Prepared for:

ATK Launch Systems
Brigham City
Utah

Open Burn Open Detonation Human Health Risk Assessment

ATK Launch Systems Promontory, Utah

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HUMAN HEALTH RISK ASSESSMENT
FOR OPEN BURNING OPEN DETONATION
AT THE PROMONTORY BURNING GROUNDS,
PROMONTORY, UTAH,

EXECUTIVE SUMMARY

Open Burn Open Detonation Human Health Risk Assessment for Promontory

ATK Launch Systems (ATK), located 30 miles west of Brigham City, Utah, currently operates open burning (OB) and open detonation (OD) units for the treatment and disposal of waste propellants and propellant-contaminated materials. The location of the Promontory Facility is shown on Figure ES-1. The units are subject to Resource Conservation and Recovery Act (RCRA), 40 CFR 264, Subpart X permitting requirements for miscellaneous treatment units, and this human health risk assessment (HHRA) is prepared under a Utah Department of Environmental Quality, Division of Solid and Hazardous Waste (Utah DSHW) approved protocol in support of a new RCRA Subpart X permit application.

This HHRA follows US Protection Agency (EPA) Human Health Risk Assessment Protocol Guidance for Incineration (HHRAP) (EPA, 2005a) as coded into a software program by Lakes Environmental of Waterloo, Canada (Lakes, 2014). This software program has been validated and approved for use at incineration facilities by the EPA, and incorporates key receptors, exposure assumptions, and methods of hazard and risk calculation. Further, the Lakes software was updated with November 2014 dose-response information and 2014a EPA default exposure assumptions to make it current. The risk assessment process is iterative; meaning, the process will make overly conservative assumptions to calculate risk, if the risks are unacceptable, the assumptions are examined to determine if they are appropriate. If the assumptions are deemed inappropriate then they are often revised to be more realistic.

The HHRA process uses the amounts and types of wastes processed, coupled with a conservative measure of the emissions of chemicals of potential concern (COPCs) from these wastes to provide inputs to air dispersion modeling, conducted by CB&I using a Utah DSHW-approved protocol (CB&I, 2014). Modeling produced short-term and long-term ambient air concentrations, and COPC deposition rates that provide the basis for media COPC concentrations. These concentrations are used to calculate hazards and risks for the assumed receptors.

This Executive Summary provides the results of the HHRA, and shows that all long-term cancer risks are less than one in one million, and hazards are less than one. These acceptable risk levels are provided by the Utah Administrative Code R-315-101-6, and are consistent with the US EPA point of departure acceptable risk levels.

Each area of the risk assessment is discussed below.

Characterizing Facility Emissions and Calculating Emission Rates

There are two emissions sources at ATK Promontory: M-136, and M-225, and two activities are undertaken: open burn (OB) and open detonation (OD). OB treatment is considered a quasi-continuous source because the treatment event is usually complete within one hour or less. OD is considered an instantaneous source because treatment is completed within milliseconds. A description of the sources, and the amount and types of materials processed is provided in Section 4 (Emission Sources and Parameters) of the CB&I modeling protocol. The total amount of material processed by ATK is 10,065,000 pounds per year (distributed amongst all sources in different proportions), and this amount was used to characterize the facility emissions, hazards and risks.

To determine emissions factors, ATK conducted emissions tests by preparing bundles of 100 percent (%) 1.3-Class ammonium perchlorate propellant (AP), 85% AP and 15 % trash, and 65% AP and 35% trash, placed the bundles on stainless steel trays and ignited the bundles with nickel-chromium wire. The emissions were captured, analyzed and the amount captured was used to give COPC emissions as pounds of waste per pound of waste. Based on the DSHW protocol, the highest emissions rate from each test was used to develop a conservative emissions rate for each COPC. Where no COPC was detected, the highest detection limit was used. For polynuclear aromatic compounds (PAH) emission rates were developed from a combination of 1.3-Class AP and 1.1-Class AP.

Emissions rates were approved by the Utah DSHW, and are provided for over 200 COPCs.

Selection of COPC

The HHRAP guidance recommends selecting the COPCs from the stack test data, which are unavailable for this OB/OD facility. The HHRAP allows for the elimination of compounds that are not processed or generated by a facility, narrowing lists of compounds quantitatively evaluated to capture the risks associated with the facility. However, this risk assessment eliminates few COPCs, and evaluates both chemicals that are detected, and those that are not detected. The selection process resulted in over 200 COPCs that are evaluated quantitatively in the risk assessment.

Approach to Air Quality Modeling

Air dispersion modeling for long-term risk assessment calculations conservatively assume that sources M-136 and M-225 are burned together in quantities that add up to the permitted annual quantity. These assumptions violate ATK's operational safety protocols that only allow one location to operate at one time, and will overestimate the emissions for short-term exposures, and therefore the short-term hazards. Short-term evaluations are initially conducted using this assumption to comply with the Utah DSHW-approved modeling protocol. For short-term exposures, the results from the model were modified to calculate air COPC concentrations

assuming only the source with the highest predicted air concentrations operates at one time to provide realistic short-term air concentrations and therefore more realistic hazards.

CB&I conducted the air quality modeling for the Promontory treatment units in August 2014. The model used was developed by them based on some initial air quality modeling conducted by TetraTech, and combined an OB/OD Model (OBODM) with the Industrial Source Complex (ISC-3) American Meteorological Society Gaussian air dispersion modeling (AERMOD) to produce an improved OBODM/AERMOD hybrid model that is believed to more accurately predict air chemical transport and dispersion for this type of facility. The output files from this model became the source files for the Lakes HHRA software for both short-term and long-term exposure hazard and risk calculations.

The model provides short-term ambient air concentration data. The highest air concentrations for the National Ambient Air Quality Standard (NAAQS) compounds from one source are used to evaluate ATK's air concentrations compared with the NAAQSs, which are not exceeded by ATK. These concentrations were also compared with Utah Toxic Screening Levels (TSLs), and these were not exceeded.

Approach to Risk Assessment

The modeling protocol assumes processing occurs at all of the burning grounds simultaneously; however, limiting simultaneous burns to M-136: A1, A2 and A3, and M-225 A or a different combination of burn locations would limit exposure to COIs and comply with ATK's operational protocols. Therefore, the HHRA includes the simultaneous burn scenarios M-136 A (A1, A2 and A3) with M-225 A, because this is consistent with ATK's typical operating protocol. While these protocols are important to maintain the health and safety of ATK's employees, they are revised from time to time, and appending them to the permit might require a permit modification when small operational changes are made. This is a cumbersome and ineffective method of operating.

It has been developed throughout the Air Dispersion Modeling Protocol, Air Dispersion Modeling Report, HHRA Protocol, and the HHRA Report that the burn scenarios for M-136 and M-225 are alternative and mutually exclusive. Meaning that any one of the M-136 scenarios A (A1, A2, A3), B, or C could occur once a day, and conversely that any one of the M-225 scenarios A or B could also occur once a day. ATK anticipates the permit conditions will be based on the combinations of burn scenarios in the HHRA, and future ERA, and those would include any one of the M 136 scenarios (A, B or C) and any one of the M-225 scenarios (A or B). For example, M-136 B and M-225 B might occur on the same day.

Chromium Speciation

Chromium (Cr) was measured as total Cr, and was not speciated into the two valent forms of Cr: hexavalent Cr (Cr(VI) and trivalent Cr (Cr(III)). In accordance with recommendations provided

in the HHRAP Guidance and considering data available from other types of facilities, or Cr sources in incineration, hazards and risks were estimated assuming 45% Cr (VI) and 55% Cr (III). Section 10.1.3 contains the details of these percentages, as well as a discussion of the relative risks assuming a range of 14% to 100% Cr (VI).

Acute (1-hour) Non-cancer Hazard Indices

In addition, short-term (1-hour) ambient air concentrations are taken from the model and compared with acceptable short-term concentrations provided by the California EPA (Cal EPA), where available, or by the Department of Defense (DOD) if not. Where no value is available from these sources, a surrogate is used. The Utah DSHW approved the short-term values, and the values were selected in accordance with the hierarchy recommended in the EPA's 2005a HHRAP Guidance Section 7.4.2 Our Recommended Hierarchical Approach. The ratio of the air concentration to the acceptable concentration is an index that should be one or less. It is called the acute hazard quotient. When added together, the sum is called the acute Hazard Index.

The acute Hazard Index exceeds the target of one for the three on-site receptors (Workers), three of the boundary/off-site hypothetical receptors, and one of the six off-site residential and farming receptors, assuming all sources are burned together. This assumption is not realistic, but it was modeled to account for the total annual permitted poundage. The results are provided in Tables ES-1, and ES-2 and ES-3, below. Nickel is the COPC that contributes most of the risk. Based on the emissions data, nickel is believed to be a test artifact contributed from the testing pans and ignition wire.

ATK's operational protocols include burning only sources M-136 A1, A2, A3 and M-225 A simultaneously. This is a more realistic scenario because it is what actually occurs at ATK. The acute Hazard Indices (HIs) calculated using only sources M-136 A1, A2, A3, and M-225 A are shown in Tables ES-1, ES-2, and ES-3. These tables also include an adjustment to the hazards associated with chromium and nickel. Under these assumptions, the HIs are all less than one. The details of the adjustment factors for chromium and nickel are presented in Sections 10.1.3 and 10.1.3, respectively.

As discussed in Section 10.1.3, Chromium and Nickel in Waste, chromium and nickel were found in the OBOD test emissions. The level of emissions from the test appears to be high relative the estimated amount of chromium and nickel in the material being processed. This high level of chromium and nickel prompted ATK to analyze their waste streams for the presence of these two analytes. AP propellant contains 16% aluminum, and based on a recent analysis of the aluminum in AP fuel (ATK, 2015a), the aluminum contains approximately 3.1 parts per million (ppm or mg/kg) chromium and 29.1 mg/kg nickel. Using these concentrations, the hazards associated with chromium and nickel were adjusted down to account for the aluminum as the source. Full details and the adjusted hazard estimates are provided in Section 10 and Appendix F.

To summarize, Tables ES-1, ES-2 and ES-3 present acute hazard estimates for the following scenarios:

- Simultaneous operation of all emission sources
- Simultaneous operation of only M-136 A1, A2, A3, and M-225 A, and
- Simultaneous operation of only M-136 A1, A2, A3, and M-225 A with an adjustment to chromium and nickel risks.

The latter scenario represents ATK's best estimate of the conservative but more likely acute hazards associated with facility operations, for the reasons stated above.

TABLE ES-1
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, ACTUAL AND FUTURE HYPOTHETICAL ON-SITE WORKERS

Receptor Location	Hazard Index	HI with Adjusted Ni and Cr[b]
Autoliv Facility using all sources[a]	4.3	
Autoliv HI using sources M-136 A1, A2, A3 and M-225 A	2.2	0.5 [b]
South Plant Main Building using all sources[a]	3.8	
S. Plant HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.9	0.5 [b]
North Plant Main Building using all sources[a]	2.0	
N. Plant HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.0	0.2 [b]
Point of Maximum On-site[c] assuming all sources	7.8	
Point of Maximum On-site[c] using M-136 A1, A2, A3 and M-225 A	3.9	9.7E-01[b]

An Index of one or less is acceptable

a All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.

b Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

c The point of maximum on-site risk is within a fenced area and has a controlled access point available only to authorized employees. This location is in an area of storage bunkers, and employees would not be in this area during a burn. The hypothetical hazards are provided for reference, but there are no receptors in this location.

TABLE ES-2
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, FOR HYPOTHETICAL BOUNDARY/OFF-SITE RESIDENTIAL/FARMER RECEPTORS

Receptor Location	Hazard Index	HI with Adjusted Ni and Cr[b]
Point of Maximum Off-site assuming all sources[a]	0.9	
Point of Maximum Off-site using M-136 A1, A2, A3 and M-225 A	0.5	0.1[b]
Blue Creek using all sources[a]	4.7	
B. Creek HI calculated using sources M-136 A1, A2, A3 and M-225 A	2.4	0.6[b]
Boundary 1 using all sources[a]	5.3	
Bound 1 HI calculated using sources M-136 A1, A2, A3 and M-225 A	2.7	0.7[b]
Boundary 2 using all sources[a]	2.3	
Bound 2 HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.2	0.3[b]
Boundary 3 using all sources[a]	0.7	
Bound 3 HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1[b]
Boundary 4 using all sources[a]	0.8	
Bound 4 HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1[b]
ATK Ranch Pond using all sources[a]	0.2	
ATK Ranch Pond HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	2.9E-02[b]

a All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.
b Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

TABLE ES-3
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, FOR ACTUAL RESIDENTIAL/FARMER OFF-SITE RECEPTORS

Receptor Location	Hazard Index	HI adjusted Ni and Cr[b]
Adams Ranch using all sources[a]	2.8	
A. Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.4	0.4[b]
Christensen Ranch using all sources[a]	0.9	
Christensen Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1 [b]
Holmgren Ranch using all sources[a]	0.5	
Holmgren Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.2	0.1 [b]
Howell Dairy using all sources[a]	0.3	
Howell Dairy HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	3.6E-02 [b]
Penrose using all sources[a]	0.3	
Penrose HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	3.4E-02 [b]
Thatcher using all sources[a]	0.3	
Thatcher HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.2	4.4E-02 [b]

Notes:
an Index of one or less is acceptable
a All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.
b Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

Risk Assessment Results

On-site and off-site discrete receptor locations are selected from within a 10 km radius. The receptors include known on-site worker locations, and maximum concentration locations, where the receptors are assumed to be hypothetical farmers and residents. The receptors off-site are known farm and residential locations, and a future hypothetical farmer and residential scenario estimated at two locations (one on-site and one off-site) of maximum concentration and deposition determined from the air dispersion modeling. These points of maximum risk are provided for reference, however, there are no receptors at these locations. In addition, there is a qualitative discussion for two recreational areas that could be frequented by hunters.

The non-cancer hazards are estimated by calculating the hazard quotient (the ratio of the calculated exposure dose to the Reference Concentration (RfC) or Reference Dose (RfD), and summing the resulting quotients to provide an index. This conservatively assumes all chemicals are toxic through the same mechanism, when they are not.

Chronic (Long-term) Hazards and Risks

On-site Worker Locations

On-site workers are exposed via inhalation. The resulting excess cancer risks and HIs are shown in Table ES-4. There were no exceedances under the Utah Administrative Code risk and hazard levels provided in R-315-101-6.

TABLE ES-4
SUMMED NON-CANCER HAZARD INDICES AND CANCER RISKS FOR ALL COPCS: ACTUAL ON-SITE INDUSTRIAL RECEPTORS

Receptor Name	Industrial Worker Cancer Risk	Industrial Worker Non-cancer HI
Autoliv[a]	8.3E-08	2.4E-02
North Plant Main Administration Building[a]	2.9E-08	8.4E-03
South Plant Main Administration Building[a]	6.9E-08	2.0E-02

Notes:
a These chronic hazard indices and cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B). This scenario is not realistic and would not occur. It represents an overestimation of risk.

Off-site Farmer and Resident Locations

The off-site resident adult and child are assumed to be exposed via inhalation of vapors and particulates, the incidental ingestion of soil, and the ingestion of homegrown produce. The subsistence farmer adult and child are assumed to be exposed via inhalation of vapors and particulates, ingestion of homegrown produce, milk, beef, chickens, eggs, and pork and incidental ingestion of soil. An off-site farmer child breast milk consumption exposure scenario is also considered.

The recreational areas are evaluated qualitatively.

Chronic (long-term, lifetime) HIs and excess lifetime cancer risks are calculated and provided in Table ES-5 and ES-6, respectively. It can be seen that these results are all below the Utah R-101 acceptable risk levels of an HI = one and a cancer risk less than one in one million for all off-site receptors.

Similar to the acute hazards, a downward adjustment was made to the chronic risks and hazards based on the assumption that the aluminum in the test pans was the source of the chromium and nickel in the OBOD tests. These adjusted results are presented in Tables 10-4 through 10-10 and the calculations are provided in Appendix F.

Hypothetical future farmer and resident scenarios at the point of maximum off-site deposition are also shown in these tables.

In addition, Tables ES-7, ES-8 and ES-9 provide a summary of all scenarios evaluated for chronic hazards and risks. The results are summarized for ease of comparison between the scenarios evaluated. Table ES-7 presents the chronic hazards and risks for the current industrial workers as well as the future hypothetical on-site worker. Table ES-8 presents chronic hazards for the existing off-site receptors and both current and future hypothetical receptors. The first scenario presented in the tables includes simultaneous burning of all sources (M-136 A, B, C and M-225 A and B). The second scenario includes the same sources, with the adjustment for chromium and nickel as discussed above. The latter scenario represents ATK's best estimate of conservative but more likely chronic hazards and risks associated with facility operations. Within Table ES-8, the non-cancer hazards are very similar for both scenarios. The adjustment for chromium and nickel did not have a big impact on the hazard estimates because these chemicals are not contributing significantly to the non-cancer hazards.

Table ES-9 presents chronic cancer risks for the existing off-site receptors and both current and future hypothetical receptors. A comparison of the two scenarios evaluated reveals that the chromium and nickel adjustment resulted in a significant reduction in the cancer risks.

The risks to an infant consuming breastmilk from an exposed subsistence farmer and resident mother are calculated and compared to the national average background exposure level of 60 picograms of dioxin toxic equivalents per kilogram per day (pg TEQ/kg/day). The highest reported dose is over 1000 times less than the national average background level, indicating that the risks associated with this pathway are negligible.

TABLE ES-5
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS:
ACTUAL OFF-SITE RECEPTORS

Receptor Name	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Adams Ranch[a]	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Christensen Ranch[a]	5.0E-03	5.0E-03	5.0E-03	5.1E-03
Holmgren Ranch[a]	2.6E-03	2.6E-03	2.6E-03	2.6E-03
Howell Dairy[a]	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Thatcher[a]	1.8E-03	1.8E-03	1.9E-03	1.9E-03
Penrose[a]	1.6E-03	1.6E-03	1.7E-03	1.7E-03
Maximum Off-site[a]	4.9E-02	4.9E-02	4.9E-02	4.9E-02

Notes:
a These chronic hazard indices were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B).
This scenario is not realistic and would not occur. It represents an overestimation of risk.

TABLE ES-6 SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS: ACTUAL OFF-SITE RECEPTORS				
Receptor Name	**Resident Adult Chronic Risk**	**Resident Child Chronic Risk**	**Farmer Adult Chronic Risk**	**Farmer Child Chronic Risk**
Adams Ranch[a]	1.6E-07	7.0E-08	3.4E-07	1.2E-07
Christensen Ranch[a]	4.7E-08	2.0E-08	9.9E-08	3.5E-08
Holmgren Ranch[a]	2.4E-08	1.0E-08	5.0E-08	1.8E-08
Thatcher[a]	1.7E-08	7.4E-09	3.7E-08	1.3E-08
Howell Dairy[a]	1.6E-08	7.0E-09	3.6E-08	1.3E-08
Penrose[a]	1.5E-08	6.3E-09	3.2E-08	1.1E-08
Maximum Off-site[a]	4.8E-07	2.1E-07	9.8E-07	3.5E-07
Notes: a These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B). This scenario is not realistic and would not occur. It represents an overestimation of risk.				

One of the conservative assumptions built into the risk assessment process is that all chemicals in the original emissions test are present at their detected concentration, or their detection limit. This is not the case and only 133 of over 200 chemicals were detected. Calculating potential cancer risks for only the 133 detected COPCs shows that non-detected chemicals account for about 9% to 34% of the potential cancer risk, depending upon the scenario. (Table not provided in this Executive Summary, See Table 9-15).

Uncertainty

Uncertainty is inherent in the risk assessment process, and there will always be uncertainty. To account for this, and in an attempt to provide a risk assessment that is health protective, conservative assumptions are made. The types of assumptions, and the degree to which they might influence this risk assessment are provided in Table ES-10. This is a qualitative analysis. Quantifying uncertainty is difficult, but US EPA's process is considered health protective and the risks calculated here should also be considered health protective.

Daycare Scenario

A daycare scenario was added to the HHRA and evaluated in December 2015, based on information that ATK intends to provide an on-site daycare facility. The details and results for the daycare scenario are presented in Appendix G. The emissions rates, COPCs, model assumptions, and approach for the daycare scenario are consistent with the main OBOD HHRA. After adjusting for the actual sources burned and the contribution of chromium and nickel from the test pans, the acute and chronic hazards are less than one and the chronic risks are all less than 1×10^{-6}.

TABLE ES-7 SUMMED NON-CANCER HAZARD INDICES AND CANCER RISKS FOR ALL COPCS: ACTUAL ON-SITE INDUSTRIAL RECEPTORS		
Receptor Name	**Industrial Worker Cancer Risk**	**Industrial Worker Non-cancer HI**
Autoliv Facility– Scenario A	8.3E-08	2.4E-02
Autoliv Facility– Scenario B	1.6E-08	2.3E-02
North Plant Main Administration Building– Scenario A	2.9E-08	8.4E-03
North Plant Main Administration Building– Scenario B	5.6E-09	8.3E-03
South Plant Main Administration Building– Scenario A	6.9E-08	2.0E-02
South Plant Main Administration Building– Scenario B	1.3E-08	2.0E-02
Future Hypothetical On-site Worker		
Maximum On-site – Scenario A	1.2E-07	3.3E-02
Maximum On-site – Scenario B	2.2E-08	3.3E-02

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE ES-8
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS: ALL SCENARIOS INCLUDED

Actual Off-Site Receptors				
Receptor Name	**Resident Adult Chronic HI**	**Resident Child Chronic HI**	**Farmer Adult Chronic HI**	**Farmer Child Chronic HI**
Adams Ranch – Scenario A	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Adams Ranch– Scenario B	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Christensen Ranch – Scenario A	5.0E-03	5.0E-03	5.0E-03	5.1E-03
Christensen Ranch– Scenario B	5.0E-03	5.0E-03	5.0E-03	5.0E-03
Holmgren Ranch – Scenario A	2.6E-03	2.6E-03	2.6E-03	2.6E-03
Holmgren Ranch– Scenario B	2.5E-03	2.5E-03	2.5E-03	2.5E-03
Howell Dairy – Scenario A	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Howell Dairy– Scenario B	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Thatcher – Scenario A	1.8E-03	1.8E-03	1.9E-03	1.9E-03
Thatcher– Scenario B	1.8E-03	1.8E-03	1.8E-03	1.8E-03
Penrose – Scenario A	1.6E-03	1.6E-03	1.7E-03	1.7E-03
Penrose– Scenario B	1.6E-03	1.6E-03	1.6E-03	1.6E-03
Future Hypothetical Resident/Farmer Receptor				
Maximum Off-site – Scenario A	4.9E-02	4.9E-02	4.9E-02	4. 9E-02
Maximum Off-site– Scenario B	4.8E-02	4.8E-02	4.8E-02	4.8E-02
Hypothetical Resident and Farmer Receptors at Boundary/Off-site Locations				
Receptor Name	**Resident Adult Chronic HI**	**Resident Child Chronic HI**	**Farmer Adult Chronic HI**	**Farmer Child Chronic HI**
Blue Creek – Scenario A	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Blue Creek– Scenario B	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Boundary 1 – Scenario A	3.2E-02	3.2E-02	3.2E-02	3.2E-02
Boundary 1– Scenario B	3.2E-02	3.2E-02	3.2E-02	3.2E-02
Boundary 2 – Scenario A	1.3E-02	1.3E-02	1.3E-02	1.3E-02

TABLE ES-8 SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS: ALL SCENARIOS INCLUDED				
Boundary 2– Scenario B	1.2E-02	1.2E-02	1.2E-02	1.2E-02
Boundary 3 – Scenario A	3. 2E-03	3.2E-03	3.2E-03	3.3E-03
Boundary 3– Scenario B	3. 2E-03	3.2E-03	3.2E-03	3.2E-03
Boundary 4 – Scenario A	3.5E-03	3.5E-03	3.5E-03	3.5E-03
Boundary 4– Scenario B	3.5E-03	3.5E-03	3.5E-03	3.5E-03
ATK Ranch Pond– Scenario A	1.1E-03	1.1E-03	1.1E-03	1.1E-03
ATK Ranch Pond– Scenario B	1.1E-03	1.1E-03	1.1E-03	1.1E-03

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE ES-9
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS: ALL SCENARIOS INCLUDED

Actual Off-site Receptor				
Receptor Name	**Resident Adult Chronic Risk**	**Resident Child Chronic Risk**	**Farmer Adult Chronic Risk**	**Farmer Child Chronic Risk**
Adams Ranch – Scenario A	1.6E-07	7.0E-08	3.4E-07	1.2E-07
Adams Ranch – Scenario B	2.1E-08	1.0E-08	1.8E-07	6.0E-08
Christensen Ranch- Scenario A	4.7E-08	2.0E-08	9.9E-08	3.5E-08
Christensen Ranch- Scenario B	7.0E-09	3.0E-09	5.3E-08	1.8E-08
Holmgren Ranch- Scenario A	2.4E-08	1.0E-08	5.0E-08	1.8E-08
Holmgren Ranch- Scenario B	3.7E-09	1.5E-09	2.7E-08	9.0E-09
Thatcher- Scenario A	1.7E-08	7.4E-09	3.7E-08	1.3E-08
Thatcher- Scenario B	2.3E-09	1.1E-09	2.0E-08	7.5E-09
Howell Dairy- Scenario A	1.6E-08	7.0E-09	3.6E-08	1.3E-08
Howell Dairy- Scenario B	2.2E-09	4.3E-09	2.0E-08	6.6E-09
Penrose- Scenario A	1.5E-08	6.3E-09	3.2E-08	1.1E-08
Penrose- Scenario B	2.6E-09	9.6E-10	1.8E-08	5.8E-09
Future Hypothetical Resident/Farmer Receptor				
Maximum Off-site- Scenario A	4.8E-07	2.1E-07	9.8E-07	3.5E-07
Maximum Off-site- Scenario B	6.9E-08	2.9E-08	5.1E-07	1.7E-07
Hypothetical Resident and Farmer Receptors at Boundary/Off-site Locations				
Receptor Name	**Resident Adult Cancer Risk**	**Resident Child Cancer Risk**	**Farmer Adult Cancer Risk**	**Farmer Child Cancer Risk**
Blue Creek- Scenario A	3.3E-07	1.4E-07	6.7E-07	2.4E-07
Blue Creek- Scenario B	5.0E-08	2.0E-08	3.5E-07	1.2E-07
Boundary 1- Scenario A	3.1E-07	1.3E-07	6.5E-07	2.3E-07
Boundary 1- Scenario B	4.3E-08	1.9E-08	3.4E-07	1.1E-07
Boundary 2- Scenario A	1.2E-07	5.2E-08	2.5E-07	8.9E-08

TABLE ES-9 SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS: ALL SCENARIOS INCLUDED				
Boundary 2- Scenario B	1.6E-08	7.5E-09	1.3E-07	4.4E-08
Boundary 3- Scenario A	3.1E-08	1.3E-08	6.4E-08	2.3E-08
Boundary 3- Scenario B	5.1E-09	1.9E-09	3.4E-08	1.1E-08
Boundary 4- Scenario A	3.3E-08	1.4E-08	6.9E-08	2.4E-08
Boundary 4- Scenario B	5.6E-09	2.0E-09	3.7E-08	1.2E-08
ATK Ranch Pond-Scenario A	9.7E-09	4.1E-09	2.1E-08	7.5E-09
ATK Ranch Pond-Scenario B	1.6E-09	6.5E-10	1.2E-08	4.0E-09

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE ES-10
SOME OF THE UNCERTAINTIES IN THE PROMONTORY OB/OD RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect of the Process	Assumption	Effect on the Risk Assessment
Emissions Tests	The worst case emissions from nine different tests is used to provide the emissions factors for the modeling of COPCs	Likely overestimates risk
Emissions Tests	The contribution from background is not subtracted from the emissions factors used to calculate COPC emissions rates	Likely overestimates risk
Emissions Tests	The chromium and nickel contribution from the stainless steel test trays likely creates artifacts that are not subtracted from the emissions factors used to calculate COPC emissions rates	Likely overestimates acute Hazard Indices
Emissions Tests	The contribution from non-detected PAH in the 1.3-Class tests was replaced with 1.1-Class emissions factors	Likely overestimates risk
Emissions Tests	The contribution from non-detected chemicals was shown to contribute an additional 7 to 25 percent of the risks	Shown to overestimate risk
Emissions Tests	Two PAH were eliminated from the COPC list	Likely neutral or potentially underestimates risk
Air Quality Modeling	Acute air concentrations are calculated assuming all sources operate at the same time	Shown to overestimate risk
Air Quality Modeling	Assumes reasonable worst case meteorological conditions at the time of processing wastes	Likely overestimates risk

TABLE ES-10
SOME OF THE UNCERTAINTIES IN THE PROMONTORY OB/OD RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect of the Process	Assumption	Effect on the Risk Assessment
Air Quality Modeling	The model has a number of complex assumptions built in to represent plume rise, air dispersion, and particulate deposition. All have uncertainty.	Could overestimate or underestimate risk
Air Quality Modeling	The model has two components: OBODM and AERMOD, the operation of these two models together has not been validated.	Could overestimate or underestimate risk
Media Concentration Models	Soil concentrations are modeled based on deposition, release of COPCs to soil. COPCs may remain on released particles and not be released to soil.	Likely overestimates risk
Media Concentration Models	COPC uptake into plants from air is based on the assumption that higher molecular weight COPCs are in the vapor phase, when they are likely to be adsorbed to particulates.	Likely significantly over estimates risk
Media Concentration Models	Plant uptake of COPCs is based on chemical specific modeling, often using physical parameters and often un-validated assumptions	Likely significantly over estimates risk
Media Concentration Models	Bio-transfer factors for COPCs from plants-to-animals, plants-to-humans, animals-to-humans, and human-to-human is based on chemical specific modeling, often using physical parameters and often un-validated assumptions	Likely overestimates risk
Exposure Assumptions	Human exposure parameter assumptions are US EPA default and are based on Reasonable Maximum Exposure, these are conservative for the majority of the population, but may be exceeded in some instances.	Could overestimate or underestimate risk

TABLE ES-10
SOME OF THE UNCERTAINTIES IN THE PROMONTORY OB/OD RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect of the Process	Assumption	Effect on the Risk Assessment
Exposure Assumptions	Human diet and intake exposure assumptions are US EPA default and are based on Reasonable Maximum Exposure, these are conservative for the majority of the population, but may be exceeded in some instances.	Will overestimate risk in this risk assessment
Exposure Assumptions	Human diet and intake exposure assumptions are unlikely at this location in Utah because the soil and water are of a quality that could not produce the assumed levels of plant and animal food for the farmer diet.	Will overestimate risk in this risk assessment
Toxicological dose-response	Risk assessment uses US EPA and other regulatory dose-response factors that are designed to be health protective for the majority of the population. By definition, these are conservative for the majority of the population, but may be exceeded in some instances.	Likely overestimates risk
Risk and Hazard Calculations	These calculations will multiply the conservative uncertainty in the parameters presented above, and will increase the uncertainty.	Likely overestimates risk

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LIST OF ACRONYMS AND ABBREVIATIONS

%	Percentage
ADAF	Age-Dependent Adjustment Factors
ADD	Average Daily Dose
AEGL-1	Acute Inhalation Exposure Guidelines-1
AERMOD	American Meteorological Society Gaussian Air Dispersion Modeling
AP	Ammonium Perchlorate
ATK	ATK Launch Systems
ATSDR	Agency for Toxic Substances and Disease Registry
BEHP	Bis-(2-ethyl hexyl) phthalate
BV_{ag}	Biotransfer factors for aboveground produce
BV_{forage}	Biotransfer factors for aboveground forage
CAF	Carcinogenic Adjustment Factor
Cal EPA	California EPA
CB&I	CB&I Environmental & Infrastructure Inc.
CERCLA	Comprehensive Environmental Response Compensation and Liability Act
CO_2	Carbon Dioxide
COPC	Chemical of Potential Concern
COPCs	Chemicals of Potential Concern
Cr	Chromium
Cr(III)	Trivalent chromium

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Cr(VI)	Hexavalent chromium
CSF	Cancer Slope Factor
csv	comma separated values
Difurans	Dibenzofurans
Dioxins	Dibenzodioxins
DNOP	Di (n-octyl) phthalate
DOD	Department of Defense
DOE	Department of Energy
DSHW	Division of Solid and Hazardous Waste
EM	Energetic Material
EPA	Environmental Protection Agency
ERPG-1	Emergency Planning Guidelines-1
g/s	gram per second
H_2O	Water
HCL	Hydrogen Chloride
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HMX	High Melting Explosive or octahydro-1,3,5,7-tetranitro-1,3,5,7-tetra
HQ	Hazard Quotient
hr	Hour
IEUBK	Integrated Exposure Uptake Bio-kinetic

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IR	Infrared
IRIS	Integrated Risk Information System
ISC-3	Industrial Source Complex-3
kg	Kilograms
km	kilometers
K_{ow}	octanol/water partition coefficients
lb per lb	pound per pound
lb	pound(s)
lb/hr	pound per hour
log K_{ow}	log octanol/water partition coefficients
LOAEL	Lowest Observed Adverse Effect Level
MAF	Mutagenic Adjustment Factor
methyl-PAH	Methyl polynuclear aromatic hydrocarbons
mg/kg	Milligrams per Kilogram
mph	miles per hour
MIDAS	Munitions Items Disposition Action System
MOA	Mode of Action
MST	Mountain Standard Time
NAAQSs	National Ambient Air Quality Standards
NASA	National Aeronautics and Space Administration
NHL	Non-Hodgkin's Lymphoma
NO_2	Nitrogen Dioxide

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NOAA	National Oceanic and Atmospheric Administration
OB	Open Burning
OBODM	Open Burning and Open Detonation Model
OD	Open Detonation
^{o}K	degrees Kelvin
OSHA	Occupational Safety and Health Association
OSWER	Office of Solid Waste and Emergency Response
PAC	Protective Action Criteria
PAHs	Polynuclear Aromatic Hydrocarbons
PG	Pasquill-Gifford
PICs	Products of Incomplete Combustion
PM	Particulate Matter
PM_{10}	Particulate Matter of 10 micrometers in diameter
$PM_{2.5}$	Particulate Matter of 2.5 micrometers in diameter
ppm	parts per million
PPRTVs	Provisional Peer Reviewed Toxicity Values
RAGS	Risk Assessment Guidance for Superfund
RCRA	Resource Conservation and Recovery Act
RDX	Royal Dutch Explosive or hexahydro-1,3,5-trinitro-1,3,5-triazine
REL	Reference Exposure Level
RfCs	Reference concentrations
RfD	Oral Reference-Dose

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RME	Reasonable Maximum Exposure
RSL	Regional Screening Level
SCAPA	Subcommittee on Consequence Assessment and Protective Actions
SERDP	Strategic Environmental Research and Development Program
SF_o	Oral Slope Factors
TCDD	Tetrachloro-dibenzo-dioxin
TCDF	Tetrachloro-dibenzo-difuran
TCDD-TE	2,3,7,8- Tetrachloro-dibenzo-dioxin-Toxic Equivalents
TCE	Trichloroethene
TDA	Toluenediamine
TDI	Toluene diisocyanate
TEEL	Temporary Emergency Exposure limits
TEF	Toxic Equivalent Factor
TNT	Trinitrotoluene
TSLs	Toxic Screening Levels
UDEQ	Utah Department of Environmental Quality
URF_i	Inhalation Unit Risk Factors
yr	Year
WDTC	West Desert Test Center

1. INTRODUCTION

ATK Launch Systems (ATK), located 30 miles west of Brigham City, Utah, currently operates open burning (OB) and open detonation (OD) units for the treatment and disposal of waste propellants and propellant-contaminated materials. These treatment units have the on-facility designation M-136 and M-225. They are subject to Resource Conservation and Recovery Act (RCRA), 40 CFR 264, Subpart X permitting requirements for miscellaneous treatment units. The location of the Promontory Facility is shown on Figure 1-1, and the location of the burning grounds is shown on Figure 1-2.

The Utah Department of Environmental Quality, Division of Solid and Hazardous Waste (Utah DSHW) requires ATK to conduct human health and ecological risk assessments in support of a new RCRA Subpart X permit application. This document is the human health risk assessment (HHRA) in support of ATK's OB/OD Subpart X permitting requirement. Before the HHRA was conducted, a number of steps are required, including: the preparation of an inventory of potential waste streams, test burning studies to quantify potential emissions from these waste streams, and air dispersion modeling analyses to evaluate the potential air quality impact from activities at the M-136 and M-225 treatment units.

For this HHRA the US Environmental Protection Agency (EPA) Incineration Risk Assessment Guidance (EPA, 2005a) was used as the guiding document. The key receptors, exposure assumptions, and methods of hazard and risk calculation in this guidance have been incorporated into a HHRA software program by Lakes Environmental of Waterloo, Canada (Lakes, 2014) and this program has been validated and approved for use at incineration facilities by the EPA. The Lakes software was updated for this HHRA to make it current with November 2014 dose-response information and EPA default exposure assumptions. The Utah DSHW-approved the updates in an August 2014 risk assessment protocol approval letter to ATK. The Lakes dose-response database is also revised based on updates to the Regional Screening Levels tables in November 2014, and again in November 2015. Also in November 2014, the Utah DSHW reviewed and approved updates to the short-term air goals for the project, as the approved protocol was found to contain values that were out of date. Further, in 2015, potentially carcinogenic chemicals with a suspected mutagenic mode of action (MOA) were re-evaluated and age-dependent adjustment factors (ADAF) were incorporated into the dose-response factors in the Lakes software, which is used to calculate the risks and hazards from the treatment units. The air dispersion modeling that provides ambient air chemical concentrations and deposition rates into the Lakes software was conducted by CB&I using a Utah DSHW approved protocol (CB&I, 2014).

The Utah DSHW-approved air dispersion modeling analysis generates air quality results for short-term (1-hour acute) and long-term (5-year annual average) meteorological conditions based on a unit emissions factor. This model is coupled with emissions factors for the waste from the OB/OD studies in the Lakes HHRA software to provide Chemical of Potential Concern (COPC) air concentrations and deposition rates, which are also used by the Lakes software to calculate COPC concentrations for other media such as soil, vegetation, and food products. The Lakes HHRA software also determines the non-cancer Hazards and Excess Lifetime Cancer Risks (Risks) (i.e., risks over background due to potential exposure from emissions) from the modeled potential emissions.

This HHRA is based on the Utah DSHW-approved HHRA Protocol (ATK, 2014a). It follows EPA, 2005a, but incorporates some site-specific assumptions. It is conservative, meaning it assumes Reasonable Maximum Exposure (RME) conditions, which is consistent for this type of risk assessment, and based on EPA statements, it is conservative, because it is likely to overestimate the risks to potentially exposed receptors (EPA, 2004a).

1.1 Report Objectives

The objectives of this report are as follows:

- To provide a brief characterization of the exposure setting for the HHRA, and provide a summary of the potential on-site and off-site receptors that might be exposed to releases from the treatment units.
- To provide references to the studies used to develop emissions factors from the treatment units.
- To select COPCs from the emissions studies and to provide a summary of the COPC emissions rates used in the modeling and risk assessment.
- To provide a brief summary of the modeling conducted to develop ambient COPC concentrations and depositions, and to provide a brief summary of the work conducted by CB&I that compared COPC air concentrations to National Ambient Air Quality Standards (NAAQSs), and to Utah Toxic Screening Levels (TSLs).
- To provide results of Acute Hazard risk calculations. That is, to document the results of a comparison of the modeled ambient COPC concentrations to Utah DSHW approved 1-hour acute Protective Action Criteria (PAC) or Acute inhalation exposure guidelines (AEGL-1) for on-site and off-site receptors.
- To provide the results of chronic Hazard and Risk calculations. That is, to document the results of the Lakes software calculations for a lifetime of

exposure to COPCs that may result in potential non-cancer Hazards and cancer Risks due to emissions from the treatment units, using EPA HHRA methods.

- Results are provided for on-site and off-site receptors.
- To provide a discussion of the uncertainty in the results of the HHRA.
- Where possible to provide an estimate of the magnitude of the uncertainty, and whether it might result in an over- or under-estimation of the risks.

1.2 Introduction and Background

ATK's Promontory Facility is located north of Utah's Great Salt Lake about 30 miles from Brigham City in a rural part of eastern Utah. The facility produces rocket motors for military and space exploration. The facility is also bordered by Autoliv, who manufacture charges for automobile safety air bags. The ATK Facility is used for the disposal of rocket motors that have reached their life expectancy and need to be removed from service. In the past the facility manufactured and tested Space Shuttle Booster motors.

The facility is located in a rural part of Utah that supports only limited farming, ranching and livestock production due to a combination of limited water resources and poor quality soil. Groundwater and surface water quality are poor, and due to these agricultural limitations the area is sparsely populated with intermittent farms and homesteads.

The Promontory Facility is one of the largest employers in the area, and the facility employs some 1400 workers. It is an important resource for the State and local economy.

1.3 Approach to the Air Quality Modeling

The air quality modeling for the Promontory treatment units was conducted by CB&I in 2014. The model used was developed by CB&I based on initial air quality modeling conducted by TetraTech (2011a), and as revised in 2011 (TetraTech, 2011b), and in 2012 (TetraTech, 2012). The CB&I model (CB&I, 2014a), and its addendum (CB&I, 2014b) combined an Open Detonation and Open Burning Model (OBODM) (WDTC, 1998a) with more standard Industrial Source Complex (ISC-3) American Meteorological Society Gaussian air dispersion modeling (AERMOD) to produce an improved OBODM/AERMOD hybrid model that is believed to more accurately predict air chemical transport and dispersion for this type of facility. The output files from this model became the source files for the Lakes HHRA software (Lakes, 2014) for both short-term and long-term exposure hazard and risk calculations.

1.4 Approach to the Risk Assessment

The HHRA is conducted based on default EPA risk assessment methodologies originally developed under the US Comprehensive Environmental Response Compensation and Liability Act (CERCLA, also known as Superfund) (EPA, 1989), and RCRA. Superfund HHRA guidance has developed substantially since 1989, and this HHRA uses EPA's Incineration Guidance (EPA, 2005a) as its primary guidance for the selection of COPCs, receptors and exposure pathways. As a result of this guidance document, Lakes Environmental developed a software product that calculated long-term hazards and risks based on the equations and parameters in EPA, 2005a (Lakes, 2014). The Lakes risk assessment model was approved by EPA for incineration projects. However, dose-response and default exposure assumptions change from time to time, and this HHRA is based on a protocol developed by ATK (ATK, 2014a) which was accepted and approved by UDEQ DSHW (UDEQ, 2015). The HHRA protocol also incorporates EPA's most recent default exposure assumptions (EPA, 2014a), dose-response information (EPA, 2014b; EPA, 2014c), and other modifications, such as the incorporation of ADAFs, that were approved by the Utah DSHW, as of December 2015, prior to conducting the HHRA. A daycare scenario was added to the HHRA and evaluated in December 2015, based on information that ATK intends to provide an on-site daycare facility. The details and results for the daycare scenario are presented in Appendix G.

1.5 Document Organization

This document is organized into eleven sections and six appendices. The body of the text provides a summary of the COPC selection process, the air quality modeling method, the exposure assumptions and the results of the acute and chronic HHRA process. The appendices contain the majority of the work including: supporting emissions factors, dose-response factors, calculations, and tables of results. Given the large volume of calculations and pages of software output, if the tables of results are compiled into the report it would result in a large and unwieldy document. Therefore, the text should be viewed as an overview of the process, with summary results, and the appendices as a compilation of individual calculations and detailed results. The EPA's Incineration Guidance provides a comprehensive description of the risk assessment assumptions, methods for receptor selection, exposure assumptions and risk assessment calculation methods, and because the Lakes software is directly based on this guidance, these methods and descriptions are not repeated in this document. A brief description of the subsequent sections is provided below:

2.0 Characterizing Facility Emissions: Provides a description of the methods for selecting COPCs, developing emissions factors and emissions rates.

