APPENDIX S

MWH Arsenic Memo

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180 SUPP. DOCS

MEMORANDUM

MWH

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To: Mr. David Sederquist
Youngdahl Consulting Group

Date: February 13, 2007

From: Lee Shull PhD
Principal

Subject: Arsenic in Soil at the Rio Del Oro Site

This memorandum provides my response to a specific comment made by Central Valley Regional Water Quality Control Board (CVRWQCB) staff regarding arsenic in soil at the Rio Del Oro site (hereafter "the Site"). This comment was one of 20 specific comments made by CVRWQCB in their preliminary set of comments on the Rio Del Oro Specific Plan, Draft EIR/EIS[1]. The CVRWQCB's comments on arsenic are the following:

General comment: "The report needs to discuss the potential adverse health affects associated with natural occurring levels of arsenic that are present on the property. Only in that manner can it be determined if mitigation measures are needed to allow construction and inhabitation of the property. A copy of pertinent sections of a report regarding the background metal concentrations on the property has been previously provided to City Planning staff. See specific comment B13, below"

Specific comment B13: "Page 3.13-6, Section 3.13.1, Site Contamination. The initial parts of this section include paragraphs on residual mercury, and lead and asbestos. An additional paragraph should be added to talk about arsenic, as there are significant concentrations of arsenic in the soils. The paragraph would be similar to those on mercury and lead by discussing the potential risks associated with arsenic. As provided in our comments on several other CEQA documents (most recently for Sunridge Specific Plan) recently being prepared by the City of Rancho Cordova, the concentrations of arsenic in the soils at the site (likely as well in properties in the vicinity of the site) are three orders of magnitude above the Preliminary Remediation Goal of 0.062 mg/kg established by USEPA and California Human Health Screening Level of 0.07 mg/kg. The average background concentration of arsenic is 6.8 mg/kg, with a maximum value of 15 mg/kg. As arsenic concentrations are naturally occurring, remediation of arsenic by those Aerojet and/or McDonnell-Douglas is not required. Nevertheless, the arsenic concentrations need to be evaluated and a determination made as to whether or not any measures need to be required prior to allowing the property to be used for residential or commercial purposes."

[1] Letter from Alexander MacDonald (CVRWQCB) to Patrick Angell (City of Rancho Cordova Planning Dept) and Anna Sutton (U.S. Army Corps of Engineers, Sacramento District), February 2, 2007.

Response

The apparent basis of the concern raised in this comment is the finding of naturally occurring concentrations of arsenic in soil at the Rio Del Oro property that exceed USEPA Region 9's "Cal/EPA modified PRG" of 0.062 mg/kg and Cal/EPA's CHHSL of 0.07 mg/kg by about two orders of magnitude[2]. Although the CVRWQCB refers to the measured soil arsenic concentrations (average of 6.8 mg/kg; maximum of 15 mg/kg) as "significant", they also rightly acknowledge that these concentrations are "background" and "naturally occurring" concentrations. The conclusion that the measured concentrations represent naturally occurring levels in California soils is supported by data from a number of surveys as follows:

BACKGROUND ARSENIC CONCENTRATIONS IN SOIL					
	Arsenic Concentration (mg/kg)				
Description of Soil	Range	Mean	Median	95th Percentile	Reference
U.S. Background	1-40				ATSDR, 2005
California Background	0.6-11	3.5			Bradford et al., 1996
Sacramento Area Background	0.8-9.6	3.7			Bradford et al., 1996
California Background-Air Force Bases			2.2	12.7	Hunter et al., 2005
California Soils	0.3-69				Dragun and Chiasson, 1991
Former Orchard, Placer County, CA	4.1-31	18			MWH, 2003

For primarily the following three reasons, I do not believe the naturally occurring arsenic concentrations measured at the Site (average of 6.8 mg/kg; maximum of 15 mg/kg) pose an unacceptable human health risk to future inhabitants of the proposed residential development. That these concentrations are about two orders of magnitude greater than either the USEPA Region 9 residential PRG (USEPA, 2004) or the Cal/EPA residential CHHSL (Cal/EPA, 2005) should not be taken as scientific evidence that soil arsenic at the Site, even though naturally occurring, poses an unacceptable cancer risk to future residents, or that mitigation measures should be taken to reduce potential human exposure.

