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From: Christine Rowe <crwhnc@gmail.com>
Sent: Thursday, June 02, 2016 2:25 AM
To: WB-RB4-losangeles
Cc: Ali, Mazhar@Waterboards; Owens, Cassandra@Waterboards
Subject: BOEING SANTA SUSANA FIELD LABORATORY NPDES NO. CA 0001309, CI-6027, HHRA Work Plan Comments - June 2, 2016
Attachments: Boeing Human Health Risk Assessment Comments from Christine L Rowe.pdf

Dear Mr. Ali and Ms. Owens,

Thank you for allowing me to submit these comments late. I know that they should have been in by yesterday at the latest. But this is a complex topic, and I am only completing my review of them now. I do hope that you will still accept them as on time so that you have to respond to them.

Respectfully submitted,

Chris Rowe

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RE: BOEING SANTA SUSANA FIELD LABORATORY NPDES NO. CA 0001309, CI-6027, HHRA Work Plan
Comments

Dear Ms. Owens,

Thank you for the opportunity to comment on the WaterBoard's ordered Human Health Risk Assessment (HHRA) Work Plan for the storm water runoff from the Santa Susana Field Laboratory (SSFL) site.

This is my tenth year as a technical stakeholder for the SSFL site. I want to thank the Water Board and the Boeing Expert Storm Water Panel for making my first SSFL site visit possible – I believe that was in around 2008? Since that time, I am guessing that I have been to the site between 40 to fifty times including for meetings of the Water Board, and tours with the Boeing Expert Storm Water Panel as well as with Water Board staff.

I am familiar with the various grades of the SSFL site. I have been to Outfall 8, and I have been to the drainages of Outfall 9. I have been to Outfalls 3 and 4 with the Federal EPA and their consultants when they were performing their radiological survey of AREA IV. I have also been to the Silvernale Pond. And I have been to various groundwater sampling locations with the Groundwater Expert Panel, so I am familiar with the Groundwater Extraction Treatment System (GETS). I have not been to the more remote locations where there are samples taken during rain events.

General Comments

I believe that I read a document many years ago that was a sampling report by the Water Board from 1947. This was probably (my assumption) just to test the groundwater for its properties so that the owners / operators would know what was in the water that they would be using for various purposes.

1. Can you please find that sampling report, or any other sampling reports from that time frame so that we can determine what was sampled for before the site was active?

2. When did the Water Board begin sampling the water from the SSFL site for chemicals of concern and radionuclides?
3. Some people believe that cleaning the site to Background or the Minimal Detectible Concentrations found in a lab will restore the SSFL site to their pre North American Aviation history. What reports do we have from back then? Since this was previously a farm, it is my assumption that there would have been more bacteria, there would have been pesticides and herbicides, chemicals from atmospheric deposition from cars and other sources as well as fallout radionuclides?
4. Was this sampling by the Waterboard for contaminants only after the Clean Water Act was expanded 1972? (1)
5. Is it possible to see the first year's annual report or the first report that would have been used by Boeing's predecessors to show what was sampled for leaving the SSFL site?
6. Why is no risk assessment being required for outfalls 3-7 and 10 when there is tremendous concern regarding what runs off to the Brandeis Bardin camp and to Runkle Canyon?
7. Is the WaterBoard considering the contaminants that the Federal EPA found within and beneath these outfalls?
8. How do we distinguish the Dioxins at the SSFL site made by anthropogenic activities v from the natural fires that have occurred here over the decades?
9. How do we know if the lead at the site is from natural occurring sources v anthropogenic uses v atmospheric deposition?
10. How much of the lead at the site is from the shooting ranges such as the one that has been remediated over the years in the Northern Drainage?
11. Several years ago, in comments I believe on Boeing's fines, I recommended that their fines be used to sample the unlined drainages beyond the outfalls and before the lined channel of the Los Angeles River was reached. I wanted to understand if the Chemicals of Potential Concern (COPC) are falling out in the sediment of the creek offsite, or if they are making their way to the lined channel of the Los Angeles River. Have the various creek's leaving the site sediment been sampled so that if there is a rain event, we would know what is there that can be caught in a rain event?
12. Has the Water Board staff looked at the most recent Background Studies for chemicals (DTSC) and Radionuclides (Federal EPA)? A report that compares chemicals and radionuclides found in background and their ranges in background soils would greatly serve to identify what are naturally occurring COPCs v COPCs which are only anthropogenic.
13. Your HHRA does not discuss a dust pathway. Around 1989, I was sent a letter that stated that I was in the prevailing winds area of the SSFL site. As you know, when they dig the soil for remediation, it is supposed to be watered down. But I have still seen dust blowing in the parking lot area that tells me that sediment is blowing.
14. Is this HHRA only for people at the SSFL site or in its drainages, or is it supposed to reflect the dust that may blow from the SSFL site when the drainages are dry? Do we have any way to measure the dust pathway?
15. On page 5, it states that soil remediation will begin in 2017. The WaterBoard has required cleanup measures for I believe, at least two decades?

