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MOFFETT FIELD
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Cal/EPA

Department of Toxic Substances Control

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January 22, 1997

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Commander
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Engineering Field Activity, West
Naval Facilities Engineering Command
Attn: Mr. Stephen Chao, Project Manager
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Dear Mr. Chao:

THE NAVY'S RESPONSE TO DTSC COMMENTS ON DRAFT FINAL SITE-WIDE ECOLOGICAL ASSESSMENT REPORT, MOFFETT FEDERAL AIRFIELD

The Department of Toxic Substances Control (DTSC) and the San Francisco Regional Water Quality Control Board (SFRWQCB) have reviewed the subject document and prepared following comments. The State did not receive the technical memoranda from the Navy until January 15, 1997. Therefore, no comments on the technical memoranda have been included in this letter.

If you have any questions, please call me at 510-540-3830.

Sincerely,

C. Joseph Chou
Remedial Project Manager
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Enclosure

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General Comments on the RTC

For most of the substantive issues on the Draft Final Phase II SWEA raised by DTSC in our letter of August 23, 1996, the Navy response appears to disagree with DTSC comments. After careful review of the RTC and our previous comments, we cannot justify a change in our position stated in our letter of August 23, 1996, or our letter of January 23, 1996. Unless substantive changes are made in the calculation of HQ estimates for birds and mammals in the Final Phase II SWEA, DTSC will find the document unacceptable to quantify and describe risks to wildlife at Moffett Federal Airfield. Many of the issues are substantive, and directly impact upon the conclusion for describing ecological risks, and therefore may directly impact upon development of risk-based remedial goals. Specific substantive issues with which we disagree include:

a. the calculation of transfer coefficients using Moffett-specific data,

b. evaluation of toxicity for dioxin-like PCBs,

c. interpretation of the high toxicity reference value (TRV) as protective of wildlife, and

d. interpretation of impacts on the endangered Salt Marsh Harvest Mouse (SMHM).

Use and Interpretation of the Toxicity Reference Values (TRVs)

The Navy's position in the Draft Final SWEA and RTC is that the high TRVs are "protective", and thus only HQ1 and HQ2 estimates greater than one are indicative of adverse effects. DTSC disagrees with this interpretation of the TRVs based upon our active involvement with the Navy/BTAG effort, the development of the Moffett TRVs, and the development of the Regional TRVs. It is DTSC's position that HQs derived from the low TRVs (HQ3 and HQ4) are the best indicators of possible adverse effects for most contaminants. The low TRVs were derived to be reasonable "low-risk" toxicity values. The low TRVs should not be viewed as overly conservative, since uncertainty factors were applied only when insufficient data were available (e.g. an unbounded lowest-observable-adverse-effect-level, (LOAEL)). No interspecies uncertainty factors were applied, nor were uncertainty factors applied to protect special-status species. HQ3 and HQ4 estimates less than one indicate there is low likelihood for adverse effects from the contaminant. HQ3 estimates greater than one indicate there is a possible adverse effect upon several individuals in the population since the dose is an average over the contaminated area. HQ4 estimates greater than one indicate

there is a possible adverse effect upon individuals exposed to hot spots of contamination, or for species with small home ranges relative to the area contaminated. When the HQ3 and HQ4 estimates are greater than one, then more evaluation is needed to refine the estimates through either toxicity testing, laboratory studies, and/or field investigations. If there is confidence in the major components used to estimate HQ3 and HQ4, the Low TRV is the appropriate toxicity value from which to derive risk-based cleanup numbers because it represents a reasonable estimate of a chronic no-observed-adverse-effect-level (NOAEL) (although the final cleanup numbers may be higher based upon the other balancing criteria). The High TRVs were developed to provide estimates of dose levels at which significant adverse impacts can be expected on individuals and are also possible at a population level (since the endpoints of the High TRVs are generally significantly increased reproductive impacts or other systemic effects on a majority of the treated animals). The HQs derived from the high TRVs (HQ1 and HQ2) should be used to indicate contaminants which are at levels high enough to warrant expedited removal actions.

