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From:	Tabor, Cynthia L
To:	Delistraty, Damon; Julie Robertson; Lyon, Jeffery; Barnes, Michael; Caggiano, Joseph; Rochette, Beth; Skorska, Maria; Whalen, Cheryl (Washington Department of Ecology)
Cc:	Bergeron, Marcel P; Aly, Alaa H; Singleton, Kristin M; Julie Robertson; Mahmudur Rahman (mrahman@intera.com)
Subject:	RE: Next Set of WMA RFI Comments
Date:	Monday, February 22, 2016 2:58:05 PM
Attachments:	Responses_To_2nd_set_Damon"s_Comments_02182016.pdf Report_Comment_19.docx Report_Comment_20.docx

Hi Damon
I am attaching some additional information regarding your below input – in order to continue discussion tomorrow.

Thank you
Cindy

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CONTRACTOR TO THE UNITED STATES DEPARTMENT OF ENERGY

From: Delistraty, Damon A. (ECY) [mailto:DDEL461@ECY.WA.GOV]
Sent: Thursday, February 18, 2016 8:54 AM
To: Tabor, Cynthia L; 'Julie Robertson'; Lyon, Jeffery; Barnes, Michael (ECY); Caggiano, Joseph; Rochette, Beth; Skorska, Maria; Whalen, Cheryl (Washington Department of Ecology)
Subject: RE: Next Set of WMA RFI Comments

Hi Cindy,

Here's my response to USDOE updated responses to my initial comments on the RFI (RPP-RPT-58339, Rev A Draft) and BRA (RPP-RPT-58329, Rev 0) that you have selected in this email.

The following comments are OK: Damon BRA 17 (dermal contact), Damon BRA 40, Damon BRA 57, Damon RFI 8 (dermal contact), Damon RFI 15, and Damon RFI 31.

The following comments relate to the "administrative fragmentation" issue (i.e., related info located in multiple reports): Damon BRA 1, Damon BRA 13, Damon BRA 17 (groundwater ingestion), Damon BRA 43, and Damon RFI 8 (groundwater ingestion). This approach decreases transparency (especially when no clear roadmap is provided) and may ignore cumulative risk across pathways within an exposure scenario.

The following comments need discussion:

Damon BRA 14, Damon BRA 16, Damon RFI 11

There is extensive precedent with Hanford risk assessments for evaluating both rad and nonrad COPCs via foodchain exposure (e.g., ingestion of plants, meat, milk, fish) for resident, farmer, fisher, and tribal receptors. USDOE's Hanford Site Risk Assessment Methodology [HSRAM] (DOE/RL-91-45, Rev 3) recommends evaluating these pathways. The following Hanford reports serve as examples, where foodchain exposure for both rad and nonrad COPCs is estimated:

1) Screening Assessment and Requirements for a Comprehensive Assessment/Columbia River Comprehensive Impact Assessment [CRCIA] (DOE/RL-96-16, Rev 1)
2) Waste Treatment Plant [WTP]/Risk Assessment Work Plan [RAWP] (24590-WTP-RPT-ENS-03-006, Rev 3)
3) Exposure Scenarios and Unit Factors for Hanford Tank Waste Performance Assessments (HNF-SD-WM-TI-707, Rev 5)
4) River Corridor Baseline Risk Assessment [RCBRA] (DOE/RL-2007-21, Rev 0).

Examples of sources of transfer factors for nonrads are USDOE's RESRAD (metals) and EPA's Human Health Risk Assessment Protocol [HHRAP] for Hazardous Waste Combustion Facilities (organics). Perhaps other useful references on transfer factors (found in RCBRA Appendix D1) are Baes et al (1984), Wang et al (1993), and Kennedy and Strenge (1992). Uncertainty due to omitting this pathway is arguably greater than uncertainty in modeling this pathway.

Damon RFI 8

RFI Figure 7-3 (Human CSM) should be the same as updated BRA Figure 3-1 (Human CSM).