3.0 Air Dispersion and Deposition Modeling: Provides a brief overview of the modeling process.

4.0 Compliance the NAAQSs: Provides a summary of the work conducted by CB&I that compared modeled air COPC concentrations to National Ambient Air Quality Standards, and the Utah Toxic Screening Levels.

5.0 Exposure Scenario Identification: Provides a description of the exposure scenarios selected for the chosen receptors.

6.0 Estimating Media Concentrations: Describes the methods for calculating media (soil, biota) COPC exposure point concentrations.

7.0 Quantifying Exposure: Briefly describes how exposures are quantified for the HHRA.

8.0 Toxicity Assessment: Describes the hierarchy of sources used to obtain the short-term (1-hour) and long-term dose-response factors for inhalation and ingestion.

9.0 Characterizing Risks and Hazards: Provides the results of the risk assessment calculations

10.0 Estimating Uncertainty for Human Health Risk Assessment: Provides a summary of the uncertainties in the HHRA

11.0 References: Provides a list of the citations used in the HHRA.

Figures

Tables

2. CHARACTERIZING OB/OD FACILITY EMISSIONS

ATK's waste profiles were relatively constant for many years, and were predominantly the results of the disposal of 1.3-Class propellant wastes with small amounts of 1.1-Class wastes, contaminated waste, and visual distress flare manufacturing wastes. Recently, the percentage of 1.3-Class propellant wastes has decreased, and the objective of re-evaluation is to provide ATK with a flexible operating permit and minimize or perhaps eliminate the need for a future permit modification during the life of the permit, based on the inclusion of higher amounts of 1.1-Class wastes. To this end, the emissions factors from OB/OD tests performed using ATK's 1.3-Class (ATK, 2009), and the 1.1-Class (ATK, 1998) wastes are considered when developing emissions factors for this risk assessment. The approved-HHRA Protocol developed a conservative, but reasonable process for selecting chemical emissions factors from these tests for use in the risk assessment.

In short, an OB/OD emissions test compiles small samples of the materials processed by ATK and burns them in a relatively closed system that captures and subsequently measures the emissions from each test. The combustion process results in gasses, particulate matter and low levels of chemical products of incomplete combustion. The contained system allows the combustion by-products to be captured and submitted to an analytical laboratory for analysis, where the amount of a chemical detected in the test is converted to an "emissions factor" that describes the amount of chemical produced in pound per pound (lb per lb) of test material burned. When a chemical is not detected it is considered present at its method detection limit to give an "upper-bound" emissions factor. The actual emissions are likely to be lower than the detection limit, and could be as low as zero.

The analytical process attempted to measure over 200 different chemicals, or COPCs, and all of which are considered at this stage of the process.

2.1 Identifying Emission Sources

The two activities in the facility are OB and OD. OB treatment is considered a quasi-continuous source because the treatment event is usually complete within one hour. OD is considered an instantaneous source because treatment is completed within milliseconds. The approach to modeling these two types of events was as follows.

2.2 Open Burn

OB results in combustion of the energetics and rapid rise of the hot combustion products due to buoyancy until a final height is reached. At this point, the emission cloud has no upward momentum and starts to disperse downwind. This event is simulated as an

elevated volume source with the stack height equal to the final cloud height predicted from OBODM.

2.3 Open Detonation

OD results in instantaneous combustion and immediate rise of the emission cloud to a final height. The cloud height is based on the reactive waste weight, wind speed, and atmospheric stability. Once elevated, this cloud disperses downwind. This event is simulated as an elevated volume source.

2.4 Estimating Emission Rates

Emission rates are estimated based on the quantity of material processed in OB/OD test events, and the emission factors are provided in Table 2-1 (and in Appendix A). Modeling to assess ambient air quality impacts is conducted using the estimated actual emission rates for each scenario. To reduce the number of required model runs, the emission rates for a single pollutant (i.e., $PM_{2.5}$) are input to the model. The single pollutant modeling results are then applied to the other pollutants that are part of the impact analysis by scaling the modeled results by the ratio of the desired pollutant emission rate to the modeled emission rate. However, modeling in support of the risk assessment is conducted at a unit emission rate of 1 gram per second (g/s) to allow for application of pollutant-specific emission rates within the risk assessment software.

The reactive waste quantities for each of the scenarios are based on the desired permit limits which are listed below.

2.5 M-136 Stations

M-136 has 14 burn stations (1 through 14) and any one of the following alternative and mutually exclusive scenarios could occur in these stations:

- A1: OB in six Burn Stations 1 through 12 at 16,000 pounds (lbs) in each station totaling 96,000 lbs reactive waste weight per event
- A2: 10,000 lbs reactive waste weight per event in Burn Station 13
- A3: 16,000 lbs reactive waste weight per event in Burn Station 14
- B: OB of 125,000 lbs of large rocket motors in Station 14
- C: OD of 600 lbs reactive waste in Stations 13 and 14 each, totaling 1,200 lbs reactive waste weight per event

At M-136 A1, burn stations 1 through 12 are clustered within 100 meters of each other. Six of the 12 stations are located closest to the western property line (Stations 1, 4, 7, 8,

10, and 11) and six are further from the boundary. The six areas near the boundary are modeled as six separate sources and used in the HHRA because their use in the model is more conservative. The other stations (Stations 2, 3, 5, 6, 9 and 12) are used by ATK but were not modeled as they are further from the boundary. Burn Stations 13 and 14 are modeled separately.

From previous modeling and from burn information provided by the facility, the following assumptions are made:

- Burn Stations 1 through 12 each consist of four adjacent burn pans. The average dimension of each burn pan is 8 feet by 13 feet, and the burn pan layout per station is approximated as an area of 16 feet by 26 feet.
- Burn Station 13 consists of two adjacent burn pans. The average dimension of each pan is 6 feet by 9 feet, and the burn pan layout for this station is approximated as an area of 9 feet by 12 feet.
- Burn Station 14 consists of four adjacent burn pans. The average dimension of each burn pan is 8 feet by 13 feet, and the burn pan layout for this station is approximated as an area of 16 feet by 26 feet.
- The dimension of the rocket motor burn area at Burn Station 14 is assumed to be 5 feet by 50 feet.
- The height of burn stations = 1.0 meter.
- The detonation will be started at ground level.

Of the potential scenarios at M-136 any one of the following alternative **and mutually exclusive scenarios** could occur in these stations. Operations are considered mutually exclusive because under ATK's operational protocols, the different scenarios do not operate at the same time, and the assumption that all units operate at the same time will lead to an over-estimate of short-term exposure. The uncertainty associated with this assumption is discussed in Section 10.

Scenario M-136-A

- A1: OB in six of Burn Stations 1 through 12 at 16,000 pounds (lb) in each station totaling to 96,000 lb reactive waste weight per event
- A2: 10,000 lb reactive waste weight per event in Burn Station 13
- A3: 16,000 lb reactive waste weight per event in Burn Station 14

Scenario M-136-B

- B: OB of 125,000 lb of large rocket motors in Station 14

Scenario M-136-C

- C: OD of 600 lb reactive waste in Stations 13 and 14 each, totaling 1,200 lb reactive waste weight per event

The source parameters for M-136 are taken from CB&I, 2013, and are presented in Section 3.

2.6 M-225 Stations

Burn Stations 1 through 4 are clustered within 100 meters and are modeled as a single source located approximately at the center of the cluster. The OD pit is modeled separately. From previous modeling and from burn information provided by the facility, the following assumptions are made:

- Burn Stations 1 through 4 each consists of one burn pan, having an average pan dimension of 6 feet by 17 feet.
- The height of burn stations = 1.0 meter.
- The detonation will be started at ground level.

Of the potential scenarios at M-225 either of the following alternative **and mutually exclusive scenarios** (meaning that under ATK's operational protocols, the different scenarios do not operate at the same time), could occur in these stations:

Scenario M-225-A

- A: OB of 1,125 lb of reactive waste in each of the Burn Stations 1 through 4 for a total of 4,500 lb reactive waste weight per event

Scenario M-225-B

- B: OD of 600 lb of reactive waste in Station 1

The source parameters for M-225 are taken from CB&I, 2013b, and are presented in Section 3.

2.7 Summary of Wastes Volumes

In summary, the air quality modeling, emissions rates and risk assessment are based on the following amounts of materials processed.

- M-136 A 8.4 million pounds

- M-136 B 1.5 million pounds
- M-136 C 0.10 million pounds

Total M-136 10.0 million pounds

- M-225 A 0.055 million pounds
- M-225 B 0.010 million pounds

Total M-225 0.065 million pounds

The total amount processed by M-136 and M-225 combined is 10.1 million pounds.

2.8 Identifying Chemicals of Potential Concern

ATK is selective about its waste streams and does not accept general commercial wastes from outside sources. However, ATK accepts wastes from their own clients, such as Department of Defense (DOD) and National Aeronautics and Space Administration (NASA), and ATK has typically originated these materials, which include unused rocket motors, and other process waste streams related to businesses at their own facilities. The primary waste stream is polymer bound perchlorate oxidizer mixed with aluminum as the fuel. Pure propellant burns around 4976°F (TetraTech, 2011b), in comparison to municipal incinerators, which are designed to reach a maximum temperature of 1,560°F, and hazardous waste incinerators, which burn at 1,400°F to 2,200°F. Aluminum burns at 6920°F; any material burned with the propellant burns quickly and at high temperatures. ATK also processes discarded rocket motors from various discontinued programs that contain highly energetic solid rocket fuel materials, and illumination flare wastes.

The COPCs are selected using the process outlined in EPA, 2005a, except as described in the approved-HHRA Protocol. The process used is summarized here.

Section 2.3 of the EPA's 2005 HHRAP guidance, which is designed to cover a range of facilities, outlines the COPC selection process. EPA (2005a) Figure 2-3, COPC Identification, maps out the compound selection process that is used to evaluate COPCs in the risk assessment. The process allows for the elimination of compounds that are not processed or generated by a facility, narrowing lists of compounds quantitatively evaluated to capture the risks associated with the facility, yet making the process more relevant to the facility under consideration. This narrowing makes the process more manageable. However, this risk assessment eliminates few COPCs, and evaluates both chemicals that are detected, and those that are not detected, assuming they are actually present at the detection limit of the test devised to measure emissions.

To summarize from the HHRAP guidance, "*We recommend selecting the risk assessment COPC from the stack test data.*" (EPA, 2005a; page 2-32) In the case of Promontory, stack tests are unavailable and the OB/OD test data serve the same function, that is, to provide a list of potential emissions from materials processed at the facility.

2.9 Step 1 Trial Burn and Fugitive Emissions

The HHRAP guidance process starts with the question, "*Was compound detected?*" If "*Yes*" the chemical is selected for quantitative evaluation in the HHRA. Tables 1-1 and 2-1 of the HHRA Protocol (ATK, 2014a) show the chemicals detected in the OB/OD 1.3- or 1.1-Class tests (ATK, 2009 & ATK, 1998). This subsection describes in more detail the nature of the detections.

2.9.1 Metals

The test bundles used to generate data in the OB/OD tests were compiled to mimic, to the extent possible, the conditions used by ATK when operating their units (at the time of testing). ATK evaluated the emissions from the test bundles and reported the results first in the *Draft Sampling Results for Emission Characterization of Open Burning Waste Propellant Materials* (ATK, 2007) and then in the final report: *ATK Sampling Results for Emission Characterization of Open Burning Waste Propellant Materials Volume I—Summary Report* (ATK, 2009) describes the process in more detail. Test bundles containing 1.3-Class propellant (with or without waste material) were placed in a test chamber on stainless steel pans and ignited. As a test bundle burned the gasses and particulates emitted into the test chamber were sampled by capturing the particulate matter, gases and volatilized chemicals in sampling "trains," or a series of vessels designed to capture different types of chemicals. In this process no metals would be destroyed they would simply be liberated from the test materials and captured in the sampling trains.

Experimentally there were some small but important differences in the way the OB/OD tests were ignited compared with actual operations. Test sample igniters used nickel-chromium electronic heating elements and black powder to ignite the test bundles. In addition; the stainless steel pans used to contain the burn inside the test chamber were fabricated with a high percentage (%) of chromium and nickel. The pans used in the OB/OD test burns were Type 316 stainless steel (see Appendix F), which contain high levels of chromium and nickel. In Type 316 steel, the chromium content is reduced from 18% found in other formulations to 16%, and the nickel content is increased from 8% to 10-16%, forming austenitic steel which also contains 2-3% molybdenum (www.bosunsupplies.com). When the igniters and pans were exposed to the high temperatures generated in the test by burning AP propellant, they contributed to the

emissions sampled in the OB/OD tests. A description of the background tests performed and the results are provided in ATK, 2009. The emissions factors provided in Table 2-1 have not been adjusted or reduced to account for any contributions of emissions from these test-related chemicals. The emissions from the test igniters will be discussed in the uncertainty section of this risk assessment, but these test-related chemicals were not subtracted from the emissions results.

While the ignition system for the test bundles is similar to those used to ignite OB/OD material at ATK, the heating elements represent a larger mass as a percentage of material in the tests, where the bundles weighed 1-3 pounds, compared with that used by ATK, where each pan may have material weighing 16,000 to 96,000 pounds, and so the ignition system has a greater influence on the test results.

Chromium (Cr) was measured as total Cr, and not speciated into the two valent forms of Cr: hexavalent Cr (Cr(VI) and trivalent Cr (Cr(III). These two Cr metal species have significantly different toxicological properties. There are no literature studies available on the amount of either Cr species present in the emissions from OB/OD facilities. In the absence of data, EPA, 2005a (Section 2.3.5.1, Chromium) recommends a maximum of 45% Cr(VI) and 55% Cr(III) be used when evaluating the emissions from facilities with stacks. This emissions ratio is used in the HHRA. The use of 100% Cr(VI) was considered to be too conservative, and 45% Cr is considered conservative when considering data available from other types of facility, or Cr sources in incineration. Additional support for the use of 45% Cr(VI): 55% Cr(III) is provided in the Section 10.1.2.8, Chromium Speciation, and in Appendix F. The risks associated with assuming 100% Cr(VI) are provided in Section 10.1.2.8, with supporting calculations provided in Appendix F.

Other detected metals include aluminum, copper, and manganese. Metals cannot be created or destroyed in the OB/OD testing process, and are either in the waste, or as with chromium and nickel, potentially generated as part of the test.

2.9.2 Dibenzo-p-dioxins and Dibenzofurans

Polychlorinated dibenzo-p-dioxins are a mix of 75 congeners (molecules with different numbers of chlorines in different positions), and polychlorinated dibenzofurans have 135 congeners. These compounds are of great interest, because of their fat solubility and potent toxicity. It is known that these chemical compounds bioaccumulate and bio-magnify in food chains, and often contribute the majority of the risk in this type of HHRA. Rather than selecting individual chemicals, these chemicals are evaluated as a classes, or group of compounds relative to the toxicity of 2,3,7,8-tetrachloro-dibenzodioxin (2,3,7,8-TCDD).

To capture the risk from so many different compounds the risk assessment process assumes that all of the congeners are present either at their detected concentration or at their detection limit, and combines the toxic potency for each molecule compared with 2,3,7,8-TCDD, to give TCDD-Toxic Equivalents (TCDD-TE).

2.9.3 Polynuclear aromatic hydrocarbons

Polynuclear aromatic hydrocarbons (PAHs) are a class of organic compounds that has been known for many years, they are aromatic hydrocarbons molecules with structures similar to benzene but with more aromatic rings (2 through 8 rings). They are found in coal tars, oils and coal. They are not manufactured or used by ATK, and are unlikely to be found in ATK's wastes. PAHs are used in chemical and biochemical research, but not in energetics or propellant research. However, certain PAHs are detected in the emissions test because PAHs are a product of incomplete combustion. They are generated via a known mechanism and under specific conditions, as discussed in greater depth below (see Section 2.11.1.3).

2.9.4 Other Chemicals

Any other detected chemicals are included as a COPC, including: combustion gasses, such as chlorine, oxides of carbon, nitrogen, and sulfur; hydrocarbons and chlorinated hydrocarbons; and low molecular weight aldehydes.

2.10 Step 2 Is Non-detected Compound Present in the Waste?

The next question from Figure 2-3 in the HHRAP guidance asks, "*Is non-detected compound present in waste?*" The chemicals not detected in the emissions from ATK's OB/OD tests are included in the list of COPCs and are listed in Table 2-1, with the exceptions noted below.

The chemicals in Table 2-2 of the Protocol are not detected in ATK's emissions. The risk assessment assumes these COPCs are present at the lower limit of quantitation, also called the Reporting Limit, which is a concentration above their detection limits. The limit of quantitation is the lowest point on the standard curve of the analytic method, and can be well in excess of the method detection limit.

2.10.1 Metals

Metals cannot be generated in the OB and OD process, and the following metals are not detected in the OB/OD tests: barium, cobalt, magnesium, mercury, selenium and thallium. These metals may only be found in ATK's emissions if they are present in the original waste stream.

Barium is a metal with low toxicity, and it is evaluated quantitatively. Cobalt is also evaluated quantitatively. Magnesium is eliminated from the risk assessment because it is considered an essential nutrient and is generally considered safe, and is not evaluated quantitatively.

In March 2014, ATK analyzed propellant for the presence of mercury and it was not detected at a method detection limit of 0.03 mg/kg or 30 micrograms per kilogram (ATK, 2014b). The most abundant form of mercury is inorganic mercury (II), and an emissions factor is determined for the quantitative evaluation of mercury. The Lakes software evaluates mercury as particulate bound, and mercury vapor, and based on the analysis of ATK's waste stream the actual amount of mercury in emissions is expected to be low. Dimethyl mercury is not expected as this is known to be a form of mercury formed by biological methylation in aquatic sediment environments, and will not be formed at ATK. Selenium is a low abundance metal that is evaluated quantitatively. Thallium is also evaluated quantitatively because there is current dose response information available.

2.10.2 Alcohols, Phenols and Ethers

Alcohols are used in many laboratories; low molecular weight alcohols are relatively volatile, and alcohols burn readily. Ethers are used as solvents and also burn easily, but the high molecular weight ethers are not used by ATK. Phenols are aromatic alcohols used in the manufacture of synthetic organic compounds, such as dyes, pharmaceuticals, and agricultural products. ATK does not use phenols in its propellant manufacturing process. Phenols are used in carbon cloths that wrap motors, and are included in the risk assessment. Some phenol and chlorophenol compounds are detected in the OB/OD studies and are also included in the HHRA. Pentachlorophenol is a wood preservative and is not used by ATK; however, this compound is evaluated quantitative. Alcohols, phenols and ethers are also evaluated quantitatively.

2.10.3 Aldehydes

The broad class of aldehydes listed in the test study analyte list is not used by ATK, and the non-detected aldehydes are evaluated quantitatively.

2.10.4 Amine, Aniline, Hydrazine and Benzidine Compounds

Amines are found in some polymer components and in some epoxy-based resins that are used in rocket motors. These polymer materials may have amines present, but only low levels of free amine would be present in these polymer matrices; however, amines are evaluated quantitatively.

Hydrazine is used as a liquid fuel in some rocket motors, specifically in the power nozzles, direction and control systems. It is not used in solid rocket fuels. Liquid propellants containing hydrazine are not treated by OB/OD at the Promontory facility. Hydrazine is highly flammable, especially in combination with other fuels, and if it were present it would be rapidly destroyed. Based on comment-responses with the Utah DSHW, hydrazine and diphenylhydrazine are not considered quantitatively in the risk assessment.

Benzidine compounds (benzidine, 3,3'-dimethylbenzidine and 3,3'-dichlorobenzidine) are not contained in ATK's wastes, nor would they be found in ATK's emissions. The predominant uses for benzidine and benzidine-compounds were in the production of dyes, especially azo-dyes in the leather, textile, and paper industries. Benzidine, and benzidine-compounds are no longer produced for commercial sale in the United States. In 1973, Occupational Safety and Health Association (OSHA) regulations banned United States production of benzidine. In addition, benzidine is no longer imported into the United States nor is it used in any significant amounts by industry (Agency for Toxic Substances and Disease Registry; ATSDR, 1995). ATK does not import or use benzidine and benzidine-compounds, and they would not be found in ATK's waste streams. Benzidine, and benzidine compounds, are reactive and relatively unstable, and would not be produced in the highly reactive conditions of a propellant incineration.

The emissions data from the OB/OD test burns using some propellant/waste bundles showed no presence of benzidine. Based on the lack of detections, and the unstable nature of the benzidine molecule, and the high temperatures involved, benzidine and benzidine-compounds are not believed to be present, and are not quantified.

2.10.5 Polynuclear Aromatic Hydrocarbons

PAHs are found in oils, coal tars and coal. They are not manufactured or used by ATK, and are unlikely to be found in ATK's wastes. PAHs are used in chemical and biochemical research, but not in energetics or propellant research. Selected lower molecular weight PAHs are detected as products of incomplete combustion (PICs) during the OB/OD process; they are discussed in greater depth below.

2.10.6 Phthalates

Phthalates are detected in ATK's OB/OD studies. They are a component of polymer formulations and are found in small quantities in ATK's manufacturing process. These compounds are used in the plastics industry, and may be found in some plastic wastes burned by ATK. Phthalates are generally used in small quantities in plastics, and because plastics only represent a small percentage of ATK's waste, they are unlikely to be present

in any significant quantity in the emissions. Small quantities of plastics are used in the manufacture of motors, and a description of the phthalates possibly used by ATK is shown in Table 2-2 of the HHRA Protocol. In earlier guidance EPA recommended always including bis (2-ethyl hexyl) phthalate (BEHP) and di (n-octyl) phthalate (DNOP) in every risk assessment. EPA no longer recommends automatically including phthalates in risk assessments (EPA, 2005a; page 259). In their guidance, EPA indicates that there is no apparent mechanism for phthalate to be formed as PICs by burning other chemical compounds. Two phthalates are detected in the OB/OD tests, and are most likely due to being present in waste or a laboratory artifact because phthalates are commonly found in analytical laboratory background. Guidance indicates that facilities that burn plastics or materials with phthalate plasticizers should carefully consider the potential for phthalate plasticizers to exist in gaseous emissions due to incomplete combustion. The phthalates BEHP and DNOP are detected in ATK's test emissions and phthalates are considered quantitatively.

2.10.7 Nitroaromatic compounds

Nitroaromatic compounds are present in different forms in energetic compounds and motor fuels. Nitroaromatic organic compounds such as 1,3-dinitrobenzene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, nitrobenzene, and pentachloronitrobenzene (or close relatives such as toluenediamine [TDA] and toluene diisocyanate [TDI]-derivatives of dinitrotoluene) are typically associated with explosives. Dinitrotoluene is used to make two products: trinitrotoluene and TDA. Nitrocarbon compounds are a significant component of RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine) and HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetra). Neither of these was detected in the OB/OD tests, and it is unlikely that nitrobenzene compounds are generated during combustion because these compounds contain no aromatic rings. However, trinitrotoluene (TNT) may be present in explosive mixtures processed by ATK. Nitroaromatic compounds are evaluated quantitatively. All of these compounds including RDX and HMX are energetically unstable. Understandably no residues of these compounds are detected. All of these compounds fuel the burning process and are likely consumed by the process. HMX and RDX are unlikely to survive the process and they are not evaluated quantitatively. Because of their reactivity, it is unlikely that HMX, RDX, and TNT residues result from incomplete treatment, and these explosives will be added to the list of analytes to be monitored under the operating permit.

2.11 Step 3 Is the Non-detected Chemical Likely to be a Product of Incomplete Combustion?

Step 3 asks, "*Does non-detect have a high potential to be emitted?*" It is hypothetically possible that PICs are released in the OB/OD process, and this subsection discusses the

possibility that non-detected COPCs might be formed. The guidance asks if a non-detected chemical has a "high potential" to be detected. Table 2-2 of the HHRA Protocol shows non-detected chemicals and whether they will be included in the risk assessment, even though the majority of these chemicals are not used by ATK, and are unlikely to be generated. Where indicated, these chemicals are included because they are on the analyte list of the OB/OD test analyses. With only a few exceptions, these chemicals are evaluated quantitatively. For those not evaluated reasons are provided in the text below.

2.11.1 Metals

Metals cannot be generated in the OB/OD process. And, ATK does not routinely burn metals. However, metals such as chromium and nickel may be found in emissions due to their presence in the igniters, and the test pans, or trace amounts in contaminated waste. The metals could be aerosolized in the heat of the test. Metals will be evaluated quantitatively in the HHRA, but nickel and chromium are believed to be test artifacts, and are discussed in the uncertainty section.

2.11.2 Polychlorinated Dibenzo(p)dioxins and Dibenzofurans

Some dibenzodioxins and dibenzofurans (dioxins and difurans) are detected in the OB/OD studies, and all dioxins and difurans are evaluated quantitatively. Consistent with EPA, 2005a, dioxins are assessed using the Toxic Equivalent Factor (TEF) method. These constituents are generated in the burning process and it will be assumed that all 210 individual congeners in the 7--dioxin congener groups and 10-difuran congener groups, for which there are TEFs, are produced.

2.11.3 Polynuclear Aromatic Hydrocarbons (PAHs)

PAHs are a broad class of chemical compounds, ranging from two aromatic ring compounds, such as naphthalene, to complex six (or more) aromatic ring compounds such as benzo(ghi)perylene. PAHs are found as by-products in many combustion processes and have been found in some of ATK's emissions.

2.11.4 1.3-Class Propellant PAH Emissions

The 1.3-Class OB/OD tests evaluated 100 % propellant, 85 percent propellant—15% trash, and 65% propellant—35% trash to determine emissions. In these tests, PAHs are generated and are found in emissions gases. The lower the number of aromatic rings the more frequently the PAHs are detected. For example, the two aromatic ring PAH naphthalene are detected in 16 of 18 samples, compared with three ring aromatic PAHs, phenanthrene (detected in 8 of 18 samples), and the three aromatic ring fluoranthene, (four rings total) was detected in 5 of 18 samples. For lower molecular PAHs the risks

and hazards associated with the detected PAHs are evaluated quantitatively using the higher emissions factor of the 1.3-or 1.1-Class propellant (ATK, 2009).

However, the data from the 1.3-Class ammonium perchlorate (AP) propellant indicates that higher molecular weight PAHs are not generated in the OB/OD tests. No chemicals with four, five or six aromatic ring PAHs are detected in ATK's OB/OD studies of 1.3-Class propellants. Based on the research provided below, the presence of aluminum and heat in the OB/OD process restricted the formation of PAHs, and/or led to the destruction of higher molecular weight PAH.

Michael P. Kramer, PhD and Senior Scientist with ATK's Explosives, Propellants and Pyrotechnics Group noted that an OD event can be described as a very rapid and efficient combustion event where the high-energy release rate and good oxygen balance of the explosive only favors the formation of small stable molecules. The entropy term for this rapid energy release drives all explosive materials to form the predicted carbon dioxide, water and nitrogen gas species. In experiments where attempts are made to capture all of the explosive products, no molecules larger than the predicted carbon dioxide (CO_2), water (H_2O) and nitrogen dioxide (NO_2) species are found except for a very small amount of solid carbon. In addition to very efficient energy release, there is also good mixing with the surrounding air, which further promoted the formation of CO_2 and H_2O species (ATK, 2013a).

In an open burning event, the high-energy release in the flame zone and the extended mixing with the surrounding air favored small product molecules. The formation of PAHs required a carbon rich environment with long heating times at temperatures typically lower than open combustion (ATK, 2013a).

ATK's OB/OD test data indicate that these lower molecular PAH compounds are related to the processing of trash because they appeared to increase with the percentage of trash in the test material. The majority of the material processed by ATK is primarily waste perchlorate propellant and high-energy material that does not contain a high volume of trash, and are unlikely to produce significant quantities of PAHs.

It is important to recognize that the mechanism of PAH formation is different from that of dioxin formation in OB and OD processes. Dioxin formation is discussed by EPA in their 2003 document on dioxin toxicity (EPA, 2003a) and is believed to involve a range of pathways and mechanisms dependent on temperature, residence time, and the presence of chlorine radicals, and oxychlorination. Chlorinated dioxins and difurans are energetically relatively stable when formed, and are not easily destroyed after formation. Conversely, PAH formation mechanisms vary with flame substrates, temperature, and combustion precursors. Research using an acetylene or benzene flame system designed

to produce high levels of PAHs indicated that "cyclopentadienyl" is a key species for naphthalene formation, a key intermediate precursor to PAH formation. The further growth process is based on hydrogen abstraction and acetylene addition (Richter, 1999; Richter, 2000). In other words, higher molecular weight PAHs are formed starting with two aromatic ring PAHs, such as naphthalene, and additional rings are added through acetylene addition. The mole fraction of higher molecular PAHs are lower (Richter, 2000) because they require the formation of lower molecular weight PAH as precursors. For example, under these test conditions the peak mole fraction in the Flame I experiments was low ($5x10^{-8}$) (Richter, 1999). Unlike dioxins, higher molecular weight PAHs are not energy sinks, and are actually destroyed at higher temperatures. In fact, a free energy barrier appears in the range 1400^{o}K to 1800^{o}K range, which increased sharply with increasing temperature (Richter, 2000, page 598). It should be noted that the experimental conditions in these tests are significantly different from ATK's open burn and open detonation process, which is deficient in hydrogen and acetylene.

In previous incineration guidance, EPA (EPA, 1994a; 1994b; 1994c; 1998a) recommended evaluating seven potentially carcinogenic PAHs as COPCs (shown in Table 2-2).

The EPA focused on these high molecular weight PAHs because these are often found in samples from incineration stacks, and are associated with soot, wood fires and tobacco smoke. However, EPA, 2005a (page 2-72), states "*Based on the toxicity and combustion chemistry of PAHs, we generally recommend that stack gas testing confirm the absence of these compounds from stack emissions*." ATK's OB/OD studies confirmed the absence of the seven potentially carcinogenic PAHs in the 1.3-Class tests.

In 1.3-Class propellant tests, only three aromatic ring PAHs are detected. As described above, the higher emissions factors of the 1.3- or 1.1-Class AP propellant will be evaluated quantitatively.

Higher molecular weight PAHs are not detected in 1.3-Class tests because incineration at temperatures above 570^{o}F (300^{o}C) with longer residence times, in the presence of aluminum significantly decreases PAHs. As described by Müller, "Naphthalene as well as 15 PAHs of the EPA priority list and some identified methyl-PAH decrease nearly exponentially with increasing aluminum proportion." (Müller, et, al., 1997)

Five and six aromatic ring PAHs are not detected in the 1.3-Class propellant tests. Higher molecular weight compounds are typically associated with burning rubber, tires and other high molecular weight petroleum products, and by comparison low levels are produced from trash (Bjorseth and Ramdahl, 1985, page. 12). They are also more predominant in lower temperature combustion processes, and are found when combustion temperatures

were lower (Bjorseth and Ramdahl, 1985, page. 4). So it is not surprising that the seven potentially carcinogenic PAHs are not detected in the high temperature open burn and open detonation process with perchlorate as the driving force.

Two recent scientific studies provide technical support and show that higher molecular weight PAHs (five and six ring) are not formed in the open burning of AP wastes and munitions. A report on small arm and light weapons ammunition destruction stated:

"With the exception of small quantities of naphthalene and its alkylated sister compounds, emission products larger than the molecules in the EM (Energetic Materials) were not found in the detonation and burn plumes. This is consistent with detonation theory and chemical kinetic mechanisms. It also confirms that collisions between CxHy- radicals (molecular fragments produced by the detonation/deflagration) are the source of the aromatic hydrocarbons. Thus, polycyclic aromatic hydrocarbons containing three or more aromatic rings are not likely to be produced by OB and OD events." (SALW, 2004, page. 4)

A study titled, "Innovative Technology Development for Comprehensive Air Quality Characterization from Open Burning" (SERDP, 2012), shows the levels and types of PAHs from the open burning of a number of five different test rockets and munitions, including Sparrow rocket motors that contained AP, M1, M26, SPCF and M31A1E1. These munitions contained both 1.1- and 1.3-Class propellants. Table 3-3 (page 20) of SERDP shows that no five or six ring PAHs are detected in emissions.

Further, as previously stated, the PW85-15 OB/OD test sample included diesel fuel. The percentage of diesel fuel present in the wastes are quite low, and as shown in analytical data previously provided to the Division, the diesel contains no four, five or six ring PAHs in the original samples. Therefore, no higher molecular weight PAHs are expected in the PW85-15 OB/OD test sample.

Other studies provide evidence that higher molecular weight PAHs are not formed. For example, a study by Mitchell and Suggs (EPA, 1998b) shows that AP mixed with other material containing four percent diesel (Study 7, page 60) are evaluated under OB conditions that captured combustion by-products, including high molecular weight PAHs (page 66) and did not detect PAHs.

It is noted in the modeling protocol (TetraTech, 2011b) that ATK uses desensitizing agents, such as shingle oil and diesel fuel. These items are included in the PW85-15 OB/OD test sample. Benzo(ghi)perylene is a six ring PAH, and is not detected in waste emission for 1.3-Class propellants. As reported by Mitchel and Suggs (EPA, 1998b) the Emission Factors for the Disposal of Energetic Materials by OB/OD, in two of the test

samples identified in Study 2 (Aluminized Propellant Manufacturing Waste Surrogate and Diesel Fuel and Dunnage Surrogate) that contained various amounts of diesel fuel, benzo(ghi)perylene is not reported as a detected compound.

Based on these results, higher molecular weight PAHs do not appear to be formed with 1.3-Class propellant, or they are destroyed, and are not evaluated quantitatively. Further, the detection limit for the 1.1-Class propellant will be evaluated quantitatively where high molecular weight PAHs are not detected.

2.11.5 1.1-Class Propellant PAH Emissions

Low levels of PAHs were detected in the one study conducted by ATK with 65% 1.1-Class propellant mixed with 35% trash. The emissions from 1.1-Class propellants with higher trash levels contained PAHs at low concentrations. These PAHs are evaluated quantitatively using the measured concentration and emissions factor for the detected chemicals. The PAHs detected in 1.1-Class propellant test samples include: anthracene, chrysene, pyrene, indeno(1,2,3,cd)pyrene, chrysene, 2-methylmaphthalene, benzo(a)anthracene, benzo(b)anthracene, benzo(k)anthracene, and benzo(ghi)perylene. These are quantitatively evaluated. Benzo[a]pyrene and dibenzo[a,h]anthracene are not detected in either the 1.3- or 1.1-Class propellant studies. It is assumed that where a PAH is not detected the detection limit from the 1.1-Class propellant study is used to provide emissions factors for quantitative evaluation. The inclusion of emissions factors for non-detected compounds from 1.1-Class emissions tests is to provide ATK flexibility with operations under their permit and minimize or perhaps eliminate the need for a permit modification during the life of the permit.

2.11.6 Methylated PAH Emissions

One low molecular weight methyl-PAH (2-methylnaphthalene) is detected in the 1.1-Class OB/OD test, and this compound is evaluated quantitatively. With the exception of this compound, no other methyl PAHs are detected due to the fact that the formation of higher molecular weight methyl-PAHs are not favored in the mechanism of PAH formation. Based on the mechanism of PAH formation, higher molecular weight methyl-PAHs are less likely to be formed in the high temperature OB/OD process because they would need to form from lower molecular weight methylated-PAH compounds by the addition of one or more PAH. The formation of dimethyl-PAH is statistically even more unlikely because two methyl-PAH would need to react to form a dimethyl PAH. This is borne out experimentally. Müller, *et. al*. (1997) investigated the presence of methyl-PAHs in the OB/OD process in the presence of aluminum and found none. Therefore, higher molecular methyl-PAH (such as dimethylbenzanthracene (a four ring PAH) are not evaluated quantitatively in this HHRA.

2.11.7 2-Acetylaminofluorene and 3-Methylcholanthrene

Another chemical listed in Table 2-2 of the Protocol that appears on the analyte list is 2-acetylaminofluorene. This chemical is a research chemical and is used in chemical synthesis as a reactive intermediate because it is a three ring compound with a reactive functional group attached to the molecule. It is not used by ATK, it is not in their wastes, and it also unlikely to be formed because the reactive functional group would bond with other molecules in the open burn and open detonation process. It is unlikely to survive the open burn and open detonation process, and has not been seen in other studies of munitions and AP wastes (US Army, 2009). This chemical is not evaluated quantitatively.

3-Methylcholanthrene is a research compound used in medical, biochemical and synthetic chemistry research. It is highly unlikely this chemical will be formed in high temperature incineration and it is not evaluated quantitatively.

2.12 Step 4 Are there: Related site-specific factors and is it possibly emitted?

From a site-specific perspective it is important to re-emphasize that ATK's process destroys material that is highly energetic, ATK's process burns rapidly with intensity by generating a hot flame under controlled burn conditions. Perchlorate is a strong oxidizer and provides a significant boost to contaminated waste incineration. The burning of trash or contaminated waste is not the objective of ATK's process; the objective is the disposal of perchlorate based propellant results from "off-specification" rocket motors and fuel, missile rocket fuel and laboratory waste contaminated with lower levels of energetic wastes. Apart from the issues discussed above, there are no site-specific factors that would lead to the inclusion of other COPCs.

Particulate emissions associated with the ATK facility are modeled for each worst-case scenario from M-136 and M-225. Particulate Matter (PM) smaller than 2.5 ($PM_{2.5}$) and 10 (PM_{10}) micrometers were modeled and compared to the NAAQS for $PM_{2.5}$ and PM_{10} (CB&I, 2013). No exceedances of the NAAQS for $PM_{2.5}$and PM_{10} were shown at the ATK property boundary when comparing the modeled concentration for $PM_{2.5}$ and PM_{10} to the respective NAAQSs (CB&I, 2014).

2.13 Category E/Flare Wastes

ATK produces three types of Military Flares; infrared illumination, visible illumination, and decoy/countermeasure flares. Waste illuminate and contaminated material resulting from the manufacturing process is treated by OB. The volume of these waste streams varies depending on the contracts requested by the Military. The quantity of these waste streams has varied from 40,000 – 165,000 lb/yr. over an eight year period.

ATK conducted a review of the Munitions Items Disposition Action System (MIDAS) database and available U.S. EPA AP-42 (EPA. 2009) ordnance emission factors. The review included a comparison of ATK flare waste constituent data to constituent data available from the MIDAS database for the ordnance items found in AP-42 Section 15.3 for large cartridges. Based on the review, ATK selected emission factors for the M816, 81-mm Infrared (IR) Illumination Cartridge. The constituent profile for the M816 Illumination Cartridge was found to be most representative of the ATK flare wastes. The M816 81-mm IR Illumination Cartridge emission factors were presented to the Utah-DSHW and approved for use as emission factors for ATK's flare waste material.

After further review of the M816 81-mm IR Illumination Cartridge emission factors, it was agreed with the Utah DSHW that the emission factors contained in Table 2-1 represented a more conservative set of emission factor then the M816 81-mm emission factors and that the emission factors contained in Table 2-1 would be representative for ATK's illuminant waste material.

ATK's illuminant waste material does contain a few different elements other than those contained in the traditional propellant material such as: boron, bismuth, cesium, indium, iron, silicon, tin, zinc and zirconium. ATK was asked to review the potential risk associated with these compounds. These compounds are discussed in the following paragraphs and in Table 2-3 Potential Elements/Compounds in Flare Wastes.

In the absence of these factors, ATK believes the use of the proposed Emissions Factors (Table 2-1) is appropriate to represent these wastes because the amount of this waste stream is very insignificant compared to the magnitude of the 1.3-propellant wastes that has been treated over the last eight year period. The maximum quantity of this waste stream is approximately one-half the maximum quantity of 1.1-Class propellant waste that has been treated over the last eight year period. And although the amount of flare wastes process by ATK is higher in 2013 than in previous years, this higher level of flare wastes is not expected to continue.

Flare formulations are both proprietary and subject to non-disclosure for security reasons. However, the primary propellant is 1.3-Class material with other components added, and these components would represent an even smaller amount of material.

Some of the wastes contain the elements, or compounds of: boron, bismuth, cesium, indium, iron, silicon, tin, zinc and zirconium. The toxicological information on many of these chemicals is unavailable, and information that is available is provided in Table 2-3. None of these chemicals appear to be carcinogenic, or carcinogenicity data is unavailable. With the exception of zirconium, the chemicals have relatively low toxicity. This is shown by comparison to iron, which has an oral reference-dose (R_fD) of 0.7 mg/kg-day,

and which is a nutritionally required element. All of the elements or compounds are in a similar range to this R_fD, with the exception of zirconium. This element would represent such a small proportion of the waste as to be trivial. See Table 2-3 Potential Elements/Compounds in Flare Wastes.

2.14 Emissions Factors

The 2006 tests for 1.3-Class propellants are published 2009, in *Sampling Results for Emissions Characterization of Open Burning Waste Propellant Materials,* (ATK, 2009). The 1997 (results published in 1998) tests using 1.1-Class propellant test samples were prepared with 65-percent 1.1-Class propellant and 35-percent waste material. To be conservative, the emissions factors from both tests are compiled and brought into the COPC selection process. The higher emissions are selected, except in very specific cases. Emissions factors were considered in light of EPA guidance (EPA, 1998a).

In addition, from time to time, illumination flare wastes are present in low amounts in ATK's wastes. As discussed in the previous section, after an analysis of available U.S. EPA AP-42 (EPA. 2009) ordnance emission factors it has been agreed that the Table 2-1 represent a more conservative analysis of these wastes. The illumination flare waste contains compounds that are evaluated by comparing the toxicity of these chemicals to the relative toxicity of other chemicals that were present in the OB/OD tests, for which risks are calculated.

The *Sampling Results for Emissions Characterization of Open Burning Waste Propellant Materials* (ATK, 2009) report provides details of the wastes processed by ATK, and describes the study used to determine the nature of the emissions from the processing of these wastes. These test results form the basis of the emissions that are modeled, as described in *Air Dispersion Modeling Protocol for Open Burning and Open Detonation at ATK Launch Systems in Promontory, Utah* (CB&I, February 2013), and its associated Addendum (CB&I, June, 2013).

With the goal of keeping this risk assessment relatively simple and understandable, yet conservative, that is, to capture a reasonable maximum exposure and risk level from ATK's process, emissions factors are selected in accordance with the following process.

In summary, one emissions factors table (Table 2-1) has been developed. It summarizes the emissions factors that are used in the process. To capture the potential emissions from a wide range of wastes, and allow for flexibility in operations, but with the aim of being also conservative the following process was used:

- Where a constituent is detected in either the 1.3- or 1.1-Class OB/OD test, the emissions factor is developed from the highest detection in all of the tests. No averaging is used.
- Where one test showed the presence of a chemical and the other did not, the chemical is assumed to be present at the level detected, and the emissions factor is based on the detected concentration.
- Where a chemical is not detected in any OB/OD test (1.3- or 1.1-Class) the highest detection limit is used to develop the emissions factor, with the following exceptions:
- Where higher molecular weight PAHs are not detected, and are not formed in 1.3-Class waste emissions, the detection limit for 1.1-Class wastes is used.
- Dioxins and difurans are evaluated as classes of compounds, and the 1.3-Class detections are used because they lead to a higher 2,3-7,8-tetrachlorodibenzo-dioxin toxic equivalent factor (TCDD-TEQ), and so higher risk.
- Benzidine, 3,3´-dimethyl/dichlorobenzidine, 2-acetylaminofluorene, 3-methylcholanthrene and 7,12-dimethylbenzanthracene are eliminated from the process for reasons discussed above. No emissions factors are provided for these constituents.
- The chromium in the emission was not speciated to separate and quantify hexavalent chromium (Cr(VI)) from trivalent chromium(Cr(III)), and it was assumed (based on the discussion provided in Section 2.9.1 that 45% of the chromium is Cr(VI) and 55% of the chromium is Cr(III).

