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MCAS EL TORO
SSIC # 5090.3

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION IX
75 Hawthorne Street
San Francisco, CA 94105

November 4, 1999

Mr. Dean Gould
BRAC Environmental Coordinator
Former U. S. Marine Corps Air Station - El Toro
P. O. Box 51718
Irvine, California 92619-1718

Re: U. S. EPA Comments on Draft Phase II Remedial Investigation Report, Operable Unit 3B, Sites 7 and 14, Marine Corps Air Station (MCAS) El Toro, CA

Dear Mr. Gould:

The United States Environmental Protection Agency (EPA) has reviewed the above referenced document. The attached comments are provided by Dr. Jeffrey Paull, EPA's toxicologist for MCAS El Toro. Please feel free to contact me if you have any questions or if you would like to arrange a meeting or conference call to discuss Dr. Paull's comments.

Sincerely,

Glenn Kistner

Glenn Kistner
Remedial Project Manager
Federal Facilities Cleanup Branch

Attachment

cc: Alice Gimeno, DTSC
Patricia Hannon, RWQCB
Gregory Hurley, RAB Co-Chair
Polin Modanlou

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION IX
75 Hawthorne Street
San Francisco, CA 94105-3901

MEMORANDUM

TO: Glenn Kistner
Remedial Project Manager
Federal Facilities Cleanup Branch, Navy Section

FROM: Jeffrey M. Paull, DrPH, CIH
Regional Toxicologist
Superfund Technical Support Team

DATE: November 3, 1999

SUBJECT: Review of Draft Phase II Remedial Investigation Report, Attachments O and P, Operable Unit 3B, Sites 7 and 14, Marine Corps Air Station, El Toro, California

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Document Reviewed

In response to the request of Glenn Kistner, EPA Remedial Project Manager (RPM) for El Toro Marine Corps Air Station (MCAS), the Superfund Technical Support Team has reviewed the *"Draft Phase II Remedial Investigation (RI) Report, Attachments O and P, Operable Unit 3B, Sites 7 and 14, MCAS El Toro."* The RI report was prepared by Bechtel National, Inc., San Diego, California, and is dated September 7, 1999. The document was reviewed by Dr. Jeffrey Paull of the USEPA Technical Support Team. We previously reviewed the MCAS El Toro *"Risk Assessment Plan"* (January 20, 1995), the *"Revised Draft Work Plan, Phase II RI/ FS Study* (May 24, 1995), the *"Draft Phase II RI Report, OU-2B, Sites 2 and 17"* (May 14, 1996), the *"Draft Phase II RI Reports, OU-2C, Sites 3 & 5"* (June 20, 1996), the *"Technical Memorandum, Revised Risk Assessment Procedures"* (July 25, 1996), and the *"Draft Final Phase II RI Report, OU-3A Sites"* (April 10, 1997).

Scope of Review

We focused our review on Section 6 of the Draft Phase II RI Report, containing the baseline human health risk assessment (HHRA) for Sites 7 and 14. The HHRA, and its associated figures, data tables, and appendices, was reviewed for scientific and technical accuracy, and for conformance with USEPA Region 9 risk assessment guidelines, policies, and procedures. We have provided specific comments on the HHRA, organized according to section and page location. Minor grammatical and typographical errors and inconsistencies in the document have been noted only to the extent that they may affect the interpretation of the risk assessment. We request that all future changes or additions to the human health risk assessment portions of the document be clearly identified.

Summary

Overall, the Draft Phase II RI Report, containing the baseline human health risk assessment (HHRA) for Sites 7 and 14 is well-written, organized, and succinct. The approach for each component of the risk assessment is clearly presented in Section 6 of the RI, and the methods and procedures used to estimate the human health risks are generally consistent with USEPA risk assessment guidance.

Although relevant exposure pathways, plausible exposure assumptions, and appropriate toxicity values and exposure factors were generally employed to estimate risks in the HHRA, there were some exceptions which may have resulted in the underestimation of site risks; these are noted in our specific comments below.

The most significant of our comments involves the exclusion of potential exposure pathways (*e.g.,* the groundwater, indoor air, and plant uptake exposure pathways) from the risk assessment. We recommend that all COPCs be carried through the entire risk assessment, and that their contribution to site risks be made more explicit. We also recommend that the uncertainty section of the HHRA make note of the potential underestimation of site risks for VOCs in soil, due to the method of sample collection and analysis (soil matrix vs. soil gas), and as a result of other data gaps and simplifying assumptions. Additionally, we recommend that exposure factors and toxicity values be revised and updated to ensure consistency with current USEPA values.