16. According to the 2007 Consent Order and the 2010 Administrative Orders on Consent the soil remediation is supposed to be COMPLETE by 2017.
17. From page 6 – Do Boeing and NASA have documented soil volumes to show how much soil has been removed under the Water Board’s Orders for ISRA and for the Imminent and Substantial Endangerment Order by DTSC around 2009?
18. Wouldn’t it be important to know when these soil volumes were removed from each outfall area so that the COPCs that were of concern in the past no longer pose a danger today?
19. From page 6, am I correct in understanding that Outfall 19 has been in place and has been used for discharges of GETS treated water, but Outfall 20 is yet to be constructed?
20. Isn’t it a violation of State Water Policy to take clean water and recontaminate it by sending it down Bell Creek which has not been remediated and to the Los Angeles River where it will also become further contaminated?
21. Doesn’t it make more sense to keep the remediated water on site for use for dust mitigation, revegetation, and for ponds for animal life to get water?
22. Page 8 – I thought that the EPA was constantly updating their guidelines. Why are we using guidelines from 1989?
23. Page 10 – Potential Exposure Routes – I am again concerned that VOCs are referenced for inhalation, but that dust is not referenced for a potential inhalation pathway.
24. At a meeting with Bell Canyon residents with Water Board staff, these residents stated that they used to fish in Bell Creek. Again, I am concerned about whether the creek has been sampled from the SSFL site to this area where there used to be enough water for fishing and potentially for swimming. Can we get the creek sediment sampled?
25. You discuss the wading exposure, but if water is deep enough to wade in, then children are very likely to fall in and splash in it. This scenario needs to be considered for the Bell Canyon area.
26. Page 12 – Exposure Point Concentrations (EPC) – Does the EPC risk imply an estimate based upon future risk after remediation or past risks from 2009 forward?
27. As I look at these EPCs, this method would require you to go to every quarterly monitoring report since 2009, and to determine what COPCs were found at each outfall and at what levels to combine them to develop a health risk for each outfall at each time there was a rain event and there was water in the drainages?
28. In reference to the discussion on EPCs, it is not made clear that a onetime acute exposure may be different from chronic exposures to COPCs which is how the cancer 1 per million is calculated – exposure over time. However, there are acute doses, for example for radionuclides – both in the industry and in medical applications – that do increase the lifetime risk of cancer. See the DOE Ionizing Radiation Dose Ranges Chart.(2) On this chart, it states under Cancer Epidemiology that there is: “Evidence for small lifetime increases in human cancer above 10 rem acute exposure, 20 rem chronic exposure.” An example of 1.5- 10 rem is a Spiral CT scan – full body.(2)
29. Page 13 – Are Preliminary Remediation Goals (PRGs) the only screening criteria? I thought that PRGs were not clean up values but rather just a tool to use for sampling purposes? (3)

Preliminary Remediation Goals (PRGs)

Initial clean-up goals that (1) are protective of human health and the environment and (2) comply with ARARs. They are developed early in the process based on readily available information and are modified to reflect results of the baseline risk assessment. They also are used during analysis of remedial alternatives in the remedial investigation/feasibility study (RI/FS).

(30) You are screening water for risk. How can we compare the surface water PRGs with the PRGs for soil Background Values found by the EPA for Radionuclides and DTSC for chemicals?

(31) Which screening values for PRGs are you using when the end use is open space / parkland?

(32) How do you compare PRGs to MCLs?

(33) Page 13 – Would it be possible to hold a meeting with Water Board staff, the Boeing Expert Storm Water Panel, OEHHA staff, and the US EPA to explain the various methods for Toxicity Assessment at the level that the average stakeholder will understand?

Ideally this location would be at Corporate Pointe in West Hills which is centrally located for most stakeholders.

(34) Page 13 – #6 – Why is the Health Effects Assessment Summary Table from 1997 – isn't that old?

(35) Page 13 - #7 – Why are we using a “National Center for Environmental Assessment (NCEA) document from 2004 – isn't there an updated document for this?