DTSC disagrees that using the High TRVs are the best point estimates of risk (HQ1 and HQ2). The Low TRV was developed as a "low risk" toxicity value. Levels above this low risk value imply there is potential risk to the receptor. The "High TRV" value was developed as a "significant adverse effect level". In a general sense for risk management purposes, the "Low TRV" is analogous to a 10-6 cancer risk level, while the "High TRV" is analogous to a 10-4 cancer risk level (keep in mind we are not evaluating cancer risk in the SWEA, but adverse noncancer levels well above those predictive of cancer). Therefore, the Navy's emphasis on using the "high TRV" estimates (HQ1 and HQ2) as the "best point estimate" of risk is analogous to deciding the 10-4 cancer risk level is the benchmark of concern instead of the 10-6 cancer risk level. Such an approach is not consistent with the objectives of the TRV working group, nor can it be defended on a toxicological basis. We cannot overemphasize the importance of this issue in the interpretation of the SWEA report; it appears to be a major area of disagreement between the Navy and regulatory agencies/ natural resource trustees.

Impacts on the Salt Marsh Harvest Mouse

Page 1, response to DTSC General Comment No. 2 states, "The Navy believes that the data and risk estimates suggest that the likelihood of impacts to individual salt marsh harvest mice is low..." DTSC disagrees with this comment. As indicated below, correct calculation of the sediment-to-pickleweed transfer coefficient (TC) for polychlorinated biphenyls (PCBs) will substantially increase the dose and risk estimates to the salt

marsh harvest mouse (SMHM). We calculate the correct value for this TC to be 3.8 (dry weight food/dry weight sediment basis). This value is 4.4 times greater than the TC of 0.86 used by the Navy. Therefore, all dose estimates to the SMHM from total PCBs should be 4.4 times greater (3.8 / 0.86 = 4.4). Using the DTSC-calculated TC, the correct HQ3 is 3.7, and the correct HQ4 is 42.7. Since adverse reproductive effects were observed in laboratory animals chronically exposed to levels around 1 mg/kg-day, average dose estimates to the SMHM (HQ3) are close to this level, and localized areas of high contamination exceed these dose estimates (HQ4). The SMHM is likely to be exposed to high localized areas of contamination due to it's small home range. Thus, DTSC believes it is likely there are impacts on individual SMHM from PCBs.

We did not calculate HQ1 and HQ2, using the high TRV, because these quotients are of limited use in evaluating risks to the SMHM from total PCBs. As we stated in our letter of August 23, 1996, the small mammal high TRV of 84 mg/kg-day greatly exceeds the level at which adverse impacts in laboratory animals chronically exposed to PCBs were observed (1 to 2 mg/kg-day).

In addition to likely impacts to the SMHM from PCBs, the SWEA also indicates lead, selenium, and other metals may impact the SMHM. DTSC disagrees with the characterization of risk in Section 11.4.1.4 and 11.4.1.5 of the SWEA. The total hazard index is greater than one for all HQ estimates. Selenium and lead are significant contributors to the total risk. For lead, HQ3 and HQ4 estimated risks are significantly greater than one, even when compared to background HQs for lead. The SMHM HQ is 3 fold greater than background under average exposure conditions (HQ3), and 23 times greater than background when exposed to the highest concentrations of lead in sediment. Thus, localized areas of lead contamination above background are likely adversely impacting the SMHM.

The high TRV for selenium is based on a study using selenate in drinking water in which there was a significant reduction (about 50%) in the number of offspring compared to controls (Schroeder and Mitchener, 1971). Therefore, levels below the High TRV would still be expected to have adverse effects on reproduction in the SMHM. The Phase II SWEA dismisses the significance of selenium exposure to SMHM by speculating that the selenium at Moffett is in a less toxic form than at Kesterson Reservoir. This statement is made without any site-specific supporting evidence. In any wetland system, the biological cycling and mobilization of selenium from sediment into the foodchain is possible, and is expected to be a major exposure pathway. It has been shown that in aquatic system, that selenium taken up by algae, plants, and zooplankton transform the selenium