The OB/OD tests are conducted with relatively small amounts of pure propellant wastes (2,130 grams (g)), and less propellant/trash wastes (873g). The processing conducted by ATK typically involves 10,000-50,000 lbs per burn, which results in more intense temperatures than seen in the OB/OD studies. Also, the OB process has adequate oxygen because there is no constriction to airflow (ATK Promontory Permit Attachment 11, January 2014 (ATK, 2014b)).

There are a number of key issues for the development of emissions factors from the test profiles, and the OB/OD testing:

- ATK profiles all of the wastes that are sent to the burning grounds to determine the nature of the wastes and to obtain a general inventory of the waste streams.
- ATK's primary waste streams do not contain the metals chromium, lead, mercury, or nickel, and ATK strives to keep these metals out of their wastes. There are some exceptions: laboratory waste of barium chromate, and lead

fulminate, these are present in such low quantities (milligram quantities in contaminated waste); and from the Group D, profile #PR49 (primary explosive with lead) which contains lead styphnate at 44% lead and lead azide at 71% lead. Even though there are more of these wastes than for lead fulminate, they are not considered consequential relative to the thousands of pounds processed by ATK in a single burn.

- An emissions factor for lead is provided, but there are no dose-response factors available for use in the Lakes model. The risks is evaluated by comparing the Lakes model calculated soil lead concentrations to the US EPA default residential lead goal of 400 milligrams per kilogram (mg/kg). Lead has been shown to have neurological effects, and young children are particularly susceptible to the effects of lead. The US EPA evaluates lead using a bio-uptake model call the Integrated Exposure Uptake Bio-kinetic (IEUBK) model, which calculates potential blood lead concentrations based on exposure to media, food and water. The estimated blood lead levels are compared with acceptable blood lead levels for children. This model was used by the EPA to calculate a soil lead level below which no adverse effects on children would be expected. This acceptable soil lead concentration is 400 mg/kg (EPA, 2002). The calculated soil lead concentrations at all locations are well below (by a factor of several thousand) than 400 mg/kg and no further processing is necessary. More details on the soil lead concentrations and the comparison to the EPA's acceptable lead level are provided in Section 9.6.

The class of chemicals called dioxins is a mixture of similarly structured compounds or congeners, with many different congeners in the class. When selecting the emissions factors for this class of compounds the data were examined to determine which class of AP propellant (1.1-Class or 1.3-Class) would have the highest risk, and selecting that Class. This was achieved by multiplying the emissions (pound of dioxin per pounds of waste processed) for each class of dioxin and difurans by the 2,3,7,8-TCDD—Toxicity Equivalence Factor (TCDD-TEF) (shown in Table 2-4), and summing the results for all compounds in the class. The emissions factors for each class, the TCDD-TEF, the method used, and the results of the analysis are shown in Appendix B.

Based on the calculation in Appendix B, the 1.3-Class propellant emissions factor has more dioxin risk than the 1.1-Class, and therefore the 1.3-Class emissions are considered the higher, and are used to represent emissions from ATK materials.

2.15 **Identifying Emission Rates**

CB&I developed the air quality model, ambient air chemical concentrations and chemical deposition rates (CB&I, 2014a) for the ATK facility based on a unit emissions rate of 1

g/s. In order to calculate the emissions for each source, the emissions factors for each chemical potentially released from the facility is calculated using the emissions rate, in pounds of chemical per pound of waste burned (lb/lb), and the amount of material processed at each station, converted to g/s for the model. Therefore, chemical-specific emissions rates are calculated for each source that is modeled in this risk assessment. The modeling report provides the quantities of waste burned per event, as well as the annual maximum permitted quantity of waste for each of the burn stations at M-136 and M-225. The emissions rates are calculated for the following:

- One-hour average acute air exposure,
- Annual deposition for chronic exposure

2.16 Short-term (1-hour) Emissions Rates

The emissions rate for one-hour acute ambient air exposure are calculated for the amount of waste burned per event for each source, in units of pound per hour (lb/hr). For this scenario, lb/hr is equivalent to a pound per event because a typical burn event lasts approximately one hour. The emissions during the event are given by the following:

$$Emissions\left(^{lb}/_{hour}\right) = lbs\ processed\ in\ 1\ hour \times EF(lb\ /\ lb)\ \ [chemical\ specific]$$

For example, at Source M-136 A1[1]: where 96,000 lb are processed (CB&I, 2014a), and assuming acenaphthene is generated based on its Emissions Factor of 5.48×10^{-7} lb/lb, the emissions would be:

$$Emissions = 96{,}000 \times 5.48 \times 10^{-7} = 5.26 \times 10^{-2}\left(^{lb}/_{hr}\right)$$

That rate is adjusted from units of pounds per hour to grams per second according to the following equation:

$$One - hour\ emissions\ rate\left(^{g}/_{s}\right) = \left[\frac{5.26 \times 10^{-2} lbs\ per\ hour * 453.6\ grams\ per\ lb}{3{,}6000\ seconds\ per\ hour}\right] = 6.63 \times 10^{-3}\left(^{g}/_{s}\right)$$

[1] Source M-136 A1 is comprised of burn stations 1 through 12. Six of the 12 stations located closest to the western property line (Stations 1, 4, 7, 8, 10, and 11) are modeled as six separate sources. For the presented example, each burn station in the source will have a one-hour emission rate of 1.1E-03 g/s.

The resulting emissions rate is used in conjunction with the air dispersion model results to calculate potential ambient air concentrations at the identified receptors. Table 2-1 provides the chemical-specific emissions factor for the project, and the amount processed at each station are provided above. The resulting emissions rates for the chemicals, as well as comma separated values (csv) files for input to the Lakes software, are provided in Appendix A.

2.17 Annual Emissions Rates

The Lakes software is designed to process continuous emissions from emissions stacks and is a 365-days per year model. The model developers (Lakes, 2014) inform us that, at this point in time, it cannot be modified to process batch data, and so is unable to process batch processes, like those at ATK. Therefore, the emissions rates are modified to give an annual emission by assuming the annual amount of material processed at one station averaged over one year.

The emissions rate for annual deposition for chronic exposures are calculated using the annual maximum permitted quantity for each source. For this scenario, the lb/yr is given by the following:

$$Emissions\left(^{lb}/_{year}\right) = lb\ processed\ per\ year \times Emissions\ Factor\left(^{lb}/_{lb}\right)[chemical\ specific]$$

For example, at Station M-136 A1[2]: where 6,720,000 lb/yr (6.72 million lb/yr, above) are processed. This would be equivalent to a daily operation of 18,410 pounds per day, and assuming acenaphthene is generated based on its Emissions Factor of 5.48x10^{-7} lb/lb, the emissions would be:

$$Emissions\left(^{lb}/_{day}\right) = \frac{6{,}720{,}000}{365} \times 5.48 \times 10^{-7} = 1.00 \times 10^{-2}\left(^{lb}/_{day}\right)$$

[2] Source M-136 A1 is comprised of burn stations1 through 12. Six of the 12 stations located closest to the western property line (Stations 1, 4, 7, 8, 10, and 11) are modeled as six separate sources. For the presented example, each burn station in the source will have an annual average emission rate of 8.83E-06 g/s.

That rate is adjusted from units of pounds per day to grams per second according to the following equation:

$$Annual\ emissions\ rate\ \left({}^{g}/_{s}\right) = \left[\frac{1.00 \times 10^{-2}\ lb\ per\ day * 453.6\ grams\ per\ lb}{86{,}400\ seconds\ per\ day}\right]$$
$$= 5.3 \times 10^{-5}\left({}^{g}/_{s}\right)$$

The resulting emissions rate is used in conjunction with the air dispersion model results, to calculate potential ambient air concentrations at the identified receptors. Table 2-1 provides the chemical-specific emissions factor for the project, and the amount processed at each station are provided above. The resulting annual emissions rates for the chemicals as well as csv files for input to the Lakes software, are provided in Appendix A.

3. AIR DISPERSION AND DEPOSITION MODELING

As noted in Section 1 of this HHRA, preliminary modeling was conducted using the OBODM (SERDP, 1998) under a Utah DSHW-approved protocol (TetraTech, 2011b). However, CB&I (2014a) made improvements to the model and a revised version of the model was approved by Utah DSHW in 2014 (CB&I, 2014b). OBODM is specifically designed to predict the air quality impact from OB and OD treatment of obsolete weapons, solid rocket propellants, and associated manufacturing wastes. The OB and OD treatment of waste propellants and propellant-contaminated materials can be classified as instantaneous events for OD treatment and as quasi-continuous events for OB treatment. The model is also designed to use either empirical emission factors such as those derived in the Dugway Proving Ground Bang Box™ studies or emissions predicted by a "products of combustion" model. OBODM calculates peak air concentration, time-weighted air concentrations, and dosage (time-integrated concentration) for OB and OD releases. It can also consider the effects on concentration and dosage of the gravitational settling and deposition of particulates.

However, OBODM has several limitations, which constrain the modeling in this application. For example, OBODM can handle only 100 receptors at a time, it cannot predict deposition in complex terrain, and it uses older algorithms for the calculation of downwind dispersion of COPCs.

To overcome these limitations, ATK proposed a hybrid approach for the air modeling using the OBODM with the American Meteorological Society/U.S. Environmental Protection Agency (EPA) Regulatory Modeling System (AERMOD) model, which is the EPA's preferred dispersion model for short range transport (up to 50-kilometers [km]). OBODM has two distinct parts. The first part simulates the OB and OD events and generates initial parameters of the emission cloud (emission rate, cloud height, cloud diameter), and the second part is the downwind dispersion of the emission cloud. The main limitation of OBODM is in the second part (i.e., dispersion). The downwind dispersion is better handled by AERMOD, which has practically no limitation on number of receptors, can easily handle complex terrain, and handles dispersion of emission clouds based on state-of-the-art understanding of atmospheric turbulence.

Therefore, the revised air quality assessment was conducted using this hybrid approach based on the emission rates and initial source parameters from OBODM and using these parameters in AERMOD to predict downwind dispersion and deposition. The details of this hybrid modeling approach are provided in CB&I, 2014b. Selected sections of this approved work plan are repeated here, or are briefly summarized.

3.1 Emissions Source Parameters

Both the OB and OD events were modeled as elevated volume sources. The source parameters required for dispersion of volume sources are:

- Emission rate
- Release height of vapor cloud
- Initial horizontal and vertical dimensions of the vapor cloud

The methodology for determining these source parameters are based on several discussions with Utah DSHW as summarized in CB&I, 2014b.

3.2 Emissions Rates

As described in Section 2, emission rates are estimated based on the quantity of emissions from the reactive waste in the OB/OD test events. Modeling to assess ambient air quality impacts was conducted using the estimated maximum emission rates from each scenario. To reduce the number of required model runs, the emission rates for a single pollutant (i.e., $PM_{2.5}$) are input to the model, and the results are then applied to the other pollutants by scaling the modeled results by the ratio of the desired pollutant emission rate to the modeled emission rate.

Modeling in support of the risk assessment is conducted at a unit emission rate of 1 g/s to allow for application of pollutant-specific emission rates within the risk assessment software.

3.3 Release Height of Vapor Cloud

3.3.1 Open Burning

The CB&I (2014b) air modeling protocol (Section 4.2) describes the modeling process and the parameters used in the model related to the release height. OB results in combustion of the energetics and rapid rise of the hot combustion products due to buoyancy until a final height is reached. At this point, the emission cloud has no upward momentum and starts to disperse downwind. This event is simulated as an elevated volume source with the release height equal to the final cloud height predicted from OBODM.

Based on several discussions with Utah DSHW and its consultant, the following approach is used for determination of cloud heights for OB events.

All of the unrestricted hours are grouped based on wind speed and stability condition. The wind speeds are grouped in four ranges as identified below:

- Category 0: 3.0 miles per hour (mph) – 5.0 mph
- Category 1: 5.0 mph – 7.5 mph
- Category 2: 7.5 mph – 10.0 mph
- Category 3: 10.0 mph – 12.5 mph
- Category 4: 12.5 mph – 15 mph

Atmospheric stabilities are grouped into six Pasquill-Gifford (PG) atmospheric stability classes for each of the hours in each of the wind speed categories listed above.

The OBODM is used to determine the vapor cloud height for each combination of the PG atmospheric stability class and wind speed categories. The vapor cloud heights are determined for the lower threshold, the higher threshold, and midpoint for each wind speed category. To ensure a conservative impact assessment, the minimum cloud height out of these three wind speeds are considered for each combination of atmospheric stability and wind speed category. This approach is presented in the March 2013 Hybrid Air Modeling Protocol, and has been discussed with and accepted by Utah DSHW (CB&I, 2014b). The approved protocol shows all vapor cloud heights determined by OBODM for each combination of the PG atmospheric stability class and wind speed categories for OB. A summary of the lowest total cloud height for each scenario is also provided in the protocol.

The procedure outlined here for determining the vapor cloud heights specific to meteorological conditions is conducted for each of the scenarios proposed for representing the OB/OD events. In the case of scenarios that consider simultaneous events at multiple burn stations, only one representative burn station was modeled in OBODM for each scenario. The assumed conditions for this burn assumed the most conservative meteorological conditions (lowest dispersion) under normal operating conditions. The resulting vapor cloud heights are then applied to each of the other identical burn stations for that scenario. The results of the open burn model are used by the air dispersion portion of the model, and the outputs are fed into the Lakes risk model (Lakes, 2014b).

3.3.2 Open Detonation

The same procedure described for OB is used to determine vapor cloud height for OD using the OBODM. Attachment 1 of the approved protocol shows all vapor cloud heights

determined by OBODM for each combination of the PG atmospheric stability class and wind speed categories for OD. A summary of the lowest total cloud height for each scenario is also provided there.

3.4 Initial Dimensions of Vapor Cloud

3.4.1 Open Burning

During rapid rise of the cloud from the OB, atmospheric air is entrained and the dimension of the cloud increases. OBODM does not calculate the initial release diameter for quasi-continuous releases, such as open burns. Therefore, site observations of burn operations are utilized in estimating this parameter. Based on videos of the open burning events, the final dimensions of the cloud at final plume height are typically four to eight times larger than the dimension of the burn pans. As a conservative estimate, the cloud diameter is based on four times the equivalent diameter of the burn pans. Because the burn stations have multiple adjacent burn pans, the equivalent diameter is based on the total area covered by the reactive waste. It is assumed that the vapor cloud plume is a sphere.

The burn pan layout is estimated to be four burn pans in a square pattern. Due to the circular shape of the cloud plume, the burn pan equivalent diameter is estimated assuming the burn pan area is circular.

Based on the Utah DSHW-approved model developed for the project by CB&I (2014) and per AERMOD guidance, the initial vertical and horizontal dimensions of an elevated volume source, such as the vapor cloud, is calculated by dividing the initial cloud diameter (i.e., four times equivalent diameter covered by reactive waste on burn pans) by a factor of 4.3. Detailed parameters for all scenarios are provided in the air modeling protocol (CB&I, 2014).

3.4.2 Open Detonation

The initial dimension of the vapor cloud is obtained directly from the OBODM output. As described earlier in this document, OBODM is used to model each combination of wind speed category and PG atmospheric stability considered in this phase of the analysis. OBODM yields the same initial vapor cloud diameter for each OD scenario modeled, regardless of the meteorological conditions considered. Calculation of vapor cloud dimensions for buoyant sources is described in Section 2.6.3 of Volume 2 of the OBODM User's Guide, (WDTC, 1998b; page 27). The initial vapor cloud diameter for detonations, as presented in the OBODM output, appears to be calculated using

Equation 2-75 of the OBODM User's Guide and is assumed to represent the initial diameter of the cloud immediately after detonation. Note that the initial diameter is the only lateral dimension reported in the OBODM output. Equation 2-75 determines the initial radius as a function of the quantity of material detonated, the effective heat content of the material detonated, and the ambient temperature, among other parameters. For this analysis, the ambient temperature used in OBODM is a default temperature of 293 degrees Kelvin (^{o}K). The modeled ambient temperature is kept constant for each combination of wind speed category and atmospheric stability evaluated. Based on a cursory analysis, variations in the ambient air temperature do not have a significant effect on the initial diameter determined by OBODM. However, at this time, OBODM is not actively supported by its developers or by any regulatory agency, and it is not entirely clear from the User's Guide how the equations presented in Section 2.6.3 are used by the model. The application of the equations for determining cloud dimensions using OBODM is considered an area of uncertainty in this analysis.

Per AERMOD guidance, the initial vertical and horizontal dimensions of an elevated volume source, such as the vapor cloud, are calculated by dividing the initial vapor cloud diameter by a factor of 4.3.

3.5 Other Source Parameters

3.5.1 M-136 Stations

Burn Stations 1 through 12 are clustered within 100 m of each other. Six of the twelve stations located closest to the western property line (Stations 1, 4, 7, 8, 10, and 11) are modeled as six separate sources. Burn Stations 13 and 14 are modeled separately. The dimensions of M-136 stations are provided in Section 2.5, and are shown on Figure 1-3.

3.5.2 M-225 Stations

M-225 Burn Stations 1 through 4 are clustered within 100 m and are modeled as a single source located approximately at the center of the cluster. The OD pit is modeled separately. The dimensions of M-225 stations are provided in Section 2.6, and are shown on Figure 1-3.

3.6 Summary of AERMOD Modeling Parameters

Based on the information, a summary of the actual parameters used for modeling are described below.

3.6.1 M-136 Stations

The source parameters for M-136 are shown in Table 3-1 of this report.

To determine the emission rates used in AERMOD, the maximum emission factors described in Section 3.0 are multiplied by the daily quantity burned for each scenario. The actual emission rates are determined based on the reactive waste for each scenario and assuming only one event would occur per hour. A sample calculation for PM_{10} for Scenario M-136 A-1 is shown Example 3-1.

Example 3-1:

- M-136 A-1 reactive waste: 96,000 lb
- Maximum PM10 emission factor from Table 3-2 of the air modeling report (CB&I, 2014): 0.12 lb/lb reactive waste

$$Emission\ Rate\ \left(\frac{lb}{event}\right) = 96{,}000\ lb\ reactive\ waste * 0.12\frac{lb}{lb\ reactive\ waste} = 11{,}520\ lb/event$$

Since AERMOD considers emissions to be continuous over one hour and based on the assumption that each event would occur within one hour, the lb per event is equal to the lb per hour emission rate.

$$Emission\ Rate\ \left(\frac{lb}{event}\right) = Emission\ Rate\ \left(\frac{lb}{hr}\right) = 11{,}520\frac{lb}{hr}$$

Converting to grams per second:

$$Emission\ Rate\ \left(\frac{g}{s}\right) = 11{,}520\frac{lb}{hr} * \frac{453.6\ g}{lb} * \frac{hr}{3600\ s} = 1{,}451.5\frac{g}{s}$$

The actual emission rates for NAAQS and air toxics are discussed in Section 4.0

3.6.2 M-225 Stations

The source parameters for Scenarios M-225 are shown in Table 3-2 of this report.

To determine the emission rates used in AERMOD, the maximum emission factors described in Section 3.0 are multiplied by the daily quantity burned for each scenario. The actual emission rates are determined based on the reactive waste for each scenario and assuming only one event would occur per hour. A sample calculation for PM_{10} for

Scenario M-225 A is shown Example 3-2.

Example 3-2:

- M-225 A reactive waste: 4,500 lb
- Maximum PM10 emission factor from Table 3-2 of the air modeling report (CB&I, 2014): 0.12 lb/lb reactive waste

$$Emission\ Rate\ \left(\frac{lb}{event}\right) = 4{,}500\ lb\ reactive\ waste * 0.12 \frac{lb}{lb\ reactive\ waste} = 540\ lb/event$$

Since AERMOD considers emissions to be continuous over one hour and based on the assumption that each event would occur within one hour, the lb per event is equal to the lb per hour emission rate.

$$Emission\ Rate\ \left(\frac{lb}{event}\right) = Emission\ Rate\ \left(\frac{lb}{hr}\right) = 540 \frac{lb}{hr}$$

Converting to grams per second:

$$Emission\ Rate\ \left(\frac{g}{s}\right) = 540 \frac{lb}{hr} * \frac{453.6\ g}{lb} * \frac{hr}{3600\ s} = 68.0 \frac{g}{s}$$

The emission rates for NAAQS and air toxics are shown in Section 4.0.

4. COMPLIANCE WITH NATIONAL AMBIENT AIR QUALITY STANDARDS AND UTAH TOXIC SCREENING LEVELS

In addition to preparing and reporting a human health risk assessment for the open burning process, ATK's emissions are required to comply with current clean air regulations, two of which apply in this case: the NAAQS and the Utah TSLs. This work was conducted by CB&I under an Utah DSHW-approved protocol, and is presented in CB&I, 2014b. However, selected tables from the CB&I report are repeated here to compile all of the associated air quality compliance issues in one location.

4.1 National Ambient Air Quality Standards

To determine if facility operations are in compliance with the NAAQSs, CB&I developed a Utah DSHW approved modeling protocol to determine ambient air concentrations of the criteria pollutants and compared these concentrations with the standards for the agreed period. A summary of the criteria pollutants considered for NAAQSs analysis is provided in Table 4-1. A summary of the sources of the NAAQSs is provided in Section 2.

Although each pollutant and averaging period has its own method to determine the design value, for this analysis, each maximum one-hour impact was averaged over the five-year period to obtain an average maximum one-hour impact for NAAQS analysis for all pollutants except PM_{10} and annual NO_2. This methodology is conservative for NAAQSs. For PM_{10} and annual NO_2, the maximum impact over the five-year period of one-hour and annual average concentrations is considered, respectively.

NAAQSs compliance is demonstrated by comparing the design-modeled concentration for all pollutants and averaging times with the respective NAAQS. No background concentrations were added to the design modeled concentration because, as mentioned in CB&I, 2014b, the emission factors used for the modeling included background concentration. For all averaging times and pollutants, sub-sources M-136 A and M-225 A had the highest impact. The design modeled cumulative results are shown in Table 4-2. Table 4-2 also shows that none of the criteria pollutants exceeded the NAAQS, and compliance was demonstrated in CB&I, 2014b.

4.2 Compliance with Utah's Toxic Screening Levels

Air toxics included in the preliminary modeling dated March 2012 (TetraTech, 2012) were compared to respective Utah TSLs. The acute toxics and corresponding TSLs considered are listed in Table 3-18 of the July 2012 preliminary modeling report. The maximum one-hour concentrations are averaged over the five-year period and compared to the acute TSLs. The chronic air toxics and corresponding TSLs are listed in Tables 3-35 and 3-52 of the preliminary modeling report dated July 2012. The maximum 24-hour

concentrations are averaged over the five-year period and compared with the chronic TSLs. The acute 1-hour and chronic 24-hour TSLs are shown in Table 4-3, and Table 4-4 respectively, with the associated air concentration and an indication of whether the COPC exceeds the TSL.

It can be seen from Table 4-3 and Table 4-4 that none of the TSLs are exceeded.

5. EXPOSURE SCENARIO IDENTIFICATION

The exposure assessment identifies the exposure scenarios that are evaluated in the HHRA to estimate the type and magnitude of human exposure to COPC emissions from the OB/OD treatment units. An exposure scenario is a combination of exposure pathways to which a single receptor may be subjected. Human receptors may come into contact with COPCs emitted to the atmosphere via two primary exposure routes, either directly via inhalation; or indirectly via subsequent ingestion of soil, vegetation, and animal products that might became contaminated by COPCs through uptake into the food chain.

Exposure to COPCs may occur via numerous exposure pathways. Each exposure pathway consists of four fundamental components: (1) a source, (2) a mechanism of COPC release and transport by environmental media; (3) a point of potential human contact with the contaminated medium; and (4) a route of entry into the human body. Humans, plants, and animals in the assessment may take up COPCs directly from the air or indirectly via the media receiving deposition, and uptake into biota that are subsequently consumed.

The exposure scenarios recommended for evaluation in EPA's HHRAP guidance are generally conservative in nature and are not intended to be entirely representative of actual scenarios at all sites. They are intended to allow for standardized and reproducible evaluation of risks across most sites and land use areas, with conservatism incorporated to ensure protectiveness of potential receptors not directly evaluated, such as special subpopulations and regionally specific land uses.

The risk assessment exposure assumptions in the Lakes software are based on the default assumptions available in 2005 when the HHRAP guidance was published. In February 2014, the Office of Solid Waste and Emergency Response (OSWER) issued Directive 9200.1-120 (EPA, 2014a), which revised a number of the default risk assessment assumptions. The assumptions that changed are incorporated into the Lakes software and are discussed in Section 7.

5.1 Characterizing the Exposure Setting

Risks are characterized for the maximum vapor phase and deposition concentration location(s) with a general grid of 10 kilometers (km) from each treatment unit and at discrete receptor locations. The general receptor grid is discussed in Section 4.6 of the air dispersion modeling protocol. The general receptor grid is used to determine the maximum 1-hour and annual vapor and deposition concentration location(s) within and beyond the ATK facility boundary. Based on prior experience modeling for OB/OD treatment units in flat and complex terrain, the location of the maximum impact always

occurs within 3 km of the source. Consequently, no general grid receptors are proposed beyond a 10 km radius from each treatment unit. It should be noted that while the general grid is extend only to 10 km, OBODM also estimates short term and annual contaminant concentrations at discrete receptor locations potentially impacted by M-136 and M-225 emissions. Discrete receptors are defined as special receptors that exist within or outside of the general grid. The discrete receptors that are evaluated this risk assessment are consistent with the types of receptors recommended in the HHRAP guidance, as well as those requested for evaluation by the Utah DSHW. The discrete receptors in this risk assessment are shown in Table 5-1 and briefly described here:

On-site Receptors

- Autoliv Facility. This is the offsite commercial business at a location between the M-136 and M-225 treatment units.
- North Plant Main Administration Building and Main Manufacturing Area at a location 2.5 miles north of M-136 and 6.7 miles north-northwest of M-225.
- South Plant Main Administration Building and Main Manufacturing Area at a location 1.8 miles south of M-136 and 3.9 miles west-northwest of M-225.

Off-site Locations without Actual Receptors

- Point of Maximum Deposition
- Four facility boundary receptors that are selected based on the annual prevailing wind directions that are measured over a five-year period (1997 through 2001) at the M-245 meteorological monitoring station.
- Blue Creek, which is a perennial stream which is an additional boundary receptor located along the western property boundary. It was originally selected as an ecological receptor. The water is poor in quality because it is high in total dissolved solids and minerals. Human receptors are not frequently in this area, however, there is the potential for a rancher to move cattle along the stream so the location was included in the HHRA.

Off-site Locations with Actual Receptors

- The Adam's Ranch, which is the closest dwelling to M-136.
- The Holmgren Ranch, which is the closest domestic dwelling to the M-225.
- The Howell Dairy Farm just north of the ATK northern property boundary.
- Christensen Residence. This residential dwelling is at a location due north of ATK.
- The Town of Thatcher is at a location about 6 miles northeast of ATK.

- The Town of Penrose at a location about 4 miles east of ATK.
- The ATK Ranch Pond, which is at a location southwest of M-225.

In addition, at the request of the Utah DSHW a qualitative evaluation of risk is performed at the following locations and presented in the uncertainty section of the HHRA:

- Salt Creek Waterfowl Management Area
- Bear River Migratory Bird Refuge

All discrete receptors listed above are shown in Figure 5-1. The spring pools (Shotgun, Pipe, Fish, etc.) located south of the facility along Highway 83 are not selected as discrete receptor points for the human health risk assessment because the water in the spring pools is not used as a drinking water source and there are no game fish present in these water bodies.

5.1.1 Current and Reasonable Potential Future land use

The current land use is considered in this risk assessment, i.e., on-site workers, and off-site residents (farmers and residents). In addition, while farmers or residents are not currently located within the facility boundaries it is possible for the area within the facility boundaries to be developed for residential or agricultural use if the facility was closed in the future. Risks would be estimated and reevaluated at some point in the future if the facility closed and on-site redevelopment took place. Therefore, future exposures considered in this HHRA include the evaluation of a farmer and resident scenario at the point of maximum risk located off-site.

5.1.2 Water Bodies and their Associated Watershed

There is a creek on the western side of the Facility, but the water is of poor quality because it has high total dissolved solids and minerals. It is a small stream that has eroded a channel approximately 20 feet deep where it runs across ATK property, making it largely inaccessible to cattle and humans. While the water in Blue Creek is potentially accessible by livestock approximately 5 months of the year in the fall and winter, it is not the only water source; ATK provides clean water to the ranches that water cattle in that area. As stated in Section 3.2 of the approved HHRA protocol, the HHRAP guidance recommends not evaluating ingestion of water by animals, because it is expected that the contribution of that pathway to the total risk is negligible compared to the contributions of the recommended exposure pathways for cattle which include ingestion of contaminated forage, silage and grain and incidental ingestion of soil. In addition, there are some small natural ponds formed from natural springs at the southern end of Promontory. These springs do not support game fish. Therefore, based on discussions with Utah DSHW

concerning Blue Creek and the springs, the decision was made to follow the HHRAP guidance concerning the recommended exposure pathways.

5.1.3 Sensitive Sub-populations

HHRA guidance recommends evaluating sensitive sub-populations that might exist in the area for particular attention in a risk assessment. Sensitive sub-populations are segments of the population that may be at higher risk due to increase sensitivity and/or increase exposure to COPCs. For example, ATK has the intention of providing daycare for the children of employees at the facility at some point in the future. This daycare, if made available, would likely be located at the South Main Plant Administration Building, and would represent a potential sensitive receptor. This potential receptor has been evaluated as a separate scenario and is provided in Appendix G to this HHRA.

5.2 Site-specific Acute Exposure Scenarios

This scenario accounts for short-term effects of exposure to maximum 1-hour concentrations of COPCs in modeled emissions. Acute exposures are evaluated for all of the discrete receptors listed in Table 5-1. These exposure estimates are unique to each of the receptor locations, and are independent of the type of receptor that exists at each location (resident, farmer, or worker).

5.3 Site-specific Chronic Exposure Scenarios

5.3.1 On-site Worker

The industrial worker scenario is evaluated to account for exposure to COPCs during a workday. The exposure pathway is direct inhalation of particulates and vapors, as shown in Table 5-2. Risks and Hazards for the industrial worker are calculated using EPA standard default exposure assumptions. It is assumed that the industrial worker works 8 hours/day, 250 days/year for 25 years. Air concentrations are calculated by the Lakes software and represent annual average concentrations. Risks are calculated for a current worker at the Autoliv facility, the North Main Plant Administration, and the South Main Plant Administration buildings. In addition, risks are calculated for a future worker at the location of the maximum on-site impact.

5.3.2 Farmer Adult and Farmer Child

A subsistence farm scenario is evaluated in this risk assessment. The farmer exposure scenario is evaluated to account for the combination of exposure pathways to which a receptor may be exposed in a farm or ranch exposure setting. The farmer is assumed to

be exposed to COPCs emitted from the facility through the following exposure pathways, also shown in Table 5-3:

- Direct inhalation of vapors and particles
- Incidental ingestion of soil
- Ingestion of homegrown produce
- Ingestion of homegrown beef
- Ingestion of milk from homegrown cows
- Ingestion of homegrown chicken
- Ingestion of eggs from homegrown chicken
- Ingestion of homegrown pork
- Ingestion of breast milk (evaluated only for dioxins/furans)

For the farmer scenario, the receptor is assumed to consume a portion from each food group to make up a total consumption rate, and all amounts consumed are assumed to be homegrown, even though the soil and water in the area will not support this assumption (see Section 10, the uncertainty section).

The exposure pathways are the same for the farmer adult and child. The primary difference is that the adult is assumed to be exposed for 40 years and the child is assumed to be exposed for 6 years. Also the consumption rates of homegrown food are assumed to be about half that for a child versus an adult.

5.3.3 Resident Adult and Resident Child

The residential scenario is evaluated to account for the combination of exposure pathways to which a receptor may be exposed in an urban or rural (nonfarm) setting. The resident is assumed to be exposed to COPCs from the emission source through the following exposure pathways, also shown in Table 5-3:

- Direct inhalation of vapors and particles
- Incidental ingestion of soil
- Ingestion of homegrown produce
- Ingestion of breast milk (evaluated only for dioxins/furans)

The exposure pathways are the same for the resident adult and child. The primary difference is that the adult is assumed to be exposed for 26 years and the child is assumed

to be exposed for 6 years. Also the consumption rates of homegrown produce are assumed to be about half that for a child versus an adult.

5.3.4 Wildlife Areas

Prior to the development of the first HHRA protocol, the areas where hunting might occur were discussed and based on the available modeling data, the distance from the source areas to the nearest hunting area, and the amount of meat that might be ingested by a hunter compared with an adult farmer it was agreed with USDHW that the risks would be *de minimis*. To further substantiate this, the Lakes model assumptions were reviewed and the farmer is assumed to ingest beef, poultry, produce, eggs, milk and pork at a total rate of 1,117 pounds (lbs) per year (lbs/yr) for a farmer adult weighing 80 kilograms (kg). This weight of food was obtained by adding up the ingestion rates (kg/kg-day) for the farmer adult listed in Table 7-1, multiplying by a body weight of 80 kg and an exposure frequency of 350 days/year. The model assumes that 100% of these foods have all taken up COPCs at the point of maximum deposition from the source, which is very conservative.

The Lakes model does not have a hunter scenario, and these assumptions are typically site specific. Based on consumer-only intake of home-produced game from the EPA Exposure Factors Handbook (Table 13-41; EPA, 2011a), it is assumed that a hunter would ingest between 59 and 197 pounds of game each year. This is significantly lower than the 1,117 lbs for the farmer. Even if the meat contained COPCs from the sources it would not increase the hunter's risk significantly. The weight of 197 lbs is based on the 95th percentile ingestion rate for a male aged 40-69 (3.19 g/kg-day or 9 ounces of deer meat per day, every day of the year), it is the highest of the rates available, is for consumers only, and it assumes ingestion over an entire year. It represents approximately 18 percent of the hunter's total diet. In contrast, the weight of 59 lbs is the mean of the same age group (40 – 69 years) and represents approximately five percent of the hunter's total diet. It does not make any allowance for hunting regulations that might limit the availability of game for hunting. Within the approximately 20,000 acres that constitute the Facility, there is no hunting allowed. The wildlife areas near ATK are further away from the Facility than areas that are currently being evaluated for an assumed Farmer or Resident. Because the wildlife locations are further away exposure to COPCs released by the Facility will be further away, and so COPC concentrations in hunted game and the associated risks, will be lower. Therefore, the risks and hazards in wildlife areas would be negligible, and are not quantified. See Section 10.6 for further discussion.

5.3.5 Scenarios Not Considered and Why

As presented in the approved HHRA protocol, and as supported by EPA, 2005, the following pathways were not included in this risk assessment:

- **Ingestion of Groundwater**. EPA (1998) found that groundwater is an insignificant exposure pathway for combustion emissions. In addition, groundwater at the site is not part of this RCRA Sub-part X Permit, and is being addressed in a separate risk assessment.
- **Inhalation of Resuspended Dust.** EPA (1990) found that risk estimates from inhalation of resuspended dust (i.e., soil and dust resuspended by wind), are insignificant. It is anticipated exposure through direct inhalation of vapor and particle phase COPCs and incidental ingestion of soil are more significant.
- **Dermal Exposure to Surface Water, Soil, or Air.** Available data indicate that the contribution of dermal exposures to soils to overall risk is typically small (EPA, 1995; 1996). For example, the risk assessment conducted for the Waste Technologies Industries, Inc., hazardous waste incinerator in East Liverpool, Ohio, indicated that the risk resulting from soil ingestion and dermal contact for an adult farmer in a subarea with high exposures was 50-fold less than the risk from any other exposure pathway and 300-fold less than the total estimated risk (EPA, 1995; 1996). Also, there are significant uncertainties associated with estimating potential COPC exposure via the dermal exposure pathway. The most significant of these uncertainties are associated with determining the impact of soil characteristics and the extent of exposure (e.g., the amount of soil on skin and the length of exposure) on estimating compound-specific absorption fractions.
- **Inhalation of COPCs and Ingestion of Water by Animals**. EPA does not recommend these animal exposure pathways when calculating animal tissue concentrations because it is expected their contribution to the total risk is negligible compared to the contributions of the recommended exposure pathways.

6. ESTIMATING MEDIA CONCENTRATIONS

The purpose of this section is to describe the equations and associated parameters necessary for estimating media concentrations. However, because they are presented in the HHRAP guidance (EPA, 2005a) and used in the Lakes software, they are not reproduced here. All media concentrations (and subsequent risk calculations) presented in this report are obtained directly from the Lakes software, and those equations are referenced below according to the exposure pathways that are relevant to this risk assessment. The references that are presented correspond to the specific equations and page numbers found in the appendices of the HHRAP guidance.

6.1 Calculating COPC Concentrations in Air for Direct Inhalation

The HHRAP guidance recommends calculating COPC concentrations in air by summing the vapor phase and particle phase air concentrations of COPCs. To evaluate long-term (chronic) exposure via direct inhalation, the Lakes software utilizes unitized yearly air parameter values to calculate air concentrations for all COPCs except mercury, as specified in App. B, Table B-5-1 (EPA, 2005a; page B-276).

To evaluate short-term (acute) exposure via direct inhalation, Lakes software utilizes unitized hourly air parameter values to calculate air concentrations for all COPCs except mercury, as specified in Appendix B, Table B-6-1 (EPA, 2005a; page B-279). The model default values are utilized in this risk assessment.

6.1.1 Mercury Wizard

Within the Lakes software, there is a tool called the Mercury Wizard. The calculated emissions rate for mercury from Appendix A is entered into the model for each sub source (M-136 A1, A2, etc), and then the model adjusts for the portion of mercury that is lost to the global cycle (51.8%), the portion of mercury that is deposited as divalent mercury (48%), and the portion that is emitted as elemental mercury (0.2%). These percentages are consistent with the information presented in Chapter 2, Section 2.3.5.3 Mercury of the HHRAP (EPA, 2005a). The model then generates emissions factors for mercuric chloride and elemental mercury using those percentages, and those emissions factors are carried forward in the calculations of media concentrations and ultimately, non-cancer hazard quotients for mercuric chloride and methyl mercury. Section 6.2 below provides a discussion of calculating mercury concentrations in soil.

6.2 Calculating COPC Concentrations in Soil

The HHRAP guidance recommends calculating COPC concentrations in soil by summing the vapor phase and particle phase deposition of COPCs to the soil, and considering wet

and dry deposition of particles and vapors. The equations presented in the guidance and used in the Lakes software account for loss of COPCs by several mechanism, including leaching, erosion, runoff, biotic and abiotic degradation and volatilization. These loss mechanisms all lower the soil concentration associated with the deposition rate. The recommended equations for calculating soil concentration and soil losses of COPCs are presented in App. B Tables B-1-1 through B-1-6 (EPA, 2005a; starting on page B-1) for land use areas. Note that the Appendix B equations contain adjustments for calculating mercury soil concentrations, and the Lakes model applies these necessary adjustments to account for each species of mercury, as described in Section 6.1.1 above. The model default values are utilized in this risk assessment.

Section 5 of the HHRAP Guidance presents a detailed discussion on calculating the following:

- Cumulative soil concentration, including site-specific parameters used in determining those (Sections 5.2.1 and 5.2.4)
- COPC soil loss constant (Section 5.2.2)
- Calculating the deposition term (Section 5.2.3)

The details of these sections are not reproduced in this report. However, default values presented in the Lakes software are used in calculating these terms.

6.3 Calculating COPC Concentrations in Produce

The HHRAP guidance states that indirect exposure resulting from ingestion of produce depends on the total concentration of COPCs in the leafy, fruit and tuber portions of the plant. Aboveground produce is evaluated differently than belowground produce. Aboveground produce is typically assumed to be contaminated by the following mechanisms:

- Direct deposition of particles – wet and dry deposition of particle phase COPCs on the leaves and fruit of plants
- Vapor transfer – uptake of vapor phase COPCs by plants through their foliage. This is known as air-to-plant transfer.
- Root uptake – root uptake of COPCs available from soil and their transfer to the aboveground portions of the plant

Belowground produce is only assumed to be affected by root uptake, so any risk estimates prepared on belowground produce would be lower than those prepared on aboveground

produce. Appendix B Table B-2-10 (EPA, 2005a; starting on page B-83) contains the equations used in calculating the COPC concentrations in belowground produce.

Aboveground exposed produce is affected by all three of these mechanisms, while aboveground-protected produce (peas, corn, and melons) is covered by a protective covering that is typically not consumed. These coverings prevent contamination of the edible portion from deposition and vapor transfer, meaning that root uptake of COPCs is the primary mechanism through which aboveground-protected produce becomes contaminated. Appendix B Tables B-2-1 through B-2-9 (EPA, 2005a; starting on page B-33) contain the equations used in calculating the COPC concentrations in aboveground produce.

The aboveground produce equations presented in the guidance and used in the Lakes software account for loss of COPCs by several mechanisms. Wind removal, water removal, and growth dilution all contribute to reduce the amount of contaminant deposited on plant surfaces.

There are several factors that need to be calculated or estimated in order to determine COPC concentration in produce. Examples include determining the length of time that the vegetation is exposed to contaminant deposition before being harvested, and the standing crop biomass, also known as productivity, which can be calculated differently for four aboveground produce classes (fruits, fruiting vegetables, legumes and leafy vegetables). Sections 5.3.1 of the HHRAP guidance discuss these and other factors to consider. The details of these sections are not reproduced in this report. However, default values presented in the Lakes software are used in calculating these terms.

6.4 Calculating COPC Concentrations in Beef and Dairy Products

The HHRAP guidance recommends estimating COPC concentrations in beef tissue and milk products on the basis of the amount of COPCs that cattle are assumed to consume through their diet. HHRAP guidance assumes the cattle's diet consists of forage (primarily pasture grass and hay), silage (forage that has been stored and fermented), and grain. Similar to aboveground produce, the mechanisms of contamination are the same:

- Direct deposition of particles – wet and dry deposition of particle phase COPCs onto forage and silage
- Vapor transfer – uptake of vapor phase COPCs by forage and silage through foliage
- Root uptake – root uptake of COPCs available from the soil and their transfer to the aboveground portions of forage, silage and grain.

Forage and silage are classified as exposed feed, but grain is classified as protected feed, and is only assumed to be contaminated through root uptake. HHRAP guidance recommends, and the model conservatively assumes that 100 percent of the plant materials eaten by cattle are grown on soil contaminated by emission sources. In addition to these feed items, it is assumed that cattle also ingest contaminated soil directly. Appendix B Tables B-3-1 through B-3-11 (EPA, 2005a) contain the equations used to determine the COPC concentrations in forage, silage, grain and soil, this is consumed by beef and dairy cattle, and the resulting COPC concentration in feed materials that will be consumed by beef cattle and dairy cattle. The equations for calculating the COPC concentration in animal tissue and milk are presented in Appendix B, Tables B-3-10 and B-3-11, and in Equation 5-22 of Section 5.4.4 and Equation 5-24 of Section 5.4.5 of the HHRAP (EPA, 2005a).

Similar to aboveground produce, plant surface contaminant loss occurs due to water removal, wind removal and growth dilution, and the equations are the same.