Damon RFI 19

For the CERCLA Residential Child, Table 7-8 shows nonrad ELCR>1E-5 (EA C and J), although below background ELCR (<5E-5). With the exception of EA F+G (HI=0.6), noncancer HI>1 for the CERCLA Residential Child for all other EAs (Table 7-8), although below background HI (<3). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).

Damon RFI 20

For the MTCA Method B resident, Table 7-9 shows ELCR>1E-5 (EA C), although equal to background ELCR (3E-5). Also, HI<1 (EA F+G) for the MTCA resident (Table 7-9). However, HI>1 at all other EAs (Table 7-9) but below background HI (2.3), with the exception of HI at EA C (HI=2.4). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).

Damon RFI 45

Except for EA C for the MTCA Method B resident (Table 7-9) and EA C and J for the CERCLA residential child (Table 7-8), nonrad ELCR<1E-5 for other EAs for MTCA and CERCLA residential exposure scenarios. Except for EA F+G for the MTCA Method B resident (Table 7-9), EA F+G for the CERCLA residential child (Table 7-8), and all EAs for the CERCLA residential adult (Table 7-8), noncancer HI>1 for other EAs for MTCA and CERCLA residential exposure scenarios. However, only HI at EA C for the MTCA Method B resident was above background (Table 7-9). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).

Damon

From: Tabor, Cynthia L [mailto:Cynthia_L_Tabor@rl.gov]
Sent: Wednesday, February 03, 2016 6:52 AM
To: 'Julie Robertson' <JulieRobertson@gofreestone.com>; Beach, Ryan E <Ryan_E_Beach@orp.doe.gov>; Johnson, Jeremy M <Jeremy_M_Johnson@orp.doe.gov>; Lyon, Jeffery (ECY) <JLYO461@ECY.WA.GOV>; Barnes, Michael (ECY) <miba461@ECY.WA.GOV>; Caggiano, Joseph (ECY) <Jcag461@ECY.WA.GOV>; Rochette, Beth (ECY) <Broc461@ECY.WA.GOV>; Delistraty, Damon A. (ECY) <DDEL461@ECY.WA.GOV>; Skorska, Maria (ECY) <msko461@ECY.WA.GOV>; Faulk, Dennis (EPA) <faulk.dennis@epa.gov>; Gerhart, Rebecca <Gerhart.rebecca@epa.gov>; Rutland, Paul L <Paul_L_Rutland@rl.gov>; Parker, Dan L (Danny) <Danny_L_Parker@rl.gov>; Radloff, Anna W <Anna_W_Radloff@rl.gov>; Robertson, Julie R <Julie_R_Robertson@rl.gov>; Bergeron, Marcel P <Marcel_P_Bergeron@rl.gov>; Singleton, Kristin M <Kristin_M_Singleton@rl.gov>; Aly, Alaa H <Alaa_H_Aly@rl.gov>; Mahmudur Rahman <MRahman@intera.com>; Wiegman, Rebecca S <IMCEAEX-_O=HANFORD_OU=HANFORD_cn=Recipients_cn=H5559735@rl.gov>; Hopkins, Andrea M <Andrea_M_Hopkins@rl.gov>
Subject: Next Set of WMA RFI Comments

Hi All
Here are the next set of comment responses for review. They are all Damon comments and are associated with the BRA or the Groundwater Screening document.

The attached files has responses for:
BRA Damon: 1, 13, 14, 16, 17, 40, 43, and 57
RFI Damon: 8, 11, 15, 19, 20, 31, and 45

In our last meeting on January 21, we discussed RFI Damon 46 and 47, which relate to the Groundwater Screening document. We provided the below response and Beth R indicated that comments associated with this document should remain open. We would like to note that RFI Damon 33, 34, 35, 38, 39, 40, 41, and 42 are also in this category. Note that RFI Damon 36 and 37 are also associated with the groundwater discussion - however these comments were not technical but rather editorial. We are considering these closed.