into organic-Se compounds (U.S. Fish and Wildlife Service, 1987; Ohlendorf, 1989; Davis, et al, 1988). It has also been shown that the organic forms of selenium are more toxic than the inorganic forms in birds (Heinz, et al., 1987); and that organic-Se accumulates in bird tissue more readily than inorganic Se forms (Heinz, et al., 1988). Similar to these effects in birds, studies on mammals have shown that organic forms of Se can accumulate and are more toxic than inorganic forms. Ferm et al. (1990) showed that organic Se is more toxic than selenite or selenate to hamsters. Pharmacokinetic studies in mammals have shown that organic Se can accumulate in mammalian tissue and be transferred to the neonate via lactation and intrauterine transfer (Hawkes, et al, 1994). Although in mammals the developing fetus may be protected from the reproductive effects of Se due to the ability of the reproductive systems to remove excess Se from the fetus (Hawkes, et al, 1994), maternal toxicity and embryotoxicity can still result in mammals exposed to excess Se (Ferm, et al, 1990). The High TRV dose is associated with significant reproductive toxicity, therefore, we disagree with the Navy's position that the High TRV for selenium is "protective". We also disagree that the selenium is in a less toxic form at Moffett Field, since no evidence other than pure speculation is presented by the Navy. For selenium hazards to the SMHM, all HQ estimates are greater than one, and are from 4 to 8 fold greater than background levels. HQ2, HQ3, and HQ4 are all significantly greater than their corresponding background HQs. For example, HQ3 risk to SMHM for selenium is 4.4 fold greater than the background HQ3 for selenium. HQ4,indicative of localized areas of contamination, is 8.4 times greater than the HQ4 background. Even HQ2 risks (representing localized areas of contamination with toxicity levels expected to cause adverse effects) for the SMHM exposed to selenium exceed one, and are double background levels. DTSC believes the majority of the uncertainty in the hazards estimates for Se come from the use of a literature-derived transfer coefficient from sediment to pickleweed. Therefore, we recommend that studies be conducted during the feasibility study (or post-remedial monitoring) to confirm the TC of selenium from sediment to pickleweed.

In summary, DTSC believes HQ3 and HQ4 risk estimates greater than one are indicative of likely adverse impacts on the SMHM from primarily PCBs and lead (and possibly selenium and other metals). Interpretation of possible effects on the SMHM should emphasize HQ3 and HQ4 estimates due to the small home range and the special status protecting individual animals, and due to the significant adverse impacts using the high TRV (HQ1 and HQ2). Therefore, we disagree with the conclusion stated in Table 11-1b of the SWEA.

Impacts on Avian Receptors

The Navy response and the SWEA claim that the High TRVs for avian receptors are the best estimates for evaluating risk to birds. We disagree, as we noted above. Our specific disagreements with the rationale provided in Chapter 11 of the SWEA for the contaminants contributing most to the hazard estimates are discussed below.

The example of the avian TRVs for DDT in the Navy response (page 16) and in the SWEA (p. 11-27-29) misrepresents the facts. There is no evidence that pelicans are "unusually " sensitive to DDT compounds. The U.S. EPA used the pelican study in their development of criteria for the Great Lakes (U.S. EPA, 1995) because it was the most appropriate peer-reviewed, long-term, field study with a dose-response for reproductive success. The pelican study was used by the U.S. EPA without any modifications for interspecies differences for the kingfisher, gull and eagle. Table 1-6 of U.S. EPA (1995) indicates this pelican study had the longest exposure duration of other species tested, and therefore, one of the lower LOAEL levels. Studies on other bird species cited by USEPA (1995) were conducted for significantly shorter time periods, were unbounded LOAELs, or were based on adult mortality instead of reproductive effects. This does NOT mean the pelican is more sensitive; but rather experimental design was more sensitive (lower dose, longer exposure, more sensitive endpoints) than studies conducted on other species. Finally, a 10 week study on chickens found a lower LOAEL (unbounded) than the longer-term pelican study. Therefore claiming the pelican is unusually sensitive is without factual basis, and cannot be used as justification for choosing the high TRV as the "best estimate of risk". Further, the "low TRV" is only three-fold less than the "high TRV". Since the "high TRV" is a based upon the lowest level at which adverse effects were observed, a three-fold lower value for the "low TRV" is not overly conservative. In short, there is no evidence to indicate the pelican is "unusually sensitive" to DDT compounds, or that the high TRV is a safe level, or that the low TRV is not an appropriate "low risk" value for bird species occupying Moffett Federal Airfield. The SWEA also claims (page 11-29) that no impact is expected on the great blue heron because population declines of this species were not reported at the peak of organochlorine (OC) pesticide usage. First, this statement gives no information on the level of exposure to the great blue heron so that a meaningful conclusion can be drawn about the toxicity of DDT to the heron. Second, the statement misses the point that the great blue heron is used as a species to <u>represent</u> the exposure to a bird species feeding on amphibians, reptiles, fish, birds and mammals (tertiary consumer) in the aquatic habitats of Moffett Field (SWRP, EDM, NC). Other birds occupying this guild in aquatic habitat would include the