6.4.1 Beef Concentration Resulting from Plant and Soil Ingestion

Once the feed concentration has been calculated, this information along with biotransfer and metabolism factors are included in the HHRAP equation for calculating the animal COPC tissue concentration, as presented in Equation 5-22 of Section 5.4.4 of the HHRAP guidance. This equation includes information such as the fraction of each plant type that is grown on contaminated soil and ingested by the animal, the quantity of each plant type (forage, silage and grain) that is eaten by the animal, the quantity of soil that is eaten by the animal each day, soil bioavailability factor, and a COPC specific biotransfer factor for beef, and a metabolism factor. Model default values are used in these calculations.

6.4.2 COPC Concentration In Milk Due to Plant and Soil Ingestion

Similar to calculating the beef concentration, Equation 5-24 of Section 5.4.5 of the HHRAP guidance presents the recommended equation for calculating the concentration of COPC in milk. Similar to Equation 5-22, this equation includes information such as the fraction of each plant type that is grown on contaminated soil and ingested by the animal, the quantity of each plant type (forage, silage and grain) that is eaten by the animal, the quantity of soil that is eaten by the animal each day, soil bioavailability factor, and a COPC specific biotransfer factor for milk, and a metabolism factor. The biotransfer factor is calculated the same as for beef, in that contaminants bioconcentrate in the fat tissues, but the multiplier is different based on the assumed fat content in milk (4%) which is lower than that in beef (19%). Appendix A2-2.13 of the HHRAP guidance discusses the biotransfer factors for animals and milk. Quantities of feed are slightly different for dairy cattle than for beef cattle, and the quantity of soil ingested is slightly less for dairy

cattle than for beef cattle, but many of the other assumptions are the same. Model default values are used in these calculations.

6.5 Calculating COPC Concentrations in Pork

Similar to Section 6.4, estimating COPC concentrations in pork tissue includes first estimating the amount of COPCs that are consumed through a diet of silage and grain. Additional COPC contamination of pork tissue may occur through ingestion of soil. Once the COPC concentrations in feed are determined, the animal COPC tissue concentration can be calculated. The equation used to determine COPC concentrations in pork is presented in Table B-3-12 of Appendix B of the HHRAP (EPA, 2005a). This equation includes information such as the fraction of each plant type that is grown on contaminated soil and ingested by the animal, the quantity of each plant type (silage and grain) that is eaten by the animal, the quantity of soil that is eaten by the animal each day, soil bioavailability factor, and a COPC specific biotransfer factor for pork, and a metabolism factor. The biotransfer factor is calculated the same as for beef, in that contaminants bioconcentrate in the fat tissues, but the multiplier is different based on the assumed fat content in pork (23%) which is higher than that in beef (19%). Appendix A2-2.13 of the HHRAP guidance discusses the biotransfer factors for animals. Model default values are used in these calculations.

6.6 Calculating COPC Concentrations in Chicken and Eggs

Similar to the previous two sections, estimates of the COPC concentration in chicken and eggs are based on the amount of COPCs that chickens consume through ingestion of grain and soil. HHRAP guidance recommends assuming that uptake of COPCs via inhalation and ingestion of water is insignificant relative to the pathways discussed here. The calculations assume that chickens are housed in a typical manner that allows for contact with soil, and that chickens consume 10 percent of their diet as soil. The remainder of the diet (90%) consists of grain grown at the exposed location, meaning that 100% of the grain consumed is contaminated.

The algorithm for aboveground produce is used to estimate the COPC concentration in grain, and grain is considered to be an aboveground protected plant, meaning it is only contaminated by root uptake. HHRAP guidance recommends assuming that a chicken eats 0.2 kg DW/day of contaminated grain, and 0.022 kg DW/day of contaminated soil (Sections 5.6.2 and 5.6.4 of the HHRAP guidance). The biotransfer factor is calculated the same as for beef, in that contaminants bioconcentrate in the fat tissues, but the multiplier is different based on the assumed fat content in chicken (14%) and the assumed fat content for eggs (8%). The biotransfer factors for chicken and eggs are presented in Appendix A2-2.13.3 of the HHRAP (EPA, 2005a). The equations used to determine

COPC concentrations in eggs and chicken are presented in Appendix B Tables B-3-13 (eggs) and B-3-14 (chicken) of the HHRAP (EPA, 2005a).

6.7 Calculating COPC Concentrations in Drinking Water and Fish

This risk assessment does not include risk estimates based on ingestion of drinking water or fish, because those are not complete/significant pathways at this site due to poor surface water quality.

6.8 Using Site-Specific vs. Default Parameter Values

HHRAP guidance suggests using site-specific values instead of default parameter values where appropriate and where their usage would provide a more representative estimate of site-specific risk. However, site-specific parameters were not used in this risk assessment. As discussed in the uncertainty section (Section 10) the amounts of vegetables, fruits, foliage and feed assumed to be produced by the area could not actually be produced near Promontory due to the poor quality of the soil and water. The intake rates used in the equations are hypothetical for this area of Utah.

Model default parameters are used in all of the media concentration calculations presented in Section 6, with the exception of the air-to-plant biotransfer factors for aboveground produce (BV_{ag}) and forage (BV_{forage}) for dibenz(a,h)anthracene. Those two values are reduced by a factor of 100, in accordance with HHRAP guidance (EPA, 2005a). Appendix A Section A-2-2.12.4, pages A-2-20 and A-2-21 presents a discussion of the methodology used to calculate those biotransfer factors and it states that the methodology overestimates the BV_{ag} and BV_{forage} and that it is appropriate to reduce them by a factor of 100 for all organics, with the exception of PCDDs and PCDFs. For COPC that are already in the Lakes software, the (BV_{ag}) and forage (BV_{forage}) values are reduced by a factor of 100 for only dibenz(a,h)anthracene. For COPC not in the HHRAP database, the (BV_{ag}) and forage (BV_{forage}) values are reduced for all of the organics. These values are presented in Appendix D of this HHRA report.

7. QUANTIFYING EXPOSURE

This section describes the factors involved in quantifying the exposure received under each of the exposure scenarios evaluated in this risk assessment. All chronic exposure are quantified in the Lakes software, which follows the HHRAP guidance. These estimates of exposure can include the following parameters: the estimated COPC media concentrations calculated as discussed in Section 6, the consumption rates of the medium, receptor body weights, and the frequency and duration of exposure. The following sections provide further details on quantifying exposure for the inhalation and ingestion pathways that are evaluated in this risk assessment, as well as a presentation of any assumptions that are modified from default values. Table 7-1 presents the exposure assumptions utilized in this risk assessment. These values are consistent with those presented in Table 3-4 of approved HHRA Protocol. Table 7-1 is also consistent with the OSWER Directive 9200.1-120, dated Feb. 6, 2014 (EPA, 2014a), which presents revised standard default exposure assumptions. Specific updated assumptions are from this latest directive are discussed below in the relevant sections.

The Lakes software includes only residential and farmer scenarios. In order to evaluate an industrial worker at Autoliv, and both the North and South Plant Main Administration buildings, the exposure assumptions for the residential adult are modified to match those of an industrial worker, as shown in Table 7-1. Inhalation is the only pathway considered for an industrial worker.

7.1 Inhalation Exposure Pathways

Direct inhalation of vapors and particulate emissions from combustion sources is a potential pathway of exposure, and is considered to be the most important pathway of exposure in this risk assessment. The Lakes software combines the COPC concentrations with the inhalation exposure assumptions shown in Table 7-1 to provide estimates of inhalation exposure. The following equation is used to estimate inhalation intakes:

$$EC = (C_{air} \times ET \times EF \times ED) / (AT \times 24 \text{ hours/day})$$

Where:

EC = exposure concentration ($\mu g/m^3$)
C_{air} = COPC concentration in air ($\mu g/m^3$); see Section 6.1 for details on C_{air}
ET = exposure time (hours per day)
EF = exposure frequency (days per year)
ED = exposure duration (years)

AT = averaging time—the period over which exposure is averaged (days); for carcinogens the averaging time is 25,550 days, based on a lifetime exposure of 70 years; for noncarcinogens, averaging time equals ED (years) multiplied by 365 days/year.

The inhalation exposure time is 24 hours per day for all of the residential and farmer scenarios evaluated. The inhalation exposure time for the industrial worker scenario evaluated for Autoliv, and both the North and South Plant Main Administration buildings is 8 hours per day. [ATK employees work a 9 hour day with every second Friday free and their actual exposure would be consistent with the 2000 hour work year used in this HHRA.]

The inhalation exposure frequency was 350 days per year for the residential and farmer scenarios, and 250 days per year for the industrial worker.

The inhalation exposure durations are specific to each exposure scenario: 26 years for the resident adult, 6 years for the resident child and farmer child, 40 years for the farmer adult and 25 years for the industrial worker. The resident adult exposure duration is modified in the Lakes software from 30 years to 26 years. Modified model parameters are exported from the model and are shown in Appendix D.

7.1.1 Soil Inhalation Resulting from Dust Resuspension

HHRAP guidance does not recommend evaluating the soil inhalation of resuspended dust exposure pathway, and the Lakes software does not include this pathway, so it is not evaluated in this risk assessment.

7.2 Ingestion Exposure Pathways

Exposure is assumed to occur over a period of time. HHRAP guidance recommends dividing the total exposure by the time period and expressing the average exposure in terms of body weight. Ingestion exposures quantified per the HHRAP uses time and body weight, presented in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day), and termed "intakes" (more accurately this is also called a "dose"). The following general equation presented as Equation 6-1 of Section 6.2 of the HHRAP (EPA, 2005a), is used to estimate ingestion intakes:

$$I = (C_{gen} \times CR \times EF \times ED) / (BW \times AT)$$

Where:

I = intake—the amount of COPC consumed by the receptor (mg/kg-day)
C_{gen} = generic COPC concentration in media of concern (food or soil) (mg/kg)
CR = consumption rate (kg/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)

AT = averaging time—the period over which exposure is averaged (days); for carcinogens the averaging time is 25,550 days, based on a lifetime exposure of 70 years; for noncarcinogens, averaging time equals ED (years) multiplied by 365 days/year.

The variables listed above are used in the Lakes software to calculate receptor-specific exposures to COPCs.

The exposures calculated in a risk assessment are intended to represent reasonable maximum exposure (RME) conditions as described in EPA's *Risk Assessment Guidance for Superfund* (RAGS) (EPA, 1989). All exposure inputs for the various receptors that are evaluated are presented in Table 7-1. Variations from the default parameters are described in Sections 7.2.1 through 7.6.

The Lakes software calculates COPC intake in accordance with HHRAP guidance. The equations for direct and indirect intake exposure pathways are not provided here, but can be found in the HHRAP Guidance Appendix C, Tables C-1-1, C-1-2, and C-1-3 (EPA, 2005a) for COPC intake from soil, intake from produce, and intake from beef, milk, pork, poultry and eggs, respectively. The intake of dioxins in breast milk is an important indirect exposure pathway and the calculation methodology of breast milk is provided in Section 3.6.1 of the Approved HHRA Protocol. The breast milk calculation is based on Table C-3-1 of EPA, 2005a.

7.2.1 Body Weight

The body weights in this risk assessment are 15 kg for a resident child and farmer child, and 80 kg for a resident adult, farmer adult, and an industrial worker. The adult body weight of 80 kg is an increase from 70 kg, the previous standard default body weight for an adult, and the value recommended in the HHRAP guidance. The value of 80 kg is from OSWER Directive 9200.1-120 (EPA, 2014a). The resident and farmer adult and industrial worker body weights are modified in the Lakes software from 70 kg to 80 kg. Modified model parameters are exported from the software and are provided in Appendix D.

7.2.2 Food (Ingestion) Exposure Pathways

Ingestion of homegrown food is a potentially complete exposure pathway for this site, and HHRAP guidance recommends determining the intake of COPCs based on the types of foods consumed, the amount of food consumed per day, the concentration of COPCs in the food, and the percentage of the diet contaminated by COPCs.

Consistent with the HHRAP guidance, the Lakes software evaluates ingestion of homegrown produce for the resident adult and child, as well as the farmer adult and child. In addition, the model evaluates ingestion of homegrown beef, milk, chicken, eggs, and pork for the farmer adult and child, and the model assumes that 100% of these items are contaminated by COPCs. This is shown in Table 7-1 as a value of one for the fraction of contaminated produce, beef, milk, chicken, eggs and pork consumed. The amounts consumed per day used in the Lakes software for each scenario are shown in Table 7-1. These values correspond to the recommended amounts provided in Table 6-1 of the HHRAP guidance, which provides these amounts in servings per week in ounces or pounds. Those values are converted to units of kg of food per kg of body weight per day (kg/kg-day) in the Lakes software, and are shown accordingly in Table 7-1. Food intakes are calculated in the model according to the equation presented above in Section 7.2.

7.2.3 Soil (Ingestion) Exposure Pathway

Soil ingestion is a potentially complete exposure pathway for this site, and the HHRAP guidance recommends evaluating this pathway. Soil ingestion rates for the resident adult and farmer adult are 100 mg per day (0.0001 kg per day). Soil ingestion rates for the resident child and farmer child are 200 mg per day (0.0002 kg per day). These values are consistent with the OSWER Directive 9200.1-120, dated Feb. 6, 2014 (EPA, 2014a). Soil intakes were calculated in the model according to the equation presented above in Section 7.2.

The soil ingestion pathway is not complete for the industrial worker at this site, because the worker works indoors.

7.2.4 Water (Ingestion) Exposure Pathways

Water ingestion pathways from groundwater and/or surface water were not included in this risk assessment, because they are not complete pathways at this site.

7.3 Dermal Exposure Pathways

HHRAP guidance does not recommend evaluating dermal exposure to COPCs through contact with soil, and the IRAP h-View model does not include dermal exposure, therefore, it is not considered in this risk assessment.

HHRAP guidance also does not typically recommend evaluating the dermal water exposure pathway, and it is not a complete pathway at this site. Therefore, it is not considered in this risk assessment.

7.4 Exposure Frequency

HHRAP guidance recommends an exposure frequency of 350 days per year for residential scenarios. This is based on the protective estimate that all receptors spend a maximum of two weeks away from the exposure scenario location. This value is used for the residential child and adult and the farmer child and adult. The industrial worker has an exposure frequency of 250 days per year, based on working five days per week for 50 weeks each year. These values are consistent with the OSWER Directive 9200.1-120, dated Feb. 6, 2014 (EPA, 2014a).

7.5 Exposure Duration

HHRAP guidance recommends exposure durations of six years for the resident and farmer child, and 40 years for the farmer adult. These values are shown in Table 7-1 and are used in the risk assessment. The HHRAP guidance recommends an exposure duration of 30 years for the resident adult, but this value is out of date and the Feb. 6, 2014 OSWER Directive recommended value is 20 years instead. However, to be consistent with the mutagenic evaluation that was conducted (see Section 8.3.4, Tables 8-8 and 8-9), 26 years was utilized for the resident adult. The value is modified in the Lakes software. An exposure duration of 25 years is used for the industrial worker at Autoliv, and the North and South Plant Main Administration buildings. This value is consistent with the OSWER Directive 9200.1-120 (EPA, 2014a). Modified model parameters were exported from the model and are shown in Appendix D.

7.6 Averaging Time

HHRAP guidance recommends using a value of the exposure duration that corresponds to the years specified for each receptor x 365 days/year as the averaging time for noncarcinogenic COPCs. The noncarcinogenic averaging time for the resident adult is modified in the Lakes software from the default of 30 years to match the updated exposure duration of 26 years. It is also modified for the industrial worker to match the exposure duration of 25 years at Autoliv, and the North and South Plant Main Administration buildings. Modified model parameters were exported from the model and are shown in Appendix D.

For carcinogenic COPCs the HHRAP guidance recommends using an averaging time of 70 years x 365 days per year. This is the default value in the Lakes software, so these values were not modified.

7.7 Breast Milk Exposure

The information on breast milk exposure is presented in Section 3.6.1 of the approved HHRA protocol, and it is repeated here for ease of reference. The Lakes software uses the equation for the calculation of the Average Daily Dose (ADD) for an infant exposed to contaminated breast milk taken from US EPA, 2005a, page C-48, Appendix C, Table C-3-2, and is as follows:

$$ADD_{infant} = \frac{C_{infant} \times f_3 \times f_4 \times IR_{milk} \times ED}{BW_{infant} \times AT}$$

Where:

ADD_{infant} = Average Daily Dose

C_{infant} = Concentration of COPC in milk fat of breast milk
(Calculated using the equation provided in EPA, 2005a, page. C-41, Appendix C, Table C-3-1.)

f_3 = Fraction of mother's breast milk that is fat
(The EPA recommendation of 0.04 from US EPA, 2005a is the default value in the model.)

f_4 = Fraction of ingested COPC that is absorbed
(The EPA recommendation of 0.9 (or 90%) from EPA, 2005a is the default value in the model.)

IR_{milk} = Ingestion rate of breast milk by the infant
(The EPA recommendation of 0.68 kg/day from US EPA, 2005a is the default value in the model.)

ED = Exposure Duration
(The EPA recommendation of 1 year from EPA, 2005a is the default value in the model.)

BW_{infant} = Infant Body Weight
(The EPA recommendation of 9.4 kg from EPA, 2005a is the default value in the model.)

AT = Averaging Time
(The EPA recommendation of 1 year from EPA, 2005a is the default value in the model.)

These values are also shown in Table 7-1. The breast milk pathway is evaluated under both the farmer and resident adult scenarios in the Lakes software, and it is automatically

calculated for any PCDDs and PCDFs that are COPC. The results are presented in Table 9-16, and are discussed in Section 9.5.

8. TOXICITY ASSESSMENT

A toxicity assessment in a risk assessment quantifies the relationship between the dose of a contaminant and its potentially toxic effects. The EPA and other regulatory and health agencies have compiled dose-response factors for use in risk assessment. This risk assessment evaluates short-term (or acute, 1-hour) inhalation hazards, and long-term (or chronic, lifetime) hazards and risks for both inhalation and oral exposure. Dose-response values for these types of exposure are selected from the sources identified in Section 7.4.2 of the HHRAP (EPA, 2005a).

8.1 Sources of Acute Dose-Response Values

The acute and chronic dose-response values used in this HHRA are provided in this section of the report. Acute values are based on a maximum 1-hour exposure time (for the conditions given in the modeling section of this HHRA), which corresponds to the time for a process burn and dispersion at the receptor of interest. The toxicological endpoints considered for acute exposure are generally all non-cancer endpoints. Chronic values are based on long-term exposure and are both non-cancer and potential cancer end-points. Based on EPA's 2005 Incineration Guidance (EPA, 2005a; Section 7), short-term ambient air concentrations are evaluated by comparison to short-term air criteria developed from short-term dose-response studies in humans and animals. The source of short-term criteria is selected based on the following agreed hierarchy:

- Cal EPA Acute Reference Exposure Levels (RELs) – the concentration in air at or below which no adverse health effects are anticipated in the general population, including sensitive individuals, for a specified exposure period (Cal EPA, 1999, or more recent version). For chemicals that had no Cal EPA REL, but had a similar structure to a chemical that had a REL, a surrogate was selected from the Cal EPA RELs (See Table 8-1 for these surrogates).
- Acute inhalation exposure guidelines (AEGL-1) – "the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure." (NOAA, 2001, or more recent version).
- Level 1 emergency planning guidelines (ERPG-1) – "the maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse effects or perceiving a clearly defined objectionable odor." (DoE 2001; SCAPA 2001, or more recent version).

- Temporary emergency exposure limits (TEEL-1) - ”the maximum concentration in air below which it is believed nearly all individuals could be exposed without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor.” (DoE, 2001; SCAPA, 2001, or more recent version).
- AEGL-2 values – “the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.” AEGL-2 values are to be used only if lower ERPG-1 or TEEL-1 values are not available. (NOAA, 2001), or more recent version.

In the evaluation of acute risk, (1-hour) modeled air concentrations were compared with the short-term (1-hour) air goal concentrations selected using this hierarchy (See Table 8-2). The most recent sources of short-term air concentrations are available from Cal EPA and the DOD. Where these sources did not contain a short-term air goal, a surrogate value was developed based on carbon chain length, structure and class of chemical (using professional judgment) (see Section 8, Table 8-1). The list of 1-hour acceptable air concentrations (Table 8-2) was developed and presented to the Utah DSHW, who agreed to these values. To determine the potential exposure to short-term emissions, estimates of acute (1-hour) hazards from air emissions are calculated. The estimated 1-hour ambient air concentrations for all 209 COPCs are generated from the air quality model and evaluated in the Lakes software (Lakes, 2014). The COPC concentration is compared with its California EPA (Cal EPA) PAC-1 concentration (Cal EPA, 2013), if available, or the Department of Defense AEGL-1 air concentration (DOE, 2012). This analysis is provided in Section 9-1 of this report.

Based on the approved HHRA Protocol (ATK, 2014a), only two sources are needed: Cal EPA RELs and NOAA AEGLs. However, not all of the COPCs had acute criteria in these two sources. These COPCs are shown in Table 8-1, and surrogates are selected for these COPCs, as shown in Table 8-1. For chemicals without acute criteria, surrogates were selected based on structural activity relationships, predominantly carbon chain length and hydrocarbon chain number. The approved acute criteria used in this risk assessment are shown in Table 8-2.

8.2 Sources of Chronic Dose-Response Values

Chronic toxicity values are generally selected based on the hierarchy provided in EPA OSWER Directive titled *Human Health Toxicity Values in Superfund Risk Assessment* (EPA, 2003b). This hierarchy is shown below:

1. Integrated Risk Information System (IRIS) (http://www.epa.gov/iris/), EPA, 2014c)
2. Provisional Peer Reviewed Toxicity Values (PPRTVs) (2004a) which are developed by the EPA Office of Research and Development/ National Center for Environmental Assessment/Superfund Health Risk Technical Support Center and available since mid March 2004 on their website at http://hhpprtv.ornl.gov/pprtv.shtml (EPA, 2004b).
3. Other sources, including the Health Effects Assessment Summary Tables (HEAST) (EPA, 1997), Regional Screening Levels (RSL) tables and the Cal EPA.

However, there are a number of exceptions to this stated hierarchy. In some cases the EPA's IRIS value was not used and it was supplanted with a more conservative dose-response value selected from the EPA's RSL tables (EPA, 2014b).

Toxicological information, such as oral reference doses (RfDs), inhalation reference concentrations (RfCs), Cancer Slope Factors (CSFs), and Inhalation Unit Risk Factors (URFs) are used in accordance with the hierarchy established above.

Toxicological dose-response information is contained in the Lakes software, based on EPA, 2005a, therefore, a number of the dose-response factors were out of date. In preparation for this risk assessment dose-response factors in the model were reviewed and updated with dose-response factors from the above hierarchy, and included in the Protocol (ATK, 2014a) and approved by the Utah DSHW. Four types of chronic dose-response values are used in the risk assessment, Inhalation RfCs, Oral RfDs, URFs and Oral Slope Factors (SF_o). The chemicals with updated toxicity criteria that are used in this risk assessment are provided in Tables 8-3, 8-4, 8-5 and 8-6, respectively.

8.2.1 Parameters for COPC not in the HHRAP Database

There are 64 COPCs listed in Table 2-1 which are not included in the HHRAP Database, and therefore, are not included in the Lakes software. Chemical specific parameters, biotransfer factors, and toxicological dose-response information are added to the Lakes software in order to estimate risks and hazards from these COPC. The values entered are consistent with those presented in Tables 3-6, 3-7 and 4-5 of the approved HHRA protocol, with the exception of m- and p-xylene and 3- and 4-methylphenol. The necessary information for these four COPCs are in the Lakes software and did not require hand entry. The chemical specific parameters and the biotransfer factors for the COPCs not in the HHRAP Database are provided in Appendix D. The toxicological dose-response information for the COPCs not included in the HHRAP Database is presented in Table 8-7.

8.3 Toxic Equivalents Factors, Species and Surrogates

8.3.1 Dibenzodioxins and Dibenzofurans

Some dioxins and difurans are detected in the OB/OD studies conducted to determine emissions, and all dioxins and difurans are evaluated quantitatively in the risk assessment. Consistent with EPA's HHRAP guidance (EPA, 2005a) dioxins are assessed using a TEF method to determine the risks associated with dioxins and furans. Rather than determine a specific toxicity factor for all 75-dibenzodioxin congeners and 135-dibenzodifuran congeners, these constituents are evaluated by grouping together congeners with similar structures into a class and assigning a relative toxicity for the class, in comparison to one member of the class. Table 2-4 shows the 7-dioxin congener groups and 10-difuran congener groups for which there are TEFs. These are the values utilized in the Lakes software and that are used in this risk assessment.

8.3.2 Polycyclic Aromatic Hydrocarbons

As presented in the approved HHRAP protocol, the relative cancer potency for the polycyclic aromatic hydrocarbons is shown in Table 2-2. The adjusted oral cancer slope factors shown in Table 2-2 are consistent with those used in the risk calculations performed in the Lakes software.

8.3.3 Assumptions Concerning Chromium

ATK's wastes do not contain the metals mercury or chromium, and do not routinely contain lead, and ATK strives to keep these metals out of their wastes. However, chromium was detected in the emissions test and the potential sources of chromium are discussed in the uncertainty section of this report. Further, the species of chromium in the emissions is not characterized and distinguishing between chromium (III) and chromium (VI) is not possible with the data currently available. However, the HHRAP Guidance provides a method for assigning a percentage of chromium (III) and (VI). This method was utilized and is discussed in Section 10.1.3.

8.3.4 Age-Dependent Adjustment Factors ADAF)

Based on EPA's Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (EPA, 2005b) on early life stage exposure, chemicals with the ability to cause cancer through actions on DNA are evaluated as having a higher potency on children. A number of these are PAH, but the list also includes Cr(VI), and some chlorinated solvents. A summary overview of the process was prepared by the Navy (2008), and is provided in Appendix D because it provides a concise summary of the issues related to early-life stage exposure to carcinogens.

The supplemental guidance recommends that, in some cases, when carcinogens have a mutagenic MOA, it may be appropriate to apply a default safety factor called an ADAF to risk calculations when evaluating cancer risk associated with exposure for children ages 0 to 16 years.

The chemicals with a mutagenic MOA were identified using the EPA's RSL tables, and where possible source documents from the RSLs or through research into the literature available. In some cases the documentation justifying a mutagenic MOA and a potential effect on DNA during early life stage exposures are poor and the mutagenic MOA for a specific exposure pathway is assumed. The literature sources identifying the chemicals with a mutagenic MOA are discussed below.

Unfortunately, ADAFs were not included in the Lakes risk assessment model in 2005, and although it was their intention to update the model in 2014, no updated model is available at this time. Further, the Lakes model does not provide a subdivision of Early-life stages for children age 0 to 16 years of age, and the recommended method of calculating risks for early-life stages in the Lakes model is to modify the Oral Cancer Slope Factor (CSF) or Inhalation Unit Risk Factor (URF) by multiplying the CSF by the default ADAF, and averaging over the exposure period, as shown in the example equations for B[a]P shown in Appendix D. This method is consistent with the method used by the EPA's RSLs.

Table 8-8 shows the SFo that will be used and Table 8-9 shows the URF. These factors will be used for the receptors in the HHRA (Farmer and Resident Child, Farmer Adult and Resident Adult with age-adjusted factors applied for 6, 40 and 26 years, respectively). In the adult scenario, the receptor is assumed to be exposed as an adult only, and these toxicity values represent standard values used in the OBOD HHRA, whereas all other exposure scenarios assume some exposure as a child, and these will incorporate the age-adjusted factors.

B[a]P

The mutagenic MOA for this chemical is based on EPA 2005b. There is a draft toxicological profile for B[a]P available from EPA (EPA, 2014d), and this profile justifies the use of age dependent adjustment factors (ADAFs) for both oral and inhalation exposure. Cancer dose-response values for both routes of exposure have been modified to include ADAFs. EPA (2006) states: "When assessing early-life exposure for PAHs using such an approach, the ADAF(s) should be applied to the B[a]P slope factor before using relative potency factors to estimate risk from exposure to other PAHs." Examples of the process by which ADAFs were derived are shown in Appendix D.

Benzo[a]anthracene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 0.1 has been used to develop the ADAF adjusted URF and CSF.

Benzo[b]fluoranthene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 0.1 has been used to develop the ADAF adjusted URF and CSF.

Benzo[k]fluoranthene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 0.01 has been used to develop the ADAF adjusted URF and CSF.

Chrysene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 0.001 has been used to develop the ADAF adjusted URF and CSF.

Dibenz[a,h]anthracene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 1.0 has been used to develop the ADAF adjusted URF and CSF.

Indeno[1,2,3-c,d]pyrene

The mutagenic MOA for this chemical is based on EPA 2005b. There is poor documentation concerning this chemicals MOA, and it is assumed mutagenic based on the action of B[a]P. A toxicity equivalency factor of 0.1 has been used to develop the ADAF adjusted URF and CSF.

Hexavalent Chromium

The mutagenic MOA for this chemical by the oral route of exposure is based on the California EPA's public health goals document (Cal EPA, 2011). There is good documentation concerning Cr (VI)'s MOA via the oral route of exposure. ADAFs were used to modify the CSF, as shown in Table 8-8.

The mutagenic MOA for Cr(VI) by the inhalation route of exposure is equivocal and this HHRA assumes Cr(VI) has a mutagenic MOA. However, a research paper by Proctor, et. al (2014), which is provided in Appendix F, states: "In vivo genotoxicity and mutagenicity data are mostly negative and do not support a mutagenic MOA. This issue is also discussed in the uncertainty section of this report.

Dichloromethane

The mutagenic MOA for this chemical by the oral route of exposure is based on the EPA IRIS file (EPA, 2011b). There is documentation concerning this chemical's MOA via the oral route of exposure using in vitro and in vivo tests, and ADAFs were used to modify the CSF, as shown in Table 8-8.

The mutagenic MOA for this chemical by the inhalation route of exposure is based on the EPA IRIS file (EPA, 2011b). There is documentation concerning this chemical's MOA via the oral route of exposure using in vitro and in vivo tests, but no clear mechanism for the MOA via the inhalation route has been demonstrated. However, ADAFs were used to modify the URF as if dichloromethane has a mutagenic MOA by this route of exposure, as shown in Table 8-9.

n-Nitrosodiethylamine

The mutagenic MOA for this chemical is based on EPA 2005b. There is a no IRIS toxicological profile available for this chemical and the mechanism by which this chemical has a mutagenic MOA in animal systems has not been elucidated. The URF and CSF include an ADAF for mutagenic MOA (Tables 8-8 and 8-9, respectively).

n-Nitrosodimethylamine

The mutagenic MOA for this chemical is based on EPA 2005b. There is a no IRIS toxicological profile available for this chemical and the mechanism by which this chemical has a mutagenic MOA in animal systems has not been elucidated. The URF and CSF include an ADAF for mutagenic MOA (Tables 8-8 and 8-9, respectively).

Trichloroethene

The mutagenic MOA for this chemical by the oral route of exposure is based on the EPA IRIS file (EPA, 2011c). There is documentation concerning the MOA for this chemical via the oral route of exposure using in vitro and in vivo tests, and a mechanism for the MOA has been provided. ADAFs, in addition to other adjustments factors, were used to modify the oral slope factor, as shown in Table 8-8.

The mutagenic MOA for this chemical by the inhalation route of exposure is based on the EPA IRIS file (EPA, 2011c). There is documentation concerning this chemicals MOA via the inhalation route of exposure using in vitro and in vivo tests, and a mechanism for the MOA has been provided. ADAFs, in addition to other adjustments factors, were used to modify the inhalation unit risk, as shown in Table 8-9.

Additional adjustment factors were used to modify the toxicity values for trichloroethene (TCE). The Regional Screening Table User's Guide recommends incorporating two separate adjustment factors, in addition to the default ADAFs, into the calculation for TCE. The factors are the Cancer Adjustment Factor (CAF) and the Mutagen Adjustment Factor (MAF).

Cancer Adjustment Factor

EPA's IRIS Chemical Assessment Summary (EPA, 2011c) identifies three carcinogenic end points for TCE via oral exposure: kidney cancer, non-Hodgkins lymphoma (NHL), and liver cancer. Two of these end-points (NHL, and liver cancer) are considered to develop through a mechanism that does not involve a mutagenic MOA. Therefore, the potency of TCE for these two outcomes is different for a lifetime of exposure compared with the kidney cancer outcome. To account for this, the proportional contribution of TCE for these two end-points is calculated separately CAF and added to that TCE's cancer potency from its mutagenic MOA MAF.

The CAF is calculated by taking the ratio of the NHL+liver cancer CSF and the adult CSF as follows:

$$CAF_o = \frac{3.7E-2\ (mg\ per\ kg-day)^{-1}}{4.6E-2\ (mg\ per\ kg-day)^{-1}} = 0.804$$

A similar calculation is undertaken to determine the CAF for the inhalation route, by taking the ratio of the NHL+liver inhalation unit risk factor and the adult inhalation unit risk factor:

$$CAF_i = \frac{3.1E-6\ (mg\ per\ kg-day)^{-1}}{4.1E-6\ (mg\ per\ kg-day)^{-1}} = 0.756$$

Mutagenic Adjustment Factor (MAF)

EPA has concluded by a weight-of-evidence evaluation, that TCE is carcinogenic by a mutagenic mode of action for induction of kidney tumors (EPA, 2011c), and those exposed to carcinogens with a mutagenic mode of action are assumed to have increased early-life susceptibility. Data for TCE are not sufficient to develop separate risk estimates for childhood exposure. The oral slope factor of 4.6 x 10^{-2} per milligram per kilogram per day (mg/kg-day)$^{-1}$, calculated from data from adult exposure, does not reflect presumed increase early-life susceptibility to kidney tumors for this chemical. Generally, the application of ADAFs is recommended when assessing cancer risks for a carcinogen with a mutagenic MOA. However, the ADAF adjustment applies only to the kidney cancer component of the total cancer risk estimate, and the MAF for the oral route is calculated by taking the ratio of the kidney CSF and the adult CSF as follows:

$$MAF_o = \frac{9.3E-3\ (mg\ per\ kg-day)^{-1}}{4.6E-2\ (mg\ per\ kg-day)^{-1}} = 0.202$$

A similar calculation is used to determine the MAF for the inhalation route, by taking a ratio of the kidney URF and the adult URF:

$$MAF_i = \frac{1.0E-6\ (\mu g/m^3)^{-1}}{4.1E-6\ (\mu g/m^3)^{-1}} = 0.244$$

The CAFs and MAFs are multiplied by the default ADAFs, to develop an overall adjustment factor for TCE. These calculations are provided in Appendix D for the Farmer Adult, Resident Adult and Farmer and Resident Child scenarios.

Vinyl Chloride

The mutagenic MOA for this chemical by the oral route of exposure is based on the EPA IRIS file (EPA, 2000). There is documentation concerning the MOA for this chemical via the oral route of exposure using in vitro and in vivo tests, and a mechanism for the MOA is provided in the IRIS file. The CSF of 7.2E-1 (mg/kg-day)$^{-1}$ shown for an adult in Table 8-8 represents continuous lifetime exposure during adulthood. The modified value of 1.4 (mg/kg-day)$^{-1}$ represents continuous lifetime exposure from birth, as presented on EPA's IRIS website (EPA, 2015). The value of 1.4 (mg/kg-day)$^{-1}$ will be used in the mutagenic calculations for the Farmer and Resident Child, Resident and Adult

Farmer. According to EPA, this is considered to be the most appropriate way to handle early life susceptibility to vinyl chloride.

The mutagenic MOA for this chemical by the inhalation route of exposure is based on the EPA IRIS file (EPA, 2000). There is documentation concerning the MOA for this chemical via the inhalation route of exposure using in vitro and in vivo tests, and a mechanism for the MOA is provided in the IRIS file. The inhalation unit risk value of 4.4E-6 $(\mu g/m^3)^{-1}$ shown for an adult in Table 8-9 represents continuous lifetime exposure during adulthood, and this value is appropriate for the Industrial Worker. The modified value of 8.8E-6 $(\mu g/m^3)^{-1}$ represents continuous lifetime exposure from birth, as presented on EPA's IRIS website (EPA, 2015). The value of 8.8E-6 $(\mu g/m^3)^{-1}$ is used to calculate risks and assumes a mutagenic MOA for inhalation exposure for the 1) Farmer and Resident Child, 2) Resident and 3) Adult Farmer.

9. CHARACTERIZING RISK AND HAZARD

As described in the EPA's risk assessment guidance documents, the next step of a risk assessment is risk characterization. This involves combining the exposure estimates developed as described in Section 7, with the toxicity dose-response information in the various EPA or other appropriate sources, to calculate the non-cancer hazards and excess lifetime cancer risks for each of the pathways and receptors identified in Section 5. Hazards and risks are then summed for each receptor, across all applicable exposure pathways, to obtain an estimate of total individual hazard and risk. The uncertainties associated with risk characterization are discussed in Section 10.

9.1 Short-term Non-Cancer Hazards in Air-Methods

As discussed above, air quality modeling is conducted and maximum COPC air concentrations are calculated using worst case meteorological conditions. Two sources are considered M-136 and M-225. It is assumed that all sub-sources within a process area (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) are operating at the same time, and the emissions from all sub-sources within an area are released over a 1-hour period. This assumption is not realistic and would not occur and represents an overestimation of risk because of safety protocols at the Facility. As discussed below, hazards were also calculated based on ATK's actual operating protocol which assumes only sources M-136 A1, A2, A3 and M-225 A were burned simultaneously. This scenario represents a more realistic assumption than including all sub-sources simultaneously.

Modeled air concentrations are divided by the short-term PAC-1 concentration provided in Table 8-2. The resulting quotient, called a Hazard Quotient (HQ) for each individual COPC is then added together for each receptor to give a Hazard Index (HI). These summed indices are shown in Table 9-1, 9-2 and 9-3 for on-site industrial workers, boundary locations where there are no receptors (hypothetical receptors are possible), and off-site locations, respectively. The majority of the emissions are contributed from M-136.

Three on-site industrial worker locations have (HIs) greater than 1 assuming all sources are burned simultaneously (Table 9-1): Autoliv (HI = 4.3), South Plant Main Building (HI = 3.8), and North Plant Main Building (HI = 2.0). In addition, the point of maximum on-site deposition has a HI greater than 1 (HI = 7.8). This point is in an area that is fenced and has a controlled access point available only to authorized employees. This location is in an area of storage bunkers, and employees would not be in this area during a burn. Table 9-1 also includes HIs calculated assuming only sources M-136 A1, A2, A3 and M-225 A are burned at the same time, and an adjustment to the hazards associated with chromium and nickel. This scenario represents ATK's best estimate of conservative but

more likely short-term hazards associated with facility operations. The details of the adjustment factors for chromium and nickel are presented in Sections 10.1.3. The HIs are all less than one using the assumptions that reflect ATK's actual operating conditions and the refinements to the hazards associated with nickel and chromium emissions. The HQs for each individual COPC at each individual receptor are provided in Appendix E.

Table 9-2 presents the acute HQs for the hypothetical receptor locations. These locations include the point of maximum off-site deposition and four boundary points selected based on the annual prevailing wind direction determined over a five-year period; Blue Creek, a perennial stream located along the western edge of the property and about half a mile west of source M-136; and ATK Ranch Pond, a non-resident location at the southwest edge of the property. None of these boundary/off-site locations have actual human receptors, so these acute results represent hypothetical exposure scenarios. The HIs assuming all sources are burned simultaneously are less than one for Boundary 3, Boundary 4, ATK Ranch Pond, and the point of maximum off-site deposition. The HIs assuming all sources are burned simultaneously are greater than 1 for: Blue Creek (HI = 4.7), Boundary 1 (HI = 5.3), and Boundary 2 (HI = 2.3). Table 9-2 also includes HIs calculated assuming only sources M-136 A1, A2, A3 and M-225 A are burned at the same time, and an adjustment to the hazards associated with chromium and nickel. This scenario represents ATK's best estimate of conservative but more likely short-term hazards associated with facility operations. The HIs are all less than one using the assumptions that reflect ATK's actual operating conditions.

Table 9-3 presents the acute HIs for the actual, off-site receptor locations. These locations include residences, a dairy farm and two small towns located near the site. The HIs assuming all sources are burned simultaneously are less than one for all of the receptors, with the exception of Adams Ranch (HI = 2.8). Table 9-3 also includes HIs calculated assuming only sources M-136 A1, A2, A3 and M-225 A are burned at the same time, and an adjustment to the hazards associated with chromium and nickel. This scenario represents ATK's best estimate of conservative but more likely short-term hazards associated with facility operations. The HIs are all less than one using the assumptions that reflect ATK's actual operating conditions.

As discussed in Section 10.1.3, Chromium and Nickel in Waste, nickel is not present in ATK's waste stream and is believed to be an experimental artifact generated from the nickel chrome wire used to ignite the OB/OD test, and from the stainless steel pan used to contain the test bundles, which is 14 percent nickel.

Chlorine is not present in ATK's waste because it is a gas, and ATK does not burn gas cylinders. However, ATK does process waste that contains 16 percent aluminum, and

ammonium perchlorate which contains chloride ion, which is likely to form chlorine gas aluminum chloride and hydrogen chloride (HCl) in the combustion process.

9.2 Long-term Non-Cancer Hazards in All Media

Standard risk assessment models assume that chemicals with non-cancer effects have a threshold response. That is, there is a level of exposure below which no adverse effects will be observed (EPA 1989), or where it is considered safe. The default approach used by EPA to assess the potential for health effects associated with this threshold relationship is set out in EPA (2005), and it involves:

1. Comparing an estimated chemical-specific air concentration to an *R*f*C* that represents a non-harmful level for direct inhalation exposures;
2. Comparing an estimate of ingested exposure dose (see Chapter 6) to an *R*f*D* that represents a non-harmful level for oral exposures.

An *R*f*C* is an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations. Similarly, an *R*f*D* is a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a 70-year lifetime (EPA, 2005a).

Two exposure durations are evaluated in this risk assessment, as described in Section 5: short-term exposure to volatiles and particulate-borne in air are evaluated for inhalation exposure, and longer-term, or chronic, exposures to chemicals in air, soil and other media (such as biota) are considered for a range of exposure pathways.

Short-term and long-term inhalation hazards are evaluated using the approach identified in the EPA's 2009 Inhalation risk assessment guidance. The estimated air concentration, adjusted for the exposure time, frequency and duration, is compared with the corresponding acceptable air concentration for the same exposure period to give a ratio, or quotient of the hazard, typically call the HQ. This is expressed as follows:

$$HQ = \frac{[C_{air}] \times \frac{ET \times EF \times ED}{AT}}{RfC_{air}}$$

Where

HQ = Hazard Quotient (unitless)

$[C_{air}]$ = Chemical Concentration of COPC in air (mg/m^3)

ET = Exposure Time (Hours)

EF = Exposure Frequency (Days)

ED = Exposure Duration (Years)

AT = Averaging Time (Hours)

RfC = Reference Concentration (mg/m^3)

The RfC for short-term exposure is different from the acceptable air concentration for long-term exposure because non-cancer effects can be target organ and duration specific. As described in Section 8, the Cal EPA RELs are used to evaluate 1-hour air concentrations. For long-term exposure, the EPA's RfCs from their IRIS database are used.

Superfund has determined that an *HQ* of less than or equal to 1 is considered acceptable (EPA, 1989). This acceptable risk level has also been adopted by the Utah DSHW as required by Utah Administrative Code R-315-101-6. When all of the HQs are added together the summed hazard is called the HI, and for both short-term and long-term exposures, it assumes that the COPC has a mode of action at the same target organ, meaning they have a common mechanism of toxicological action, when this clearly not the case.

9.2.1 Quantitative Non-cancer Hazards for All COPCs and Receptors

This sub-section provides the results of risk calculations for chemicals with non-carcinogenic effects. On-site worker risks are followed by the risks at the discrete Boundary locations assuming off-site exposure pathways and then for actual off-site discrete receptor locations. The results show HIs less than one in all cases even when no adjustments are made for Cr and Ni contributions from the test pans. Chronic non-cancer hazards for all chemicals are presented in Tables 9-4 through 9-6. They are calculated in the Lakes software as described in Section 9.2.