Appreciate everyone's support in these reviews. Damon (or anyone else) - please let us know if you have problems with the attached responses. Thank you very much Cindy

"Discussion on Groundwater Screening Report: RPP-RPT-58297, *Screening-Level Evaluation of Groundwater Monitoring Data Collected in Vicinity of WMA C*, was developed to support the WMA C Phase 2 RFI because the 200-BP-5 and 200-PO-1 Remedial Investigation (RI) reports had not been completed (i.e., WMA C Phase 2 RFI provided to Ecology 12/14 and RIs provided to Ecology 8/15). The BP-5 and PO-1 RI reports, which contain groundwater risk assessment information and identify those constituents from WMA C impacting groundwater, are now available, and this information will be summarized in the revised WMA C Phase 2 RFI. The screening report, which was developed to provide necessary groundwater information, will not be updated since the BP-5 and PO-1 RI reports will be used to support the revised WMA C Phase 2 RFI.

Note that the information from this report was additionally used in various sections of the WMA Phase 2 RFI (e.g. Section 5, 6, and 7). Comments on this referenced information, contained in the WMA C Phase RFI, will be discussed in subsequent comment response meetings. It is anticipated that a majority of these comments will be resolved by indicating that the revised WMA C Phase 2 RFI will summarize information from BP-5 and PO-1, as appropriate."

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Comment From (ECY)	Item	Comment (s) (Provide technical justification for the comment and detailed recommendation of the action required to correct/resolve the discrepancy/ problem indicated.)	Doc	Updated Response based on Damon Email 02_18_16
Damon	Damon BRA 14, Damon BRA 16, Damon RFI 11	There is extensive precedent with Hanford risk assessments for evaluating both rad and nonrad COPCs via foodchain exposure (e.g., ingestion of plants, meat, milk, fish) for resident, farmer, fisher, and tribal receptors. USDOE's Hanford Site Risk Assessment Methodology [HSRAM] (DOE/RL-91-45, Rev 3) recommends evaluating these pathways. The following Hanford reports serve as examples, where foodchain exposure for both rad and nonrad COPCs is estimated: 1) Screening Assessment and Requirements for a Comprehensive Assessment/Columbia River Comprehensive Impact Assessment [CRCIA] (DOE/RL-96-16, Rev 1) 2) Waste Treatment Plant [WTP]/Risk Assessment Work Plan [RAWP] (24590-WTP-RPT-ENS-03-006, Rev 3) 3) Exposure Scenarios and Unit Factors for Hanford Tank Waste Performance Assessments (HNF-SD-WM-TI-707, Rev 5) 4) River Corridor Baseline Risk Assessment [RCBRA] (DOE/RL-2007-21, Rev 0). Examples of sources of transfer factors for nonrads are USDOE's RESRAD (metals) and EPA's Human Health Risk Assessment Protocol [HHRAP] for Hazardous Waste Combustion Facilities (organics). Perhaps other useful references on transfer factors (found in RCBRA Appendix D1) are Baes et al (1984), Wang et al (1993), and Kennedy and Strenge (1992). Uncertainty due to omitting this pathway is arguably greater than uncertainty in modeling this pathway.		Concur. Foodchain pathways were considered during chemical risk assessment for subsistence farmer, various recreational and tribal receptor scenarios at Hanford Sites. However, each of those BRA reports identified the calculations associated with the exposure concentrations in foods, particularly garden produce as a major source of uncertainty for the risk assessment. A linear plant uptake model was applied to soil concentrations during the calculation of concentration in foods. Uncertainty in produce concentrations is attributable to intrinsic variability related to soil conditions, plant species and tissue type, harvest time, and other environmental variables. None of those factors were considered during the calculation of concentration of chemicals in food. In addition, almost all of the nonradiological COPCs at WMA C are metals. Few VOC COPCs are present. Baes et al (1984), Wang et al (1993), Kennedy and Strenge (1992) and RESRAD model presented transer factors for radionuclides. RESRAD model includes transfor factors for produce, milk and beef for few metals. However, those transfer factors for produce are based on 20% assumption of wet to dry ratio. Due to these uncertainties associated with the transfer factors, food chain pathways were not considered during the chemical risk assessment. By obmitting the foodchain pathways, the chemical risk assessment assessment underestimated the total risk . Text will be added in the in the uncertainty assessment to address this issue.
Damon	Damon RFI 8	RFI Figure 7-3 (Human CSM) should be the same as updated BRA Figure 3-1 (Human CSM).		Concur. Figure 3-1 will be updated as updated BRA Figure 3-1.