1. **Both the PRG and CHHSL values for soil arsenic are overly conservative screening-level indicators of theoretical upper-bound cancer risk, and, as such, should be used with caution in risk management decision making.**

   It is extremely important that users of these screening values understand the underlying science involved in their development. Both USEPA Region 9's "Cal/EPA modified PRG" of 0.062 mg/kg and Cal/EPA's CHHSL of 0.07 mg/kg were derived using similar generic

[2] Instead of three orders of magnitude above the PRG and CHHSL values as stated by the CVRWQCB, the measured background concentrations at the Site are approximately two orders magnitude above these screening values.

default exposure assumptions for three exposure pathways; soil ingestion, soil dermal contact, dust inhalation generated from local soil. Although a number of factors contribute to the conservatism of these values, by far the oral cancer slope factor (CSF_o) for arsenic has the greatest influence on the conservativeness of these values.

CSF_o values for arsenic have been developed by the U.S. Environmental Protection Agency (USEPA) and the California Office of Environmental Health Hazard Assessment (OEHHA), and the National Research Council (NRC).

- *USEPA*: 1.5 $(mg/kg/day)^{-1}$
- *OEHHA*: 9.5 $(mg/kg/day)^{-1}$

Although the OEHHA value was used in calculating both the Cal/EPA modified PRG and the CHHSL, both CSF_o values are based on antiquated cancer assessment guidance, which accounts for their extreme conservatism. Both of these CSF_o values were developed under the now outmoded federal *Guidelines for Carcinogenic Risk Assessment* (51 FR 33992, September 24, 1986). A fundamental assumption in these guidelines is that a linear relationship exists between dose and cancer causation. That is, at any exposure level (dose), no matter how low, a probability of cancer exists (*i.e.,* there is no dose below which arsenic would have zero probability of cancer causation). This assumption is termed "non-threshold carcinogenesis" or "linear dose-response relationship." Whereas the evidence that arsenic is a human carcinogen is unequivocal[3], there is now substantial scientific evidence that the dose response for induction of cancer by arsenic is "non-linear" (i.e., a threshold instead of assumed non-threshold dose-response relationship exists for arsenic and cancer causation). The two primary contributing factors underlying this evidence are the following:

- Absorbed arsenic is metabolized and rapidly excreted in the urine and to a lesser extent in the feces (ATSDR, 2000) (*i.e.,* arsenic does not accumulate in body tissues as many known organic carcinogens do (e.g., PCBs, organochlorine pesticides, polycyclic aromatic hydrocarbons).

- The mode of action of arsenic carcinogenesis is now postulated to involve indirect, rather than direct, interactions with DNA (*e.g.,* Germolec et al., 1996; Zhao et al., 1997; Shimizu et al., 1998; Barchowski et al., 1999; Kaltreider et al., 1999; Kerkpatrick et al., 2000; Menzel et al., 2000; Trouba et al., 2000; Andrew et al., 2003; Kitchin and Ahmed, 2003). Among the evidence for this view is the fact that arsenic does not cause point mutations in experimental systems (*i.e.,* it is not mutagenic).

Therefore, because the levels of arsenic actually absorbed and retained in body tissues would

[3] The USEPA has classified arsenic as a known oral and inhalation human carcinogen; the former (oral) based on largely on increased mortality from multiple internal organ cancers (liver, kidney, lung, bladder) and an increased incidence of skin cancer in epidemiological studies of Taiwanese populations consuming high levels of arsenic (several hundred micrograms per day) in drinking water (IRIS, 2007), and the latter (inhalation) based on increased lung cancer mortality observed in multiple studies of inhalation exposure exposures to arsenic by smelter workers (IRIS, 2007).

have to be quite high for a sufficient period of time to induce the types of indirect DNA effects (*e.g.*, inhibition of enzymes that repair DNA damaged from spontaneous mutations) known to occur, the postulated mode of carcinogenic action would be expected to have a non-linear dose-response relationship (*i.e.*, low doses would be expected to have no adverse effect, and are more likely than not to have zero cancer risk).