Specific Comments

(1) **Soil Gas Survey, §1.3.5, p. O1-9:** In 1994, a soil gas survey was conducted at Sites 24 and 25 in the southwestern quadrant of the base. During this investigation, both soil gas and soil samples were collected from approximately 15 and 30 feet bgs in 465 locations. Both soil gas and soil samples were analyzed for VOCs. However, there is no information presented in the RI indicating that the two sets of VOC data were ever correlated or compared with one another. With respect to the evaluation of VOCs in soil, this type of comparison is important to conduct, since it has been well established that a significant fraction of VOCs are lost from soil matrix samples prior to analysis. The results from the soil gas analyses could have been use to validate the soil matrix samples, or to correct the soil matrix data for sample losses.

(2) **Summary of Phase 1 Sampling Results, §4.1, p. O4-2:** Six VOCs were reported at concentrations ranging from 2 to 64 µg/kg in shallow soil samples collected from 13 shallow borings in Units 1, 3, 4, and 5. However, estimated risks for exposure to VOCs are not presented in the text portion of the HHRA. The RI should clarify which VOCs were identified as COPCs in the text (as well as listing them in the Appendix), and present the estimated risks for all VOCs detected (particularly benzene), rather than presenting estimated risks for only the risk drivers at each site.

(3) **Summary of Phase 1 Sampling Results, §4.1, p. O4-2:** Four SVOCs and 11 PAHs were

reported at concentrations ranging from 240 to 6900 µg/kg in shallow-soil samples collected from 14 borings in Units 1 through 5. However, estimated risks for only two of these PAHs (benzo(a)pyrene and dibenz(ah)anthracene) are presented in the text portion of the HHRA. The RI should clarify which PAHs were identified as COPCs in the text (as well as listing them in the Appendix), and present the estimated risks for all PAHs detected, rather than presenting estimated risks for only the risk drivers at each site.

(4) **Summary of Phase 1 Sampling Results, §4.1, p. O4-2:** Seven pesticides, including chlordane, DDD, DDE, DDT, endrin, methoxychlor, were reported at concentrations ranging from 0.186 to 200 µg/kg in shallow-soil samples from nine borings in Units 1, 3, 4, and 5. However, estimated risks for exposure to pesticides are not presented in the text portion of the HHRA. The RI should clarify which pesticides were identified as COPCs in the text (as well as listing them in the Appendix), and present the estimated risks for all pesticides detected, rather than presenting estimated risks for only the risk drivers at each site.

(5) **Summary of Phase 1 Sampling Results, §4.1, p. O4-13:** The RI states that: *"The findings of the Phase II RI OU-2A VOC source area investigation and the Phase I RI investigation for this site have demonstrated that Site 7 is not a source of regional groundwater contamination. Groundwater contamination beneath Site 7 is being addressed by the OU-2A work, and therefore, a discussion of the Phase I RI groundwater analytical results is not presented here."*

However, the fact that Site 7 is not a source of groundwater contamination does not preclude the assessment of human health risks for the groundwater pathway, given that the groundwater beneath the site represents a viable drinking water aquifer. Moreover, the purpose of a baseline risk assessment is to evaluate risks to all potential receptors for all potential exposure pathways. Hence, there appears to be insufficient justification for the exclusion of groundwater analytical results from the baseline risk assessment for OU-3B, which renders the baseline human health risk assessment incomplete.

(6) **Summary of Phase 2 Sampling Results, §4.2, p. O4-14:** The one-page summary of laboratory analytical results for shallow-soil samples and for the deeper subsurface-soil samples collected from Units 1 through 5 in Phase II, similar to that prepared for the Phase I results (see p. O4-2), appears to have been inadvertently omitted from the RI.

(7) **Nature and Extent of Contamination, §4.3, p. O4-14 to O4-76:** This section should include a discussion of the treatment of nondetect Phase 1 and Phase II sampling results, including treatment of elevated detection limits or sample quantitation limits which may exceed health-based levels. Reported nondetect results do not defensibly demonstrate, in and of themselves, that the potential for adverse health risks is unlikely. In a screening of contaminants of potential concern, the risk assessment should specify how it addresses contaminants which are reported as nondetect, but which may have elevated method or instrument detection limits or sample quantitation limits (SQLs), and which may exceed contaminant-specific health-based screening levels.