Damon	Damon RFI 19	For the CERCLA Residential Child, Table 7-8 shows nonrad ELCR>1E-5 (EA C and J), although below background ELCR (<5E-5). With the exception of EA F+G (HI=0.6), noncancer HI>1 for the CERCLA Residential Child for all other EAs (Table 7-8), although below background HI (<3). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).		Based on the Ecology's suggested response for Comment No 18, the following changes were made for risk characterization process. 1. Based on 40 CFR Part 300, the ELCRs below 1E-6 are considered acceptable risks whereas ELCRs above 1E-4 are considered unacceptable risks for CERCLA receptors. Risks between 1E-4 to 1E-6 are generally referred to as the "acceptable risk range." 2. For noncancer hazard, the EPA acceptable target HI is 1. An HI above 1 is considered unacceptable risk. The HI may exceed 1 even if all of the individual HQs are less than 1. In this case, the chemicals may be segregated by similar mechanisms of toxicity and toxicological effects. Separate HIs may then be derived based on mechanism and effect. In addition, no background risk evaluation was performed during the risk characterization process. Due to those changes, following changes were observed. Text will be updated to incorporate such changes into the appropriate section. For the CERCLA Residential Child, Table 7-8 shows that the total ELCR for each EA is within CERCLA acceptable risk range of 1E-6 to 1E-4 and is less than CERCLA unacceptable risk level of 1E-4. Therefore, no risk contributor was identified. Table 7-8 also shows that with the exception of EA F+G, the HIs for all EAs are greater than the acceptable target HI of 1. Aluminum, antimony, arsenic, cadmium, chromium, cobalt, iron, lithium, manganese, and vanadium were identified as hazard contributors. Therefore, an evaluation was performed for each EA to segregate the HIs associated with those hazard contributors by similar mechanisms of action (critical effect) and toxicological effects. When the HI based on similar mechanism of action is greater than 1, those hazard contributors will be retained. The results of the risk evaluations are presented in the following attachment- "Report_Comment_19). However, the results of risk evaluation showed that the HI based on similar mechanism of action is less than one. Therefore, no COPCs were retained as hazard contributors.

Damon	Damon RFI 20	For the MTCA Method B resident, Table 7-9 shows ELCR>1E-5 (EA C), although equal to background ELCR (3E-5). Also, HI<1 (EA F+G) for the MTCA resident (Table 7-9). However, HI>1 at all other EAs (Table 7-9) but below background HI (2.3), with the exception of HI at EA C (HI=2.4). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).		As mentioned in response to comment no 19, background evaluation was eliminated from the risk characterization. Therefore, text will be updated to include the following changes. For carcinogenic COPCs, the cumulative ELCR at EA C is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173 340 708(5)]) cumulative risk threshold of 1E-5. EA C reports a cumulative ELCR of 3E-5; the primary contributor to risk is arsenic (3E-5; 100 percent contribution). For noncarcinogenic COPCs, all EAs report an HI greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173 340 708(5)]) target HI of 1. Aluminum, antimony, arsenic, cadmium, chromium, cobalt, iron, lithium, manganese, and vanadium were identified as hazard contributors. Therefore, an evaluation was performed for each EA to segregate the HIs associated with those hazard contributors by similar mechanisms of action (critical effect). When the HI based on similar mechanism of action is greater than 1, those hazard contributors will be retained. The results of the risk evaluations are presented in the following attachment- "Report_Comment_20). Based on the results, no COPCs were retained as hazard contributors.
Damon	Damon RFI 45	Except for EA C for the MTCA Method B resident (Table 7-9) and EA C and J for the CERCLA residential child (Table 7-8), nonrad ELCR<1E-5 for other EAs for MTCA and CERCLA residential exposure scenarios. Except for EA F+G for the MTCA Method B resident (Table 7-9), EA F+G for the CERCLA residential child (Table 7-8), and all EAs for the CERCLA residential adult (Table 7-8), noncancer HI>1 for other EAs for MTCA and CERCLA residential exposure scenarios. However, only HI at EA C for the MTCA Method B resident was above background (Table 7-9). Note, however, comparison of EA vs background (for ELCR and HI) is apparently being eliminated (see Damon RFI 15).		Please see responses to Damon RFI Comment Response 19 and Damon RFI Comment Response 20.