Northern Harrier and the loggerhead shrike, as examples. Therefore, the hazards to birds occupying the tertiary consumer trophic level at Moffett Field aquatic habitat cannot be dismissed.

The SWEA (page 11-30) also states that the High TRV for birds exposed to PCBs is emphasized as the appropriate benchmark because the TRV was "...based on studies with chickens, a species that appears to be unusually sensitive to the effects of PCBs.... the mallard appears to be relatively insensitive to the reproductive affects of PCBs". This misrepresents the dataset upon which the TRVs are based. The chicken appears to be one of the most sensitive species tested; the reason is largely because of limitations in the experimental design of the studies conducted on other species (exposure duration and sensitivity of endpoints). This does NOT mean that the chicken is unusually sensitive. Also, the mallard is used as a species to represent the exposure to a bird species feeding on aquatic vegetation at Moffett Field. Other birds occupying this guild are expected to have similar exposure as the mallard, and the TRV is chosen to evaluate the toxicity of the exposure. Finally, the High TRV of 131.5 mg/kg-day approaches the acute dose which is lethal to half the animals (LD50 doses), and levels significantly below the High TRV caused increased mortality in mallard ducklings after only 10 days of exposure to PCBs in combination with a virus. For this reason, the high TRV for birds exposed to PCB has been significantly revised as part of the Regional TRVs after evaluating the studies more thoroughly. The revised High TRV is 1.27 mg/kg-day, or two orders of magnitude lower than the High TRV for Moffett (Draft Regional TRVs, October 10, 1996). Even at 1.27 mg/kg-day, significant effects on reproduction were observed at only 9 weeks exposure in the chicken (hatchability decreased to 1.8% for Aroclor 1248, and 69% for Aroclor 1254 compared to controls of 95%).

Lead exposure to birds is also a significant contributor to hazard. We disagree that the High TRV should be emphasized as the appropriate benchmark in judging the likelihood of impacts to individual birds, because it is not a protective dose. Significant reduction in egg production occurred at the high TRV of 8.75 mg/kg-day in the chicken after 10 weeks of exposure (14 % reduction compared to controls). At doses significantly lower than the High TRV for lead, significant reproductive effects were observed in the quail: a dose of 0.12 mg/kg-day resulted in a 23% drop in egg production, and a dose of 1.1 mg/kg-day of Pb resulted in a 37% drop in egg production compared to controls, after only 5 weeks of exposure (Edens and Garlich, 1983). These results are corroborated in another study in which quail hatchlings dosed for 12 weeks at similar dose levels also had decreased egg production (Edens, et al., 1976).

We also disagree that the hazards to birds from chlordane are unlikely (p. 11-26, 27 and Table 11-1b of SWEA). We believe hazards from chlordane for birds at Moffett Field are unknown, due to the lack of avian TRVs.

In summary DTSC disagrees with Table 11-1b of the SWEA as the likelihood of adverse effect on individual birds and mammals because the analysis is based on the use of HQ1, which uses the High TRVs. We also disagree that adverse effects on populations is unlikely for many of the birds and mammals.

Specific Comments on the RTC

The numbers below correspond to the Navy's responses to DTSC Specific Comments, which start on page 4 of the subject letter.

1. Comment noted. DTSC still recommends post-remedial monitoring in the eastern diked marsh, Lindberg Avenue stormdrain, and Northern Channel to verify remediation of the Lindberg Avenue drain remedial action was successful.