(36) Page 15 – References to other documents not incorporated in this document from Geosyntec such as the SRAM requires the reader to be familiar with each of these documents and to be able to comprehend documents that are highly technical in nature. I do not believe that the majority of the SSFL stakeholders have this technical expertise to read these documents let alone to interpret them unless they have a science background in risk and remediation.

(37) Page 15 – Paragraph 2 – At which screening level are you considering – Backyard garden, Suburban residential, open space?

(38) Page 15 – Paragraph 3 – Does this mean that our drinking water in California is blended to a 1 in 10,000 risk range? Are these screening levels based on an adult male's exposure?

(39) Page 16 – American Cancer Society risk of cancer – I believe this is old data. I think it also requires clarification.

“In the US, the lifetime risk of developing cancer is 42% (1 in 2) in men and 38% (1 in 3) in women (Table 6, page 14). These probabilities are estimated based on the overall experience of the general population and may overestimate or underestimate individual risk because of differences in exposures (e.g., smoking), family history, and/or genetic susceptibility.”(4)

Furthermore, it is important to note in the 2016 American Cancer Society document:

- 1) That California has the highest number of expected cases of cancer in 2016 – this is estimated at 173,200 new cases.

- 2) The number one cause of cancer in California is female breast cancer with an estimated 26,730 new cases expected in 2016. However, the expected mortality in female breast cancer is only 4,400 cases which shows that this cancer if caught by screening can be treated.

“Risk factors: Potentially modifiable factors associated with increased breast cancer risk include weight gain after the age of 18 and/or being overweight or obese (for postmenopausal breast cancer), use of menopausal hormone therapy (combined estrogen and progestin), physical inactivity, and alcohol consumption. In addition, recent research indicates that long-term, heavy smoking may also increase breast cancer risk, particularly among women who start smoking before their first pregnancy. The International Agency for Research on Cancer has concluded that shift work, particularly at night (i.e., that disrupts sleep patterns), may be associated with an increased risk of breast cancer.

Non-modifiable factors associated with increased breast cancer risk include older age; a personal or family history of breast or ovarian cancer; inherited mutations (genetic alterations) in *BRCA1*, *BRCA2*, or other breast cancer susceptibility genes; certain benign breast conditions (such as atypical hyperplasia); a history of ductal or lobular carcinoma in situ; high-dose radiation to the chest at a young age (e.g., for cancer treatment); high breast tissue density (the amount of glandular tissue relative to fatty tissue measured on a mammogram); high bone mineral density (evaluated during screening for osteoporosis); and type 2 diabetes (independent of obesity). Reproductive factors that increase risk include a long menstrual history (menstrual periods that start early and/or end later in life), recent use of oral contraceptives, never having children, having one’s first child after age 30, and high natural levels of sex hormones.”⁽⁴⁾

These factors are important because based on the California Cancer Registry data, there is elevated incidence of breast cancer in Eastern Ventura County and Western Los Angeles County. However, based on the above risk factors, I believe that breast cancer should not be correlated with storm water runoff.

While in the Morgenstern study on Cancer Incidence in the Community Surrounding the Rocketdyne Facility in Southern California (2007), he found a correlation between ionizing radiation and female breast cancer, how would people exposed to storm water be exposed to ionizing radiation in doses that would cause breast cancer? (5)

In the 2014 PowerPoint called “Cancer Occurrence in Offsite Neighborhoods Near the Santa Susana Field Laboratory “ by Dr. Thomas Mack of USC at the DTSC Open House, Dr. Mack showed the plotting of breast cancer incidence around the SSFL site. In each of three maps for various time periods, the plottings did not show a correlation (in my opinion) with the SSFL site.

Please see Dr. Mack’s presentation regarding cancer incidence around the SSFL site as the most recent epidemiological study done for this area.

From Morgenstern 2007:

“The main methodologic limitation of this study is the absence of data—either environmental or individual-level—for measuring exposures to ionizing radiation or toxic

chemicals. Distance from SSFL is a very crude proxy that does not take into consideration the fate and transport of hazardous substances migrating offsite, local geological and meteorological conditions, and the behavior of residents that would affect their levels of exposure. It might be possible to generate better indicators of environmental exposures by applying the models of Cohen et al. (2006) for predicting geographic-specific exposure concentrations; but this approach would probably not allow us to separate the effects of different exposures (due to collinear relations), and it still has major limitations for the study of cancers that have long induction and latent periods (from first exposure of individuals to disease detection). The main problem is substantial population mobility before and during the follow-up period, especially in the Hispanic population. It is likely that some new cancers detected in the vicinity of SSFL between 1988 and 2002 occurred among residents who did not live in that area very long and therefore could not have been exposed to offsite contaminants; conversely, some new cancers detected in the reference region (>5 miles from SSFL) might have previously lived in the region closest to SSFL; and it is likely that some persons potentially exposed before 2002 may have moved away from the two-county area so that subsequent cancer occurrences would not be identified in this study.

