effects.

6.0 CLEANUP GOAL & SCREENING LEVEL RATIONALE

A human health risk screening of the subject property shows that the theoretical upper-bound incremental lifetime cancer risk for future potential receptors at the subject property exceeds the desired risk range (10^{-6} to 10^{-4}) used by the U.S. EPA as acceptable risk for Superfund Cleanup Sites. The resident child hazard index within portions of the site also exceeds the maximum acceptable non-cancer index of 1.0 when maximum concentrations are used as EPCs (Table C-6). Figure 4 in the body of the RAW presents an Arsenic Concentration Map showing the locations of site samples collected with emphasis on arsenic and a 16.0 mg/kg isocontour line used as a preliminary screening number to delineate elevated arsenic concentrations. Figure 5 in the body of the RAW shows the location of OCP concentrations detected on the site.

Previous site sampling and analysis yielded mean and maximum background arsenic concentrations of 2.1 and 4.4 mg/kg respectively. Current shallow surface site sampling and analysis yielded overall site mean and maximum values of 15.6 and 68 mg/kg respectively.

An arsenic cleanup goal of 8 mg/kg at the 95% UCL for the overall site was conservatively established following derivation of an arsenic cleanup goal in accordance with the DTSC guidance document titled *Arsenic Strategies: Determination of Arsenic Remediation Development of Arsenic Cleanup Goals for Proposed and Existing School Sites (DTSC, 2007).* Option 2 methodology was utilized in accordance with the referenced guidance, which provides for development of cleanup goals using site specific data, and an approach incorporating both visual evaluation of the data plots (graphical evaluation) and statistical calculations (statistical evaluation). These evaluations are presented below.