2. <u>Sediment to Pickleweed Transfer Coefficients (TC):</u>

a. DTSC's main point is that the Phase II sampling locations are not representative of the Phase I sampling data. The Navy's response (middle paragraph, page 6) centers on the Aroclor 1260 results, and ignores the complete absence of detected Aroclor 1254 in the sediments of SSRP-33 and -34 (Phase II sampling). This is done in spite of the fact that Tables I-4 and 5-11 (Phase I sampling) indicate Aroclor 1254 predominates in the SWRP and in the areas where SMHM will likely inhabit. A comparison of total PCB concentrations in Table I-4 (SMHM habitat), to Table 5-11 (SWRP habitat), to the collocated samples from Phase II (Table 5-22), reveals that the collocated samples are not representative of the SWRP or the sediments to which the SMHM is exposed. The absence of Aroclors in the SSWRP-34 sample means that ratios between Aroclor data and congener-specific data cannot be obtained. We suggest that the SSRP-34 congener-specific data be used to estimate dioxin-like toxicity of PCBs to the SMHM (in addition to the risk estimates for total PCBs). Please refer to recommendations made in our January 16, 1996 memorandum. The preferred alternative to our recommendations for salvaging the PCB congener data is to resample the SMHM habitat areas during the feasibility study and do both Aroclor and congener-specific PCBs analysis at all sample locations.

b. DTSC believes that there is considerable uncertainty in using transfer coefficients from Travis and Arms because they were developed for a human-food chain system that has not been shown to be applicable to wildlife habitat. The Travis and Arms

regression equations cannot even be considered conservative estimates, since a regression equation would give a best-fit estimate, rather than an upper-bound estimate. The result is a significant gap in the data needed to develop site-specific transfer coefficients for organochlorine pesticides. Many of these compounds bioaccumulate, so transfer to food of the SMHM is possible. Although these pesticides may be detected infrequently in the SWRP as a whole, localized areas of contamination may be impacting individual SMHM. The preferred recommendation is to resample the SMHM habitat areas during the feasibility study (refer to above recommendation for PCBs) so that transfer coefficients for organochlorine compounds can be better estimated. If resampling is not done, we recommend it be included in the post-remedial monitoring.

c. The Navy's response compares the mean concentration of contaminants in the collocated sediment samples to the ranges of contaminant concentrations in the SWRP sediments. DTSC maintains that comparing average to ranges is not a valid comparison. Average concentrations in the collocated samples are significantly less than the average concentrations in SWRP samples for the metals noted in our original comment. Similarly, maximum values detected in the collocated samples are significantly less than maximum concentrations in SWRP sediments. DTSC maintains that the collocated samples are not representative of levels found in the SWRP, and recommends that transfer coefficient determination be included in the feasibility study or the post-remedial monitoring. Because Phase I and Phase II data do not agree, and because TC are not necessarily linear across a concentration gradient, DTSC believes there is a high degree of uncertainty. This uncertainty is best addressed during the feasibility study or post-remedial monitoring.

d. DTSC's comment regarding lipid-normalization of data in our memorandum of December 8, 1995 on the Draft SWEA was referring to evaluating bioaccumulation from transfer coefficient data. This was in response to a Navy statement that bioaccumulation was not occurring. The Draft SWEA upon which our comment was based did not contain any calculations for estimating dose, and the food ingestion rate calculations were incorrect. Therefore, the manner in which the Navy intended to perform the dose calculations, specifically the estimation of contaminants in the diet based upon transfer coefficients, was not indicated in the Draft SWEA.

The data presented in Table I-49b, indicate that the sediment to pickleweed transfer coefficient that should be used with the dry weight food ingestion rate is 3.8. We arrived at this value by dividing the pickleweed wet weight concentrations of 2.7 ug/kg total PCBs by 0.06 (the dry weight fraction assumed

for pickleweed in Table I-49a) to derive a total PCB pickleweed concentration of 45 ug/kg dry weight. The transfer coefficient we determined by dividing the total PCB pickleweed concentration (dw basis) by the total PCB sediment concentration (dw basis). Using a sediment total PCB concentration of 11.9 ug/kg from Table I-49b, the transfer coefficient on a dry weight basis is 3.8 (45 divided by 11.9). This dry weight TC for total PCBs is 4.4 times greater than that calculated using a TOC and lipid normalized TC (3.8 divided by 0.862 = 4.4). To DTSC, this is a significant difference in transfer coefficients. Since dietary sources are the predominant contributors to the HQs for the SMHM, this will translate into approximately 4 fold greater hazard quotient (HQ) estimates than is represented in the Draft Final SWEA. We consider this a substantial difference.