(8) **Fixed-Base Laboratory Results, Volatile Organic Compounds, §4.3.1.1, p. O4-31:** There is an apparent conflict between the summary data which reported a total of *six*

VOCs detected in Units 1, 3, 4, and 5, and the data presented in this section, which reported that *eight* VOCs (acetone, benzene, carbon tetrachloride, chloroform, 1,2-dichlorobenzene, methylene chloride, toluene, and xylene) were detected in 22 shallow-soil samples collected from nine boring locations in Unit 1 alone.

(9) **Fixed-Base Laboratory Results, Semivolatile Organic Compounds/Polynuclear Aromatic Hydrocarbons, §4.3.1.1, p. O4-31:** There is an apparent conflict between the summary data which reported *four* SVOCs and *11* PAHs detected in Units 1 - 5, and the data presented in this section, which reported that *six* SVOCs and *15* PAHs were detected in 25 shallow-soil samples collected from eight boring locations in Unit 1 alone. The highest reported PAH concentration of 7000 µg/kg for fluoranthene and pyrene appears to differ slightly from the highest value of 6900 µg/kg reported in the summary data.

(10) **Transformation Processes, Volatile Organic Compounds, §5.2.2, p. O5-10:** Here it is stated: *"Eleven VOCs were reported at low concentrations (less than 73 µg/kg) in surface and shallow-soil samples at Site 7. Because of the low concentrations, these VOCs are not addressed further in this section."* It is not surprising that VOCs were reported at low concentrations in surface soils due to volatilization and evaporative loss. However, the method of sample collection and analysis for VOCs, involving bulk sample analysis of the soil matrix, rather than soil gas analysis, can result in significant losses of VOCs from the soil samples. The RI should address the potential for underestimating VOC concentrations in soil due to the sampling and analysis method for VOCs, and the potential underestimation of health risk which may result (see Comment #1).

(11) **Transformation Processes, Tables 5-2, & 5-3, p. O5-11:** An explanation is needed for why data for only two COPCs (benzo(a)pyrene and dibenz(a,h)anthracene) is provided in Table 5.2, *"Physiochemical Properties of Selected Organic Compounds at Site 7"*, and Table 5.2, *"Estimates of Mobility and Persistence of Selected Chemicals at Site 7."* Generally this type of data is provided for all chemicals of potential concern in a risk assessment, rather than for just a select subset of COPCs which are found to be risk drivers.

(12) **Conceptual Model, §5.1, & Figure 5-2, p. O5-1 to O5-4:** The conceptual site model (CSM) does not show the indoor air exposure pathway for VOCs in soil gas, which should be evaluated for the future resident, and occupational worker. Additionally, the CSM does not indicate direct contact with surface and subsurface soils as a potential exposure pathway for the future resident; direct contact via ingestion and dermal contact is presented only for workers. Also missing from the CSM is ingestion, inhalation, and dermal absorption via the groundwater exposure pathway (see Comment #5), and ingestion via the plant uptake exposure pathway.

(13) **Chemicals of Potential Concern, §6.1, p. O6-1:** Although chemicals of potential concern (COPCs) are presented in Appendix I, they should be brought forward in the document, and be presented in the body of the HHRA.
We recommend presenting a summary table for each OU site, listing each COPC detected, it's exposure point concentration, and the corresponding risk and/or hazard index.

(14) **Data Evaluation Process, §6.1.1, p. O6-1 to O6-2:** Although it is stated that: *"All data were evaluated for the data quality indicators (precision, accuracy, representativeness, comparability, and completeness),"* no mention is made of the evaluation of the adequacy of sample detection limits, and the treatment of non-detect data (see Comment #7).

(15) **Receptor Analysis, §6.2.1, p. O6-3 to O6-4:** To ensure clarity, this section should note that the residential receptor analysis is divided into adult and child exposure evaluations. In addition, this section should describe all potential receptor populations considered (*e.g.;* construction/utility worker, current and future off-site resident, resident trespasser) and provide a rationale for elimination of these receptors from consideration in the risk assessment. Where the direct soil exposure pathway exists, we generally recommend evaluating the construction/utility worker receptor for contact with both surface and subsurface soil in the risk assessment. This receptor is assumed to be a worker who spends all of their time working outdoors in construction-related activities, and has considerable potential for exposure to surface and subsurface soil.