Risk Characterization Results Related to HI for CERCLA Residential Child

(Table 7-8)

For noncarcinogenic COPCs, all EAs report an HI greater than the target HI of 1. Aluminum, antimony, arsenic, cadmium, chromium, cobalt, iron, lithium, manganese, and vanadium were identified as hazard contributors. Therefore, an evaluation was performed for each EA to segregate the HIs associated with those hazard contributors by similar mechanisms of action (critical effect) and toxicological effects. When the HI based on similar mechanism of action is greater than 1, those hazard contributors will be retained. The mechanisms of action (critical effect) for each of the hazard contributors are as follows:

- Aluminum – neurological effects
- Antimony - longevity, blood glucose, and cholesterol
- Arsenic – hyperpigmentation, keratosis, and possible vascular complications
- Cadmium - significant proteinuria
- Chromium – nasal septum atrophy
- Cobalt –blood effects
- Iron – gastrointestinal (GI) tract effects
- Lithium - nervous system and kidney effects
- Manganese – central nervous system effects
- Vanadium – decreased hair cysteine

The results of the evaluations for each EA are summarized in more detailed in the following section.

EA A plus B - The HI for EA A plus B is 1.4 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.097; 6.8 percent contribution), arsenic (HQ = 0.17; 12 percent contribution), chromium (HQ = 0.041; 2.9 percent contribution), cobalt (HQ = 0.39; 27 percent contribution), iron (HQ=0.44; 31 percent contribution), lithium (HQ = 0.053; 3.8 percent contribution), manganese (HQ = 0.032; 2.3 percent contribution), and vanadium (HQ = 0.15; 11 percent contribution).

Among all hazard contributors for EA A plus B, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.18, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA A plus B.

EA C - The HI for EA C is 2.5 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.12; 4.8 percent contribution), arsenic (HQ = 0.98; 39 percent contribution), chromium (HQ = 0.055; 2.2 percent contribution), cobalt (HQ = 0.47; 19 percent contribution), iron (HQ=0.52; 21 percent contribution), lithium (HQ = 0.077; 3.1 percent contribution), manganese (HQ = 0.041; 1.6 percent contribution), and vanadium (HQ = 0.20; 8.0 percent contribution).

Among all hazard contributors for EA C, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.24, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI

is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA C.

EA E - The HI for EA E is 1.6 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.095; 6.1 percent contribution), arsenic (HQ = 0.24; 15 percent contribution), cadmium (HQ = 0.046; 3.0 percent contribution), chromium (HQ = 0.073; 4.7 percent contribution), cobalt (HQ = 0.38; 24 percent contribution), iron (HQ = 0.44; 28 percent contribution), lithium (HQ = 0.058; 3.7 percent contribution), manganese (HQ = 0.045; 2.9 percent contribution), and vanadium (HQ = 0.15; 9.6 percent contribution).

Among all hazard contributors for EA E, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.2, which is less than the target HI of 1. In addition, exposure to cadmium and lithium result in kidney effects, summing the hazard quotients for these two analytes results in a HQ of 0.10, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA E.