The use of a TOC and lipid normalized transfer coefficient with a food ingestion rate that is based upon dry weight is not correct. DTSC maintains that the transfer coefficient for total PCBs used in the Draft Final SWEA is not correct. The use of this incorrect TC will reduce the dose estimates which will result in HQs that are not truly reflective of the risk.

3. The Navy's response states that it is "likely that the majority of sediments in the SWRP are nondetect for Aroclor 1254 based on this low frequency of detection" (11.7% frequency of detection). We disagree that the available data demonstrates that PCBs are infrequently detected in the SWRP because very high detection limits for many of the analytes will bias the frequency of detection. DTSC has repeatedly commented on this problem throughout the Phase I SWEA.

Using the data presented in Table I-47b (Aroclor 1260) and I-47c (PCB congeners) for calculating the sediment to polychaete transfer coefficients on a dry weight basis, we obtain TCs of 2.4 and 0.09, respectively. These are similar to TCs obtained using the incorrect TCs (TOC/lipid-normalized basis). This is coincidental in that the TOC/lipid normalized factor of 0.20 is equal to the dry weight fraction for polychaetes. Therefore, while DTSC agrees that the HQ estimates for birds are adequate, we maintain that the calculation is not correct and should be corrected in the Final SWEA.

4. No response is necessary.

5. DTSC disagrees that assuming a transfer coefficient equal to one for PAHs and metals is likely conservative. Similarly, use of Travis and Arms equations for transfer coefficients have not been shown to be conservative for upland or aquatic habitats. The results of the SWEA indicate that food-chain pathways are dominant pathways for many metals. We continue to recommend that

the risk characterization uncertainty sections discuss more explicitly how the various methods of calculating transfer coefficients impacts the risk characterization.

6. No response is necessary.

7. DTSC agrees that the "...exposure point concentrations, dose estimates, and hazard quotients for DDTR may be potentially underestimated by as much as 21%" due to omission of o,p'-DDTR isomers from the sample analysis. DTSC however disagrees that it is likely the underestimate of DDTR (total of all isomers of DDT, DDD, DDE) is lower than 21%. The Navy response contends that the p,p'-isomer ratio to the o,p' isomer ratio in the environment will be less than that of the technical formulation. The ATSDR (1994, p. 35) indicates the p,p'-isomers account for approximately 85% of the total DDTR, which is approximately the same percentage of p,p' to o,p'-isomers in the technical grade DDT. ATSDR (1994, p. 92) cites a U.S. National Soils Monitoring Program on the overall pattern of DDTR that also indicates the proportion of p,p' to o,p'-isomers is approximately that of the technical grade DDT. Until site-specific information is available, no conclusions can be drawn regarding the proportion of p,p' isomers to o,p'-isomers.

The Navy also contends that o,p'-isomers are less toxic than p,p'-isomers. We disagree that o,p'-isomers are less toxic. ATSDR (1994, p. 59, 60) states, "p,p'-DDT as well as o,p'-DDT can have adverse effects on reproduction after short term or longer term exposure of rats...". Further, regarding developmental effects, "These effects reflect the estrogenic activity of o,p'-DDT and o,p'-DDD on the developing reproductive system. These effects were not observed with p,p'-DDT, o,p-DDT, or p,p'-DDE or o,p'-DDE." It is true that at much higher acute lethal doses of DDT compounds, that the p,p'-DDT is more toxic than o,p'-DDT (Smith, 1991, p. 746). However, in this evaluation, we are primarily concerned with chronic exposures to lower levels of DDTR, and particularly concerned with reproductive and developmental effects.

Finally, all of the TRVs are based upon reproductive effects (low and high TRVs; bird and mammal) using total DDTR or technical grade DDT, which contains both o,p' and p,p'-isomers. We recommend the proposed change to the SWEA simply state the hazard quotients for DDTR are likely underestimated due to analysis of only the p,p'-isomers. DTSC recommends that risk-based remediation goals be developed for total DDTR, including the o,p'-isomers, and that verification sampling methodology include all isomers of DDTR.