(16) **Exposure Pathways, §6.2.2, & Figure 6-1, p. O6-4 to O6-5:** Additional explanation or justification is required for elimination of the groundwater exposure pathway for metals, and the indoor air exposure pathway for VOCs in soil gas, which should be evaluated for both the future resident, and occupational worker. Also missing from the HHRA is consideration of the plant uptake exposure pathway for the future resident. (see Comments #5 & #12).

(17) **Exposure Point Concentration, §6.2.3, p. O6-4 to O6-7:** The HHRA states that: *"For the resident child and adult (residential scenario), soil concentrations (0 to 10 feet bgs) were used to calculate exposure-point concentrations."* However, averaging of all of the data between 0 and 10 feet bgs may not be the most representative or health-protective basis for determining residential soil exposures. Although it is reasonable to assume that future residents could have contact with subsurface soil via a spectrum of residential land use, it is also true that the vast majority of soil exposure will be attributable to surficial soils.

The combination of all available soil data down to a depth below a standard building footing depth may be problematic at sites where surficial releases are of predominant concern, particularly from contaminants which are unlikely to leach to significant depths (*e.g.,* lead, PAHs). The risk assessment should therefore consider and discuss whether risks and hazards due to residential exposures should be based on contact with two exposure point concentrations--one based on surficial soil, and a second based on combined surface and subsurface data. Risk managers may also find risk and hazard estimates based on two exposure point concentrations to be beneficial in fostering a better understanding of environmental conditions at the base. This is particularly important for sites being proposed for no further action.

(18) **Estimation of Dose Rate, §6.2.4, p. O6-7 to O6-9:** The exposure assumptions for the adult and child receptors used in the HHRA are derived from EPA default exposure factors from the 1989 Exposure Factors Handbook. The EPA has updated these values in the 1997 Exposure Factors Handbook, and we recommended that these updated values be employed in the risk assessment.

These updated values include 75 years rather than 70 years for average life expectancy, and 71.8 kg for mean adult body weight rather than 70 kg. A soil ingestion rate of 400 mg/day for children is now recommended, instead of the 200 mg/day used in the risk assessment, while the soil ingestion rate for adults has been reduced from 100 mg/day to 50 mg/day. Updated dermal adherence factors of 0.08 mg/cm^2 for the adult, and 0.3 mg/cm^2 for the child were utilized in the risk assessment (however, the source for these updated dermal adherence values was omitted from the References presented in §8). Use of these updated exposure factors will be likely to result in changes in the quantitative risk estimates. The net effect will most likely be a decrease in estimated risks for the adult receptor, and an increase in estimated risks for the child receptor.

(19) **Source of Toxicity Criteria, §6.3.3, p. O6-10 to O6-11:** The toxicity values used in the risk assessment were obtained from the 1998 table of PRGs published by USEPA Region 9. The USEPA has since updated its PRG values, and we recommend that the toxicity values from the current (1999) EPA Region 9 PRG table be utilized in the risk assessment.

(20) **Toxicity Criteria for COPCs Without Assigned Criteria, §6.3.6, p. O6-12:** For surface soil, the concentration of lead was compared to the USEPA PRG of 1000 mg/kg in the risk assessment. In the event that the PRG was exceeded, the Cal-EPA pharmacokinetic (LeadSpread) model was used to evaluate potential lead exposure. We do not concur with this approach, as both the adult, and particularly the child residential receptor are most often exposed to lead via contact with surficial soils. Assuming exposure to soils from 0-10 feet bgs for the residential receptor may result in underestimating both exposure and risk for many immobile COPCs such as lead and PAHs (see Comment #17). We recommend using the Cal-EPA LeadSpread model to evaluate potential lead exposure for the adult and child resident whenever lead exposure point concentrations exceed the EPA residential PRG of 400 mg/kg in surficial soils.

(21) **Incremental Risk, §6.4.3, p. O6-13 to O6-14:** Incremental carcinogenic risk was calculated in the HHRA by subtracting background risk for metals from their corresponding total lifetime risk. We recommend that the risk assessment present the risks and hazards associated with these background concentrations in the RI. This information would provide useful site-specific risk information to the risk manager.