EA H plus I - The HI for EA H plus I is 1.6 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.090; 5.6 percent contribution), arsenic (HQ = 0.23; 14 percent contribution), chromium (HQ = 0.072; 4.5 percent contribution), cobalt (HQ = 0.43; 27 percent contribution), iron (HQ = 0.48; 30 percent contribution), lithium (HQ = 0.048; 3.0 percent contribution), manganese (HQ = 0.037; 2.3 percent contribution), and vanadium (HQ = 0.17; 11 percent contribution).

Among all hazard contributors for EA H plus I, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.18, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA H plus I.

EA J - The HI for EA J is 1.7 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.099; 6.0 percent contribution), antimony (HQ = 0.069; 4.2 percent contribution), arsenic (HQ = 0.36; 22 percent contribution), chromium (HQ = 0.049; 3.0 percent contribution),cobalt (HQ = 0.35; 21 percent contribution), iron (HQ = 0.44; 27 percent contribution), lithium (HQ = 0.055; 3.4 percent contribution), manganese (HQ = 0.035; 2.1 percent contribution), and vanadium (HQ = 0.14; 8.4 percent contribution).

Among all hazard contributors for EA J, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.19, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA J.

EA L1 plus L2 - The HI for EA L1 plus L2 is 1.3 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.082; 6.3 percent contribution), arsenic (HQ = 0.17; 13 percent contribution), chromium (HQ = 0.041; 3.2 percent contribution), cobalt (HQ = 0.35; 27 percent contribution), iron (HQ

= 0.38; 30 percent contribution), lithium (HQ = 0.045; 3.5 percent contribution), manganese (HQ = 0.033; 2.5 percent contribution), and vanadium (HQ = 0.15; 12 percent contribution).

Among all hazard contributors for EA L1 plus L2, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.16, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA L1 plus L2.

EA P - The HI for EA P is 1.6 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.091; 5.8 percent contribution), arsenic (HQ = 0.23; 15 percent contribution), chromium (HQ = 0.053; 3.4 percent contribution), cobalt (HQ = 0.39; 25 percent contribution), iron (HQ = 0.46; 30 percent contribution), lithium (HQ = 0.053; 3.4 percent contribution), manganese (HQ = 0.033; 2.1 percent contribution),and vanadium (HQ = 0.20; 13 percent contribution).

Among all hazard contributors for EA P, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.18, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA P.

EA R - The HI for EA R is 1.7 which is greater than the target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.11; 6.4 percent contribution), arsenic (HQ = 0.27; 16 percent contribution), cadmium (HQ = 0.049; 2.9 percent contribution), chromium (HQ = 0.048; 2.8 percent contribution), cobalt (HQ = 0.45; 27 percent contribution), iron (HQ = 0.47; 28 percent contribution), lithium (HQ = 0.058; 3.4 percent contribution), manganese (HQ = 0.037; 2.2 percent contribution), and vanadium (HQ = 0.16; 9.7 percent contribution).

Among all hazard contributors for EA E, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.2, which is less than the target HI of 1. In addition, exposure to cadmium and lithium result in kidney effects, summing the hazard quotients for these two analytes results in a HQ of 0.11, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA R.

EA U - The HI for EA U is 1.5 which is greater than target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.11; 6.9 percent contribution), arsenic (HQ = 0.32; 21 percent contribution), chromium (HQ = 0.043; 2.8 percent contribution), cobalt (HQ = 0.37; 25 percent contribution), iron (HQ = 0.42; 28 percent contribution), lithium (HQ = 0.059; 3.9 percent contribution), manganese (HQ = 0.033; 2.2 percent contribution), and vanadium (HQ = 0.13; 8.7 percent contribution).

Among all hazard contributors for EA U, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.2, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Even though, their combined HI

is greater than 1, the HQ for each hazard contributor is less than 1. Therefore, no hazard contributors were retained for EA R.