Another methodologic limitation is the lack of information on potential confounders, i.e., other cancer risk factors that are associated with exposure status in the population (Rothman & Greenland, 1998). We were able to control only for the potentially confounding effects of age, gender, and race/ethnicity. It is possible that differences in cancer rates between the three regions were partly due to the effects of other cancer risk factors, such as cigarette smoking for lung, bladder, and upper-aerodigestive-tract cancers (Thun & Henley, 2006), air pollution for lung, bladder, and childhood cancers (Samet & Cohen, 2006), diet for colon, breast, and prostate cancers (Willett, 2006), and socioeconomic status and various occupational exposures for several cancers (Kawachi & Kroenke, 2006; Siemiatycki et al., 2006). Unfortunately, the only effective method of controlling for the effects of these variables involves measuring them accurately in all members of the two-county study population or in random samples of all geographic groups.

Conclusions

Despite the methodologic limitations discussed above, our findings suggest there may be elevated incidence rates of certain cancers near SSFL that have been linked in previous studies with hazardous substances used at Rocketdyne, some of which have been observed or projected to exist offsite. Since there are several alternative explanations for our findings, including chance and bias, it is tempting to recommend extending our study to include additional information on environmental exposures and potential confounders and the use of more sophisticated Bayesian methods of statistical analysis (Elliott et al., 2000; Banerjee et al., 2004). It is not clear, however, if this ecologic approach will yield more informative and less biased results. Even if average levels of environmental exposures and covariates are measured accurately for small areas such as census block groups, the distributions of those variables will be heterogeneous within groups and their joint distributions within groups will be missing. Therefore, estimates of exposure effects on cancer incidence may be severely distorted by ecologic bias; moreover, controlling for confounders could increase bias (Morgenstern, 1998). In addition, if only small proportions of the groups were exposed to any SSFL-related hazard, estimation of that exposure effect would be made even more difficult.

An alternative approach for learning more about environmental risk factors for cancers in the communities near SSFL is to conduct an observational study at the individual level, e.g., a cohort or case-control study. Unfortunately, this approach would be costly, and it would still be subject to problems of exposure measurement, population mobility, and relatively small numbers

of exposed residents.” (5)

Cancer of the lung and bronchus are expected to be the number two cause of cancers in California in 2016 with 18,140 expected cases. (4) There will be 12,230 expected deaths in California in 2016 from lung and bronchus.(4)

Cancers of the colon and rectum are expected to be the third highest in incidence in California in 2016 with an expected incidence of 13,770 cases. (4) The expected number of deaths from this disease in 2016 is 5,180 cases. (4)

So while Morgenstern studied cancers of the breast, lung and bronchus, and the colon and rectum in relation to the SSFL site, it should be noted, in my opinion, that these three cancers are the highest in incidence state wide. (5)

“The results from this study suggest little or no association between residential distance from SSFL and the incidence of total cancers or the group of (radiosensitive) malignancies thought to be affected by ionizing radiation. There was, however, a weak inverse association during both follow-up periods between distance from SSFL and the group of (chemosensitive) malignancies thought to be affected by exposure to chemicals used at Rocketdyne and found or projected by others to exist offsite (Cohen et al., 2006; SSFL Advisory Panel, 2006).”(5)

Page 17 – Risk Characterization

40) Cancer risk is correlated based upon exposure to the chemicals or radionuclides over time to the best of my understanding except as where I referenced earlier acute toxicity such as in high levels of radiation exposure or high doses of some chemical.