On-site Workers

The hazards presented in Table 9-4 are calculated for the three on-site locations that have workers: Autoliv, and the North and South Plan Main Administrative buildings. The highest HI is 0.024 for Autoliv. This value is well below 1, indicating that the chronic hazards to industrial workers are low, and acceptable.

Off-site Boundary Locations

The hazards presented in Table 9-5 are for off-site Boundary locations and the point of maximum deposition. They are calculated assuming a hypothetical resident and a hypothetical farmer scenario at the boundary/off-site locations. The highest HI is 4.9E-02 for the point of maximum deposition off-site. This value is well below 1, indicating that the chronic hazards to hypothetical receptors at the boundary/off-site locations are low, and acceptable.

Off-site Actual Receptor Locations

The hazards presented in Table 9-6 are calculated for the actual off-site receptors. The table contains hazards for a resident adult and child, and a farmer adult and child. The highest HI is 0.017 for Adams Ranch. This value is well below 1, indicating that the chronic hazards to off-site receptors are low, and acceptable.

The hazards presented in Tables 9-4 through 9-6 were calculated assuming all sources are burned simultaneously. This presents a worst case estimate, but it is not realistic and represents an overestimation of the actual hazards. Section 10.1.2.7 presents ATK's best estimate of conservative but more likely chronic hazards associated with the facility operations.

9.3 Long-term Excess Lifetime Cancer Risks for All Media

Risk estimates represent the incremental or increased probability that an individual will develop cancer over a lifetime, over and above their existing, or background risk, as a result of their specific exposure to a carcinogenic chemical from the site or Facility (EPA 1989). HHRAP guidance recommends calculating these risks as follow:

Inhalation Cancer Risk

$$Cancer\ Risk = EPC \times URF$$

Where

URF = Inhalation Unit Risk Factor – COPC specific (µg/m^3)

EPC = Exposure Point Concentration (µg/m^3), calculated below

$$EPC = ([C\_air\ ] \times ET \times EF \times ED)/(AT)$$

Where

C_{air} = the COPC specific air concentration based on the fraction in vapor phase and the fraction in particle phase (equation for C_{air} is provided in Table B-5-1 of the HHRAP guidance)

ET = Exposure Time (hours/day)

EF = Exposure Frequency (days/ year)

ED = Exposure Duration (years)

AT = Averaging time (70 years x 365 days/year x 24 hours/day = 613,200 hours)

Ingestion Cancer Risk

$$Cancer\ Risk = \frac{I \times\ EF \times ED\ \times SFo}{AT \times 365\ days/year}$$

Where

I = Daily intake of COPC from soil, produce, beef, milk, chicken, eggs or pork (mg COPC/kg BW-day) (the equation to develop I is provided in Table C-1-6 of the HHRAP guidance)

EF = Exposure Frequency (days/year)

ED = Exposure Duration (years)

SFo = Oral Cancer Slope Factor – COPC specific $(mg/kg\text{-}day)^{-1}$

AT = Averaging time (years)

It is possible for a receptor to be exposed to multiple COPCs within an individual exposure pathway. HHRAP guidance recommends summing the individual risks for all COPCs through a single exposure pathway and summing the risks from individual pathways such as ingestion or inhalation. This provides an estimate of cumulative risk posed to each receptor. In addition, a receptor might be exposed to emissions from multiple sources, and the HHRAP guidance recommends summing the risks from all modeled sources for each receptor at each location. The Lakes software provides cumulative risk estimates as well as by pathway, and by source. The cumulative results are presented in Section 9.3.1 for an estimate of total risk from all pathways, all COPCs and all sources. The risks broken down by COPC are provided for each receptor and each location in Appendix E.

9.3.1 Quantitative Excess Lifetime Cancer Risk for All COPCs and Receptors

This sub-section provides the results of risk calculations for chemicals with potentially carcinogenic effects. On-site worker risks are followed by the risks at the discrete Boundary locations assuming off-site exposure pathways and then for actual off-site discrete receptor locations. All of the ELCRs shown are less than one in one million. The cancer risks presented in Tables 9-4, 9-7 and 9-8 were calculated assuming all sources are burned simultaneously. This presents a worst case estimate, but it is not realistic and represents an overestimation of the actual risks. Section 10.1.3 presents ATK's best estimate of conservative but more likely chronic cancer risks associated with the facility operations.

On-site Workers

Table 9-4 presents the ELCRs to an industrial worker at the three on-site locations Autoliv, and the North and South Plant Main Administrative buildings. The highest cancer risk presented in Table 9-4 is 8.3E-08 for the industrial worker at Autoliv. The risks shown in Table 9-4 are associated with the inhalation pathway, and are all less than one in one million.

Off-site Boundary Locations

Table 9-7 presents the cancer risks to a hypothetical resident receptor and a hypothetical farmer receptor at the boundary/off-site locations and the point of maximum deposition. All of the calculated risks are less than one in one million. The highest cancer risk presented in Table 9-7 is 9.8E-7 for the hypothetical future farmer adult at the point of maximum deposition. The second highest cancer risk is 6.7E-07 for the hypothetical farmer adult at Blue Creek, which is a perennial stream running along the western edge of the site property and located about one half mile west of source M-136. As noted in Section 5.1, Blue Creek was originally selected as an ecological receptor and there is a limited potential for a rancher to move cattle along the creek. The risks by pathway for the hypothetical farmer adult are shown in Table 9-11. The farmer adult is assumed to be exposed via inhalation, ingestion of homegrown produce, beef, milk, chickens, eggs, pork and incidental ingestion of soil. Table 9-11 shows that the pathway associated with the highest risk is inhalation, followed by ingestion of milk. These two pathways account for over 85% of the total risk to the farmer adult at Blue Creek and the other receptors shown in Table 9-11. The remaining pathways (ingestion of produce, beef, chickens, eggs, pork and incidental ingestion of soil) make up the other 15% of the risk.

After the farmer adult, the next highest summed excess lifetime cancer risk in Table 9-7 is 4.8E-7 for the hypothetical future resident adult at the point of maximum deposition.

The risks by pathway for the resident adult are shown in Table 9-9. The resident adult is assumed to be exposed via inhalation, ingestion of homegrown produce and incidental ingestion of soil. Table 9-9 shows that the pathway associated with the highest risk is inhalation, which accounts for over 96% of the total risk for the resident adult at the point of maximum deposition and the other receptors shown in Table 9-9. The cancer risk to a hypothetical resident adult is about one-third that of the cancer risk to a hypothetical farmer adult for all of the receptors. The risks to hypothetical residents and farmers at the other boundary/off-site locations are also less than one in one million, and they are less than those estimated for the point of maximum deposition.

Off-site Actual Receptor Locations

Table 9-8 presents the cancer risks to the actual off-site receptors. All of the calculated risks are less than one in one million. The highest cancer risk presented in Table 9-8 is 3.4E-7 for the farmer adult at Adams Ranch. The risks by pathway for the farmer adult are shown in Table 9-11, and this table shows that the risks due to inhalation and ingestion of milk account for about 88% of the total risk to the farmer adult at Adams Ranch. The next highest summed cancer risk in Table 9-8 is 1.6E-7 for the resident adult at Adams Ranch. Table 9-9 shows that the pathway associated with the highest risk is inhalation, which accounts for nearly all of the total risk for the resident adult at Adams Ranch. The cancer risk to the resident adult is about one-third that of the cancer risk to the farmer adult. The risks to actual residents and farmers at the other off-site locations are also less than one in one million, and they are less than those estimated for Adams Ranch.

9.3.2 Quantitative Excess Lifetime Risks and Hazards for Detected Chemicals Only

Using the list of detected chemicals from Table 1-1 of the approved HHRAP protocol, risks were estimated using only the 133 detected COPC (provided in Appendix F), to determine the percentage of total risk that is contributed by including the non-detected chemicals. This analysis, presented in Table 9-15, is conducted for the discrete receptor locations that showed the highest risks for the on-site industrial worker (Autoliv), the hypothetical future resident and farmer (point of maximum deposition), and the actual off-site resident and farmer (Adams Ranch). This analysis revealed that the detected chemicals account for anywhere from 66% of the total risk to the farmer child to 91% of the total risk to the industrial worker. In other words, the non-detected chemicals account for 9% to 34% of the total cancer risk.

The non-cancer hazard quotients are not included in Table 9-15 as they did not change appreciably between all COPC and detects only. The HQs are driven by chlorine, aluminum, manganese, hydrogen chloride and nickel, which are all detected chemicals.

9.4 Breast Milk

The Lakes software calculates the Average Daily Dose (ADD) of PCDDs and PCDFs for a nursing infant under both the farmer and residential scenarios. The results are calculated for the individual dioxins and furans and a summed value is presented in Table 9-16 for each receptor location. The results for the individual dioxins and furans are presented in Appendix E. In accordance with the HHRAP guidance, the ADD is compared to the national average background exposure level of 60 picograms of dioxin toxic equivalents per kilogram per day (pg TEQ/kg/day). The summed ADD for all of the receptors is well below the national average background level of 60 pg TEQ/kg/day. The highest reported ADD is 4.7E-02 pg TEQ/kg/day for the infant of a hypothetical farmer located at Blue Creek. This value is over 1000 times less than the national average background level, indicating that the risks associated with this pathway are negligible.

9.5 Hypothetical Future Scenarios

The HHRA process assumes that ATK continues to operate for the 40 years a farmer might be exposed and that COPC concentrations do not decrease over that time. However, for the purpose of this future scenario, it is assumed that the on-site and off-site points of maximum risk are used for estimating risks to potential future receptors. The discrete receptors are evaluated for potential current exposures, and all known residences within the 10 km radius are included. However, for future development it is assumed that new residences are built off-site at the points of maximum risk, as determined by this risk assessment. These points were determined using the coordinates from the CB&I modeling report that represent the points of maximum annual and 1-hour concentration and annual deposition from sources M-136 and M-225. These points are shown on Figures A5-1 and A5-2 of the CB&I modeling report (CB&I, 2014). There are 42 unique sets of coordinates represented on these two figures and those coordinates were entered in the Lakes software as receptors. Risks were estimated for all 42 locations to determine which of the on-site or off-site locations have the highest risks. The points of maximum risk are shown on Figure 9-9. The on-site location is then evaluated for a future worker scenario, and the off-site location is evaluated for a future residential and farmer scenario. The results are shown in Tables 9-17 and 9-18 and the details of the risks and hazards, listed by COPC, are provided in Appendix E.

All estimated risks for future scenarios are less than one in one million. The highest estimated risk is 9.8E-7 for a future farmer adult at the maximum off-site location. The risk to the future on-site worker is 1.2E-07, which is slightly higher than the maximum estimated risk to a current industrial worker at Autoliv. The HIs are well below one for all of the future potential scenarios.

9.6 Lead in Soil

As presented in the approved HHRA protocol, an emissions factor was calculated for lead, but there are no dose-response factors available for use in the Lakes software. Therefore, the maximum and average lead soil concentrations were calculated at each discrete receptor location and were exported from the Lakes software. These lead values, along with the soil concentrations for the other COPCs, are presented in Appendix C. Lead has been shown to have neurological effects, and young children are particularly susceptible to the effects of lead. The US EPA evaluates lead using a bio-uptake model call the Integrated Exposure Uptake Bio-kinetic (IEUBK) model, which calculates potential blood lead concentrations based on exposure to media, food and water. The estimated blood lead levels are compared with acceptable blood lead levels for children. This model was used by the EPA to calculate a soil lead level below which no adverse effects on children would be expected. This acceptable soil lead concentration is 400 mg/kg (EPA, 2002). The maximum lead concentrations range from 6E-11 mg/kg at ATK Ranch Pond to 2E-09 mg/kg at Blue Creek, and are many orders of magnitude below the EPA default residential goal of 400 mg/kg.

10. ESTIMATING UNCERTAINY FOR HUMAN HEALTH RISK ASSESSMENT

Uncertainty occurs because risk assessment is a complex process, requiring the integration of the following area:

1. Estimates of the volume and types of chemicals processed,
2. Estimates of the amounts and types of chemicals released into the environment,
3. The fate and transport of these chemicals, in a variety of different and variable environments, by processes that are often poorly understood or too complex to quantify accurately,
4. Estimates of magnitude of exposure to the chemicals at the point of exposure,
5. The potential for adverse health effects in humans, as extrapolated from animal studies, and
6. The probability of adverse effects in a human population that is highly variable genetically, and in age, activity level, and lifestyle.

Uncertainty is inherent in the process even when using the most accurate data and the most sophisticated models. The method recommended in the HHRA guidance relies on a combination of point values—some protective and some typical—yielding a point estimate of exposure and risk that falls at an unknown percentile of the full distributions of exposure and risk. For this reason, the degree of conservatism in risk estimates cannot be known, but an evaluation of this degree of uncertainty is important. This section discusses the types of uncertainty and the areas in which uncertainty can be introduced into this assessment.

Variability is often used interchangeably with the term “uncertainty,” but this is not strictly correct. “Variability” is tied to variations in physical and biological processes. Variability can’t be reduced with additional research or information, although it may be known with greater certainty (for example, the age distribution of a population may be known and represented by the mean age and its standard deviation). “Uncertainty” is a description of the imperfect knowledge of the true value of a particular variable, or its real variability in an individual or a group.

In general, uncertainty is reducible by additional information-gathering or analysis activities (that is, better data or better models), whereas real variability does not change (although it may be known more accurately) as a result of better or more extensive measurements (Hattis and Burmaster, 1994).

Finkel (1990) classified all uncertainty into four types:

- Variable uncertainty,

- Model uncertainty
- Decision-rule uncertainty, and
- Variability

The topics identified in this uncertainty analysis are taken from EPA, 2005a (Section 8).

The goal of the uncertainty analysis is to identify important uncertainties and limitations associated with the HHRA. Uncertainties are associated with all elements of the risk assessment process from selection of COPCs through the exposure and toxicity assessment and risk/hazard characterization steps. In most cases, the methodology used to prepare the HHRA incorporates conservative assumptions with the goal of limiting the potential to underestimate receptor-specific exposures, risks, and hazards. The following discussion identifies some of the key uncertainties in the HHRA process.

10.1 Materials Processed by ATK

10.1.1 Types of Materials Processed

As part of its waste processing, ATK keeps records of the amounts and types of material processed at M-136 and M-225. They have conducted chemical analyses of the AP wastes, the rocket motor and their components, and have inventories of waste material from laboratories that are sent to the process units. These procedures have tracked waste profiles over recent years. While waste profiles have changed over the years of operations, ATK prepared test bundles for the OB/OD testing that were believed to represent a range of possible waste streams. The waste bundles contained AP waste, which typically constitutes the major waste stream, and included wastes such as paper, wood, plastics, and propellants. The composition of these test bundles is provided in the Characterization of the OB/OD Emissions Report (ATK, 2009; see Table 1, 2 and 3). These wastes may contain low levels of metals as trace components in trash, packaging, containers and housings, and metals were detected in the emissions from the OB/OD tests. As the materials processed by ATK have changed, more Flare wastes have been incorporated into the waste stream. Some unique metals are also present in proprietary flare wastes, and these were not contained in the OB/OD test bundles. The major metal additives present in these flares are identified specifically in Table 2-3, and are discussed below in Section 10.1.3.

The types of wastes may vary with each process, but the overall annual composition and volumes are known with some degree of certainty. By selecting conservative emission factors, the degree of variability will not be reduced, but the uncertainty is likely to be shifted towards the conservative, or heath protective, because it is assumed that receptors are likely to be exposed to more COPCs than are actually emitted from the process areas.

If this is correct, the risk assessment is likely to over-estimate risk. If not, the risks may be over- or under-estimated.

10.1.2 Selection of Emissions Factors

The emissions factors in Table 2-1 are selected based on the OB/OD tests performed for ATK at Dugway proving ground, as described in the approved HHRA Protocol. Emissions factors are selected, as described in Section 2, to represent the emissions from all classes of propellants. To capture the potential emissions from a wide range of wastes, and allow for flexibility in operations, but with the aim of also being conservative, and as described in Section 2.14, the following process is used:

- Where a constituent is detected in either the 1.3- or 1.1-Class OB/OD test, the emissions factor is developed from the highest detection in all of the tests. No averaging is used.
- Where one test showed the presence of a chemical and the other did not, the chemical is assumed to be present at the level detected, and the emissions factor is based on the detected concentration.
- Where a chemical is not detected in any OB/OD test (1.3- or 1.1-Class) the highest detection limit is used to develop the emissions factor, with the following exceptions:
- Where higher molecular weight PAHs are not detected, and are not formed in 1.3-Class waste emissions, the detection limit for 1.1-Class wastes is used.
- Dioxins and difurans are evaluated as classes of compounds, and the 1.3-Class detections are used because they lead to a higher 2,3,7,8-tetrachlorodibenzo-dioxin toxic equivalent factor (TCDD-TEQ), and so higher risk (See Appendix F).
- Benzidine, 3,3´-dimethyl/dichlorobenzidine, 2-acetylaminofluorene, 3-methylcholanthrene and 7,12-dimethylbenzanthracene are eliminated from the process for reasons discussed below. No emissions factors are provided for these constituents.
- The chromium in the emissions was not speciated to separate and quantify hexavalent chromium (Cr(VI)) from trivalent chromium(Cr(III)), and it was assumed (based on the discussion provided in Section 2.9.1 that 45% of the chromium is Cr(VI) and 55% of the chromium is Cr(III).

Detected COPCs

Except for the noted exceptions, this process used the highest detected emissions factors to develop a chemical's emissions factor from any of the tests conducted:

- Regardless of whether it is pure AP propellant or AP propellant plus waste;
- Regardless of whether it is 1.1-Class or 1.3-Class propellant, and
- Regardless of whether there were tests that gave lower emissions factors.

This means that, for the most part, the highest chemical emissions from any of nine tests are used to develop the COPC's emissions factor. This is considered a conservative process for selecting these emissions factors.

Where a chemical is detected in a test, but not detected in others, the detected quantity is used to develop the emissions factor. This may over- or under-estimate the risk depending on whether the non-detected concentration is higher or lower than the actual detected level of the COPC.

Non-detected COPCs

Based on discussions with the Utah DSHW, the emissions factors for non-detected compounds were included in the HHRA process. The emissions factor is developed from the method detection limit, based on the idea that a non-detected COPC is actually present at its method detection limits. This is conservative, as the chemical could be absent (at a concentration of zero) or present at any concentration up to the method detection limit. Historically, in cases where a chemical was not detected, one half of the method detection limit was used in the risk assessment process. Currently, the U.S. EPA's ProUCL statistical package (EPA, 2013), and the associated guidance document are used to determine a value to represent the non-detected chemical because risk assessment conducted under Superfund and other hazardous waste programs use a 95 percent upper confidence limit of the mean concentrations (EPA, 2013). This risk assessment uses the maximum emissions, coupled with the highest method detection limit. This approach is likely to over-estimate the concentration of the COPC.

Inclusion of Blanks and Background in the Emissions Tests

The OB/OD tests were conducted in an attempt to mimic the process units. However, there are some important differences between the tests and the actual processing; some of the key differences were the amount of material, and method of ignition of the test bundles and the test container that is sampled. In an attempt to account for these differences, a "blank" test is conducted to determine the contribution of the igniter to the

overall test. The tests were ignited with black powder and ignition wire (made of chromium-nickel), the bundles of test AP propellant or AP propellant plus wastes were contained in stainless pans, and the test container is semi-enclosed. By conducting the test without the AP or AP plus waste, a blank, or background amount could be determined.

In addition, the COPCs were trapped on sorption tubes and in reagent solutions, each of which might have its own blank, or background level of COPC present from the air or from laboratory contamination that would potentially increase the amount of chemical considered to come from the test, but in actuality are present due to the test methods.

It should be noted that there are two types of blank/background being considered: a method blank/background from the test method, and a solution blanks derived from the chemical analyses (called the "reagent blank") where the trapping solution may show a chemical to be present when it is due to test solution blank or background. In both cases no blank or background correction is made and both blanks are assumed to be due to the presence of a chemical in the waste.

The HHRA is conducted without deducting any background or blank levels of chemicals from the chemical concentrations used to develop emissions factors. This is likely to over-estimate risk because the ignition material and reagents can all contribute to background, that is, they may show the presence of a chemical when none is actually present. The areas where there is a contribution of Ni and Cr from the test method are discussed below.

10.1.3 Uncertainties Due to Chromium and Nickel in Waste

Chromium (Cr) and nickel (Ni) are good examples of where the ODOB test method may have contributed to the overall risks in the risk assessment.

Uncertainty due to Chromium in the OBODi Test

Chromium was found in the OBOD test emissions, and as shown in Table 2-1. The level of emissions from the test appears to be high relative the amount of chromium in the material being processed. This high level of chromium prompted ATK to analyze their waste streams for the presence of chromium. Ammonium perchlorate (AP) propellant contains 16% aluminum, and based on a recent analysis of the aluminum in AP fuel (ATK, 2015a), the aluminum contains approximately 3.1 parts per million (ppm or mg/kg) chromium. If it were assumed that aluminum contains 3.1 mg/kg chromium, AP waste and the aluminum is the source of all of the chromium in the waste (i.e., none in the trash), the feed material in the OBOD test, and material being processed, would contain up to 0.5 mg/kg chromium for 100% AP waste (shown below).

$$Chromium\ \ in\ AP\ waste = 3.1\frac{mg}{kg} \times \frac{16}{100} = 0.5\,{}^{mg}/_{kg}$$

With 85% AP waste mixed with 15% trash (assuming the trash contains no chromium) the waste would contain 0.42 mg/kg chromium.

$$Chromium\ \ in\ AP\ waste = 3.1\frac{mg}{kg} \times \frac{16}{100} \times \frac{85}{100} = 0.42\,{}^{mg}/_{kg}$$

With 65% AP waste mixed with 35% trash (assuming the trash contains no chromium) the waste would contain 0.32 mg/kg chromium.

$$Chromium\ \ in\ AP\ waste = 3.1\frac{mg}{kg} \times \frac{16}{100} \times \frac{65}{100} = 0.32\,{}^{mg}/_{kg}$$

100% AP Waste Assumptions

Using the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 7) the amount of chromium released from the waste in the test can be calculated for the 100% AP waste as follows:

$$Cr\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction\left(\frac{10^6 mg}{kg}\right)$$

$$Cr = 1.3 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 3.1 \times \left(\frac{100}{100}\right) \times 10^6 = 6.4\ \left({}^{mg}/_{kg}\right)\ Cr$$

$$Cr = 1.3 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 3.1 \times 1 \times 10^6 = 6.4\ \left({}^{mg}/_{kg}\right)\ Cr$$

Where:

$1.3\text{x}10^{-5}\ \left(\frac{lb}{lb}\right)$ is the emissions rate for chromium from 100% AP/0% trash,

Or $1.3\text{x}10^{-5}\ \left(\frac{kg}{kg}\right)$

$\frac{16}{100}$ is 16% aluminum in the AP waste

$3.1\ \left(\frac{mg}{kg}\right)$ is the chromium content in the aluminum in the AP waste

1.0 is the 100% AP waste in the test burn

10^6 is a conversation factor from kg to mg

85% AP Waste/15% Trash Assumptions

Using the same equation and the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 8) for the 85% AP waste, 15% trash, the amount of chromium released from the waste in the test can be calculated as follows:

$$Cr\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction \left(\frac{10^6 mg}{kg}\right)$$

$$Cr = 2.0 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 3.1 \times \left(\frac{85}{100}\right) \times 10^6 = 8.4\ \left({}^{mg}/_{kg}\right)\ Cr$$

$$Cr = 2.0 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 3.1 \times 0.85 \times 10^6 = 8.4\ \left({}^{mg}/_{kg}\right)\ Cr$$

The resulting amount of chromium in waste is 8.4 mg/kg, which is greater than 0.42 mg/kg estimated to be in the waste, indicating there is more chromium released than is available in the AP waste (assuming its only source is the aluminum in the AP waste). This emissions factor was used in the risk assessment, and based on the above calculation the HHRA assumes there is 20 times more chromium released than is present in the AP waste.

If the emissions factor of 2.0×10^{-5} lb Cr per lb of waste were adjusted to be consistent with the amount of chromium in the waste (from the analysis above) one would expect to have an emissions factor given by the following equation:

$$Cr\ Emissons\ factor = 2.0 \times 10^{-5} \times \frac{0.42}{8.2} = 9.75 \times 10^{-7}\left(\frac{lb}{lb}\right)$$

The emissions factor is given by multiplying by a factor of ${}^{0.42}/_{8.4}$, or 0.05.

65% AP Waste/35% Trash Assumptions

And finally, using the same equation and the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 9) for the 65% AP waste, 35% trash, the amount of chromium released from the waste in the test can be calculated as follows:

$$Cr\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction \left(\frac{10^6 mg}{kg}\right)$$

$$Cr = 1.6 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 3.1 \times \left(\frac{65}{100}\right) \times 10^{6} = 5.2\ \left({}^{mg}/_{kg}\right)\ Cr$$

$$Cr = 1.6 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 3.1 \times 0.65 \times 10^{6} = 5.2\ \left({}^{mg}/_{kg}\right)\ Cr$$

All of these calculations lead to higher levels of Cr than would be expected.

The highest chromium emissions rate (2.0×10^{-5}) from the 2007 OB/OD tests was used in the HHRA. This value represents the 85% AP waste 15% trash test. The amount of trash in the waste stream does not appear to significantly impact the value of the Cr emission factor. This can be seen from the fact that as the amount of trash in the waste increases from 15% to 35%, the Cr emission factor decreases from 2.0×10^{-5} to 1.6×10^{-5}. The calculations are shown in Appendix F.

Uncertainty due to Nickel in the OBODi Test

The above calculations are repeated here for nickel, which was also found in the OBOD test emissions, as shown in Table 2-1, at levels that appear to be high relative to the amount of nickel in the material being processed. ATK also analyzed their AP waste streams for the presence of nickel. Ammonium perchlorate (AP) propellant contains 16% aluminum, and this aluminum contains 29.1 parts per million (ppm or mg/kg) nickel (ATK, 2015a). If it were assumed that aluminum contains 29.1 mg/kg nickel, AP waste and the aluminum is the source of all of the nickel in the waste (i.e., none in the trash), the feed material in the OBOD test, and material being processed, would contain up to 4.7 mg/kg nickel for 100% AP waste (shown below).

$$Nickel\ \ in\ 100\%\ AP\ waste = 29.1\frac{mg}{kg} \times \frac{16}{100} = 4.7\ {}^{mg}/_{kg}$$

With 85% AP waste mixed with 15% trash (assuming the trash contains no nickel) the waste would contain 4.0 mg/kg nickel.

$$Nickel\ \ in\ 85\%\ AP\ waste = 29.1\frac{mg}{kg} \times \frac{16}{100} \times \frac{85}{100} = 4.0\ {}^{mg}/_{kg}$$

With 65% AP waste mixed with 35% trash (assuming the trash contains no nickel) the waste would contain 3.0 mg/kg nickel.

$$Nickel\ \ in\ 65\%\ AP\ waste = 29.1\frac{mg}{kg} \times \frac{16}{100} \times \frac{65}{100} = 3.0\ {}^{mg}/_{kg}$$

100% AP Waste Assumptions

Using the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 7) the amount of nickel released from the waste in the test can be calculated for the 100% AP waste as follows:

$$Ni\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction \left(\frac{10^6 mg}{kg}\right)$$

$$Ni = 3.6 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 29.1 \times \left(\frac{100}{100}\right) \times 10^6 = 167.6\ \left({}^{mg}/_{kg}\right)$$

$$Ni = 3.6 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 29.1 \times 1 \times 10^6 = 167.6\ \left({}^{mg}/_{kg}\right)$$

Where:

$3.6x10^{-5}\ \left(\frac{lb}{lb}\right)$ is the emissions rate for chromium from 100% AP/0% trash,

Or $3.6x10^{-5}\ \left(\frac{kg}{kg}\right)$

$\frac{16}{100}$ is 16% aluminum in the AP waste

$29.1\ \left(\frac{mg}{kg}\right)$ is the nickel content in the aluminum in the AP waste

1.0 is the 100% AP waste in the test burn

10^6 is a conversation factor from kg to mg

The resulting amount of nickel in waste is 170 mg/kg, which is greater than the 4.7 mg/kg that was calculated assuming that the aluminum in the AP waste is the only source of nickel in the waste stream.

85% Waste/15% Trash Assumptions

Using the same equation and the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 8) for the 85% AP waste, 15% trash, the amount of chromium released from the waste in the test can be calculated as follows:

$$Ni\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction \left(\frac{10^6 mg}{kg}\right)$$

$$Ni = 5.8 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 29.1 \times \left(\frac{85}{100}\right) \times 10^{6} = 229.5\ \left({}^{mg}/_{kg}\right)$$

$$Ni = 5.8 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 29.1 \times 0.85 \times 10^{6} = 229.5\ \left({}^{mg}/_{kg}\right)$$

The resulting amount of nickel in waste is 230 mg/kg, which is greater than the 4.0 mg/kg that was calculated assuming that the aluminum in the AP waste is the only source of nickel in the waste stream. This emissions factor was used in the risk assessment, and based on the above calculation the HHRA assumes there is over 57 times more nickel released than is actually available in the AP waste.

If the emissions factor of 5.8x10^{-5} lb Ni per lb of waste were adjusted to be consistent with the amount of nickel in the waste (from the analysis above) one would expect to have an emissions factor given by the following equation:

$$Ni\ Emissons\ factor = 5.8 \times 10^{-5} \times \frac{4.0}{230} = 1.0 \times 10^{-6}\left(\frac{lb}{lb}\right)$$

The emissions factor is given by multiplying by a factor of ${}^{4.0}/_{230}$, or 0.017.

65% Waste/35% Trash Assumptions

And finally, using the same equation and the emissions factor from the 2007 OB/OD test report (ATK, 2007; Table 9) for the 65% AP waste, 35% trash, the amount of chromium released from the waste in the test can be calculated as follows:

$$Ni\ in\ waste = Emissions\ \left(\frac{kg}{kg}\right) \times Source\ \left(\frac{mg}{kg}\right) \times Unit\ Correction\left(\frac{10^{6} mg}{kg}\right)$$

$$Ni = 1.8 \times 10^{-5}\left({}^{lb}/_{lb}\right) \times \left(\frac{16}{100}\right) \times 29.1 \times \left(\frac{65}{100}\right) \times 10^{6} = 54.5\ \left({}^{mg}/_{kg}\right)$$

$$Ni = 1.8 \times 10^{-5}\left({}^{kg}/_{kg}\right) \times 0.16 \times 29.1 \times 0.65 \times 10^{6} = 54.5\ \left({}^{mg}/_{kg}\right)\ Cr$$

All of these calculations lead to higher levels of Ni than would be expected.

The highest nickel emissions rate (5.8x10^{-5}) from the 2007 OB/OD tests was used in the HHRA. This represents the 85% AP waste 15% trash test. The calculations are shown in Appendix F.

Volatilization of Chromium and Nickel

The burn pans in which the test was conducted contain about 15 percent chromium and nickel in the steel (ATK, 2015b), and, with the nickel-chromium ignition wire are thought to be the source for the additional chromium found in the emissions. The chromium in the stainless steel pans has been shown to aerosolize during welding. In 2003, welding fumes were sampled during health and safety monitoring (for worker protection) during steel pan welding operations (ATK, 2003). The air during welding was found to contain 0.6 to 3.3 micrograms of chromium per cubic meter of air. The temperatures attained during the open burn and open detonation process are high, and TetraTech, 2011b (page 4-6) indicates flame temperatures of 4976^{o}F (100% AP propellant), 2950^{o}F (85% AP propellant), and 2260^{o}F (65% AP propellant). These temperatures are higher than temperatures found in steel welding operations (1500^{o}F) (NiDI, 1988; page 10), and where welding data from ATK shows that nickel is generated during welding the stainless steel pans used by ATK in the OB/OD test Chamber can reasonably be assumed to be a source (ATK, 2003).

A 2010 UK study found chromium in air during welding ranged from ND at 100 μg/m^3 to a detected level of 620 μg/m^3 (HSE, 2010). This study also found nickel from ND at 10 μg/m^3 to a high detected level of 350 μg/m^3. Chromium and nickel were found at high levels in confined spaces during welding steel in a petroleum plant (Wilson, et al, 1981), and this study has been confirmed by many others, including a study by Abrahams (1983) that showed 30 to 40 percent of welders have experienced metal fume fever at some time.

The risk assessment process does not make adjustments or subtractions for contributions from the equipment used in the test. However, there was an attempt to determine if the testing equipment contributed to the test by conducting a "blank" test; that is, to determine contributions to the test from the ignition wire contained in the testing pan alone. This blank test did not actually simulate or release and capture emissions from the testing equipment under actual burn conditions because of the difference in the temperatures involved. While the temperatures were not measured, it is important to recognize that the temperatures reached during the blank burn (ignition wire only) would be significantly lower than those reached during the tests because there is no material to combust, and therefore no significant combustion source available to generate the heat. Whereas the test burns, with AP waste burn at a higher temperature because of the combusted material. It is unlikely that chromium from the burn pan would be aerosolized during the blank test but chromium would aerosolize during the actual tests because of the heat involved. Therefore, the aerosolization of chromium from the pans will lead to an overestimation of chromium risk from ATK's emissions, and this would not be indicated by simply evaluating the blank data.

The estimated potential short-term hazard for On-site 1-hour nickel is above a short-term hazard index of one, if it is assumed that all of the burning ground-sub units are operated at one time. Although this is unlikely because ATK's operational protocol prevents this, it is important to recognize that the hazard index is an over-estimate. When ATK's procedures are followed, only M-136 A-1, A-2 and A-3, and M-225 A are used, the emissions are lower and below one.

The over-estimation of chromium and nickel in the OB/OD tests is important because chromium is estimated to have an elevated cancer risk and non-cancer hazard, and although these are not above the acceptable threshold of one in one million at an actual receptor, it is likely that they are over-estimated. The degree to which this occurs has been estimated below.

Chromium and Nickel - Acute Hazards Based on Adjusted Emissions

The HHRA results (Section 9.0) are based on the assumption that the chromium and nickel emissions are due entirely to their presence in AP waste plus trash (85:15), and the highest of any emissions factors is selected to characterize emissions. As described in Section 10.1.3, elevated chromium and nickel levels may be from the OBOD test. If it is assumed that chromium emissions are over-estimated by some 20-fold (see Section 10.1.3), and the emission and risks due to direct exposure should be correspondingly lower; the uncertainty can be quantified by adjusting the hazards by the factor of 0.05, as described in Section 10.1.3. Similarly for nickel, if it is assumed that the emissions are over-estimated by approximately 57-fold (see Section 10.1.3), the uncertainty can be quantified by adjusting the hazards by the factor of 0.017, as described in Section 10.1.3. Below is a discussion of the calculation of the acute hazards, using the adjustment factors of 0.05 for chromium and 0.017 for nickel.

Acute Inhalation Hazard Quotients

Section 9.0 describes how the Acute Inhalation Hazard Quotients are calculated by dividing the modeled 1-hour air concentrations by the short-term PAC-1 (1-hour) concentration provided in Table 8-2. The resulting quotient, called a Hazard Quotient (HQ) for each individual COPC is then added together for each receptor to give a Hazard Index (HI). These summed indices are shown in Tables 9-1, 9-2 and 9-3 for on-site industrial workers, boundary/off-site locations where there are no receptors (hypothetical receptors), and off-site locations representing actual receptors, respectively. The values were calculated assuming the following:

- Scenario 1: All sources (M-136 A, B, C, M-225 A and B) are burned simultaneously, and

- The original assumptions for chromium and nickel that potentially overestimate the amounts present in waste by a factor of 20-fold and 57-fold, respectively.

After discussions with ATK and UDSHW, these HQs were recalculated assuming the following:

- Scenario 2: Sources M-136 A1, A2, A3 and M-225 A are burned simultaneously, and
- Scenario 3: The chromium amounts were adjusted by multiplying by a factor of 0.05, and
- The nickel amounts were adjusted by multiplying by a factor of 0.017.

These three scenarios are presented in Tables 9-1, 9-2 and 9-3. Scenario 3 represents ATK's best estimate of conservative but more likely acute hazards associated with facility operations. It is also more realistic because it doesn't include simultaneous burning of all sources. These summed indices are shown in Table 10-1, 10-2 and 10-3 for on-site industrial workers and hypothetical future workers, boundary/off-site locations where there are no receptors (hypothetical receptors), and off-site locations representing actual receptors. The HIs are below one for all receptors and scenarios. The HIs range from 0.2 to 0.5 for the on-site workers as shown in Table 10-1. The HIs for the boundary/off-site hypothetical receptors are presented in Table 10-2, and range from 2.9E-02 to 0.7; the latter of which is the maximum HI for the discrete receptors and represents a hypothetical receptor located at Boundary 1. The HIs range from 3.4E-02 to 0.4 for the actual, off-site receptors, as presented in Table 10-3. The Acute HQs for each individual COPC at each individual receptor are provided in Appendix F.

It can be seen from these tables that the HIs have been reduced significantly due to the reduction in the HQ for both chromium and nickel.

Chromium and Nickel – Chronic Hazards and Risks Based on Adjusted Emissions

Similar to the process described in the previous section, the chronic non-cancer hazards and excess lifetime cancer risks presented in Tables 9-4 through 9-14, 9-17 and 9-18, were calculated assuming the following:

- All sources (M-136 A, B, C, M-225 A and B) are burned simultaneously, and
- The original assumptions for chromium and nickel that potentially overestimate the amounts present in waste by a factor of 20-fold and 57-fold, respectively.

Applying the same logic that was used to adjust the chromium and nickel in the Acute Hazards, the chronic risks and hazards were recalculated assuming the following:

- All sources (M-136 A, B, C, M-225 A and B) are burned simultaneously, and
- The inhalation risks due to chromium were adjusted by multiplying by a factor of 0.05, and
- The inhalation risks due to nickel were adjusted by multiplying by a factor of 0.017.

This scenario represents ATK's best estimate of conservative but more likely chronic hazards and risks associated with facility operations. The chronic risks and hazards address long term exposures that are summed and evaluate combined exposures from multiple sources, therefore specific sub sources are not selected as they were in addressing acute hazards. Also, the chromium and nickel adjustment factors were only applied to the inhalation pathway. An example of the linear adjustment made to the chromium inhalation risk is shown below. The values represent the Industrial Worker scenario at Autoliv, where inhalation is the only pathway via which workers are potentially exposed to emissions.

Original Cr Inhalation Risk X Cr Adjustment Factor = Adjusted Cr Inhalation Risk

1.5E-07 X 0.05 = 7.7E-09

The original inhalation risk for chromium was multiplied by a factor of 0.05 to obtain the adjusted risk estimate. Similarly, the original inhalation risk for nickel was multiplied by a factor of 0.017 to obtain the adjusted risk estimate.

1.4E-09 X 0.017 = 2.4E-11

Chromium was a risk driver for the inhalation pathway, so the chromium adjustment has a greater impact on the total risk than the nickel adjustment.

A similar calculation was done for all of the receptor types (Adult and Child Farmer and Adult and Child Resident) for all of the discrete receptor locations. Also, the same calculation was performed for the non-cancer hazards. The results of the adjusted risks and hazards are presented in Tables 10-4 through 10-8. Tables 10-9 and 10-10 present the adjusted risks and hazards for the future scenarios at the maximum on-site and maximum off-site locations, respectively. The points of maximum on-site and off-site risk are provided for reference. However, there are no receptors at these locations. The details of these adjusted chronic risk and hazard calculations are presented in Appendix F.

Summary of Chronic Hazard and Risk Estimates

Tables 10-11, 10-12 and 10-13 present a summary of all scenarios evaluated for chronic hazards and risks. The results are summarized for ease of comparison between the scenarios evaluated. Table 10-11 presents the chronic hazards and risks for the current industrial workers as well as the future hypothetical on-site worker. Table 10-12 presents chronic hazards for the existing off-site receptors and both current and future hypothetical receptors. The first scenario (Scenario A) presented in the tables includes simultaneous burning of all sources (M-136 A, B, C and M-225 A and B). The second scenario (Scenario B) includes the same sources, with the adjustment for chromium and nickel as discussed above. Scenario B represents ATK's best estimate of conservative but more likely chronic hazards and risks associated with facility operations. Within Table 10-12, the hazards are very similar for both scenarios. The adjustment for chromium and nickel did not have a big impact on the hazard estimates because these chemicals are not contributing significantly to the non-cancer hazards.

Table 10-13 presents chronic cancer risks for the existing off-site receptors and both current and future hypothetical receptors. A comparison of the two scenarios evaluated reveals that the chromium and nickel adjustment resulted in a significant reduction in the cancer risks, as discussed above.

Chromium Speciation

In the original tests, chromium was measured as total Cr, and not speciated into the two valent forms of Cr: Cr(VI) and Cr(III). These two Cr metal species have significantly different toxicological properties. There are no literature studies available on the amount of either Cr species present in the emissions from OB/OD facilities. In the absence of data, EPA, 2005a (Section 2.3.5.1) states the following:

"We generally recommend using the following method (developed by us through interpretation of data available in the MACT database, as documented in Appendix D) to generate a default speciation:

- When the measured amount of total chromium is <10 micrograms per dry standard Cubic meter (μg/dscm), we recommend a default of 5 μg/dscm hexavalent chromium.
- When the measured amount of total chromium is in the range of 10 μg/dscm to 100 μg/dscm, we recommend assuming 45 percent is hexavalent chromium.
- When the measured amount of total chromium is >100 μg/dscm, we recommend assuming 30 percent is hexavalent chromium."

The total Cr emissions measured in the OB/OD test are greater than 100 μg/dscm, and the ratio of 30% Cr(VI) and 70% Cr(III) would be recommended when evaluating the emissions from facilities with stacks, based on the above. The percentage of Cr(VI) was established by EPA based on documentation described in Appendix D of EPA, 2005a.

For the purpose of understanding the range of uncertainty associated with the chromium speciation, the range of risk and hazards are calculated for an assumed adult farmer scenario assuming Cr(VI) is present at 100%, 45% and 14.3%, the latter of which was selected based on available data in a literature search. The point of maximum deposition was selected even though this location is not occupied because it corresponds to the highest cancer risk estimates. The results of these calculations are provided in Table 10-14 and Appendix F. The acute and chronic non-cancer hazards did not change based on the percentage of Cr(VI), therefore, those results are not included in Table 10-14. The cancer risks ranged from 6.5E-7 to 1.6E-6 assuming 14.3% and 100% Cr(VI), respectively. However, after adjusting for the amount of Cr that originates from the test pans, the cancer risks are all very similar and are approximately 5E-7, regardless of the percentage of Cr(VI) that is assumed. This process demonstrates that assuming 45% Cr(VI) is reasonable and will not significantly underestimate risks.

Cr and Ni Metal Sequestration

In addition, ATK is interested in the fate of aluminum and other metals released during the use of AP fuels and in a recent paper (ATK, 2015) they have demonstrated that aluminum forms small spheres. During the use of AP fuel these small Aluminum spheres are available for contact in air, and in soil. Aluminum is a relatively non-toxic metal and is used extensively in food and beverage storage and distribution (ATSDR, 2008). ATK's paper (ATK, 2015) shows that metal impurities, such as chromium and nickel, are distributed within the small aluminum beads and not on the surface, and so are likely to have low bio-availability in soil ingestion and plant uptake pathways. Therefore, the assumption that Cr(VI) is present in secondary pathways, such as milk, meat, and vegetables is conservative and likely to lead to an over-estimate of risk. For direct pathways, such as inhalation and soil ingestion, risks from Cr(VI) may also be over-estimated because Cr(VI) is bound into an aluminum matrix and less available than the 100 percent availability assumed in the HHRA.