(22) **Risk Characterization Results, §6.5, p. O6-14 to O6-21:** The following statement in the RI paraphrases the National Oil and Chemical Substances Pollution Contingency Plan (NCP): *"The NCP states that for known or suspected carcinogens acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between one in ten thousand (10^{-4}) and one in one million (10^{-6})."* The RI subsequently refers to this as the "acceptable risk range."
The USEPA does not share this interpretation of the NCP, and does not consider 10^{-6} to 10^{-4} as an acceptable risk range. Rather, EPA recommends that risks in the 10^{-6} to 10^{-4} range be carefully evaluated for remediation, depending on the frequency and duration of exposure, the population potentially exposed, the weight of evidence of carcinogenicity, and other factors, including feasibility and cost of remediation. Hence, a more appropriate term for the 10^{-6} to 10^{-4} range would be the "risk management range." USEPA considers a 10^{-6} risk as the point of departure for considering remediation of risks in this range.

(23) **Risk Characterization Results, §6.5, p. O6-21:** Here it is stated that, *"The cancer risk for the adult resident is slightly higher than for the child."* However, estimated cancer risks for the child will probably increase once updated exposure factors are used to recalculate risks. The child is a susceptible subpopulation with respect to cancer, due to the child's higher relative intake rates, smaller body mass, less mature immune system, and potentially greater latency period.

(24) **Site 7, Unit 1, North Pavement Edge, Industrial Scenario, §6.5.1, p. O6-31:** We recommend eliminating the word *"threshold"* from the term *"background threshold risk."* We find its usage in this term to be somewhat confusing, and unnecessary in this context.

(25) **Site 7, Unit 1, North Pavement Edge, Industrial Scenario, §6.5.1, p. O6-31:** The RI states that, *"The risk for lead is considered acceptable based on a comparison of the U.S.EPA industrial PRG for lead (1,000 mg/kg) and the 95 percent UCL for lead in surface soils of Unit 5 (931 mg/kg). "* We do not concur with this conclusion, and recommend that lead be evaluated for the adult and child residential receptors at this site using the Cal-EPA LeadSpread model, for the reasons presented in Comments #17 and #20 above.

(26) **Uncertainty Analysis, Data Evaluation, §6.6.1, p. O6-37 to O-39:** This section should include a discussion of the uncertainties in the risk assessment which may result from the underestimation of VOCs in soil, due to the method of sample collection and analysis (see Comment #1). Uncertainties introduced by high sample detection limits (at or near health-based limits such as USEPA PRG values) should also be discussed (see Comment #7).

(27) **Uncertainty Analysis, Exposure Assessment, §6.6.2, p. O6-39:** This section should include a discussion of the uncertainties in the risk assessment which may result from the underestimation of site risks due to the elimination of indirect, and secondary exposure pathways from the risk assessment (See Comment #16).

(28) **Uncertainty Analysis, Toxicity Assessment, §6.6.3, p. O6-39 to O-40:** According to EPA risk assessment guidelines, route-to-route extrapolation is generally contraindicated for substances which display a direct local toxic effect at the point of application. However, the route-to-route extrapolation methodology appears to have been applied across the spectrum of potential contact for all COPCs. For example, the RI presents dermal risk estimates based on an oral cancer slope factor for PAHs, although PAHs are known to initiate malignant skin tumors through direct action at the point of application. The uncertainty section of the risk assessment should be revised to discuss the appropriateness of the route-to-route extrapolation methodology for PAHs and any other COPCs which cause a direct local toxic effect.

(29) **Site 14, Unit 1, Residential Scenario, §6.6.2, p. P6-24:** In contrast to the way in which lead was evaluated at Site 7, Unit 1, here it is stated: *"Because the Cal-EPA residential soil PRG for lead (130 mg/kg) was exceeded at this area of potential concern (923 mg/kg), the Cal-EPA pharmacokinetic model was utilized to estimate the blood lead concentration for a resident child and an adult exposed to lead in the shallow soils at Unit 1."* The results of this LeadSpread analysis showed predicted blood lead concentrations for the child receptor above the 10µg/dL threshold at the 90th, 95th, 98th, and 99th percentile. We would expect a similar result for the evaluation of lead risk at Site 7, Unit 1 (see Comment #24).

cc: John Christopher, Cal-EPA/DTSC

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