Risk Characterization Results Related to HI for MTCA Method B Receptor related to Unrestricted Uses

(Table 7-9)

For noncarcinogenic COPCs, all EAs report an HI greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. Aluminum, antimony, arsenic, cadmium, chromium, cobalt, iron, lithium, manganese, and vanadium were identified as hazard contributors. Therefore, an evaluation was performed for each EA to segregate the HIs associated with those hazard contributors by similar mechanisms of action (critical effect). When the HI based on similar mechanism of action is greater than 1, those hazard contributors will be retained. The mechanisms of action (critical effect) for each of the hazard contributors are as follows:

- Aluminum – neurological effects
- Antimony - longevity, blood glucose, and cholesterol
- Arsenic – hyperpigmentation, keratosis, and possible vascular complications
- Cadmium - significant proteinuria
- Chromium – nasal septum atrophy
- Cobalt –blood effects
- Iron – gastrointestinal (GI) tract effects
- Lithium - nervous system and kidney effects
- Manganese – central nervous system effects
- Vanadium – decreased hair cysteine

The results of the evaluations for each EA are summarized in more detailed in the following sections.

1 Exposure Area A plus B

The HI for EA A plus B is 1.4 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.094; 7 percent contribution), arsenic (HQ = 0.16; 11 percent contribution), chromium (HQ = 0.04; 3 percent contribution), cobalt (HQ = 0.38; 28 percent contribution), iron (HQ=0.43; 31 percent contribution), lithium (HQ = 0.05; 4 percent contribution), manganese (HQ = 0.03; 2 percent contribution), and vanadium (HQ = 0.15; 11 percent contribution).

Among all hazard contributors for EA A plus B, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.18, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA A plus B.

2 Exposure Area C

The HI for EA C is 2.4 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.12; 5 percent contribution), arsenic (HQ = 0.88; 37 percent contribution), chromium (HQ = 0.053; 2 percent contribution), cobalt (HQ = 0.46; 19 percent contribution), iron (HQ=0.51; 21 percent contribution),

lithium (HQ = 0.076; 3 percent contribution), manganese (HQ = 0.04; 2 percent contribution), and vanadium (HQ = 0.2; 8.0 percent contribution).

Among all hazard contributors for EA C, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.23, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA C.

3 Exposure Area E

The HI for EA E is 1.5 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.093; 6 percent contribution), arsenic (HQ = 0.22; 14 percent contribution), cadmium (HQ = 0.041; 3 percent contribution), chromium (HQ = 0.072; 5 percent contribution), cobalt (HQ = 0.37; 24 percent contribution), iron (HQ = 0.43; 29 percent contribution), lithium (HQ = 0.057; 4 percent contribution), manganese (HQ = 0.044; 3 percent contribution), and vanadium (HQ = 0.15; 10 percent contribution).

Among all hazard contributors for EA E, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.19, which is less than the target HI of 1. In addition, exposure to cadmium and lithium result in kidney effects, summing the hazard quotients for these two analytes results in a HQ of 0.10, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA E.

4 Exposure Area F plus G

The HI for EA F plus G is 1.3 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.085; 6 percent contribution), arsenic (HQ = 0.15; 11 percent contribution), chromium (HQ = 0.037; 3 percent contribution), cobalt (HQ = 0.34; 26 percent contribution), iron (HQ = 0.43; 32 percent contribution), lithium (HQ = 0.046; 3 percent contribution), manganese (HQ = 0.035; 3 percent contribution), and vanadium (HQ = 0.17; 13 percent contribution).

Among all hazard contributors for EA F plus G, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.17, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA F plus G.

5 Exposure Area H plus I

The HI for EA H plus I is 1.6 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.088; 6 percent contribution), arsenic (HQ = 0.21; 14 percent contribution), chromium (HQ = 0.07; 5 percent contribution), cobalt (HQ = 0.42; 27 percent contribution), iron (HQ = 0.47; 30 percent contribution), lithium (HQ = 0.047; 3.0 percent contribution), manganese (HQ = 0.037; 2 percent contribution), and vanadium (HQ = 0.17; 11 percent contribution).