Mercury

Mercury was detected in one sample of emissions from pure AP propellant. This too is likely to be an artifact of the testing process and the presence of low levels of mercury in the test pans, the ignition sources because mercury has not been found in ATK's waste stream at a method detection limit of 0.03 mg/kg or 30 micrograms per kilogram (ATK,

2014b). The Lakes model evaluates mercury as particulate bound, and mercury vapor, and the conservative assumption is made that it is present as both.

Other Metals

ATK processes flare wastes that contain a number of metals, as shown in Table 2-3. There are a number of metals used in the production of flares that are present in ATK's waste but were not present in the OBOD test bundles. These metals include: bismuth, boron, cesium, indium, iron, lead, silicon, strontium, tin, zinc and zirconium. As these chemicals are processed but do not have emissions factors the risk assessment will potentially underestimate risk. Risk is a combination of the level of exposure to a chemical coupled with the chemical's toxicity. The metals identified in Table 2-3 are relatively non-toxic, as indicated by their relatively high Reference Doses. They are a similar order of magnitude as iron, a metal essential for human health. Therefore, with the exception of zirconium, the risk from these metals would be low. While the oral toxicity of zirconium is high, there is no inhalation Reference Concentration available at this time.

Polynuclear Aromatic Hydrocarbons

Due to the high temperatures and highly oxidizing environment in the thermal destruction processes for propellants, the formation of high molecular PAHs and methylated PAHs has been shown to be unlikely (see Section 2). However, high molecular weight PAHs have been included in the HHRA. This inclusion of chemicals that may not be present is likely to over-estimate risk.

The exclusion of methylated PAHs from the process, if they are in fact formed could lead to an underestimation of the risks.

Benzidine Compounds

Benzidine compounds are typically associated with the manufacture of azo-dyes. These compounds have been banned from the US since the 1970s and it is highly unlikely that they are to be found in the US, and it is even more unlikely that they are present in ATK's waste stream. They are more a remnant of the US history of manufacturing than a real threat to human health at ATK. In addition, they are unstable compounds that are unlikely to be generated, or to be stable in the high temperature, oxidizing conditions of thermal destruction. Therefore, they have been eliminated from the risk assessment. If they were present, the risk assessment would underestimate the risk from these chemicals.

10.2 Modeled Air Concentrations and Deposition

The air quality modeling process contains a number of assumptions, some are thought to overestimate and some to underestimate ambient air concentrations, and deposition.

The air quality model is a hybrid model of OBODM and AERMOD, and while these models are thought to be conservative models that will potentially over predict the air concentration of pollutants, but this has not been verified because this specific application of the models has not been tested. The original OBODM air dispersion model was developed by the West Desert Test Center, Dugway, Utah (WDTC, 1998a), and was used by TetraTech in 2011 for project scoping. In 2014, the OBODM was updated by CB&I Environmental & Infrastructure, Inc. (CB&I) in conjunction with the air dispersion portion of the AERMOD model to take maximum advantages of advances in air dispersion modeling science. The approach used by CB&I, while less conservative than the TetraTech approach, better represents the process and is still considered conservative. However, in their modeling protocol CB&I stated the following concerning the OBODM: at this time, OBODM is not actively supported by its developers or by any regulatory agency.

The models use meteorological data from the facility, and the protocol was approved by the Utah DSHW. There are a number of components to the model, as discussed in Section 3. These include: emissions source parameters (such as, emissions rates and plume rise characteristics), air dispersion parameters, and depositional characteristics. The uncertainty associated with these components is summarized below.

10.2.1 Emissions Source Parameters

Source emissions are estimated based on the amount of material processed (pounds per year) and emissions of compounds from the burning process (combustion by products, and products of incomplete combustion). The HHRA is based on the amount of material processed per year by ATK, and is provided in the modeling protocol. This amount is used in the HHRA and represents the maximum amount that may be processed by ATK under their permit. In the past few years ATK has processed significantly less material than allowed in the permit, and therefore, the actual emissions during those years were lower. Current and future operations will not exceed the permitted amount, and so the risk assessment is intended to represent an over-estimate of emissions, and thus, provide a conservative estimate of risk.

The emissions factors are discussed above in Section 10.1.2.

ATK uses two burning grounds M-136 and M-225, and within each of these areas there are designated locations for processing different types and/or amounts of materials.

M-136 is the primary processing area, and it has three operational scenarios, M 136-A, M 136 B and M-136 C. Scenario M-136 A is further sub-divided into A1, A2 and A3. ATK uses M-136 A to process the majority of the AP-related waste, whereas M-136 B is used to process single motors, and M 136 C is used to treat waste material through open detonation. The three scenarios are not used on the same day for safety reasons. It would be hazardous to employees to have all treatment scenarios contain materials for processing at the same time. The initial HHRA modeling protocol assumed all scenarios would operate at the same time; however, the model assumed each source operates independently, and the results are summed together. This approach over-estimates risk, whereas adjusting for burn scenarios that cannot occur on the same day calculates risks for the actual operating situation.

This issue of selecting specific sub-sources does not affect the chronic aspect of the risk assessment because it addresses long term exposures that are summed to evaluate combined exposures from multiple sources.

The initial assumption of all treatment scenarios operating together would over-estimate the acute air concentrations because it combines estimated air concentrations from sources M-136 A, M-136 B and M-136 C together, to give a summed potential air chemical concentrations at each receptor, when in fact this does not occur. Due to the operating conditions at M-136, acute air chemical concentrations would come from processing at either M-136 A, or M-136 B, or M-136 C and not from the sources added together.

Based on discussions with the Utah DSHW, the initial protocol was modified to reflect actual operating conditions and not the hypothetical situation of all grounds operating together. Therefore, the HHRA includes the simultaneous burn scenarios M-136 A (A1, A2 and 3 with M-225 A, because this is consistent with ATK's typical operating protocol. ATK anticipates the permit conditions will be based on the combinations of burn scenarios in the HHRA, and future ERA, and those would include any one of the M 136 scenarios (A, B or C) and any one of the M-225 scenarios (A or B). For example, M-136 B and M-225 B might occur on the same day.

10.2.2 Emissions Rates

The emission rate is a combination of the emissions factor and the amount processed. The modeling protocol provides emissions at a unit rate in grams per second (g/s), which is multiplied by the compound specific emission factor to give the amount released. ATK uses a batch process and emissions occur within a short period of time. There is some uncertainty; however, the use of conservative emissions factors selected in the modeling is designed to help provide results that tend to over-estimate exposure, and risk. When

modeling short-term air chemical concentrations, the amount burned in a single batch will have a direct impact on potential exposure.

Modeling was conducted by modeling single burns using a unit emissions rate in grams per second, and adjusting the air concentration by the emissions factor. The single burn uses conservative factors to give the highest air concentration for calculation of the short-term inhalation Hazard Index. This is more conservative for the estimation of acute risk because it is based on exposure and not the amount processed.

When estimating the long-term (chronic) hazards and risks, the overall amount processed is more important because the summed accumulation of exposure is important, not one particular burn cycle. In the chronic case, the risks are based on the total amount processed, as discussed above.

The OBODM is designed to model air emissions from open burning/open detonation process. There are a number of components to the model:

- The location of the burning ground relative to the local terrain
- The type of source release (quasi-continuous or instantaneous)
- The area of the source
- The release height relative to the local topography
- The heat of combustion and associated plume rise

Some of these parameters can be measured with relative precision, such as the height of the burning grounds, and other may vary, such as the combustion temperature and the estimated plume rise. The more uncertainty present in a parameter, the greater the uncertainty in the final result. At the time of its development through the present day, the OBODM portion of the modeling process is a listed EPA alternative air dispersion model, and is thought to be conservative. The combustion temperature is one of the key parameters in the model as it affects gaseous plume rise, and so affects the height above the ground where dispersion starts. At Promontory, the plume dimension and volume was verified through video monitoring, and the volume was found to be four to eight times the size of the burn pan. A volume of four times the burn pan was used, to be conservative.

10.2.3 Air Dispersion Parameters

The AERMOD dispersion model was used by CB&I. This model calculated the dispersion of the gas cloud under the assumed meteorological conditions, and the

associated theoretical air chemical concentration at the locations on a receptor grid. The following parameters were used in the model:

- Emission rate of 1 g/s
- Release height predicted from OBODM using five wind speed categories covering a range of 3.0 mph – 15 mph
- Initial volume source diameter of 28.06 m (four times the size of the burn pan)
- The events will occur only between the hours of 9:00 a.m. Mountain Standard Time (MST) and 6:00 p.m. MST
- The wind speed during the events will be between 3 mph and 15 mph
- The Clearing Index during the events will be 500 or higher

There is uncertainty in parameters, and the values were selected by CB&I to be conservative. The Stability Class was selected based on Meteorological data to provide the lowest dispersion under ATK's normal operating conditions.

The dispersion modeling conducted with AERMOD utilized hourly meteorological data files. Five years (1997 to 2001) of on-site meteorological data obtained from the site were used in the modeling entitled "Addendum Air Dispersion Modeling Report for Open Burning and Open Detonation at ATK Launch Systems in Promontory Utah" dated August 2014. The hourly meteorological data were obtained from the site in CD-144 format and included wind speed, wind direction, temperature, and barometric pressure monitored at the site along with concurrent ceiling height and opaque cloud cover from Hill Air Force Base. These data may not precisely represent the meteorological conditions at the facility, but are believed to constitute the best available representation of conditions at the burning grounds.

For NAAQS and air toxics analysis, an off-site receptor grid was used to determine the maximum off-site ground level concentrations. The layout of the receptors was placed along the property fence line at 100-m intervals. A Cartesian receptor grid starting from the property line extended up to 10 km in all directions with a receptor grid spaced at 100-m intervals to a distance of 3 km from the facility and at 500-m intervals between 3 km and 10 km from the facility. This grid spacing was considered sufficiently small to represent the area.

The uncertainties in the air modeling approach are discussed in CB&I's 2014 modeling report. The model was considered to be sufficiently conservative by the Utah DSHW, who accepted the protocol in November 2014.

10.2.4 Depositional Characteristics

In addition to calculating hourly, 24-hour and annual ambient air concentrations, the model calculated deposition of particulate matter. Within AERMOD, the gas phase dry deposition was modeled using a conservative deposition velocity of 0.03 meter per second (m/sec), which is the highest of the default values specified in the HHRAP guidance. This velocity is consistent with the value used in the preliminary modeling performed by Tetra Tech and proposed in ATK Launch Systems Waste Characterization and Air Dispersion Final Modeling Protocol for Use in the Human Health and Ecological Risk Assessments, dated 2011. UDHSW accepted the proposed value as part of the technical review of the protocol document.

10.3 Chemical Uptake, Food Chain Modeling and Dose Estimates

Plant uptake and food chain models are used to predict the potential for a chemical to translocate from one medium to another. This process is most common with hydrophobic, and lipophilic molecules such as dioxins, difurans, PCBs and some pesticides. It is also possible with other selected metals, such as mercury, which can bioconcentrate as Hg(0) or elemental mercury, methyl mercury in aquatic systems and selenium in certain circumstances.

The lipophilic nature of these bioaccumulative chemicals is used in the translocation process and measures such as octanol/water partition coefficients (K_{ow}) of the log of this value (log K_{ow}) is used as surrogates for bioaccumulation or bio-translocation. Therefore, the ability of this value to represent the uptake process becomes a key uncertainty in the bio-uptake and bio-accumulation estimation process.

10.3.1 Volatile versus Particle Bound Biotransfer Factors

Low Molecular Weight and Volatile COPC

Air quality modeling estimates the COPC concentrations as either gas or particle bound. For low molecular weight COPCs it is reasonable to assume that the chemical is present in the plume rising from the incineration process as a volatile constituent that travels as a gas. The gas would disperse with other hot gasses and be present at a receptor in this form, where it might be inhaled by a human receptor, or absorbed into plant matter. The HHRA assumes a COPC will adsorb into plant matter or adsorb onto soil by deposition. Plant uptake is assumed to be governed by physical chemical partition parameters based on K_{ow}. For low molecular weight compounds this is a reasonable assumption and may over- or under-estimate risk.

High Molecular Weight and Volatile COPC

For high molecular weight COPCs it is also assumed that the chemical is present in the plume rising from the incineration process as a volatile constituent that travels as a gas, and that the gas will disperse with other hot gasses to be present at a receptor as a gas where it might be inhaled by a human receptor, or absorbed into plant matter. The HHRA assumes a COPC will adsorb into plant matter or adsorb onto soil by deposition. Plant uptake is assumed to be governed by physical chemical partition parameters based on octanol/water partition coefficients. For high molecular weight compounds this not necessarily a reasonable assumption. Higher molecular weight compounds typically have higher melting points and may be a solid at higher temperatures, or are more likely to attach to particulate matter present in the plume when the temperature of the hot gasses cool, or when the vapor high molecular compounds come in contact with particles. Therefore, assuming these COPCs are available for direct absorption into plant matter is a very conservative assumption, and one that drives the risk assessment process for these COPC. There is a large difference between the plant tissue concentration of COPC that is present through direct leaf absorption and through the alternative absorption route of deposition onto soil, soil mixing, root uptake from soil, and distribution through the plant. This difference may be as high as four to six orders of magnitude, or 10,000 to 1,000,000, and has a significant impact on the risk assessment process for bioaccumulative compounds.

10.3.2 Food Chain Models

The EPA (2005a), and the Lakes model, provides foliage chemical bioaccumulation equations for the calculation of plant and animal tissue chemical concentrations based on a chemical's lipophilic properties (or K_{ow}) because these chemicals have a propensity to bio-accumulate. If a COPC's physical chemical parameters are unknown the uptake factor is assumed to be one (meaning 100%).

One of the key factors driving the risk assessment process is a COPC's leaf uptake factor, which represents the amount of COPC absorbed into the leaf from the COPC's concentration in air. This factor introduces additional uncertainty into the risk assessment process because it magnifies concentrations when moving up the food chain from plants to animals, and from one animal to another (e.g., from beef to humans).

Where possible, bio-uptake and bio-accumulation factors are taken from the EPA's Incineration Guidance (EPA, 2005a). However, to the extent possible, default factors are evaluated to determine if more scientifically accurate and appropriate factors can be used in the risk assessment process. Appendix D contains the chemical specific parameters and biotransfer factors for the COPC not in the HHRAP Database and for those chemicals

that have changed based on these modifications. The uncertainty associated with the selection and use of uptake, biotransfer and bio-accumulation factors is high.

10.4 Dose-response Modeling

A dose-response relationship describes the likelihood and severity of adverse health effects (the responses) related to the amount and condition of exposure to an agent (the dose provided). Dose-response relationship principles generally apply for studies where the exposure is to a concentration of the agent (e.g., airborne concentrations applied in inhalation exposure studies), and the resulting information is referred to as the "concentration-response" relationship.

Typically, as the dose increases, the measured response also increases. One of the key issues for risk assessment is that toxicology experiments often require high doses to force an effect, whereas, typical exposures encountered under environmental conditions are low. In these cases, the dose-response model extrapolates a response at low doses from high dose animal experiments using conservative, or health protective assumptions.

For cancer effects, a dose-response curve with no threshold assumes a straight line is drawn from the point of departure (lowest effect) for the observed data to the origin (where there is zero dose and zero response). The slope of this straight line, called the slope factor or cancer slope factor, is use to estimate risk at exposure levels that fall along the line. When linear dose-response is used to assess cancer risk, EPA calculates excess lifetime cancer risk (i.e., probability that an individual will contract cancer over a lifetime) resulting from exposure to a contaminant by considering the degree to which individuals were exposed, as compared to the slope factor.

There is uncertainty in this process, as there is often only two or three points on the line, in addition to the origin or zero point, and these may not form a straight line. Slope factors are drawn to provide a conservative measure of the probability that cancer might result from exposure, that the human response is the same as in animals, and that the exposure will cause cancer when there might be a level that will not.

For non-cancer effects a Reference dose approach is used. The reference dose (RfD) is an oral or dermal dose derived from the No Observed Adverse Effect Level, Lowest Observed Adverse Effect Level (LOAEL) or Benchmark Dose Level with the application of uncertainty factors, (generally order-of-magnitude) that will not have an adverse effect. These uncertainty factors take into account the variability and uncertainty that are reflected in possible differences between test animals and humans (generally 10-fold or 10x) and variability within the human population (generally another 10x); the uncertainty factors are multiplied together: 10 x 10 = 100x. If a LOAEL is used, another uncertainty

factor, generally 10x, is also used. In the absence of key toxicity data (duration or key effects), an extra uncertainty factor(s) may also be employed. This approach is also conservative (health protective).

10.4.1 Acute Non-cancer Hazards

The acute Cal EPA PAC are developed from a range of dose-response experiments, or epidemiology studies in humans. Each chemical was evaluated based on the data available. DOD values were developed using the same approach.

The chemical with the highest acute Hazard Index is nickel, which is calculated using the Cal EPA Reference Exposure Level of 0.2 $\mu g/m^3$ nickel. This is based on a study by Graham, (1978) supported by Adkin (1979), who demonstrated increased mortality in mice exposed to nickel chloride aerosol followed by streptococcal infection. Nickel dosing levels were 0, 100, 250, 375 and 490 $\mu g/m^3$ and were adjusted using a benchmark dose approach from a level of 165 $\mu g/m^3$ to 0.2 $\mu g/m^3$. This is a conservative approach and was supported by immunological responses in humans.

10.4.2 Chronic Non-cancer Hazards

The non-cancer Hazard Index is calculated by dividing the dose by the RfD, or the air concentration by the RfC. There is uncertainty in this calculated value because there is uncertainty in the exposure dose or concentration, as described above, and uncertainty in the RfD and RfC.

Therefore, the uncertainty could be magnified by multiplying the uncertainty in each value.

10.4.3 Excess Lifetime Cancer Risks

The excess lifetime cancer risks are calculated by multiplying the dose by the cancer slope factor. Each of these factors has uncertainty and when multiplied the uncertainty would also be magnified.

Chemicals with a mutagenic MOA are considered to act on DNA and form adducts or cause damage that may be more potent in children compared with adults. EPA has provided guidance on methods that should be used to modify cancer potency dose-responses for children. For the chemical compounds identified in EPA, 2005b, mutagenicity is often determined from *in vitro* experiments in cell systems, or extrapolated from *in vivo* experiments from other compounds in the same chemical class, or with similar chemical structures because direct experimental data is unavailable. This is particularly true for PAHs other than B[a]P. This has the potential to over-estimate

risk. For chemicals other than PAHs the cancer potency was adjusted using methods consistent with the EPA's IRIS file for each chemical as discussed in Appendix D. There is less uncertainty concerning these chemicals because the dose-response information is more specific to each chemical rather than making an assumption for the overall class of chemicals.

10.5 Overall Risk Estimates

There is uncertainty in the overall risk assessment process, and there is uncertainty in the final risk estimates. It is important to understand which assumptions have a potentially large impact on the overall risk assessment results and which do not. There may be key assumptions that drive the risks, and implementing regulatory requirements based on an assumption may not be the most appropriate method of regulating the Facility. For example, if a chemical is not detected, it is assumed to be present, and its emissions factor is based on an elevated method detection limit, the risk assessment would be based on an assumption rather than on real data.

Table 10-15 summarizes many of the uncertainties in the HHRA and the risk assessment process. It also demonstrates that the final risk estimates will likely overestimate the potential risks because they multiply conservative uncertainties assumed in the various parameters and compound it. While some of the items listed in Table 10-15 demonstrate that risks may be underestimated in some cases, the majority of the assumptions lead to an overestimation of the final risks and hazards. For example, the use of the highest emissions factors from the OB/OD tests, the addition of non-detected chemicals, the use of worst case meteorological conditions at the time wastes are burned or detonated, the use of conservative biotransfer and uptake factors, the use of human exposure and diet and intake factors that represent the Reasonable Maximum Exposure, and finally the use of toxicological dose-response factors that contain safety factors and are designed to be health protective of the majority of the population, all lead to an overestimation of risk.

10.6 Hunters at Salt Creek Waterfowl Management Area and Bear River Migratory Bird Refuge

Hunters at the Salt Creek Waterfowl Management Area and Bear River Migratory Bird Refuge could be exposed to constituents of concern via inhalation of gases and particulates, and the ingestion of contaminated birds. This exposure pathway is not evaluated quantitatively in the HHRA because there are no standard default exposure assumptions for this pathway, exposure periods are typically short because hunters only spend a limited amount of time hunting, and the locations for this scenario are quite far removed from the source. In addition, the incremental risk associated with the ingestion of game by hunters is expected to be less than the dietary exposure risk quantified for a

resident farmer (adult and child) due to the relatively low ingestion rate for home prepared game as described in Section 5.3.4. Consequently, the risks are expected to be low, and a quantitative evaluation was not performed.

- Figure 9-3 shows the location of the Salt Creek Waterfowl Management Area and Bear River Migratory Bird Refugee relative to M-136 and M-225. These two locations are approximately 6.0 and 6.7 miles, respectively, from the closest source area, M-225. The nearest location with the highest impacts is Blue Creek which is only 0.5 miles from the source. The potential impacts from OB/OD operations at the two sites are low because of the distance from these sources.

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FIGURES

Figure 1-1
Promontory Facility Location Map

Figure 1-2
M-136 and M-225 Location Map, Promontory Facility

Figure 1-3
Layout of Sub-Sources in M-136 and M-225
Promontory Facility Location Map

Figure 5-1
Locations of All Receptors
Promontory Facility Location Map

Figure 9-1
Short-Term (1-Hour) Air Hazard Indices for On-Site Receptors

Figure 9-2
Short-Term (1-Hour) Air Hazard Indices for Boundary/Off-Site Receptors

Figure 9-3
Short-Term (1-Hour) Air Hazard Indices for Off-Site Receptors

Figure 9-4
Actual On-Site Industrial Receptors Non-Cancer Hazard Indices and Cancer Risks [All COPCs]

Figure 9-5
Non-Cancer Hazard Indices for All COPCs: Hypothetical Boundary/Off-Site Resident and Farmer Receptors

Figure 9-6
Cancer Risks for All COPCs: Hypothetical Boundary/Off-Site Resident and Farmer Receptors

Figure 9-7
Cancer Risks for All COPCs: Actual Off-Site Resident and Farmer Receptors

Figure 9-8
Non-Cancer Hazard Indices for All COPCs: Actual Off-Site Resident and Farmer Receptors

Figure 9-9
Summed Risks and Hazard Indices for All COPCs: Future Resident/Farmer at Point of Maximum Off-site Risk and Future Worker at Point of Maximum On-site Risk

TABLES

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
83-32-9	Acenaphthene	5.48E-07
208-96-8	Acenaphthylene	3.08E-06
75-07-0	Acetaldehyde	9.30E-05
67-64-1	Acetone	2.40E-05
75-05-8	Acetonitrile	1.90E-05
98-86-2	Acetophenone	2.68E-06
107-13-1	Acrylonitrile	1.60E-05
100-44-7	alpha-Chlorotoluene	5.70E-07
7429-90-5	Aluminum	4.00E-02
92-67-1	Aminobiphenyl, 4-	1.10E-05
62-53-3	Aniline	8.00E-06
120-12-7	Anthracene	1.30E-07
7440-36-0	Antimony	2.90E-05
7440-38-2	Arsenic	5.50E-07
7440-39-3	Barium	3.90E-07
100-52-7	Benzaldehyde	3.80E-05
71-43-2	Benzene	1.20E-04
56-55-3	Benzo(a)anthracene	5.86E-07
50-32-8	Benzo(a)pyrene	7.69E-08
205-99-2	Benzo(b)fluoranthene	1.15E-06
191-24-2	Benzo(ghi)perylene	4.55E-07
207-08-9	Benzo(k)fluoranthene	1.15E-06
65-85-0	Benzoic acid	6.24E-05
100-51-6	Benzyl alcohol	7.77E-07
111-91-1	bis(2-Chloroethoxy)methane	5.48E-07
111-44-4	bis(2-Chloroethyl)ether	6.13E-07
117-81-7	bis(2-Ethylhexyl)phthalate	1.17E-06
75-27-4	Bromodichloro methane	7.80E-07
75-25-2	Bromoform	1.30E-06
74-83-9	Bromomethane	1.20E-07
101-55-3	Bromophenyl phenyl ether, 4-	5.48E-07
78-93-3	Butanone (MEK), 2-	3.90E-06
106-98-9	Butene, 1-	2.20E-05
590-18-1	Butene, cis-2-	1.70E-06
624-64-6	butene, trans-2-	7.70E-06
85-68-7	Butyl benzyl phthalate	1.50E-07
7440-43-9	Cadmium	4.70E-08

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
86-74-8	Carbazole	7.01E-07
75-15-0	Carbon Disulfide	9.80E-06
56-23-5	Carbon Tetrachloride	1.50E-05
59-50-7	Chloro-3-methylphenol, 4-	6.79E-07
107-14-2	Chloroacetonitrile	1.10E-06
106-47-8	Chloroaniline, 4-	1.55E-07
108-90-7	Chlorobenzene	2.50E-06
75-00-3	Chloroethane	4.40E-07
67-66-3	Chloroform	6.10E-06
74-87-3	Chloromethane	1.40E-05
90-13-1	Chloronaphthalene, 1-	5.48E-07
91-58-7	Chloronaphthalene, 2-	5.48E-07
95-57-8	Chlorophenol, 2-	1.92E-06
7440-47-3	Chromium (III)	1.1E-05
18450-29-9	Chromium (VI)	9.00E-06
218-01-9	Chrysene	7.23E-07
7782-50-5	Cl2	1.18E-02
7440-48-4	Cobalt	1.20E-07
7440-50-8	Copper	2.50E-05
4170-30-3	Crotonaldehyde	3.20E-06
98-82-8	Cumene	4.20E-07
110-82-7	Cyclohexane	2.50E-06
53-70-3	Dibenz(a,h)anthracene	1.02E-07
132-64-9	Dibenzofuran	5.48E-07
124-48-1	Dibromochloromethane	8.80E-07
106-93-4	Dibromoethane (EDB), 1,2-	8.90E-07
95-50-1	Dichlorobenzene, 1,2-	5.59E-07
541-73-1	Dichlorobenzene, 1,3-	6.24E-07
106-46-7	Dichlorobenzene, 1,4-	5.81E-07
75-34-3	Dichloroethane, 1,1-	3.20E-07
107-06-2	Dichloroethane, 1,2-	5.40E-07
75-35-4	Dichloroethene, 1,1-	4.30E-07
156-59-2	Dichloroethene, cis-1,2-	1.20E-07
156-60-5	Dichloroethene, trans-1,2-	7.20E-07
120-83-2	Dichlorophenol, 2,4-	9.26E-07
87-65-0	Dichlorophenol, 2,6-	5.48E-07
78-87-5	Dichloropropane, 1,2-	3.70E-07

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
10062-01-5	Dichloropropene, cis-1,3-	1.30E-06
10061-02-6	Dichloropropene, trans-1,3-	6.10E-07
84-66-2	Diethyl phthalate	8.00E-07
105-05-5	Diethylbenzene, 1,4-	6.70E-07
105-67-9	Dimethyl phenol, 2,4-	6.90E-06
131-11-3	Dimethyl phthalate	5.48E-07
60-11-7	Dimethylaminoazobenzene, p-	5.48E-07
5779-94-2	Dimethylbenzaldehyde, 2,5-	2.70E-05
75-83-2	Dimethylbutane, 2,2-	1.40E-06
79-29-8	Dimethylbutane, 2,3-	3.50E-06
565-59-3	Dimethylpentane, 2,3-	1.40E-05
108-08-7	Dimethylpentane, 2,4-	5.20E-06
84-74-2	Di-n-butyl phthalate	1.10E-05
534-52-1	Dinitro-2-methylphenol, 4,6-	9.53E-06
99-65-0	Dinitrobenzene, 1,3-	5.70E-07
51-28-5	Dinitrophenol, 2,4-	2.41E-05
121-14-2	Dinitrotoluene, 2,4-	5.48E-07
606-20-2	Dinitrotoluene, 2,6-	5.63E-07
117-84-0	Di-n-octyl phthalate	3.70E-06
123-91-1	Dioxane, 1,4-	6.40E-07
122-39-4	Diphenylamine	5.48E-07
100-41-4	Ethyl Benzene	1.10E-05
60-29-7	Ethyl Ether	2.50E-06
97-63-2	Ethyl Methacrylate	1.60E-06
611-14-3	Ethyltoluene, 2-	4.50E-07
620-14-4	Ethyltoluene, 3-	4.80E-06
622-96-8	Ethyltoluene, 4-	5.30E-06
206-44-0	Fluoranthene	2.63E-06
86-73-7	Fluorene	6.53E-07
50-00-0	Formaldehyde	4.70E-05
7647-01-0	HCl	1.78E-02
118-74-1	Hexachlorobenzene	4.66E-06
87-68-3	Hexachlorobutadiene	8.11E-07
77-47-4	Hexachlorocyclopentadiene	1.10E-05
67-72-1	Hexachloroethane	5.91E-07
1888-71-7	Hexachloropropene	7.89E-07
110-54-3	Hexane	9.80E-06

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
591-78-6	Hexanone, 2-	2.00E-06
35822-46-9	HpCDD, 1,2,3,4,6,7,8-	2.90E-11
67562-39-4	HpCDF, 1,2,3,4,6,7,8-	7.30E-10
55673-89-7	HpCDF, 1,2,3,4,7,8,9-	1.90E-10
39227-28-6	HxCDD, 1,2,3,4,7,8-	3.50E-12
57653-85-7	HxCDD, 1,2,3,6,7,8-	8.90E-12
19408-74-3	HxCDD, 1,2,3,7,8,9-	6.10E-12
70648-26-9	HxCDF, 1,2,3,4,7,8-	2.60E-10
57117-44-9	HxCDF, 1,2,3,6,7,8-	1.60E-10
72918-21-9	HxCDF, 1,2,3,7,8,9-	1.20E-10
60851-34-5	HxCDF, 2,3,4,6,7,8-	1.90E-10
193-39-5	Indeno(1,2,3-cd)pyrene	3.98E-07
78-59-1	Isophorone	5.48E-07
7439-92-1	Lead	4.10E-05
7439-96-5	Manganese	9.40E-05
7439-97-6	Mercury	7.40E-08
126-98-7	Methacrylonitrile	5.90E-06
80-62-6	Methyl Methacrylate	1.60E-06
1634-04-4	Methyl tert-butyl ether	1.30E-05
108-10-1	Methyl-2-pentanone, 4-	8.20E-07
108-87-2	Methylcyclohexane	1.20E-05
75-09-2	Methylene Chloride	2.40E-04
540-84-1	Methylheptane, 2-	2.40E-05
589-81-1	Methylheptane, 3-	3.50E-06
591-76-4	Methylhexane, 2-	1.70E-05
589-34-4	Methylhexane, 3-	2.20E-05
91-57-6	Methylnaphthalene, 2-	7.47E-06
107-83-5	Methylpentane, 2-	1.10E-05
96-14-0	Methylpentane, 3-	7.10E-06
95-48-7	Methylphenol, 2-	3.29E-06
91-20-3	Naphthalene	9.16E-05
134-32-7	Naphthylamine, 1-	1.10E-05
91-59-8	Naphthylamine, 2-	1.10E-05
7440-02-0	Nickel	5.80E-05
88-74-4	Nitroaniline, 2-	5.48E-07
99-09-2	Nitroaniline, 3-	2.19E-06
100-01-6	Nitroaniline, 4-	2.19E-06

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
98-95-3	Nitrobenzene	6.24E-07
88-75-5	Nitrophenol, 2-	4.71E-06
100-02-7	Nitrophenol, 4-	3.61E-06
55-18-5	N-Nitrosodiethylamine	5.48E-07
62-75-9	N-Nitrosodimethylamine	5.58E-07
924-16-3	N-Nitrosodi-n-butylamine	5.48E-07
621-64-7	N-Nitrosodi-n-propylamine	5.48E-07
86-30-6	N-Nitrosodiphenyl amine	9.75E-08
10595-95-6	N-Nitrosomethylethyl amine	9.09E-07
59-89-2	N-Nitrosomorpholine	5.48E-07
40321-76-4	PeCDD, 1,2,3,7,8-	6.70E-12
57117-41-6	PeCDF, 1,2,3,7,8-	8.00E-11
57117-31-4	PeCDF, 2,3,4,7,8-	1.60E-10
608-93-5	Pentachlorobenzene	5.48E-07
76-01-7	Pentachloroethane	6.98E-07
82-68-8	Pentachloronitrobenzene	5.81E-07
87-86-5	Pentachlorophenol	2.74E-05
14797-73-0	Perchlorate	4.90E-07
85-01-8	Phenanthrene	3.17E-06
108-95-2	Phenol	2.98E-06
7723-14-0	Phosphorus	1.10E-04
123-38-6	Propanal	5.20E-05
103-65-1	Propylbenzene	4.60E-06
115-07-1	Propylene	4.90E-05
129-00-0	Pyrene	2.25E-06
110-86-1	Pyridine	8.11E-07
7782-49-2	Selenium	1.60E-06
7440-22-4	Silver	1.20E-06
100-42-5	Styrene	1.30E-06
1746-01-6	TCDD, 2,3,7,8-	2.30E-12
51207-31-9	TCDF, 2,3,7,8-	4.00E-11
95-94-3	Tetrachlorobenzene, 1,2,4,5-	5.48E-07
79-34-5	Tetrachloroethane, 1,1,2,2-	4.20E-07
127-18-4	Tetrachloroethene	2.50E-06
58-90-2	Tetrachlorophenol, 2,3,4,6-	7.12E-07
109-99-9	Tetrahydrofuran	9.00E-07
529-20-4	Tolualdehyde-o	4.00E-05

TABLE 2-1
EMISSIONS FACTORS USED IN THE PROMONTORY OB/OD HUMAN HEALTH RISK ASSESSMENT

CAS	Name	Emission Factor
108-88-3	Toluene	2.80E-05
95-53-4	Toluidine, o-	7.01E-06
120-82-1	Trichlorobenzene, 1,2,4-	6.46E-07
71-55-6	Trichloroethane, 1,1,1-	2.70E-07
79-00-5	Trichloroethane, 1,1,2-	7.30E-07
79-01-6	Trichloroethene	9.40E-07
95-95-4	Trichlorophenol, 2,4,5-	1.42E-06
88-06-2	Trichlorophenol, 2,4,6-	1.31E-06
526-73-8	Trimethylbenzene, 1,2,3-	4.20E-07
95-63-6	Trimethylbenzene, 1,2,4-	2.50E-05
108-67-8	Trimethylbenzene, 1,3,5-	1.90E-05
565-75-3	Trimethylpentane, 2,3,4	8.20E-06
99-35-4	Trinitrobenzene, 1,3,5-	5.48E-07
1120-21-4	Undecane	1.20E-05
75-01-4	Vinyl Chloride	7.60E-06
95-47-6	Xylene, o-	1.30E-05
7440-66-6	Zinc	5.60E-05
3268-87-9	OCDD	3.70E-11
39001-02-0	OCDF	5.30E-10
108-38-3	Xylene, m-	1.10E-05
106-42-3	Xylene, p-	1.10E-05
108-39-4	Methylphenol, 3-	1.29E-07
106-44-5	Methylphenol, 4-	1.29E-07

TABLE 2-2 TOXICITY EQUIVALENCY FACTORS FOR CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS		
Compound	**Toxicity Equivalent Factor**	**Oral Slope Factor multiplied by Relative Potency**
Benzo(a)pyrene	1.0	7.3
Benz(a)anthracene	0.1	0.73
Benzo(b)fluoranthene	0.1	0.73
Benzo(k)fluoranthene	0.01	0.073
Chrysene	0.001	0.007
Dibenz(a,h)anthracene	1.0	7.3
Indeno(1,2,3-c,d)pyrene	0.1	0.73
Source: EPA, 2014b; EPA, 1993		

TABLE 2-3 POTENTIAL ELEMENTS/COMPOUNDS FOUND IN FLARE WASTES, AND ASSOCIATED DOSE-RESPONSE INFORMATION				
Materials	**Oral Reference Dose (mg/kg-day)**	**Inhalation Reference Conc. ($\mu g/m^3$)**	**Cancer Ingestion Slope Factor**	**Inhalation Unit Risk**
Bismuth	NA	NA	NA	NA
Boron	0.02	2.00E-02	--	--
Cesium	NA	NA	NA	NA
Indium	NA	NA	NA	NA
Iron	0.7		Nutritional Element	
Lead azide and styphnate	NA	NA	NA	NA
Silicon	Essentially Non toxic			
Strontium	0.6			
Tin	0.6	--	--	--
Triacetyl tin	8.0	--	--	--
Zinc Powder	0.3	--	Nutritional Element	
Zirconium	8.00E-05			

TABLE 2-4
TETRACHLORO-DIBENZODIOXIN AND TETRACHLORO-DIBENZODIFURAN—
TOXICITY EQUIVALENCY FACTORS

Compound	Toxicity Equivalent Factor	Oral Slope Factor multiplied by Relative Potency
2,3,7,8-Tetrachlorodibenzo(p)dioxin	1	1.30E+05
1,2,3,7,8-Pentachlorodibenzo(p)dioxin	1	1.30E+05
1,2,3,4,7,8-Hexachlorodibenzo(p)dioxin	0.1	1.30E+04
1,2,3,6,7,8-Hexachlorodibenzo(p)dioxin	0.1	1.30E+04
1,2,3,7,8,9-Hexachlorodibenzo(p)dioxin	0.1	1.30E+04
1,2,3,4,6,7,8-Heptachlorodibenzo(p)dioxin	0.01	1.30E+03
1,2,3,4,6,7,8,9-Octachlorodibenzo(p)dioxin*	0.0003	3.90E+01
2,3,7,8-Tetrachlorodibenzofuran	0.1	1.30E+04
1,2,3,7,8-Pentachlorodibenzofuran*	0.03	3.90E+03
2,3,4,7,8-Pentachlorodibenzofuran*	0.3	3.90E+04
1,2,3,4,7,8-Hexachlorodibenzofuran	0.1	1.30E+04
1,2,3,6,7,8-Hexachlorodibenzofuran	0.1	1.30E+04
1,2,3,7,8,9-Hexachlorodibenzofuran	0.1	1.30E+04
2,3,4,6,7,8-Hexachlorodibenzofuran	0.1	1.30E+04
1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.01	1.30E+03
1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.01	1.30E+03
1,2,3,4,6,7,8,9-Octachlorodibenzofuran*	0.0003	3.90E+01

Source: EPA, 2005a, Incineration Guidance, p. 2-69
World Health Organization, 1998
Van De Berg, et al., 1998
* EPA 2013 Update

TABLE 3-1
M-136 SOURCE PARAMETERS

Parameter	M-136-A1	M-136-A2	M-136-A3	M-136B	M-136-C
Daily Quantity Burned (lb reactive waste)	96,000	10,000	16,000	125,000	1,200
Annual Quantity Burned (lb reactive waste)	6,720,000	840,000	840,000	1,500,000	100,000
Burn Duration	15-45 min	15-45 min	15-45 min	15-45 min	5 sec
Burn Pan Area (m^2)	38.65	10.03	38.65	23.23	--
Burn Pan Equivalent Diameter (m)	7.01	3.57	7.01	5.44	--
Volume Source Diameter (m)	28.06	14.3	28.06	21.75	19.51
Initial Sigma Y (m)	6.53	3.32	6.53	5.06	4.54
Initial Sigma Z (m)	6.53	3.32	6.53	5.06	4.54
Release height (m)	238	219.3	238	297	189.7

Abbreviations:
lb pounds m Meters m^2 Square meters min minutes sec seconds

TABLE 3-2
M-225 SOURCE PARAMETERS

Parameter	M-225-A	M-225-B
Daily Quantity Burned (lb reactive waste)	4,500	600
Annual Quantity Burned (lb reactive waste)	55,000	10,000
Burn Duration	15-45 min	5 sec
Burn Pan Area (m^2)	9.48	--
Burn Pan Equivalent Diameter (m)	3.47	--
Volume Source Diameter (m)	13.89	19.51
Initial Sigma Y (m)	3.23	4.54
Initial Sigma Z (m)	3.23	4.54
Release height (m)	148.8	189.7

Abbreviations:
lb pounds m Meters m^2 Square meters min minutes sec seconds

TABLE 4-1
CRITERIA POLLUTANTS CONSIDERED IN NAAQS COMPLIANCE DEMONSTRATION

Criteria Pollutant	Source	NAAQS averaging time	Design Model Concentration	Method of Determination of Design Value
PM_{10}	(a)	24-Hour	150 µg/m^3	Sixth highest of 5 years of meteorological data
$PM_{2.5}$	(a)	24-Hour	35 µg/m^3	Average of first highest of 5 years of meteorological data
$PM_{2.5}$	(a)	Annual	12 µg/m^3	Average of first highest of 5 years of meteorological data
SO_2	(a)	1-Hour	75 ppb (195 µg/m^3)	Five-year average of the 99th percentile (4th highest) of the annual distribution of daily maximum 1-hour average concentrations
SO_2	(a)	3-Hour	1,300 µg/m^3	Five-year average of 2nd highest (not to be exceeded once per year)
NO_2	(a)	1-Hour	100 ppb (189 µg/m^3)	Five-year average of the 98th percentile (8th highest) of the annual distribution of daily maximum 1-hour average concentrations
NO_2	(a)	Annual	100 µg/m^3	Maximum over 5 years of meteorological data
CO	(a)	1-Hour	40,000 µg/m^3	Average of first highest of 5 years of meteorological data
	(a)	8-Hour	10,000 µg/m^3	Average of first highest of 5 years of meteorological data
Lead	(b)	3-Month Rolling Average	0.15 µg/m^3	100th percentile; maximum over 3 years of meteorological data

Abbreviations:

PM_{10} = Particulate matter (10 micrometers in diameter)

$PM_{2.5}$ = Particulate matter (2.5 micrometers in diameter)

NO_2 = Nitrogen dioxide.

SO_2 = Sulfur dioxide.

CO = Carbon monoxide

ppb = Parts per billion.

$\mu g/m^3$ = Micrograms per cubic meter

(a) From: CB&I, 2014b, Addendum to the Revised Air Dispersion Modeling Report for Open Burning and Open Detonation at ATK Launch Systems in Promontory, Table 9-1

(b) From: TetraTech, 2012b, Revised Air Dispersion Modeling Assessment Report for Open Burn and Open Detonation Treatment Units at ATK Launch Systems, Table 3-69 and Table 3-71.