One of the most significant revisions to the federal Cancer Assessment Guidelines (USEPA, 2005) promulgated by USEPA in 2005 was prompted by recognition that not all carcinogens cause cancer in the same way (*i.e.*, same mechanism), and that non-linear dose-response relationships (*i.e.*, threshold carcinogenesis) exist for some substances, such as arsenic. However, neither the USEPA nor the OEHHA have yet re-assessed arsenic under these new guidelines. Whereas the USEPA is currently in the process of re-evaluating it's CSF_o under the revised guidelines, OEHHA is not.

Both the OEHHA and USEPA CSF_o's were derived using the linearized multistage (LMS) high-to-low extrapolation model. This model assumes a linear dose-response relationship which, as discussed above, is not an appropriate assumption for arsenic. USEPA based it's CSF_o on the occurrence of skin cancer in a Taiwanese population exposed to arsenic in drinking water. USEPA stated in its development of this CSF that "The risk at low doses may be much lower than the current estimates, as low as zero, due to such factors as the metabolism or pharmacokinetics of arsenic" (USEPA, 1988). The agency also acknowledged that this value carries considerable uncertainty, which is reflected by the following quotation taken from IRIS:

"This assessment is based on prevalence of skin cancer rather than mortality because the types of skin cancer studied are not normally fatal. However, competing mortality from Blackfoot disease in the endemic area of Taiwan would cause the risk of skin cancer to be underestimated. Other sources of inorganic arsenic, in particular those in food sources have not been considered because of lack of reliable information. There is also uncertainty on the amount of water consumed/day by Taiwanese males (3.5 L or 4.5 L) and the temporal variability of arsenic concentrations in specific wells was not known. The concentrations of arsenic in the wells was measured in the early 1960s and varied between 0.01 and 1.82 ppm. For many villages 2 to 5 analyses were conducted on well water and for other villages only one analysis was performed; ranges of values were not provided. Since tap water was supplied to many areas after 1966, the arsenic-containing wells were only used in dry periods. Because of the study design, particular wells used by those developing skin cancer could not be identified and arsenic intake could not be assigned except by village. Several uncertainties in exposure measurement reliability existed and subsequent analysis of drinking water found fluorescent substances in water that are possible confounders or caused synergistic effects.

Because of the controversy and uncertainty surrounding the carcinogenicity and toxicity of ingested arsenic, further revisions to the CSF are anticipated.

Historically, the primary basis behind the assumption of linearity in the dose-response relationship in cancer assessment is direct genotoxicity mechanisms (such as, interference with DNA synthesis or repair) of carcinogenicity. Genotoxic effects may result from either

direct interaction with DNA or indirect DNA effects. In the case of arsenic, the findings of animal studies on the carcinogenicity of inorganic arsenic are generally uniformly negative (NRC, 2001). This line of evidence is supportive of a non- or indirect genotoxic mode of action for arsenic carcinogenicity. It is likely that arsenic carcinogenicity is based on chromosomal alterations, most likely as a result of cellular housekeeping processes (that is, via an indirect non-genotoxic mode of action). Such indirect effects are predicted to lead to sublinear (or possibly threshold) dose responses (NRC, 2001). Therefore, for arsenic carcinogenicity, the most plausible mode of action is via indirect non-genotoxic effects, which lead to a sublinear, not linear, dose-response relationship. This is contrary to how the current CSF was established.

In comparison, the basis for the OEHHA CSF_o (9.5 $(mg/kg/day)^{-1}$) is the mortality of arsenic-induced lung and urinary bladder cancers observed in epidemiological studies of populations in Taiwan, Chile, and Argentina (OEHHA, 2003). The agency used this CSF_o to derive a proposed public health goal (PHG) of 0.004 µg/L (4 ppt) for arsenic in drinking water. Similar to USEPA's CSF_o, the OEHHA value is based on a low-dose linear extrapolation approach using the aforementioned LMS model. As discussed above, the primary source of uncertainty in the derivation of CSF values for arsenic is the shape of the dose-response curve. If this is the case, the assumption of linearity would overestimate risks.