8. No response is necessary.

9. No response is necessary.

10. Burrowing owl exposure to VOCs in the subsurface: There appears to be a about 4 to 5 orders of magnitude difference in concentrations of VOCs measured in the 1988 soil-gas survey results around Site 5 (depth approximately 5 feet bgs), and the monitoring of the burrows conducted for the Phase II SWEA (it is unclear from the RTC at what depth bgs VOC sampling was conducted, or whether sampling 2 feet inside the burrow opening is at the terminus of the burrow). From the information presented in the SWEA, no remediation of the subsurface has been conducted around Site 5. The question then becomes, what happened to the high VOCs in the subsurface? Are the results of the burrow samples representative? Are VOCs in the subsurface moving away from Site 5, and if so, in what direction? Because the discrepancy between the measured values is so great, DTSC recommends continued monitoring of the soil-gas around the animal burrows before conclusions can be drawn concerning potential ecological impacts on the burrowing owl. We continue to recommend that Professor Lynne Trulio evaluate her data on burrowing owls at Moffett Federal Airfield against the available contamination data (including soil-gas survey, burrow sampling data, and areas with surface soil contamination). Refer to our original recommendation in our letter of August 23, 1996. Appendix F, Table F-7 - "Chemicals detected in the Upland Soil Vapor" does not indicate that TCE or it's breakdown products were evaluated.

11. DTSC disagrees that the high TRVs are the best point estimates of risk. Refer to our comments above under the heading "Use and Interpretation of the Toxicity Reference Values (TRVs)". Also, in our above comments regarding impacts on the SMHM and avian receptors, DTSC has indicated how toxicological data used to derive specific contaminant High TRVs are being misinterpreted by the Navy.

The Navy's example of TRVs for DDT and mercury, which are based directly upon the U.S. EPA (1995) document on the Great Lakes without modification, are two exceptions to the approach taken by the workgroup in developing TRV s for Moffett Federal Airfield. In general, the workgroup developed the High TRVs to be levels at which significant adverse effects on growth, reproductive success, or survival are expected to occur. As we have noted above in our comments, the "high TRV" are levels at which significant effects are expected. Essentially, the Navy is choosing only levels above the "high TRV" (i.e., HQ 1 and HQ2) as being indicative of adverse effects. We do not support the use of "high TRV" values as the best point estimates of risk in the manner in which the Navy is using them in the SWEA.

Response to Recommendations

1. The Navy misinterprets our memorandum of January 16, 1996, and misrepresents the facts surrounding evaluation of TRVs for PCBs. The January 16, 1996 memorandum was in response to a lengthy discussion in a conference call held on December 7, 1995, in which DTSC disagreed that the toxicity data evaluated for several different mixtures and specific congeners adequately evaluated the dioxin-like PCBs. PRC staff requested that DTSC outline our recommendations, which we did in our memorandum of January 16, 1996, which was transmitted to the Navy. At that point, it was the Navy's responsibility to formally disagree with DTSC recommendations by raising the issue with the TRV workgroup. This was not done.

The Navy's response cites a Metcalfe and Haffner (1995) reference that suggests insufficient information is available to develop appropriate avian TEFs for risk assessment. However, in the same paper, those authors cite numerous examples in the literature, including application to avian species, where avian TEFs were used. These authors further state that all the various studies using TEFs concluded that "...coplanar PCBs were responsible for more than 90% of dioxin-like toxicity".

DTSC acknowledges the limited PCB congener-specific data available at Moffett Federal Airfield. At the working meetings held to develop the Phase II SWEA work, DTSC wanted all the collocated samples to be analyzed for both PCB congener analysis and Aroclor analysis, but the Navy believed this to be too costly. The Navy wanted only one sample of each media to have PCB congener analysis done. If the Navy now believes these data are too limited, then additional collocated samples can be collected by the Navy prior to finalizing this SWEA. Otherwise, it is DTSC's position that these are the only data currently available, and they should be used to calculate dioxin-like PCB toxicity as described in our January 16, 1996 memorandum.