TABLE 4-2
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225
CRITERIA POLLUTANTS

COPC	Source	Averaging Time	Sub-source	Rank	Design Model Conc. ($\mu g/m^3$)	NAAQS ($\mu g/m^3$)	Percent of NAAQS	Exceedance of NAAQS? (Yes/No)
$PM_{2.5}$	(a)	24-Hour	M-136 A	1st	25.00	35	71%	No
			M-225 A	1st	1.48	35	4.2 %	No
			Total	1st	26.49	35	75.7%	No
		Annual	M-136 A	1st	5.75	12	48%	No
			M-225 A	1st	0.05	12	0.4%	No
			Total	1st	5.81	12	48.4%	No
PM_{10}	(a)	24-Hour	M-136 A	1st	57.14	150	38.1%	No
			M-225 A	1st	3.65	150	2.4%	No
			Total	1st	60.79	150	40.5%	No
NO_2	(a)	1-Hour	M-136 A	1st	64.01	189	33.9%	No
			M-225 A	1st	3.79	189	2.0%	No
			Total	1st	67.80	189	35.9%	No
		Annual	M-136 A	1st	0.70	100	0.7%	No
			M-225 A	1st	0.007	100	0.1%	No
			Total	1st	0.71	100	0.7%	No
SO_2	(a)	1-Hour	M-136 A	1st	5.00	195	2.6%	No
			M-225 A	1st	0.30	195	0.2%	No

TABLE 4-2
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225
CRITERIA POLLUTANTS

COPC	Source	Averaging Time	Sub-source	Rank	Design Model Conc. ($\mu g/m^3$)	NAAQS ($\mu g/m^3$)	Percent of NAAQS	Exceedance of NAAQS? (Yes/No)
			Total	1st	5.30	195	2.7%	No
		3-Hour	M-136 A	1st	1.67	1300	0.1%	No
			M-225 A	1st	0.10	1300	0.1%	No
			Total	1st	1.77	1300	0.1%	No
CO	(a)	1-Hour	M-136 A	1st	64.01	40,000	0.2%	No
			M-225 A	1st	3.79	40,000	0.1%	No
			Total	1st	67.80	40,000	0.8%	No
		8-Hour	M-136 A	1st	8.00	10,000	0.1%	No
			M-225 A	1st	0.47	10,000	0.1%	No
			Total	1st	8.48	10,000	0.1%	No
Lead	(b)	3-Month	M-136, Sources 1-3 (comparable to M-136 A)	1st	0.03	0.15	20%	No
			M-136, Source 4 (comparable to M-136 C)	1st	0.05	0.15	33.3%	No
			M-225, Source 1 (comparable	1st	0.01	0.15	6.7%	No

TABLE 4-2
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225
CRITERIA POLLUTANTS

COPC	Source	Averaging Time	Sub-source	Rank	Design Model Conc. ($\mu g/m^3$)	NAAQS ($\mu g/m^3$)	Percent of NAAQS	Exceedance of NAAQS? (Yes/No)
			to M-225 A)					

Abbreviations:

1-Hr = 1-Hour
24-Hr = 24-Hour
8-Hr = 8-Hour
1st = First
Conc. = Concentration
COPC = Chemical of Potential Concern
PM_{10} = Particulate matter (10 micrometers)
$PM_{2.5}$ = Particulate matter (2.5 micrometers)
NO_2 = Nitrogen dioxide.
SO_2 = Sulfur dioxide.
ppb = Parts per billion.
$\mu g/m^3$ = Micrograms per cubic meter
% = Percent
NAAQS = National Ambient Air Quality Standard

Notes:

(a) From: CB&I, 2014b , Addendum to the Revised Air Dispersion Modeling Report for Open Burning and Open Detonation at ATK Launch Systems in Promontory, Table 9-1
(b) From: From: TetraTech, 2012b, Revised Air Dispersion Modeling Assessment Report for Open Burn and Open Detonation Treatment Units at ATK Launch Systems.

TABLE 4-3
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225 – ACUTE ONE-HOUR AIR TOXICS[1]

Chemical of Potential Concern	Acute 1-Hr TSL Value	Scenario M-136 A			Scenario M-225 A			Total (M-136 A and M-225 A)		
		1-Hr Conc.	% of TSL	Exceed TSL? (Y/N)	1-Hr Conc.	% of TSL	Exceed TSL? (Y/N)	1-Hr Conc.	% of TSL	Exceed TSL? (Y/N)
	$\mu g/m^3$	$\mu g/m^3$			$\mu g/m^3$			$\mu g/m^3$		
Isophorone	2,826	0.006	0.0002%	No	0.0003	0.00001%	No	0.006	0.0002%	No
Formaldehyde	37	0.470	1.27%	No	0.028	0.08%	No	0.498	1.35%	No
Hydrogen Chloride	298	180.034	60.41%	No	10.662	3.58%	No	190.696	63.99%	No
Hydrogen Cyanide	520	0.220	0.04%	No	0.013	0.003%	No	0.233	0.04%	No
1,2,4,-Trichlororbenzene	3,711	0.013	0.0004%	No	0.001	0.00002%	No	0.014	0.0004%	No

Notes:
1. This table is taken from CB&I's, 2014, Air Quality Modeling Report

Abbreviations:
NAAQS = National Ambient Air Quality Standard
1-Hr = 1-Hour
1st = First
% = Percent
Conc. = Concentration
ppb = Parts per billion

PM_{10} = Particulate matter (10 micrometers)
$PM_{2.5}$ = Particulate matter (2.5 micrometers)
NO_2 = Nitrogen dioxide.
SO_2 = Sulfur dioxide
TSL = Toxic Screening Level
µg/m3 = Micrograms per cubic meter

TABLE 4-4
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225 – CHRONIC 24-HOUR AIR TOXICS[1]

Chemical of Potential Concern	Chronic 24-Hr TSL Value	Scenario M-136 A			Scenario M 225 A			Total (M-136 A and M-225 A)		
		24-Hr Conc.	% of TSL	Exceed TSL? (Yes/No)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)
	µg/m^3	µg/m^3			µg/m^3			µg/m^3		
1,4-Dichlorobenzene	2004	3.04E-04	0.00%	No	1.80E-05	0.00%	No	3.22E-04	0.00%	No
2,4-Dinitrotoluene	7	2.29E-04	0.00%	No	1.36E-05	0.00%	No	2.43E-04	0.00%	No
o-Toluidine	292	2.92E-03	0.00%	No	1.73E-04	0.00%	No	3.09E-03	0.00%	No
Phenol	642	1.00E-03	0.00%	No	5.92E-05	0.00%	No	1.06E-03	0.00%	No
Cl2	48	5.00E+00	10.42%	No	2.96E-01	0.62%	No	5.30E+00	11.04%	No
1,1,2-Trichloroethane	1819	3.04E-04	0.00%	No	1.80E-05	0.00%	No	3.22E-04	0.00%	No
1,3-Butadiene	49	1.00E-02	0.02%	No	5.92E-04	0.00%	No	1.06E-02	0.02%	No
1,4-Dioxane	2402	2.67E-04	0.00%	No	1.58E-05	0.00%	No	2.83E-04	0.00%	No
Acrylonitrile	48	6.67E-03	0.01%	No	3.95E-04	0.00%	No	7.06E-03	0.01%	No
Benzene	3	1.96E-02	0.04%	No	1.16E-03	0.00%	No	2.07E-02	0.04%	No
Bromoform	172	5.42E-04	0.00%	No	3.21E-05	0.00%	No	5.74E-04	0.00%	No
Carbon Tetrachloride	350	6.25E-03	0.00%	No	3.70E-04	0.00%	No	6.62E-03	0.00%	No
Chlorobenzene	1535	1.04E-03	0.00%	No	6.17E-05	0.00%	No	1.10E-03	0.00%	No
Chloroform	1628	2.54E-03	0.00%	No	1.51E-04	0.00%	No	2.69E-03	0.00%	No
cis-1,3-Dichloropropene	151	5.42E-04	0.00%	No	3.21E-05	0.00%	No	5.74E-04	0.00%	No

TABLE 4-4
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225 – CHRONIC 24-HOUR AIR TOXICS[1]

Chemical of Potential Concern	Chronic 24-Hr TSL Value	Scenario M-136 A			Scenario M 225 A			Total (M-136 A and M-225 A)		
		24-Hr Conc.	% of TSL	Exceed TSL? (Yes/No)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)
	µg/m^3	µg/m^3			µg/m^3			µg/m^3		
Cumene	8193	1.75E-04	0.00%	No	1.04E-05	0.00%	No	1.85E-04	0.00%	No
Styrene	2840	4.13E-04	0.00%	No	2.44E-05	0.00%	No	4.37E-04	0.00%	No
Toluene	2512	7.92E-03	0.00%	No	4.69E-04	0.00%	No	8.39E-03	0.00%	No
Vinyl Chloride	28	3.17E-03	0.01%	No	1.88E-04	0.00%	No	3.35E-03	0.01%	No
Antimony	17	1.21E-02	0.07%	No	7.16E-04	0.00%	No	1.28E-02	0.08%	No
Arsenic	0.33	2.29E-04	0.07%	No	1.36E-05	0.00%	No	2.43E-04	0.07%	No
Cadmium	0.02	2.54E-04	1.27%	No	1.51E-05	0.08%	No	2.69E-04	1.35%	No
Chromium	0.11	8.33E-03	7.58%	No	4.94E-04	0.45%	No	8.83E-03	8.03%	No
Cobalt	0.77	2.54E-04	0.03%	No	1.51E-05	0.00%	No	2.69E-04	0.03%	No
Manganese	6.7	3.92E-02	0.58%	No	2.32E-03	0.03%	No	4.15E-02	0.62%	No
Mercury	0.33	3.08E-05	0.01%	No	1.83E-06	0.00%	No	3.27E-05	0.01%	No
Nickel	1.11	2.42E-02	2.18%	No	1.43E-03	0.13%	No	2.56E-02	2.31%	No
Phosphorus	3.3	4.58E-02	1.39%	No	2.71E-03	0.08%	No	4.86E-02	1.47%	No
Selenium	6.7	6.67E-04	0.01%	No	3.95E-05	0.00%	No	7.06E-04	0.01%	No

Notes and Abbreviations:
1. This table is taken from CB&I's, 2014, Air Quality Modeling Report
NAAQS = National Ambient Air Quality Standard

PM_{10} = Particulate matter (10 micrometers)

TABLE 4-4
RESULTS OF CUMULATIVE IMPACT FOR M-136 AND M-225 – CHRONIC 24-HOUR AIR TOXICS[1]

Chemical of Potential Concern	Chronic 24-Hr TSL Value	Scenario M-136 A			Scenario M 225 A			Total (M-136 A and M-225 A)		
		24-Hr Conc.	% of TSL	Exceed TSL? (Yes/No)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)	24-Hr Conc.	% of TSL	Exceed TSL? (Y/N)
	µg/m^3	µg/m^3			µg/m^3			µg/m^3		

24-Hr = 24-Hour
1st = First
% = Percent
Conc. = Concentration
ppb = Parts per billion
$PM_{2.5}$ = Particulate matter (2.5 micrometers)
NO_2 = Nitrogen dioxide.
SO_2 = Sulfur dioxide
TSL = Toxic Screening Level
µg/m3 = Micrograms per cubic meter

TABLE 5-1
SUMMARY OF DISCRETE RECEPTORS CONSIDERED IN THE PROMONTORY OB/OD HHRA

Location	Coordinates	Reason Selected
Off-site Boundary Locations (Without Actual Receptors)		
Offsite Point of Maximum Deposition	UTM E = 379600 UTM N = 4616500	An offsite location west of the facility near the South Plant, west of Blue Creek on the side of a hill north of Adams Ranch. The location is not easily accessed, and cannot be reached by automobile because of the steep terrain and there is no road
Blue Creek	UTM E = 379909 UTM N = 4615872	A perennial stream which runs along the western property boundary and is 0.53 miles west of M-136
Boundary 1	UTM E = 382606 UTM N = 4616686	Model Defined- selected based on the annual prevailing wind direction measured over a five-year period (1997 through 2001) at the M-245 meteorological monitoring station. Boundary 1 is located 1.27 miles northeast of M-136
Boundary 2	UTM E = 379527 UTM N = 4612623	Model Defined- selected based on the annual prevailing wind direction measured over a five-year period (1997 through 2001) at the M-245 meteorological monitoring station. Boundary 2 is located 2.13 miles south-southwest of M-136
Boundary 3	UTM E = 387811 UTM N = 4610223	Model Defined- selected based on the annual prevailing wind direction measured over a five-year period (1997 through 2001) at the M-245 meteorological monitoring station. Boundary 3 is located 1.38 miles east-northeast of M-225
Boundary 4	UTM E = 386804 UTM N = 4608540	Model Defined- selected based on the annual prevailing wind direction measured over a five-year period (1997 through 2001) at the M-245 meteorological monitoring station. Boundary 4 is located 1.19 miles south-southeast of M-225
On-site Receptors		
Onsite Point of Maximum Deposition	UTM E = 380450.07 UTM N = 4616958.63	An onsite location directly north of source M-136. This location has no structure nor workers.
Autoliv Facility	UTM E = 381612 UTM N = 4614016	Off-Site commercial business located 1.25 miles southeast of M-136
North Plant Main Administration Building	UTM E = 381071 UTM N = 4619888	Actual Worker Scenario-Facility located 2.53 miles north-northeast of M-136
South Plant Main Administration Building	UTM E = 380425 UTM N = 4613562	Actual Worker Scenario-Facility located 1.42 miles south-southwest of M-136
Off-site Receptors		
Adams Ranch	UTM E = 378193 UTM N = 4613689	Closest domestic dwelling to M-136, and 2.07 miles southwest of M-136

TABLE 5-1
SUMMARY OF DISCRETE RECEPTORS CONSIDERED IN THE PROMONTORY OB/OD HHRA

Location	Coordinates	Reason Selected
Holmgren Ranch	UTM E = 388309 UTM N = 4608872	Closest domestic dwelling to the M-225 and located 1.83 miles southeast of M-225
Howell Dairy Farm	UTM E = 380246 UTM N= 4627858	Immediately north of the ATK northern property boundary, and 7.49 miles north-northwest of M-136
Christensen Residence	UTM E = 381309 UTM N = 4622372	Residential dwelling located due north of ATK and 4.08 miles north-northeast of M-136
Town of Thatcher	UTM E =392258 UTM N = 4616803	Located approximately 5.91 miles northeast of M-225
Town of Penrose	UTM E = 391564 UTM N = 4612029	Located approximately 3.91 miles east northeast of M-225
ATK Ranch Pond	UTM E = 376606 UTM N = 4599685	Non-resident location 8.51 miles southwest of M-225
Salt Creek Waterfowl Area	Qualitative	Hunting/Recreational area located 6 miles east-northeast of M-225
Bear River Bird Refuge	Qualitative	Hunting/Recreational area located about 6.69 miles south-southwest of M-225

TABLE 5-2
SUMMARY OF COMPLETE EXPOSURE PATHWAYS FOR ON-SITE RECEPTORS

Receptor	Worker Exposure Pathway
Autoliv Facility	Acute and Chronic Vapors and Particles Inhalation
North Plant Main Administration Building	Acute and Chronic Vapors and Particles Inhalation
South Plant Main Administration Building	Acute and Chronic Vapors and Particles Inhalation
On-site Maximum for Hypothetical Worker	Acute and Chronic Vapors and Particles Inhalation

TABLE 5-3
SUMMARY OF COMPLETE EXPOSURE PATHWAYS FOR ACTUAL OFF-SITE RECEPTORS AND HYPOTHETICAL MAXIMUM OFF-SITE RECEPTOR

Receptor	Direct Exposure Pathways	Direct Exposure Pathways
Subsistence Farming Family (Adult)	Acute and Chronic Vapor and Particulates Inhalation Soil Ingestion	Produce Ingestion Beef Ingestion Milk Ingestion Chicken Ingestion Egg Ingestion Pork Ingestion

TABLE 5-3
SUMMARY OF COMPLETE EXPOSURE PATHWAYS FOR ACTUAL OFF-SITE RECEPTORS AND HYPOTHETICAL MAXIMUM OFF-SITE RECEPTOR

Receptor	Direct Exposure Pathways	Direct Exposure Pathways
Subsistence Farming Family (Child)	Acute and Chronic Vapor and Particulates Inhalation Soil Ingestion	Produce Ingestion Beef Ingestion Milk Ingestion Chicken Ingestion Egg Ingestion Pork Ingestion Breast Milk Ingestion
Resident Family (Adult)	Acute and Chronic Vapor and Particulates Inhalation Soil Ingestion	Produce Ingestion
Resident Family (Child)	Acute and Chronic Vapor and Particulates Inhalation Soil Ingestion	Produce Ingestion

TABLE 7-1
SUMMARY OF EXPOSURE ASSUMPTIONS

RECEPTOR	Resident Adult[(1)]	Resident Child[(1)]	Farmer Adult[(1)]	Farmer Child[(1)]	Industrial Worker[(2)]	Units
All Exposures						
Averaging time for carcinogens	70	70	70	70	70	yr
Averaging time for noncarcinogens	26	6	40	6	25	yr
Exposure duration	26	6	40	6	25	yr
Exposure frequency	350	350	350	350	250	day/yr
Body weight	80	15	80	15	80	kg
Time period at the beginning of combustion	0	0	0	0	0	yr
Length of exposure duration	26	6	40	6	25	yr
Inhalation						
Inhalation exposure duration	26	6	40	6	25	yr
Inhalation exposure frequency	350	350	350	350	250	day/yr
Inhalation exposure time	24	24	24	24	8	hr/day
Drinking Water						
Fraction of contaminated drinking water	NA	NA	NA	NA	NA	--
Consumption rate of drinking water	NA	NA	NA	NA	NA	L/day
Incidental Ingestion of Soil						
Fraction of contaminated soil	1	1	1	1	0	--
Consumption rate of soil	0.0001	0.0002	0.0001	0.0002	0 (3)	kg/d
Ingestion of Poultry						
Fraction of contaminated poultry	1	1	1	1	0	--
Consumption rate of poultry	0	0	0.00066	0.00045	0	kg/kg-day FW

TABLE 7-1
SUMMARY OF EXPOSURE ASSUMPTIONS

RECEPTOR	Resident Adult[(1)]	Resident Child[(1)]	Farmer Adult[(1)]	Farmer Child[(1)]	Industrial Worker[(2)]	Units
Ingestion of Produce						
Fraction of contaminated produce	1	1	1	1	0	--
Consumption rate of aboveground produce	0.00032	0.00077	0.00047	0.00113	0	kg/kg-day DW
Consumption rate of protected aboveground produce	0.00061	0.0015	0.00064	0.00157	0	kg/kg-day DW
Consumption rate of belowground produce	0.00014	0.00023	0.00017	0.00028	0	kg/kg-day DW
Ingestion of Beef						
Fraction of contaminated beef	1	1	1	1	0	--
Consumption rate of beef	0	0	0.00122	0.00075	0	kg/kg-day FW
Ingestion of Eggs						
Fraction of contaminated eggs	1	1	1	1	0	--
Consumption rate of eggs	0	0	0.00075	0.00054	0	kg/kg-day FW
Ingestion of Milk						
Fraction of contaminated milk	1	1	1	1	0	--
Consumption rate of milk	0	0	0.01367	0.02268	0	kg/kg-day FW
Ingestion of Pork						
Fraction of contaminated pork	1	1	1	1	0	--
Consumption rate of pork	0	0	0.00055	0.00042	0	kg/kg-day FW
Ingestion of Breast Milk						

TABLE 7-1
SUMMARY OF EXPOSURE ASSUMPTIONS

RECEPTOR	Resident Adult(1)	Resident Child(1)	Farmer Adult(1)	Farmer Child(1)	Industrial Worker(2)	Units
Body weight - infant		9.4				kg
Exposure duration - infant		1				year
Proportion of ingested dioxin that is stored in fat		0.9				--
Proportion of mother's weight that is fat		0.3				--
Fraction of fat in breast milk		0.04				--
Fraction of ingested contaminant that is absorbed		0.9				--
Half-life of dioxin in adults		2555				days
Ingestion rate of breast milk		0.688				kg/day
DW - Dry weight of soil or plant/animal tissue.						
FW - Fresh weight (or whole/wet weight) of plant or animal tissue.						
NA - Not applicable						
1 - Values from EPA's Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, September 2005.						
2 - Values are from EPA's Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, December 2002.						
3 - It is assumed an industrial worker is inside a building						

TABLE 8-1
SURROGATES FOR CHEMICALS WITHOUT CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) OR DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS

Chemical Without PAC-1 Value	CAS Number	Surrogate Chemical Name	PAC-1 Value ($\mu g/m^3$)
1,4-Diethylbenzene	105-05-5	4-Ethyltoluene	12,000
2,3,4-Trimethylpentane	565-75-3	2,2,4-Trimethylpentane	14,000,000
2,3-Dimethylbutane	79-29-8	3-Methylpentane	1,800,000
2,3-Dimethylpentane	565-59-3	3-Methylpentane	1,800,000
2,4-Dimethylpentane	108-08-7	3-Methylhexane	1,800,000
2,5-Dimethylbenzaldehyde	5779-94-2	Benzaldehyde	17,000
3-Ethyltoluene	620-14-4	4-Ethyltoluene	12,000
3-Methylheptane	589-81-1	2-Methylheptane	1,400,000
2-Methylhexane	591-76-4	3-Methylpentane	1,800,000
3-Methylhexane	589-34-4	3-Methylpentane	1,800,000
Chromium (VI)	18540-29-9	Chromium (VI) hydroxide (CAS # 12626-43-6)	89
n-Nitrosodibutylamine	924-16-3	n-Nitrosodipropylamine	2,000
n-Nitrosodiethylamine	55-18-5	n-Nitrosodimethylamine	1,700
n-Nitrosomethylethylamine	10595-95-6	n-Nitrosodimethylamine	1,700
o-Tolualdehyde	534-52-1	Benzaldehyde	17,000
Perchlorate	14797-73-0	Potassium Perchlorate (7778-74-7)	23
Perchlorate	14797-73-0	Ammonium Perchlorate (7790-98-9)	1,700
Tetrachlorophenol 2,3,4,6	58-90-2	2,4-Dichlorophenol	1,300

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
1,2,3-Trimethylbenzene	526-73-8	690,000	
1,2,4-Trimethylbenzene	95-63-6	690,000	
1,4-Diethylbenzene	105-05-5	12,000	
1-Butene	106-98-9	1,200,000	
1-Chloronapthalene	90-13-1	4,600	
1-Naphthylamine	134-32-7	210	
2,2,4-Trimethylpentane	540-84-1	14,000,000	
2,2-Dimethylbutane	75-83-2	1,800,000	
2,3,4-Trimethylpentane	565-75-3	14,000,000	
2,3-Dimethylbutane	79-29-8	1,800,000	
2,3-Dimethylpentane	565-59-3	1,800,000	
2,4-Dimethylpentane	108-08-7	180,000	
2,5-Dimethylbenzaldehyde	5779-94-2	17,000	
2,6-Dichlorophenol	87-65-0	8,800	
2-Ethyltoluene	611-14-3	12,000	
2-Hexanone	591-78-6	41,000	
2-Methylhexane	591-76-4	1,800,000	
2-Methylnaphthalene	91-57-6	3,000	
2-Methylpentane	107-83-5	1,800,000	
2-Naphthylamine	91-59-8	320	
3-Ethyltoluene	620-14-4	12,000	
3-Methylheptane	589-81-1	1,400,000	
3-Methylhexane	589-34-4	1,800,000	
3-Methylpentane	96-14-0	1,800,000	
4,6-Dinitro-2-methylphenol	534-52-1	200	
4-Aminobyphenyl	92-67-1	490	
4-Ethyltoluene	622-96-8	12,000	
Acenaphthene	83-32-9	3,600	
Acenaphthylene	208-96-8	10,000	
Acetaldehyde	75-07-0	81,000	470
Acetone	67-64-1	470,000	
Acetonitrile	75-05-8	22,000	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
Acetophenone	98-86-2	10,000	
Acrylonitrile	107-13-1	10,000	
Aluminum	7429-90-5	3,000	
Aniline	62-53-3	30,000	
Anthracene	120-12-7	270	
Antimony	7440-36-0	500	
Arsenic	7440-38-2	30	0.2
Barium	7440-39-3	1,500	
Benzaldehyde	100-52-7	17,000	
Benzene	71-43-2	170,000	27
Benzo(a)anthracene	56-55-3	1,200	
Benzo(a)pyrene	50-32-8	600	
Benzo(b)fluoranthene	205-99-2	31	
Benzo(g,h,i)perylene	191-24-2	30,000	
Benzo(k)fluoranthene	207-08-9	19	
Benzoic acid	65-85-0	2,800	
Benzyl alcohol	100-51-6	130,000	
Benzyl chloride	100-44-7	5,200	240
Bis(2-chlorethyl)ether	111-44-4	58,000	
Bis(2-chloroethoxy)methane	111-91-1	920	
Bromodichloromethane	75-27-4	260	
Bromoform	75-25-2	15,000	
Bromophenyl-phenylether, 4-	101-55-3	290	
Butylbenzylphthalate	85-68-7	15,000	
Cadmium	7440-43-9	100	
Carbazole	86-74-8	660	
Carbon disulfide	75-15-0	40,000	6,200
Carbon tetrachloride	56-23-5	280,000	1,900
Chlorine	7782-50-5	1,400	210
Chloro-3-methylphenol, 4-	59-50-7	5,500	
Chloroacetonitrile	107-14-2	9,000	
Chloroaniline, p-	106-47-8	2,200	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
Chlorobenzene	108-90-7	46,000	
Chloroethane	75-00-3	260,000	
Chloroform (Trichloromethane)	67-66-3	9,800	150
Chloronaphthalene, 2-	91-58-7	600	
Chlorophenol, 2-	95-57-8	1,300	
Chromium, hexavalent	18540-29-9	89	
Chromium, trivalent	7440-47-3	1,500	
Chrysene	218-01-9	600	
Cis-1,3-dichloropropene	10062-01-5	600	
cis-2-Butene	590-18-1	150,000,000	
Cobalt	7440-48-4	180	
Copper	7440-50-8	1,000	100
Cresol, m-	108-39-4	20,000	
Cresol, o-	95-48-7	20,000	
Cresol, p-	106-44-5	20,000	
Crotonaldehyde	4170-30-3	540	
Cumene	98-82-8	250,000	
Cyclohexane	110-82-7	340,000	
Dibenz(a,h)anthracene	53-70-3	34	
Dibenzofuran	132-64-9	30,000	
Dibromochloromethane	124-48-1	7,400	
Dichlorobenzene, 1,2-	95-50-1	300,000	
Dichlorobenzene, 1,3-	541-73-1	16,000	
Dichlorobenzene, 1,4-	106-46-7	60,000	
Dichloroethane 1,1-	75-34-3	650,000	
Dichloroethane, 1,2	107-06-2	200,000	
Dichloroethylene 1,1-	75-35-4	180,000	
Dichloroethylene, cis-1,2-	156-59-2	560,000	
Dichloroethylene-1, 2 (trans)	156-60-5	1,100,000	
Dichlorophenol, 2,4-	120-83-2	1,300	
Dichloropropane, 1,2-	78-87-5	140,000	
Diethyl phthalate	84-66-2	5,000	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
Dimethyl phthalate	131-11-3	15,000	
Dimethylphenol, 2,4-	105-67-9	4,500	
Di-n-butyl phthalate	84-74-2	15,000	
Dinitrobenzene, 1,3-	99-65-0	1,000	
Dinitrophenol, 2,4-	51-28-5	220	
Dinitrotoluene, 2,4-	121-14-2	600	
Dinitrotoluene, 2,6-	606-20-2	600	
Di-n-octylphthalate	117-84-0	6,800	
Dioxane, 1,4-	123-91-1	61,000	3,000
Diphenylamine	122-39-4	30,000	
Ethyl methacrylate	97-63-2	13,000	
Ethylbenzene	100-41-4	140,000	
Ethylene Dibromide	106-93-4	130,000	
Ethylether	60-29-7	1,500,000	
Ethylhexyl phthalate, bis-2-	117-81-7	10,000	
Fluoranthene	206-44-0	1,500	
Fluorene	86-73-7	6,600	
Formaldehyde	50-00-0	1,100	55
HeptaCDD, 1,2,3,4,6,7,8-	35822-46-9	3	
HeptaCDF, 1,2,3,4,6,7,8-	67562-39-4	45	
HeptaCDF, 1,2,3,4,7,8,9-	55673-89-7	60	
HexaCDD, 1,2,3,4,7,8-	39227-28-6	0.07	
HexaCDD, 1,2,3,6,7,8-	57653-85-7	0.75	
HexaCDD, 1,2,3,7,8,9-	19408-74-3	4	
HexaCDF, 1,2,3,4,7,8-	70648-26-9	0.07	
HexaCDF, 1,2,3,6,7,8-	57117-44-9	0.04	
HexaCDF, 1,2,3,7,8,9-	72918-21-9	30	
HexaCDF, 2,3,4,6,7,8-	60851-34-5	0.49	
Hexachloro-1,3-butadiene	87-68-3	11,000	
Hexachlorobenzene	118-74-1	6	
Hexachlorocyclopentadiene	77-47-4	110	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
Hexachloroethane (Perchloroethane)	67-72-1	29,000	
Hexachloropropene	1888-71-7	905	
Hexane	110-54-3	1,100,000	
Hydrogen chloride	7647-01-0	2,700	2,100
Indeno(1,2,3-cd) pyrene	193-39-5	15	
Isophorone	78-59-1	23,000	
Lead	7439-92-1	150	
Manganese	7439-96-5	3,000	
Mercuric chloride	7487-94-7	2,700	
Methacrylonitrile	126-98-7	2,700	
Methyl bromide	74-83-9	74,000	
Methyl chloride	74-87-3	210,000	
Methyl ethyl ketone	78-93-3	590,000	13,000
Methyl isobutyl ketone	108-10-1	310,000	
Methyl tert-butyl ether	1634-04-4	180,000	
Methylcyclohexane	108-87-2	1,600,000	
Methylene chloride	75-09-2	690,000	14,000
Methylmethacrylate	80-62-6	70,000	
Naphthalene	91-20-3	79,000	
Nickel	7440-02-0	4,500	0.20
Nitroaniline, 2-	88-74-4	4,800	
Nitroaniline, 3-	99-09-2	110	
Nitroaniline, 4-	100-01-6	9,000	
Nitrobenzene	98-95-3	9,100	
Nitrophenol, 2-	88-75-5	1,000	
Nitrophenol, 4-	100-02-7	1,200	
Nitroso-di-n-butylamine, n-	924-16-3	2,000	
Nitrosodiphenylamine, N-	86-30-6	1,400	
Nitrosodipropylamine, n-	621-64-7	2,000	
n-Nitrosodiethylamine	55-18-5	1,700	
n-Nitrosodimethylamine	62-75-9	1,700	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
n-Nitrosomethylethylamine	10595-95-6	1,700	
n-Nitrosomorpholine	59-89-2	74	
OctaCDD, 1,2,3,4,6,7,8,9-	3268-87-9	3	
OctaCDF, 1,2,3,4,6,7,8,9-	39001-02-0	75	
o-Tolualdehyde	529-20-4	17,000	
p-Dimethylaminoazobenzene	60-11-7	670	
PentaCDD, 1,2,3,7,8-	40321-76-4	0.10	
PentaCDF, 1,2,3,7,8-	57117-41-6	0.24	
PentaCDF, 2,3,4,7,8-	57117-31-4	0.07	
Pentachlorobenzene	608-93-5	1,600	
Pentachloroethane	76-01-7	320,000	
Pentachloronitrobenzene	82-68-8	1,500	
Pentachlorophenol	87-86-5	1,400	
Perchlorate	14797-73-0	23	
Phenanthrene	85-01-8	760	
Phenol	108-95-2	58,000	5,800
Phosphorous	7723-14-0	270	
Propanal	123-38-6	110,000	
Propylbenzene	103-65-1	5,400	
Propylene	115-07-1	860,000	
Pyrene	129-00-0	150	
Pyridine	110-86-1	9,700	
Selenium	7782-49-2	200	
Silver	7440-22-4	100	
Styrene	100-42-5	85,000	21,000
TetraCDD, 2,3,7,8-	1746-01-6	0.00003	
TetraCDF, 2,3,7,8-	51207-31-9	0.07	
Tetrachlorobenzene, 1,2,4,5-	95-94-3	240	
Tetrachloroethane, 1,1,2,2-	79-34-5	7,000	
Tetrachloroethylene	127-18-4	240,000	20,000
Tetrachlorophenol, 2,3,4,6-	58-90-2	1,300	
Tetrahydrofuran	109-99-9	290,000	

TABLE 8-2
CALIFORNIA SHORT-TERM AIR REFERENCE EXPOSURE LEVELS (RELS) AND DEPARTMENT OF ENERGY 1-HOUR PROTECTIVE ACTION CRITERIA (PAC-1) CONCENTRATIONS FOR SCREENING

Chemical of Potential Concern	CAS Number	PAC-1 ($\mu g/m^3$)	Cal EPA RELs
Toluene	108-88-3	750,000	37,000
Toluidine, o-	95-53-4	8,800	
Trans-1,3-dichloropropene	10061-02-6	75,000	
trans-2-Butene	624-64-6	1,000,000	
Trichlorobenzene, 1,2,4-	120-82-1	3,400	
Trichloroethane, 1,1,1-	71-55-6	1,300,000	
Trichloroethane, 1,1,2-	79-00-5	81,000	
Trichloroethylene	79-01-6	700,000	
Trichlorophenol, 2,4,5-	95-95-4	7,400	
Trichlorophenol, 2,4,6-	88-06-2	1,800	
Trimethylbenzene, 1,3,5-	108-67-8	690,000	
Trinitrobenzene, 1,3,5	99-35-4	530	
Undecane	1120-21-4	1,500	
Vinyl Chloride	75-01-4	640,000	180,000
Xylene, m-	108-38-3	650,000	22,000
Xylene, o-	95-47-6	650,000	22,000
Xylene, p-	106-42-3	650,000	22,000
Zinc	7440-66-6	1,900	

TABLE 8-3
CHEMICALS WITH UPDATED INHALATION REFERENCE CONCENTRATIONS (RFC) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Inhalation RfC (mg/m^3)	Reference
Acetone	67-64-1	3.1E+01	A
Ammonia	7664-41-7	1.0E-01	I
Arsenic, Inorganic	7440-38-2	1.5E-05	C
Benzyl Chloride	100-44-7	1.0E-03	P
Cadmium (Diet)	7440-43-9	1.0E-05	A
Cadmium (Water)	7440-43-9	1.0E-05	A
Carbon Tetrachloride	56-23-5	1.0E-01	I
Chlorine	7782-50-5	1.5E-04	A
Chlorobenzene	108-90-7	5.0E-02	P
Chloroform	67-66-3	9.8E-02	A
Chromium (VI)	18540-29-9	1.0E-04	I
Cresol, o-	95-48-7	6.0E-01	C
Dichloroethane, 1,2-	107-06-2	7.0E-03	P
Dichloroethylene, 1,2-trans-	156-60-5	6.0E-02	P
Dioxane, 1,4-	123-91-1	3.0E-02	I
Ethyl Methacrylate	97-63-2	3.0E-01	P
Hexachloroethane	67-72-1	3.0E-02	I
Isopropanol	67-63-0	7.0E+00	C
Methacrylonitrile	126-98-7	3.0E-02	P
Methylene Chloride	75-09-2	6.0E-01	I
Nickel Soluble Salts	7440-02-0	9.0E-05	A
Nitroaniline, 2-	88-74-4	5.0E-05	X
Nitroaniline, 4-	100-01-6	6.0E-03	P
Nitrobenzene	98-95-3	9.0E-03	I
TCDD, 2,3,7,8-	1746-01-6	4.0E-08	C
Tetrachloroethylene	127-18-4	4.0E-02	I
Tetrahydrofuran	109-99-9	2.0E+00	I
Toluene	108-88-3	5.0E+00	I
Trichlorobenzene, 1,2,4-	120-82-1	2.0E-03	P

TABLE 8-3
CHEMICALS WITH UPDATED INHALATION REFERENCE CONCENTRATIONS (RFC) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Inhalation RfC (mg/m^3)	Reference
Trichloroethane, 1,1,1-	71-55-6	5.0E+00	I
Trichloroethane, 1,1,2-	79-00-5	2.0E-04	X
Trichloroethylene	79-01-6	2.0E-03	I

Abbreviations:
A ATSDR
C Cal EPA
P PPRTV
RfC Reference Concentration
I IRIS
X PPRTV Appendix – screening value

TABLE 8-4
CHEMICALS WITH UPDATED ORAL REFERENCE DOSES (RFD) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Oral RfD (mg/kg-day)	Reference
Acrylonitrile	107-13-1	4.0E-02	A
Aluminum	7429-90-5	1.0E+00	P
Aniline	62-53-3	7.0E-03	P
Barium	7440-39-3	2.0E-01	I
Benzyl Alcohol	100-51-6	1.0E-01	P
Benzyl Chloride	100-44-7	2.0E-03	P
Cadmium (Water)	7440-43-9	5.0E-04	I
Carbon Tetrachloride	56-23-5	4.0E-03	I
Dichlorobenzene, 1,4-	106-46-7	7.0E-02	A
Dichloroethane, 1,1-	75-34-3	2.0E-01	P
Dichloroethane, 1,2-	107-06-2	6.0E-03	X
Dichloroethylene, 1,2-cis-	156-59-2	2.0E-03	I
Dichloropropane, 1,2-	78-87-5	9.0E-02	A
Dinitrotoluene, 2,6-	606-20-2	3.0E-04	X
Dioxane, 1,4-	123-91-1	3.0E-02	I
Dioxins (TCDD-2,3,7,8)-	1746-01-6	7.0E-10	I
Hexachlorobutadiene	87-68-3	1.0E-03	P
Hexachloroethane	67-72-1	7.0E-04	I
Methyl Acrylate	96-33-3	3.0E-02	H
Methylene Chloride	75-09-2	6.0E-03	I
Nitroaniline, 2-	88-74-4	1.0E-02	X
Nitroaniline, 4-	100-01-6	4.0E-03	P
Nitrobenzene	98-95-3	2.0E-03	I
Octyl Phthalate, di-N-	117-84-0	1.0E-02	P
Pentachlorophenol	87-86-5	5.0E-03	I
Tetrachloroethane, 1,1,2,2-	79-34-5	2.0E-02	I
Tetrachloroethylene	127-18-4	6.0E-03	I
Tetrahydrofuran	109-99-9	9.0E-01	I

TABLE 8-4
CHEMICALS WITH UPDATED ORAL REFERENCE DOSES (RFD) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Oral RfD (mg/kg-day)	Reference
Toluene	108-88-3	8.0E-02	I
Trichloroethane, 1,1,1-	71-55-6	2.0E+00	I
Trichloroethylene	79-01-6	5.0E-04	I
Trichlorophenol, 2,4,6-	88-06-2	1.0E-03	P
Trichloropropane, 1,2,3-	96-18-4	4.0E-03	I
Trimethylbenzene, 1,3,5-	108-67-8	1.0E-02	X

Abbreviations:

A	ATSDR
I	IRIS
C	Cal EPA (2005b)
P	PPRTV
X	PPRTV Appendix – screening value
RfD	Reference Dose

TABLE 8-5
CHEMICALS WITH UPDATED INHALATION UNIT RISK FACTORS (URF) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Inhalation URF $(\mu g/m^3)^{-1}$	Reference
Bis(2-ethylhexyl)phthalate	117-81-7	2.4E-06	C
Bromodichloro methane	75-27-4	3.7E-05	C
Cadmium (Water)	7440-43-9	1.8E-03	I
Carbon Tetrachloride	56-23-5	6.0E-06	I
Chromium (VI)	18540-29-9	8.4E-02	S
Dibromochloro methane	124-48-1	2.7E-05	C
Dioxane, 1,4-	123-91-1	5.0E-06	I
Ethylbenzene	100-41-4	2.5E-06	C
Hexachloroethane	67-72-1	1.1E-05	C
Methylene Chloride	75-09-2	1.0E-08	I
Naphthalene	91-20-3	3.4E-05	C
Nickel Soluble Salts	7440-02-0	2.6E-04	C
Nitrobenzene	98-95-3	4.0E-05	I
Nitrosodimethylamine, N-	62-75-9	1.4E-02	I
Nitrosodiphenylamine, N-	86-30-6	2.6E-06	C
Pentachlorophenol	87-86-5	5.1E-06	C
TCDD, 2,3,7,8-	1746-01-6	3.8E+01	C
Tetrachloroethylene	127-18-4	2.6E-07	I
Trichloroethylene	79-01-6	4.1E-06	I
Vinyl Chloride	75-01-4	4.4E-06	I

Abbreviations:
A ATSDR
C Cal EPA
I IRIS
P PPRTV
S RSLs, see user guide section 5

TABLE 8-6
CHEMICALS WITH UPDATED ORAL SLOPE FACTORS (CSF) USED IN THE RISK ASSESSMENT

Chemical of Potential Concern	CAS Number	Oral SF $(mg/kg\text{-}day)^{-1}$	Reference
Butyl Benzyl Phthlate	85-68-7	1.9E-03	P
Carbon Tetrachloride	56-23-5	7.0E-02	I
Chloroaniline, p-	106-47-8	2.0E-01	P
Chloroform	67-66-3	3.1E-02	C
Chromium (VI)	18540-29-9	5.0E-01	NJ
Dinitrotoluene, 2,6-	606-20-2	1.5E+00	P
Dioxane, 1,4-	123-91-1	1.0E-01	I
Ethylbenzene	100-41-4	1.1E-02	C
Hexachloroethane	67-72-1	4.0E-02	I
Methylene Chloride	75-09-2	2.0E-03	I
Nitroaniline, 4-	100-01-6	2.0E-02	P
Nitrosodimethylamine, N-	62-75-9	5.1E+01	I
Pentachlorophenol	87-86-5	4.0E-01	I
TCDD, 2,3,7,8-	1746-01-6	1.3E+05	C
Tetrachloroethylene	127-18-4	2.1E-03	I
Trichlorobenzene, 1,2,4-	120-82-1	2.9E-02	P
Trichloroethylene	79-01-6	4.6E-02	I
Vinyl Chloride	75-01-4	7.2E-01	I

Abbreviations:
A ATSDR
C Cal EPA
I IRIS
NJ New Jersey
P PPRTV

TABLE 8-7
TOXICITY CRITERIA FOR CHEMICALS OF POTENTIAL CONCERN
NOT IN THE LAKES DATABASE

CAS No.	COPC	Oral RfD (mg/kg-day)	Oral SF $(mg/kg\text{-}day)^{-1}$	Inhalation RfC (mg/m^3)	Inhalation URF $(\mu g/m^3)^{-1}$
526-73-8	1,2,3-Trimethylbenzene	ND	ND	5.00E-03	ND
95-63-6	1,2,4-Trimethylbenzene	ND	ND	7.00E-03	ND
105-05-5	1,4-Diethylbenzene	1.00E-01	1.10E-02	1.00E+00	2.50E-06
106-98-9	1-Butene	ND	ND	3.00E+00	ND
90-13-1	1-Chloronaphthalene	8.00E-02	ND	ND	ND
134-32-7	1-Naphthylamine	ND	1.80E+00	ND	5.14E-04
540-84-1	2,2,4-Trimethylpentane	4.00E-02	ND	2.00E-01	ND
75-83-2	2,2-Dimethylbutane	4.00E-02	ND	2.00E-01	ND
565-75-3	2,3,4,-Trimethylpentane	4.00E-02	ND	2.00E-01	ND
79-29-8	2,3-Dimethylbutane	4.00E-02	ND	2.00E-01	ND
565-59-3	2,3-Dimethylpentane	4.00E-02	ND	2.00E-01	ND
108-08-7	2,4-Dimethylpentane	4.00E-02	ND	2.00E-01	ND
5779-94-2	2,5-Dimethylbenz	1.00E-01	ND	ND	ND
87-65-0	2,6-Dichlorophenol	3.00E-03	ND	ND	ND
611-14-3	2-Ethyltoluene	1.00E-01	1.10E-02	1.00E+00	2.50E-06
591-78-6	2-Hexanone	5.00E-03	ND	3.00E-02	ND
562-27-6	2-Methylheptane	4.00E-02	ND	2.00E-01	ND
591-76-4	2-Methylhexane	6.00E-02	ND	7.00E-01	ND
91-57-6	2-Methylnaphthalene	4.00E-03	ND	ND	ND
107-83-5	2-Methylpentane	4.00E-02	ND	2.00E-01	ND
91-59-8	2-Naphthylamine	ND	1.80E+00	ND	5.14E-04
620-14-4	3-Ethyltoluene	1.00E-01	1.10E-02	1.00E+00	2.50E-06
589-81-1	3-methylheptane	4.00E-02	ND	2.00E-01	ND
96-14-0	3-methylpentane	4.00E-02	ND	2.00E-01	ND
589-34-4	3-Methylhexane	4.00E-02	ND	2.00E-01	ND
108-39-4	Cresol,m-(3-	5.00E-02	ND	6.00E-01	ND
106-44-5	Cresol,p- (4-	1.00E-01	ND	6.00E-01	ND
534-52-1	4,6-Dinitro-2-	8.00E-05	ND	ND	ND
92-67-1	4-Aminobiphenyl	ND	2.10E+01	ND	6.00E-03
622-96-8	4-Ethyltoluene	1.00E-01	1.10E-02	1.00E+00	2.50E-06
208-96-8	Acenaphthylene	6.00E-02	ND	ND	ND
7429-90-5	Aluminum	1.00E+00	ND	5.00E-03	ND
191-24-2	Benzo(g,h,i)perylene	3.00E-02	ND	ND	ND
111-91-1	bis(2-Chloroethoxy)	3.00E-03	ND	ND	ND
86-74-8	Carbazole	ND	2.00E-02	ND	ND
107-14-2	Chloroacetonitrile	ND	ND	6.00E-02	ND
10062-01-5	cis-1,3-Dichloropropene	3.00E-02	1.00E-01	2.00E-02	4.00E-06

TABLE 8-7
TOXICITY CRITERIA FOR CHEMICALS OF POTENTIAL CONCERN
NOT IN THE LAKES DATABASE

CAS No.	COPC	Oral RfD (mg/kg-day)	Oral SF $(mg/kg\text{-}day)^{-1}$	Inhalation RfC (mg/m^3)	Inhalation URF $(\mu g/m^3)^{-1}$
590-18-1	cis-2-Butene	NA	NA	3.00E+00	NA
7440-48-4	Cobalt	3.00E-04	NA	6.00E-06	9.00E-03
7440-50-8	Copper	4.00E-02	NA	NA	NA
4170-30-3	Crotonaldehyde	NA	1.90E+00	NA	NA
110-82-7	Cyclohexane	NA	NA	6.00E+00	NA
132-64-9	Dibenzofuran	1.00E-03	NA	NA	NA
122-39-4	Diphenylamine	2.50E-02	NA	NA	NA
60-29-7	Ethyl Ether	2.00E-01	NA	NA	NA
1888-71-7	Hexachloropropene	7.00E-04	4.00E-02	3.00E-02	1.10E-05

Abbreviations:
NA Not Available

TABLE 8-8
ORAL SLOPE FACTORS† FOR CHEMICALS WITH A MUTAGENIC MODE OF ACTION FOR USE IN THE LAKES MODEL AND OBOD HHRA AT PROMONTORY

COPC	Adult – Standard Toxicity (26 Years as adult)	Farmer and Resident Child (0-6 Years)	Resident (26 Years)	Adult Farmer (40 Years)
Age-dependent Adjustment Factor	1.0	5.3	2.8	2.2
Benzo[a]pyrene	7.3	38.7	20	16
Benzo[a]anthracene	0.73	3.9	2	1.6
Benzo[b]fluoranthene	0.73	3.9	2	1.6
Benzo[k]fluoranthene	7.3E-2	3.9E-1	2.0E-1	1.6E-1
Chrysene	7.3E-3	3.9E-2	2.0E-2	1.6E-2
Dibenz(a,h)anthracene	7.3	38.7	20	16
Indeno[1,2,3-c,d]pyrene	0.73	3.87	2	1.6
Hexavalent Chromium	0.5	2.7	1.4	1.1
Methylene Chloride	2.0E-3	1.1E-2	5.6E-3	4.4E-3
n-Nitrosodiethylamine	150	795	420	330
n-Nitrosodimethylamine	51	270	143	112
Trichloroethylene[1]	4.6E-2	5.1E-2	6.4E-2	5.5E-2
Vinyl Chloride[2]	7.2E-1	1.4	1.4	1.4

† All units are mg/kg/day

[1] The oral slope factors for TCE have been calculated as shown in Appendix D.