Another important factor that supports the position that the carcinogenic effects of arsenic can not be accurately predicted by linear extrapolation from high doses to low doses is the scientific evidence that sub-toxic levels of arsenic appear to be protective by inducing inherent protective mechanisms in the body (Wang et al., 1994; Barchowski et al., 1999; Snow et al., 1999; Pott et al., 2000; Romach et al., 2000; Snow et al., 2000).

Although epidemiological evidence exists for human carcinogenesis at high exposure levels, all of the studies have been similarly criticized for not providing adequate information for estimating the magnitude of risk at specific arsenic concentrations (Brown and Beck, 1996). There is also no evidence of increased incidence of skin cancer based on studies of U.S. populations consuming relatively high concentrations (range of 0.1 to 0.2 mg/L) in drinking water (ATSDR, 2000).

In summary, both the USEPA and OEHHA CSF_o values are outmoded, are not based on present-day scientific methods for establishing carcinogenic potency of substances that behave in the body as arsenic does, and should be re-evaluated by both OEHHA and USEPA as soon as possible. Thus, in my opinion, Region 9's Cal/EPA modified PRG and Cal/EPA's CHHSL, which are based on OEHHA CSF_o, likely overestimate theoretical upperbound carcinogenic risk by at least one to two orders of magnitude. Furthermore, both the USEPA Region 9 and OEHHA recognize the conservatism of the Cal/EPA modified PRG and CHHSL values, and has advised accordingly in their respective guidance documents. As shown in the Table above, values are well below naturally occurring concentrations of arsenic in virtually all California soils, and should not be applied as regulatory cleanup standards. Rather, both agencies use these screening values only to provide preliminary indication whether further evaluation at a site is warranted (*i.e.*, measured concentrations less than a screening value provides strong assurance that no further evaluation is needed).

2. **Because the bioavailability of naturally occurring arsenic in soil is generally low, the amount of arsenic absorbed into the body is thereby reduced resulting in lowered potential for adverse health impacts.**

For arsenic to cause systemic toxicity, it must be absorbed into the body (*i.e.,* enter the blood stream). Therefore, the fraction of naturally occurring arsenic available for absorption (*i.e.,* the "bioavailability") is an important factor is evaluating the health implications of the arsenic in soil at the Site. Both USEPA's Cal/EPA modified PRG and Cal/EPA's CHHSL assume that 100% of the arsenic that enters the gastrointestinal tract (GIT) will be absorbed into the body. Moreover, the CSF_o values are based on human dose-response data in which people ingested high levels of ionic arsenic (arsenate and/or arsenite in drinking water). Unlike naturally occurring arsenic in soil, arsenic in water is 100% bioavailable, and at high enough levels can overwhelm the protective mechanisms afforded by metabolism and excretion.

The factors influencing the bioavailability of arsenic in the portals of entry (GIT, skin, lungs) are well understood. Only ionic arsenic is absorbed in the GIT of animals including humans (ATSDR, 2000). The primary determinant of arsenic bioavailability is the dissolution rate of arsenic in the fluids of the GIT (*i.e.,* rate that the ionic form is generated from naturally occurring chemical complexes in the GIT). Ionic forms that can bind to soil particles and less soluble mineral forms are less bioavailable than more soluble forms such as sodium arsenate and arsenic trioxide. Less soluble forms include sulfide minerals, complex oxides, and mineral forms in which arsenic is covalently bonded with iron, manganese and phosphate (ATSDR, 2000; Kelley et al., 2002). The less soluble forms of arsenic are reported to be one-half to one-tenth as bioavailable as the more soluble forms of arsenic (DoD, 2003). Arsenopyrite, a complex of arsenic, sulfur, iron, and phosphate, is one of the most common chemical forms in soil and rock in the Sacramento area.

Numerous *in vivo*[4] and *in vitro*[5] studies have been conducted on the bioavailability of ingested arsenic in soil and various waste materials (Freeman et al., 1993 & 1995; Groen et al., 1994; Borch et al., 1994, Boyce et al., 1996, USEPA, 1996b, Casteel et al., 1997 & 2003; Hamel et al., 1999; Kees et al., 1994, Ng et al., 1998, Rodriquez et al., 1991, Ruby et al., 1999; Roberts et al., 2002). In a recent symposium hosted by the California Department of Toxic Substances Control (DTSC)[6], the scientific consensus was reached that *in vitro* arsenic bioavailability alone are not reliable (*i.e.,* do not correlate well with *in vivo* measurements). Therefore, for purposes of this memorandum, only *in vivo* study results will be discussed.