Among all hazard contributors for EA H plus I, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.17, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA H plus I.

6 Exposure Area J

The HI for EA J is 1.6 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.097; 6.0 percent contribution), antimony (HQ = 0.068; 4 percent contribution), arsenic (HQ = 0.33; 21 percent contribution), chromium (HQ = 0.048; 3.0 percent contribution),cobalt (HQ = 0.34; 22 percent contribution), iron (HQ = 0.43; 27 percent contribution), lithium (HQ = 0.054; 3 percent contribution), manganese (HQ = 0.034; 2 percent contribution), and vanadium (HQ = 0.14; 9 percent contribution).

Among all hazard contributors for EA J, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.19, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA J.

7 Exposure Area L1 plus L2

The HI for EA L1 plus L2 is 1.3 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.08; 6 percent contribution), arsenic (HQ = 0.15; 12 percent contribution), chromium (HQ = 0.04; 3 percent contribution), cobalt (HQ = 0.34; 27 percent contribution), iron (HQ = 0.38; 30 percent contribution), lithium (HQ = 0.044; 4 percent contribution), manganese (HQ = 0.032; 3 percent contribution), and vanadium (HQ = 0.15; 12 percent contribution).

Among all hazard contributors for EA L1 plus L2, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.16, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA L1 plus L2.

8 Exposure Area P

The HI for EA P is 1.5 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.089; 6 percent contribution), arsenic (HQ = 0.21; 14 percent contribution), chromium (HQ = 0.052; 3 percent contribution), cobalt (HQ = 0.38; 25 percent contribution), iron (HQ = 0.45; 30 percent contribution), lithium (HQ = 0.052; 3 percent contribution), manganese (HQ = 0.033; 2 percent contribution),and vanadium (HQ = 0.19; 13 percent contribution).

Among all hazard contributors for EA P, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.17, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such

it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA P.

9 Exposure Area R

The HI for EA R is 1.6 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.10; 6 percent contribution), arsenic (HQ = 0.24; 15 percent contribution), cadmium (HQ = 0.043; 3 percent contribution), chromium (HQ = 0.047; 3 percent contribution), cobalt (HQ = 0.44; 27 percent contribution), iron (HQ = 0.46; 28 percent contribution), lithium (HQ = 0.057; 3 percent contribution), manganese (HQ = 0.037; 2 percent contribution), and vanadium (HQ = 0.16; 10 percent contribution).

Among all hazard contributors for EA R, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes result in a HQ of 0.2, which is less than the target HI of 1. In addition, exposure to cadmium and lithium result in kidney effects, summing the hazard quotients for these two analytes results in a HQ of 0.08, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA R.

10 EA U

The HI for EA U is 1.5 which is greater than the 2007 MTCA ("Human Health Risk Assessment Procedures" [WAC 173-340-708(5)]) target HI of 1. The primary contributors to noncancer HI (those analytes that contribute greater than 1 percent of the total HI) are aluminum (HQ = 0.10; 7 percent contribution), arsenic (HQ = 0.29; 20 percent contribution), chromium (HQ = 0.042; 3 percent contribution), cobalt (HQ = 0.36; 25 percent contribution), iron (HQ = 0.41; 28 percent contribution), lithium (HQ = 0.058; 4 percent contribution), manganese (HQ = 0.033; 2 percent contribution), and vanadium (HQ = 0.13; 9 percent contribution).

Among all hazard contributors for EA U, exposure to aluminum, lithium, and manganese result in neurological effects, summing the hazard quotients for these three analytes results in a HQ of 0.19, which is less than the target HI of 1. Other hazard contributors for this EA result in a different critical effect, as such it is appropriate to segregate the contributions of each hazard contributor. Therefore, no hazard contributors were retained for EA U.