“EPA classifies all radionuclides as Group A carcinogens. The Radionuclide Table on this website, formerly HEAST Table 4, lists ingestion, inhalation and external exposure cancer slope factors (risk coefficients for total cancer morbidity) for radionuclides in conventional units of picocuries (pCi). (7) Ingestion and inhalation slope factors are central estimates in a linear model of the age-averaged, lifetime attributable radiation cancer incidence (fatal and nonfatal cancer) risk per unit of activity inhaled or ingested, expressed as risk/pCi. External exposure slope factors are central estimates of lifetime attributable radiation cancer incidence risk for each year of exposure to external radiation from photon-emitting radionuclides distributed uniformly in a thick layer of soil, and are expressed as risk/yr per pCi/gram soil. When combined with site-specific media concentration data and appropriate exposure assumptions (8), slope factors can be used to estimate lifetime cancer risks to members of the general population due to radionuclide exposures.”(6)

“For linear carcinogens, EPA's current process of estimating cancer risk is based on the unit risk estimate (URE) for inhalation, and the carcinogenic potency slope (CPS) for ingestion. The URE represents the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent over a lifetime at a concentration of 1 µg/m³ in air. The interpretation of the URE would

be as follows: if the URE = $1.5 \times 10^{-6} \mu\text{g}/\text{m}^3$, no more than 1.5 excess tumors are expected to develop per 1,000,000 people if exposed all day, every day for a lifetime to a concentration of 1 μg of the chemical per cubic meter of air. The CPS is an upper bound, usually approximating a 95% confidence limit, on the increased cancer risk from a lifetime oral exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg/day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than 1 in 100. The URE and CPS are plausible upper-bound estimates of the risk (i.e., the actual risk is likely to be lower, but may be greater). However, because the URE and CPS reflect unquantifiable assumptions about effects at low doses, their upper bounds are not true statistical confidence limits. The tabulated UREs and CPSs were developed by EPA and the California EPA, and were selected for use by a priority system.”(7)

- (41) Many of the COPCs could have been caused by natural fires that have burned through the SSFL site over the decades of site history. Many local residents who lived in the Chatsworth area to the SSFL and beyond could have been exposed to the ash from the 2005 fire which burned 70 – 80 % of the SSFL site and to other fires. How do we know which of the contaminants were the result of the 2005 fire?
- (42) Can we please see the list of the chemicals that were found at each outfall prior to the 2005 fire and after the 2005 fire to potentially identify COPCs that may be the result of a new burn?
- (43) I was at NASA SSFL both prior to the ISRA remediation in an oak grove with both NASA and WaterBoard personnel. I have before and after photos of these trees that clearly show evidence of being burned in an area that was known to have Dioxins. While I do recognize that Dioxins are a potential carcinogen, why is the SSFL site required to remove Dioxins to MCLs or risk based levels when the adjacent properties such as Sage Ranch and Ahmanson Ranch would not be subject to this cleanup?
- (44) After remediation to the levels necessary to comply with Boeing’s NPDES permit, what happens if the COPCs from other properties which are elevated above the SSFL site drain onto the SSFL site?

Table 1: Number of Samples for Constituents Detected in NPDES Permit Monitoring Surface Water Samples Santa Susana Field Laboratory

- (45) These lists of COPCs by Outfall number do not tell the reader in what year they were found, at what level over the MCL that they were found, and therefore, we do not know when they were found in place and time.
- (46) For a more accurate document to show risk, you would need to show how many rain periods there were per year that were sampled, what the number of exceedances were at each outfall and which COPCs were exceedances at each outfall. We should probably know the number of inches of rainfall during each event to understand if this water is just being absorbed into the soil, or if there was a large enough volume to run off of the site.

(47) We do know that the number of rain events has decreased over the last five years, and that as a result of remediation and Best Management Practices (BMPs) (hopefully) under ISRA, that the COPCs have reduced in numbers and in their levels of toxicity.

Respectfully submitted,

Christine L. Rowe

West Hills' resident of 38 years

- (1) Clean Water Act: <https://www.epa.gov/laws-regulations/summary-clean-water-act>
- (2) Ionizing Radiation Dose Ranges:
https://www.remm.nlm.gov/DOE_PosterShowingRadiationDoses_Part1.pdf
- (3) Risk Assessment Guidance for Superfund Volume 1:
https://www.epa.gov/sites/production/files/2015-09/documents/contents_0.pdf
- (4) Cancer Facts and Figures 2016:
<http://www.cancer.org/acs/groups/content/@research/documents/document/acspc-047079.pdf>
- (5) Cancer Incidence in the Community Surrounding the Rocketdyne Facility in Southern California (Morgenstern):
http://www.etec.energy.gov/environmental_and_health/Documents/CancerStudies/Final_Epi_Report.pdf
- (6) USER'S GUIDE: RADIONUCLIDE CARCINOGENICITY:
https://www.epa.gov/sites/production/files/2015-02/documents/heat_ug_0401.pdf
- (7) Risk Assessment for Carcinogens: <https://www.epa.gov/fera/risk-assessment-carcinogens>
- (8) Learn about Dioxin: <https://www.epa.gov/dioxin/learn-about-dioxin>