In vivo relative bioavailability[7] of arsenic in soil and waste materials has been measured in a number of animal models including rats, swine, rabbits, dogs and monkeys. Several USEPA regions and states have relied upon the results of these studies to adjust the bioavailability value used in estimating human health risks, and deriving site-specific cleanup levels. In

[4] Studies in animal models.

[5] Studies done in test tubes.

[6] This symposium entitled "Bioavailability of Lead and Arsenic: Using 'in vivo' and 'in vitro' Measurements", Sacramento, CA, September 13, 2005.

[7] 'Relative bioavailability' is a measure of the difference in absorption between different forms of a chemical (e.g., arsenic as sodium arsenate in water [100%] vs soil).

comparison to other animal models, the primate bioavailability studies generally show values for soil-borne arsenic less than 30%. For example, the relative arsenic bioavailability in soil from a copper smelter was 20% in primates (Freeman et al., 1995). Blood data from the same primate study showed estimates for both soil and house dust of 10-12% relative bioavailability. In another primate study, arsenic bioavailability in soil from five different sites ranged from 11 to 25% based on urinary arsenic excretion in primates (Roberts et al., 2002).

In summary, the bioavailability of the naturally occurring soil-borne arsenic at the Site in the human GIT is highly likely to be less than 30%, perhaps lower. Therefore, the amount of arsenic that might be absorbed and distributed to potential cancer target sites in the body (e.g., skin, lungs, urinary bladder) from incidentally ingested soil is decreased even more; to levels that are even more likely to be maintained at below critical levels at target cancer sites in tissues.

3. **In accordance with USEPA guidance and the National Contingency Plan (NCP), human exposure levels to carcinogenic substances that result in a theoretical upperbound cancer risks of less than 1 x 10^{-4} do not pose an unacceptable risk.**

The measured soil arsenic concentrations at the Site (average of 6.8 mg/kg; maximum of 15 mg/kg) are approximately two orders of magnitude above the USEPA Region 9's Cal/EPA modified PRG of 0.062 mg/kg and Cal/EPA's CHHSL of 0.07 mg/kg. Because these screening values are based on a theoretical upperbound target cancer risk level of 10^{-6}, concentrations equivalent to other target risk levels are the following:

Target Cancer Risk Level	USEPA Region 9 Cal/Modified PRG (mg/kg)	Cal/EPA CHHSL (mg/kg)
10^{-6}	0.062	0.07
10^{-5}	0.62	0.7
10^{-4}	6.2	7.0

As stated in the Federal Register 56:20, page 3535, "...a target (cancer) risk range of 10^{-4} to 10^{-6} is considered by EPA to be safe and protective of human health." Most regulatory agencies consider a 10^{-6} theoretical upper-bound cancer risk as "a point of departure", rather than a final risk management criterion. Therefore, it can be argued that the concentrations of arsenic at the site, even without consideration of the conservatism inherent in the regulatory screening values discussed above, are in the range of concentrations considered by the USEPA to be safe and protective of human health.

In summary, it is my firm opinion that the naturally occurring concentrations of arsenic measured at the subject Site would not pose an unacceptable risk to future residents that may inhabit the Site. This opinion is based on the combined conservatism in the derivation of USEPA Region 9's Cal/EPA modified PRG and Cal/EPA's CHHSL. The two primary sources of this conservatism are:

- The underlying methods and assumptions involved in the derivation of the CSFo values,

for which I estimate a minimum of one to two orders of magnitude overestimation of risk, and

- The assumed 100% bioavailability of arsenic in the human GIT, for which I estimate one-half to one order of magnitude overestimation of risk.

Even if these sources of conservatism are discounted, the concentrations of measured arsenic in Site soil is in the range of theoretical upper-bound cancer risk considered by EPA to be safe and protective of human health.

REFERENCES