It is our recommendation (stated in our January 16, 1996 memorandum) to evaluate PCB toxicity using <u>both</u> the total PCB TRV and also dioxin-like PCBs using the approach outlined for the congener-specific data and avian and mammalian TEFs. The approach recommended by DTSC for evaluating dioxin-like PCB toxicity, in addition to the evaluation of the total PCB toxicity already evaluated in the SWEA (with modifications noted above), will provide a fuller description of potential hazards to wildlife at Moffett Federal Airfield. This will aid the risk managers in evaluating possible remedial actions. Due to the selective retention of PCB congeners by biota, and because certain PCB congeners are potent dioxin-like mimics, reliance on the Aroclor data alone would provide a misleading representation

of the potential PCB hazards to wildlife. DTSC believes the TEF calculations are a useful screening tool to determine whether more extensive investigation, such as measuring EROD induction in field-collected specimens, are necessary. If the Navy chooses not to do these screening TEF calculations for PCB dioxin-like toxicity, then we strongly recommend that EROD-induction in field collected specimens be included in the post-remedial monitoring.

2. and 3. No response is necessary.

4. Refer to our comments above on the burrowing owl evaluation.

5. No response is necessary.

6. No response is necessary.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1994. *Toxicological Profile for 4,4'-DDT, 4,4'-DDE, 4,4'-DDD (Update), TP-93/05*. U.S. Department of Health and Human Services, Public Health Service. Atlanta, Georgia.

Davis, E.A., K.J. Maier, A.W. Knight. 1988. *The biological consequences of selenium in aquatic ecosystems*. California Agricultural. January-February 1988 pp. 18-29.

Edens, P.W., and J.D. Garlich. 1983. *Lead-Induced Egg Production Decrease in the Leghorn and Japanese Quail Hens*. Poultry Science, Vol. 62, pp. 1757-1763.

Edens, F.W., E. Benton, S.J. Bursian, G.W. Morgan. 1976. *Effect of Dietary Lead on Reproductive Performance in Japanese Quail, Coturnix coturnix japonica*. Tox. and Applied Pharmacology, 38:307-314.

Smith, A.G. 1991. *Chapter 15 Chlorinated Hydrocarbon Insecticides*. In: Handbook of Pesticide Toxicology, Volume 2 Classes of Pesticides, Eds., Hayes, W.J. and E.R. Laws. Academic Press, Inc. San Diego, California.

Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, D.M. G. Weller. 1987. *Reproduction in Mallards fed Selenium*. Environ. Toxicol. and Chem. , Vol. 6, pp. 423-433.

Heinz, G.H., D.J. Hoffman, and L.G. Gold. 1988. *Toxicity of Organic and Inorganic Selenium to Mallard Ducklings*. Arch. Environ. Contam. Toxicol. Vol. 17, pp. 561-568.

Lemly, D.A. 1987. *Aquatic Cycling of Selenium: Implications for Fish and Wildlife*. United States Department of the Interior, Fish and Wildlife Service, Fish and Wildlife Leaflet 12. Washington, D.C.

Metcalfe, C.D. and G.D. Haffner. 1995. *The ecotoxicology of coplanar polychlorinated biphenyls*. Environ. Rev. ,3: 171-190.

Ohlendorf, H.M. 1989. *Bioaccumulation and Effects of Selenium in Wildlife*. In: Selenium in Agriculture and the Environment, Soil Science Society of America and American Society of Agronomy Special Publication No. 23, pp. 133-177. Madison, Wisconsin.

Schroeder, H.A., and M. Mitchener. 1971. *Toxic Effects of Trace Elements on the reproduction of Mice and Rats*. Arch. Environ. Health, Vol. 23, pp 102-106.

United States Environmental Protection Agency (U.S. EPA) 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife, DDT, Mercury, 1,2,7,8-TCDD, PCBs*. Office of Water, Washington, D.C. EPA-820-B-95-008.

U.S. Fish and Wildlife Service. 1987. *Aquatic Cycling of Selenium: Implications for Fish and Wildlife*. United States Department of the Interior, Fish and Wildlife Service, Fish and Wildlife Leaflet 12. Washington, D.C.