[2] The oral slope factor for vinyl chloride has been adjusted in accordance with the IRIS file (EPA 2000) to account for continuous lifetime exposure from birth.

TABLE 8-9
UNIT RISK FACTORS† FOR CHEMICALS WITH A MUTAGENIC MODE OF ACTION FOR USE IN THE LAKES MODEL AND OBOD HHRA AT PROMONTORY

COPC	Industrial Worker (25 Years as adult)	Farmer and Resident Child (0-6 Years)	Resident (26 Years)	Adult Farmer (40 Years)
Age-dependent Adjustment Factor	1.0	5.3	2.8	2.2
Benzo[a]pyrene	1.1E-03	5.8E-03	3.1E-03	2.4E-03
Benzo[a]anthracene	1.1E-04	5.8E-04	3.1E-04	2.4E-04
Benzo[b]fluoranthene	1.1E-04	5.8E-04	3.1E-04	2.4E-04
Benzo[k]fluoranthene	1.1E-04	5.8E-04	3.1E-04	2.4E-04
Chrysene	1.1E-05	5.8E-05	3.1E-05	2.4E-05
Dibenz(a,h)anthracene	1.2E-03	6.4E-03	3.4E-03	2.6E-03
Indeno[1,2,3-c,d]pyrene	1.1E-04	5.8E-04	3.1E-04	2.4E-04
Hexavalent Chromium	8.4E-2	4.5E-1	2.4E-1	1.8E-1
Methylene Chloride	1.0E-8	5.3E-08	2.8E-08	2.2E-08
n-Nitrosodiethylamine	4.3E-02	2.3E-01	1.2E-01	9.5E-02
n-Nitrosodimethylamine	1.4E-02	7.4E-02	3.9E-02	3.1E-02
Trichloroethylene[1]	4.1E-06	5.3E-6	5.7E-6	5.3E-6
Vinyl Chloride[2]	4.4E-06	8.8E-06	8.8E-06	8.8E-06

† All units are $(\mu g/m^3)^{-1}$

[1] The URFs for TCE have been calculated as shown in Appendix D.

[2] The URF for vinyl chloride has been adjusted in accordance with the IRIS file (EPA 2000) to account for continuous lifetime exposure from birth.

TABLE 9-1
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, ACTUAL ON-SITE WORKERS

Receptor Location (a)	Hazard Index	HI with Adjusted Ni and Cr(a)
Autoliv Facility using all sources(a)	4.3	
Autoliv HI using sources M-136 A1, A2, A3 and M-225 A	2.2	0.5 [b]
South Plant Main Building using all sources[a]	3.8	
S. Plant HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.9	0.5 [b]
North Plant Main Building using all sources[a]	2.0	
N. Plant HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.0	0.2 [b]
Point of Maximum On-site Deposition using all sources(a)	7.8	
Point of Maximum On-site Deposition using M-136 A1, A2, A# and M-225 A	3.9	9.7E-1[b]

An Index of one or less is acceptable
(a) All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.
(b) Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

TABLE 9-2
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, FOR HYPOTHETICAL DISCRETE OFF-SITE BOUNDARY RECEPTORS

Receptor Location (a)	Hazard Index	HI with Adjusted Ni and Cr(b)
Point of Maximum Off-site Deposition assuming all sources	0.9	
Point of Maximum Off-site Deposition using M-136 A1, A2, A3 and M-225 A	0.5	0.1
Blue Creek using all sources	4.7	
B. Creek HI calculated using sources M-136 A1, A2, A3 and M-225 A	2.4	0.6
Boundary 1 using all sources	5.3	
Bound 1 HI calculated using sources M-136 A1, A2, A3 and M-225 A	2.7	0.7
Boundary 2 using all sources	2.3	
Bound 2 HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.2	0.3
Boundary 3 using all sources	0.7	
Bound 3 HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1
Boundary 4 using all sources	0.8	

TABLE 9-2
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, FOR HYPOTHETICAL DISCRETE OFF-SITE BOUNDARY RECEPTORS

Receptor Location (a)	Hazard Index	HI with Adjusted Ni and Cr(b)
Bound 4 HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1
ATK Ranch Pond using all sources	0.2	
ATK Ranch Pond HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	2.9E-02

<0.25 Less than 0.25, an Index of one or less is acceptable

(a) All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.

(b) Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

TABLE 9-3
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ALL COPCS, FOR ACTUAL RESIDENTIAL/FARMER OFF-SITE RECEPTORS

Receptor Location (a)	Hazard Index	HI adjusted Ni and Cr(b)
Adams Ranch using all sources	2.8	
A. Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	1.4	0.4
Christensen Ranch using all sources	0.9	
Christensen Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.4	0.1
Holmgren Ranch using all sources	0.5	
Holmgren Ranch HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.2	0.1
Howell Dairy using all sources	0.3	
Howell Dairy HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	3.6E-02
Penrose using all sources	0.3	
Penrose HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.1	3.4E-02
Thatcher using all sources	0.3	
Thatcher HI calculated using sources M-136 A1, A2, A3 and M-225 A	0.2	4.4E-02

<0.25 Less than 0.25, an Index of one or less is acceptable
(a) All sources includes M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic and would not occur. It represents an overestimation of risk.
(b) Sources M-136 A1, A2, A3 and M-225 A were selected to represent actual operating conditions, and the hazards associated with Cr VI and Ni were adjusted by factors of 0.05 and 0.017, respectively.

TABLE 9-4
SUMMED NON-CANCER HAZARD INDICES AND CANCER RISKS FOR ALL COPCS: ACTUAL ON-SITE INDUSTRIAL RECEPTORS

Receptor Name	Industrial Worker Cancer Risk	Industrial Worker Non-cancer HI
Autoliv Facility	8.3E-08	2.4E-02
North Plant Main Administration Building	2.9E-08	8.4E-03
South Plant Main Administration Building	6.9E-08	2.0E-02

TABLE 9-5
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS:
HYPOTHETICAL RESIDENT AND FARMER RECEPTORS AT BOUNDARY/OFF-SITE LOCATIONS

Receptor Name (a)	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Blue Creek	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Boundary 1	3.2E-02	3.2E-02	3.2E-02	3.2E-02
Boundary 2	1.3E-02	1.3E-02	1.3E-02	1.3E-02
Boundary 3	3. 2E-03	3.2E-03	3.2E-03	3.3E-03
Boundary 4	3.5E-03	3.5E-03	3.5E-03	3.5E-03
ATK Ranch Pond	1.1E-03	1.1E-03	1.1E-03	1.1E-03
Maximum Off-site	4.9E-02	4.9E-02	4.9E-02	4.9E-02

(a) These scenario are not realistic as there are no receptors at these locations.

TABLE 9-6
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS:
ACTUAL OFF-SITE RECEPTOR

Receptor Name (a)	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Adams Ranch	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Christensen Ranch	5.0E-03	5.0E-03	5.0E-03	5.1E-03
Holmgren Ranch	2.6E-03	2.6E-03	2.6E-03	2.6E-03
Howell Dairy	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Thatcher	1.8E-03	1.8E-03	1.9E-03	1.9E-03
Penrose	1.6E-03	1.6E-03	1.7E-03	1.7E-03

(a) Assumes all sources at the permit limit amounts.

TABLE 9-7
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS:
HYPOTHETICAL RESIDENT AND FARMER RECEPTORS AT BOUNDARY/OFF-SITE LOCATIONS

Receptor Name (a)	Resident Adult Cancer Risk	Resident Child Cancer Risk	Farmer Adult Cancer Risk	Farmer Child Cancer Risk
Blue Creek	3.3E-07	1.4E-07	6.7E-07	2.4E-07
Boundary 1	3.1E-07	1.3E-07	6.5E-07	2.3E-07
Boundary 2	1.2E-07	5.2E-08	2.5E-07	8.9E-08
Boundary 3	3.1E-08	1.3E-08	6.4E-08	2.3E-08
Boundary 4	3.3E-08	1.4E-08	6.9E-08	2.4E-08
ATK Ranch Pond	9.7E-09	4.1E-09	2.1E-08	7.5E-09
Maximum Off-site	4.8E-07	2.1E-07	9.8E-07	3.5E-07

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) This scenario is not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-8
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS:
ACTUAL OFF-SITE RECEPTOR

Receptor Name (a)	Resident Adult Chronic Risk	Resident Child Chronic Risk	Farmer Adult Chronic Risk	Farmer Child Chronic Risk
Adams Ranch	1.6E-07	7.0E-08	3.4E-07	1.2E-07
Christensen Ranch	4.7E-08	2.0E-08	9.9E-08	3.5E-08
Holmgren Ranch	2.4E-08	1.0E-08	5.0E-08	1.8E-08
Thatcher	1.7E-08	7.4E-09	3.7E-08	1.3E-08
Howell Dairy	1.6E-08	7.0E-09	3.6E-08	1.3E-08
Penrose	1.5E-08	6.3E-09	3.2E-08	1.1E-08

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B).

TABLE 9-9
SUMMARY OF SUMMED HAZARDS AND RISKS FOR ALL COPCS BY PATHWAY FOR RESIDENTIAL ADULT RECEPTORS

	Inhalation		Soil Ingestion		Produce Ingestion		Total	
Receptor (a)	**Hazard Index**	**Cancer Risk**	**Hazard Index**	**Cancer Risk**	**Hazard Index**	**Cancer Risk**	**Total Hazard Index**	**Total Cancer Risk**
Actual Receptor – Off-site								
Adams Ranch	1.7E-02	1.6E-07	4.1E-10	4.1E-13	3.5E-06	4.5E-09	1.7E-02	1.6E-07
Christensen Ranch	5.0E-03	4.6E-08	1.1E-10	1.1E-13	1.0E-06	1.3E-09	5.0E-03	4.7E-08
Holmgren Ranch	2.6E-03	2.3E-08	7.0E-11	7.2E-14	5.6E-07	6.7E-10	2.6E-03	2.4E-08
Howell Dairy	1.9E-03	1.6E-08	4.5E-11	4.5E-14	3.9E-07	5.0E-10	1.9E-03	1.6E-08
Penrose	1.6E-03	1.4E-08	4.9E-11	5.1E-14	3.7E-07	4.4E-10	1.6E-03	1.5E-08
Thatcher	1.8E-03	1.7E-08	4.0E-11	4.0E-14	3.6E-07	4.9E-10	1.8E-03	1.7E-08
Hypothetical Receptor (b) – Boundary/Off-site Locations								
Blue Creek	3.3E-02	3.2E-07	9.5E-10	9.6E-13	7.4E-06	8.7E-09	3.3E-02	3.3E-07
Boundary 1	3.2E-02	3.1E-07	8.1E-10	8.1E-13	6.8E-06	8.5E-09	3.2E-02	3.1E-07
Boundary 2	1.3E-02	1.2E-07	2.9E-10	2.8E-13	2.5E-06	3.3E-09	1.3E-02	1.2E-07
Boundary 3	3.2E-03	3.0E-08	8.8E-11	9.0E-14	7.0E-07	8.5E-10	3. 2E-03	3.1E-08
Boundary 4	3.5E-03	3.2E-08	8.2E-11	8.2E-14	7.1E-07	9.2E-10	3.5E-03	3.3E-08
ATK Ranch Pond	1.1E-03	9.4E-09	2.8E-11	2.9E-14	2.4E-07	3.0E-10	1.1E-03	9.7E-09
Maximum Off-site	4.9E-02	4.7E-07	1.0E-09	1.0E-12	9.5E-06	1.3E-08	4.9E-02	4.8E-07

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic because there are no receptors at these locations.

TABLE 9-10
SUMMARY OF SUMMED HAZARDS AND RISKS FOR ALL COPCS BY PATHWAY FOR RESIDENTIAL CHILD RECEPTORS

	Inhalation		Soil Ingestion		Produce Ingestion		Total	
Receptor (a)	**Hazard Index**	**Cancer Risk**	**Hazard Index**	**Cancer Risk**	**Hazard Index**	**Cancer Risk**	**Total Hazard Index**	**Total Cancer Risk**
Actual Receptor – Off-site								
Adams Ranch	1.7E-02	6.8E-08	4.4E-09	1.1E-12	8.4E-06	2.5E-09	1.7E-02	7.0E-08
Christensen Ranch	5.0E-03	1.9E-08	1.2E-09	3.0E-13	2.4E-06	7.5E-10	5.0E-03	2.0E-08
Holmgren Ranch	2.6E-03	9.9E-09	7.5E-10	1.9E-13	1.3E-06	3.8E-10	2.6E-03	1.0E-08
Howell Dairy	1.9E-03	6.7E-09	4.8E-10	1.2E-13	9.3E-07	2.8E-10	1.9E-03	7.0E-09
Penrose	1.6E-03	6.1E-09	5.2E-10	1.4E-13	9.0E-07	2.5E-10	1.6E-03	6.3E-09
Thatcher	1.8E-03	7.2E-09	4.2E-10	1.1E-13	8.7E-07	2.8E-10	1.8E-03	7.4E-09
Hypothetical Receptor (b) – Boundary/Off-site Locations								
Blue Creek	3.3E-02	1.4E-07	1.0E-08	2.6E-12	1.8E-05	4.9E-09	3.3E-02	1.4E-07
Boundary 1	3.2E-02	1.3E-07	8.7E-09	2.2E-12	1.6E-05	4.8E-09	3.2E-02	1.3E-07
Boundary 2	1.3E-02	5.0E-08	3.1E-09	7.5E-13	6.1E-06	1.9E-09	1.3E-02	5.2E-08
Boundary 3	3.2E-03	1.3E-08	9.4E-10	2.4E-13	1.7E-06	4.8E-10	3.2E-03	1.3E-08
Boundary 4	3.5E-03	1.3E-08	8.7E-10	2.2E-13	1.7E-06	5.2E-10	3.5E-03	1.4E-08
ATK Ranch Pond	1.1E-03	4.0E-09	3.0E-10	7.7E-14	5.7E-07	1.7E-10	1.1E-03	4.1E-09
Maximum Off-site	4.9E-02	2.0E-07	1.1E-08	2.7E-12	2.3E-05	7.2E-09	4.9E-02	2.1E-07

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-11
SUMMARY OF SUMMED CANCER RISKS FOR ALL COPCS BY PATHWAY FOR FARMER ADULT RECEPTORS

Receptor (a)	Inhalation	Soil	Vegetables	Beef	Chicken	Eggs	Milk	Pork	Total Cancer Risk
Actual Receptor – On-site									
Adams Ranch	1.9E-07	6.3E-13	1.0E-08	3.1E-08	2.0E-13	1.3E-13	1.1E-07	1.2E-09	3.4E-07
Christensen Ranch	5.4E-08	1.7E-13	3.0E-09	9.1E-09	5.6E-14	3.6E-14	3.3E-08	3.5E-10	9.9E-08
Holmgren Ranch	2.7E-08	1.1E-13	1.5E-09	4.6E-09	3.5E-14	2.3E-14	1.7E-08	1.8E-10	5.0E-08
Howell Dairy	1.9E-08	7.0E-14	1.1E-09	3.4E-09	2.3E-14	1.5E-14	1.2E-08	1.3E-10	3.6E-08
Penrose	1.7E-08	8.0E-14	9.8E-10	3.0E-09	2.5E-14	1.6E-14	1.1E-08	1.2E-10	3.2E-08
Thatcher	2.0E-08	6.2E-14	1.1E-09	3.4E-09	2.0E-14	1.3E-14	1.2E-08	1.3E-10	3.7E-08
Hypothetical Receptor (b) – Boundary/Off-site Locations									
Blue Creek	3.8E-07	1.5E-12	1.9E-08	6.0E-08	4.7E-13	3.0E-13	2.1E-07	2.3E-09	6.7E-07
Boundary 1	3.6E-07	1.3E-12	1.9E-08	5.9E-08	4.0E-13	2.6E-13	2.1E-07	2.2E-09	6.5E-07
Boundary 2	1.4E-07	4.3E-13	7.4E-09	2.3E-08	1.4E-13	9.1E-14	8.1E-08	8.7E-10	2.5E-07
Boundary 3	3.5E-08	1.4E-13	1.9E-09	5.9E-09	4.4E-14	2.9E-14	2.1E-08	2.2E-10	6.4E-08
Boundary 4	3.7E-08	1.3E-13	2.1E-09	6.3E-09	4.1E-14	2.7E-14	2.3E-08	2.4E-10	6.9E-08
ATK Ranch Pond	1.1E-08	4.5E-14	6.7E-10	2.1E-09	1.4E-14	9.2E-15	7.4E-09	7.9E-11	2.1E-08
Maximum Off-site	5.5E-07	1.6E-12	2.9E-08	8.8E-08	5.2E-13	3.3E-13	3.2E-07	3.4E-09	9.8E-07

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-12
SUMMARY OF SUMMED HAZARD INDICES FOR ALL COPCS BY PATHWAY FOR FARMER ADULT RECEPTORS

Receptor (a)	Inhalation	Soil	Vegetables	Beef	Chicken	Eggs	Milk	Pork	Total Hazard Index
Actual Receptor – Off-site									
Adams Ranch	1.7E-02	4.1E-10	5.1E-06	9.4E-06	3.0E-11	2.0E-11	3.4E-05	3.6E-07	1.7E-02
Christensen Ranch	5.0E-03	1.1E-10	1.5E-06	2.8E-06	8.5E-12	5.6E-12	1.0E-05	1.1E-07	5.0E-03
Holmgren Ranch	2.6E-03	7.0E-11	8.2E-07	1.4E-06	4.9E-12	3.2E-12	5.1E-06	5.4E-08	2.6E-03
Howell Dairy	1.9E-03	4.5E-11	5.6E-07	1.0E-06	3.3E-12	2.2E-12	3.7E-06	4.0E-08	1.9E-03
Penrose	1.6E-03	4.9E-11	5.5E-07	9.2E-07	3.4E-12	2.2E-12	3.3E-06	3.5E-08	1.7E-03
Thatcher	1.8E-03	4.0E-11	5.3E-07	1.0E-06	3.1E-12	2.0E-12	3.7E-06	3.9E-08	1.9E-03
Hypothetical Receptor (b) – Boundary/Off-site Locations									
Blue Creek	3.3E-02	9.5E-10	1.1E-05	1.8E-05	6.5E-11	4.3E-11	6.6E-05	7.0E-07	3.3E-02
Boundary 1	3.2E-02	8.1E-10	9.9E-06	1.8E-05	5.9E-11	3.8E-11	6.4E-05	6.8E-07	3.2E-02
Boundary 2	1.3E-02	2.9E-10	3.7E-06	6.9E-06	2.1E-11	1.4E-11	2.5E-05	2.6E-07	1.3E-02
Boundary 3	3.2E-03	8.8E-11	1.0E-06	1.8E-06	6.2E-12	4.1E-12	6.4E-06	6.8E-08	3.2E-03
Boundary 4	3.5E-03	8.2E-11	1.0E-06	1.9E-06	6.1E-12	4.0E-12	6.9E-06	7.4E-08	3.5E-03
ATK Ranch Pond	1.1E-03	2.8E-11	3.5E-07	6.3E-07	2.1E-12	1.4E-12	2.3E-06	2.4E-08	1.1E-03
Maximum Off-site	4.9E-02	1.0E-09	1.4E-05	2.7E-05	8.0E-11	5.2E-11	9.6E-05	1.0E-06	4.9E-02

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-13
SUMMARY OF SUMMED CANCER RISKS FOR ALL COPCS BY PATHWAY FOR FARMER CHILD RECEPTORS

Receptor (a)	Inhalation	Soil	Vegetables	Beef	Chicken	Eggs	Milk	Pork	Total Cancer Risk
Actual Receptor – Off-site									
Adams Ranch	6.8E-08	1.1E-12	3.7E-09	4.5E-09	2.2E-14	1.5E-14	4.4E-08	2.1E-10	1.2E-07
Christensen Ranch	1.9E-08	3.0E-13	1.1E-09	1.3E-09	6.1E-15	4.2E-15	1.3E-08	6.4E-11	3.5E-08
Holmgren Ranch	9.9E-09	1.9E-13	5.6E-10	6.9E-10	3.8E-15	2.6E-15	6.6E-09	3.2E-11	1.8E-08
Howell Dairy	6.7E-09	1.2E-13	4.1E-10	5.1E-10	2.4E-15	1.7E-15	4.9E-09	2.4E-11	1.3E-08
Penrose	6.1E-09	1.4E-13	3.6E-10	4.5E-10	2.6E-15	1.8E-15	4.3E-09	2.1E-11	1.1E-08
Thatcher	7.2E-09	1.1E-13	4.0E-10	5.0E-10	2.2E-15	1.5E-15	4.8E-09	2.3E-11	1.3E-08
Hypothetical Receptor (b) – Boundary/Off-site Locations									
Blue Creek	1.4E-07	2.6E-12	7.2E-09	8.8E-09	5.0E-14	3.4E-14	8.5E-08	4.2E-10	2.4E-07
Boundary 1	1.3E-07	2.2E-12	7.0E-09	8.7E-09	4.4E-14	3.0E-14	8.4E-08	4.1E-10	2.3E-07
Boundary 2	5.0E-08	7.5E-13	2.7E-09	3.4E-09	1.5E-14	1.0E-14	3.2E-08	1.6E-10	8.9E-08
Boundary 3	1.3E-08	2.4E-13	7.1E-10	8.7E-10	4.7E-15	3.2E-15	8.4E-09	4.1E-11	2.3E-08
Boundary 4	1.3E-08	2.2E-13	7.6E-10	9.4E-10	4.5E-15	3.1E-15	9.1E-09	4.4E-11	2.4E-08
ATK Ranch Pond	4.0E-09	7.7E-14	2.5E-10	3.1E-10	1.5E-15	1.1E-15	3.0E-09	1.4E-11	7.5E-09
Maximum Off-site	2.0E-07	2.7E-12	1.1E-08	1.3E-08	5.6E-14	3.9E-14	1.3E-07	6.2E-10	3.5E-07

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-14
SUMMARY OF SUMMED HAZARD INDICES FOR ALL COPCS BY PATHWAY FOR FARMER CHILD RECEPTORS

Receptor (a)	Inhalation	Soil	Vegetables	Beef	Chicken	Eggs	Milk	Pork	Total Hazard Index
Actual Receptor – Off-site									
Adams Ranch	1.7E-02	4.4E-09	1.2E-05	5.8E-06	2.0E-11	1.4E-11	5.6E-05	2.7E-07	1.7E-02
Christensen Ranch	5.0E-03	1.2E-09	3.5E-06	1.7E-06	5.8E-12	4.0E-12	1.7E-05	8.1E-08	5.1E-03
Holmgren Ranch	2.6E-03	7.5E-10	2.0E-06	8.7E-07	3.4E-12	2.3E-12	8.4E-06	4.1E-08	2.6E-03
Howell Dairy	1.9E-03	4.8E-10	1.4E-06	6.4E-07	2.3E-12	1.6E-12	6.2E-06	3.0E-08	1.9E-03
Penrose	1.6E-03	5.2E-10	1.3E-06	5.7E-07	2.3E-12	1.6E-12	5.5E-06	2.7E-08	1.7E-03
Thatcher	1.8E-03	4.2E-10	1.3E-06	6.3E-07	2.1E-12	1.5E-12	6.1E-06	3.0E-08	1.9E-03
Hypothetical Receptor (b) – Boundary/Off-site Locations									
Blue Creek	3.3E-02	1.0E-08	2.6E-05	1.1E-05	4.4E-11	3.1E-11	1.1E-04	5.3E-07	3.3E-02
Boundary 1	3.2E-02	8.7E-09	2.4E-05	1.1E-05	4.0E-11	2.8E-11	1.1E-04	5.2E-07	3.2E-02
Boundary 2	1.3E-02	3.1E-09	8.9E-06	4.3E-06	1.4E-11	1.0E-11	4.1E-05	2.0E-07	1.3E-02
Boundary 3	3.2E-03	9.4E-10	2.5E-06	1.1E-06	4.2E-12	2.9E-12	1.1E-05	5.2E-08	3.3E-03
Boundary 4	3.5E-03	8.7E-10	2.5E-06	1.2E-06	4.2E-12	2.9E-12	1.2E-05	5.6E-08	3.5E-03
ATK Ranch Pond	1.1E-03	3.0E-10	8.3E-07	3.9E-07	1.4E-12	9.8E-13	3.8E-06	1.8E-08	1.1E-03
Maximum Off-site	4.9E-02	1.1E-08	3.4E-05	1.7E-05	5.4E-11	3.8E-11	1.6E-04	7.8E-07	4.9E-02

(a) These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B) at the permit limit amounts
(b) These scenarios are not realistic and would not occur. It represents an overestimation of risk.

TABLE 9-15
COMPARISON OF CHRONIC RISKS USING ALL COPCS VERSUS DETECTED CHEMICALS ONLY

Receptor Name	Chronic Risk All COPCs	Chronic Risk Detects Only	Percent attributable to Detects
Autoliv Facility			
Industrial Worker	8.3E-08	7.2E-08	86%
Maximum Offsite			
Hypothetical Resident Adult	4.8E-07	4.3E-07	90%
Hypothetical Resident Child	2.1E-07	1.9E-07	91%
Hypothetical Farmer Adult	9.8E-07	6.9E-07	70%
Hypothetical Farmer Child	3.5E-07	2.3E-07	67%
Adams Ranch			
Resident Adult	1.3E-07	1.1E-07	90%
Resident Child	7.0E-08	6.4E-08	91%
Farmer Adult	3.4E-07	2.4E-07	70%
Farmer Child	1.2E-07	7.9E-08	66%
All COPCs includes 209 chemicals, detects only includes 133 chemicals			

TABLE 9-16
RESULTS FOR NURSING INFANTS DUE TO INGESTION OF BREAST MILK

Receptor Name	Infant ADD [pg COPC/kg BW-day] Resident Scenario	Infant ADD [pg COPC/kg BW-day] Farmer Scenario
Adams Ranch	1.2E-04	2.4E-02
ATK Ranch Pond	7.6E-06	1.6E-03
Blue Creek	2.4E-04	4.7E-02
Boundary 1	2.3E-04	4.6E-02
Boundary 2	8.8E-05	1.8E-02
Boundary 3	2.3E-05	4.6E-03
Boundary 4	2.4E-05	5.0E-03
Christensen Ranch	3.5E-05	7.1E-03
Holmgren Ranch	1.8E-05	3.6E-03
Howell Dairy	1.3E-05	2.7E-03
Penrose	1.1E-05	2.4E-03
Thatcher	1.3E-05	2.6E-03

Infant ADD = Average Daily Dose

TABLE 9-17 SUMMED RISKS AND HAZARDS FOR ALL COPCS: FUTURE ON-SITE WORKER		
Receptor Name	**Industrial Worker Cancer Risk**	**Industrial Worker Non-cancer HI**
Maximum On-site	1.2E-07	3.3E-02

TABLE 9-18 SUMMED RISKS AND HAZARD INDICES FOR ALL COPCS: FUTURE RESIDENT/FARMER				
Receptor Name	**Resident Adult Chronic HI**	**Resident Child Chronic HI**	**Farmer Adult Chronic HI**	**Farmer Child Chronic HI**
Maximum Off-site	4.9E-02	4.9E-02	4.9E-02	4.9E-02
Receptor Name	**Resident Adult Chronic Risk**	**Resident Child Chronic Risk**	**Farmer Adult Chronic Risk**	**Farmer Child Chronic Risk**
Maximum Off-site	4.8E-07	2.1E-07	9.8E-07	3.5E-07

TABLE 10-1
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES ACTUAL ON-SITE AND HYPOTHETICAL FUTURE WORKERS, SUMMING ALL COPCS WITH ADJUSTED CHROMIUM AND NICKEL EMISSIONS TO REMOVE POTENTIAL CONTRIBUTION FROM THE STAINLESS STEEL TESTING PANS

Receptor Location	Hazard Index with Limited Sources Active(a)	Hazard Index with Adjusted Nickel and Chromium(b)
Autoliv Facility	2.2	0.5
South Plant Main Building	1.9	0.5
North Plant Main Building	1.0	0.2
Point of Maximum On-site Deposition	3.9	9.7E-01

(a) Hazard Indices calculated assuming sources M-136 A1, A2, A3 and M-225 A are active at the same time. This scenario is more representative of actual operating conditions.
(b) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-2
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR HYPOTHETICAL BOUNDARY/OFF-SITE RECEPTORS (RESIDENTIAL/FARMER) SUMMING ALL COPCS WITH ADJUSTED CHROMIUM AND NICKEL EMISSIONS TO REMOVE POTENTIAL CONTRIBUTION FROM THE STAINLESS STEEL TESTING PANS

Receptor Location	Hazard Index with Limited Sources Active(a)	Hazard Index with Adjusted Nickel and Chromium(b)
Blue Creek	2.4	0.6
Boundary 1	2.7	0.7
Boundary 2	1.2	0.3
Boundary 3	0.4	0.1
Boundary 4	0.4	0.1
ATK Ranch Pond	0.1	2.9E-02
Point of Maximum Off-site Deposition	0.5	1.2E-01

(a) Hazard Indices calculated assuming sources M-136 A1, A2, A3 and M-225 A are active at the same time. This scenario is more representative of actual operating conditions.
(b) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-3
SHORT-TERM (1-HOUR) NON-CANCER HAZARD INDICES FOR ACTUAL OFF-SITE RECEPTORS (RESIDENTIAL/FARMER) SUMMING ALL COPCS WITH ADJUSTED CHROMIUM AND NICKEL EMISSIONS TO REMOVE POTENTIAL CONTRIBUTION FROM THE STAINLESS STEEL TESTING PANS

Receptor Location	Hazard Index with Limited Sources Active(a)	Hazard Index with Adjusted Nickel and Chromium(b)
Adams Ranch	1.4	0.4
Christensen Ranch	0.4	0.1
Holmgren Ranch	0.2	0.1
Howell Dairy	0.1	3.6E-02
Penrose	0.1	3.4E-02
Thatcher	0.2	4.4E-02

(a) Hazard Indices calculated assuming sources M-136 A1, A2, A3 and M-225 A are active at the same time. This scenario is more representative of actual operating conditions.
(b) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-4
SUMMED NON-CANCER HAZARD INDICES AND CANCER RISKS FOR ALL COPCS: ACTUAL ON-SITE INDUSTRIAL RECEPTORS (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name	Industrial Worker Cancer Risk	Industrial Worker Non-cancer HI
Autoliv Facility(a)	1.6E-08	2.3E-02
North Plant Main Administration Building(a)	5.6E-09	8.3E-03
South Plant Main Administration Building(a)	1.3E-08	2.0E-02

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-5
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS:
ACTUAL OFF-SITE RECEPTOR (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Adams Ranch	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Christensen Ranch	5.0E-03	5.0E-03	5.0E-03	5.0E-03
Holmgren Ranch	2.5E-03	2.5E-03	2.5E-03	2.5E-03
Howell Dairy	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Thatcher	1.8E-03	1.8E-03	1.8E-03	1.8E-03
Penrose	1.6E-03	1.6E-03	1.6E-03	1.6E-03

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-6
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS:
HYPOTHETICAL RESIDENT AND FARMER RECEPTORS AT BOUNDARY/OFF-SITE LOCATIONS (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Blue Creek	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Boundary 1	3.2E-02	3.2E-02	3.2E-02	3.2E-02
Boundary 2	1.2E-02	1.2E-02	1.2E-02	1.2E-02
Boundary 3	3. 2E-03	3.2E-03	3.2E-03	3.2E-03
Boundary 4	3.5E-03	3.5E-03	3.5E-03	3.5E-03
ATK Ranch Pond	1.1E-03	1.1E-03	1.1E-03	1.1E-03

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-7
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS:
HYPOTHETICAL RESIDENT AND FARMER RECEPTORS AT BOUNDARY/OFF-SITE LOCATIONS (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Resident Adult Cancer Risk	Resident Child Cancer Risk	Farmer Adult Cancer Risk	Farmer Child Cancer Risk
Blue Creek	5.0E-08	2.0E-08	3.5E-07	1.2E-07
Boundary 1	4.3E-08	1.9E-08	3.4E-07	1.1E-07
Boundary 2	1.6E-08	7.5E-09	1.3E-07	4.4E-08
Boundary 3	5.1E-09	1.9E-09	3.4E-08	1.1E-08
Boundary 4	5.6E-09	2.0E-09	3.7E-08	1.2E-08
ATK Ranch Pond	1.6E-09	6.5E-10	1.2E-08	4.0E-09

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-8
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS:
ACTUAL OFF-SITE RECEPTOR (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Resident Adult Chronic Risk	Resident Child Chronic Risk	Farmer Adult Chronic Risk	Farmer Child Chronic Risk
Adams Ranch	2.1E-08	1.0E-08	1.8E-07	6.0E-08
Christensen Ranch	7.0E-09	3.0E-09	5.3E-08	1.8E-08
Holmgren Ranch	3.7E-09	1.5E-09	2.7E-08	9.0E-09
Thatcher	2.3E-09	1.1E-09	2.0E-08	7.5E-09
Howell Dairy	2.2E-09	4.3E-09	2.0E-08	6.6E-09
Penrose	2.6E-09	9.6E-10	1.8E-08	5.8E-09

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-9
SUMMED RISKS AND HAZARDS FOR ALL COPCS:
FUTURE ON-SITE WORKER (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Industrial Worker Cancer Risk	Industrial Worker Non-cancer HI
Maximum On-site	2.2E-08	3.3E-02

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-10
SUMMED RISKS AND HAZARD INDICES FOR ALL COPCS:
FUTURE RESIDENT/FARMER (ADJUSTED NICKEL AND CHROMIUM)

Receptor Name (a)	Resident Adult Chronic HI	Resident Child Chronic HI	Farmer Adult Chronic HI	Farmer Child Chronic HI
Maximum Off-site	4.8E-02	4.8E-02	4.8E-02	4.8E-02
Receptor Name (a)	**Resident Adult Chronic Risk**	**Resident Child Chronic Risk**	**Farmer Adult Chronic Risk**	**Farmer Child Chronic Risk**
Maximum Off-site	6.9E-08	2.9E-08	5.1E-07	1.7E-07

(a) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-11
SUMMED NON-CANCER HAZARD INDICES AND CANCER RISKS FOR ALL COPCS: ACTUAL ON-SITE INDUSTRIAL RECEPTORS

Receptor Name	Industrial Worker Cancer Risk	Industrial Worker Non-cancer HI
Autoliv Facility– Scenario A	8.3E-08	2.4E-02
Autoliv Facility– Scenario B	1.6E-08	2.3E-02
North Plant Main Administration Building– Scenario A	2.9E-08	8.4E-03
North Plant Main Administration Building– Scenario B	5.6E-09	8.3E-03
South Plant Main Administration Building– Scenario A	6.9E-08	2.0E-02
South Plant Main Administration Building– Scenario B	1.3E-08	2.0E-02
Future Hypothetical On-site Worker		
Maximum On-site – Scenario A	1.2E-07	3.3E-02
Maximum On-site – Scenario B	2.2E-08	3.3E-02

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE 10-12
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS: ALL SCENARIOS INCLUDED

Actual Off-Site Receptors				
Receptor Name	**Resident Adult Chronic HI**	**Resident Child Chronic HI**	**Farmer Adult Chronic HI**	**Farmer Child Chronic HI**
Adams Ranch – Scenario A	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Adams Ranch– Scenario B	1.7E-02	1.7E-02	1.7E-02	1.7E-02
Christensen Ranch – Scenario A	5.0E-03	5.0E-03	5.0E-03	5.1E-03
Christensen Ranch– Scenario B	5.0E-03	5.0E-03	5.0E-03	5.0E-03
Holmgren Ranch – Scenario A	2.6E-03	2.6E-03	2.6E-03	2.6E-03
Holmgren Ranch– Scenario B	2.5E-03	2.5E-03	2.5E-03	2.5E-03
Howell Dairy – Scenario A	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Howell Dairy– Scenario B	1.9E-03	1.9E-03	1.9E-03	1.9E-03
Thatcher – Scenario A	1.8E-03	1.8E-03	1.9E-03	1.9E-03
Thatcher– Scenario B	1.8E-03	1.8E-03	1.8E-03	1.8E-03
Penrose – Scenario A	1.6E-03	1.6E-03	1.7E-03	1.7E-03
Penrose– Scenario B	1.6E-03	1.6E-03	1.6E-03	1.6E-03
Future Hypothetical Resident/Farmer Receptor				
Maximum Off-site – Scenario A	4.9E-02	4.9E-02	4.9E-02	4. 9E-02
Maximum Off-site– Scenario B	4.8E-02	4.8E-02	4.8E-02	4.8E-02
Hypothetical Resident and Farmer Receptors at Boundary/Off-site Locations				
Receptor Name	**Resident Adult Chronic HI**	**Resident Child Chronic HI**	**Farmer Adult Chronic HI**	**Farmer Child Chronic HI**
Blue Creek – Scenario A	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Blue Creek– Scenario B	3.3E-02	3.3E-02	3.3E-02	3.3E-02
Boundary 1 – Scenario A	3.2E-02	3.2E-02	3.2E-02	3.2E-02
Boundary 1– Scenario B	3.2E-02	3.2E-02	3.2E-02	3.2E-02

TABLE 10-12
SUMMED NON-CANCER HAZARD INDICES FOR ALL COPCS: ALL SCENARIOS INCLUDED

Boundary 2 – Scenario A	1.3E-02	1.3E-02	1.3E-02	1.3E-02
Boundary 2– Scenario B	1.2E-02	1.2E-02	1.2E-02	1.2E-02
Boundary 3 – Scenario A	3. 2E-03	3.2E-03	3.2E-03	3.3E-03
Boundary 3– Scenario B	3. 2E-03	3.2E-03	3.2E-03	3.2E-03
Boundary 4 – Scenario A	3.5E-03	3.5E-03	3.5E-03	3.5E-03
Boundary 4– Scenario B	3.5E-03	3.5E-03	3.5E-03	3.5E-03
ATK Ranch Pond– Scenario A	1.1E-03	1.1E-03	1.1E-03	1.1E-03
ATK Ranch Pond– Scenario B	1.1E-03	1.1E-03	1.1E-03	1.1E-03

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE 10-13
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS: ALL SCENARIOS INCLUDED

Actual Off-site Receptor				
Receptor Name	**Resident Adult Chronic Risk**	**Resident Child Chronic Risk**	**Farmer Adult Chronic Risk**	**Farmer Child Chronic Risk**
Adams Ranch – Scenario A	1.6E-07	7.0E-08	3.4E-07	1.2E-07
Adams Ranch – Scenario B	2.1E-08	1.0E-08	1.8E-07	6.0E-08
Christensen Ranch-Scenario A	4.7E-08	2.0E-08	9.9E-08	3.5E-08
Christensen Ranch-Scenario B	7.0E-09	3.0E-09	5.3E-08	1.8E-08
Holmgren Ranch-Scenario A	2.4E-08	1.0E-08	5.0E-08	1.8E-08
Holmgren Ranch-Scenario B	3.7E-09	1.5E-09	2.7E-08	9.0E-09
Thatcher- Scenario A	1.7E-08	7.4E-09	3.7E-08	1.3E-08
Thatcher- Scenario B	2.3E-09	1.1E-09	2.0E-08	7.5E-09
Howell Dairy-Scenario A	1.6E-08	7.0E-09	3.6E-08	1.3E-08
Howell Dairy-Scenario B	2.2E-09	4.3E-09	2.0E-08	6.6E-09
Penrose- Scenario A	1.5E-08	6.3E-09	3.2E-08	1.1E-08
Penrose- Scenario B	2.6E-09	9.6E-10	1.8E-08	5.8E-09
Future Hypothetical Resident/Farmer Receptor				
Maximum Off-site-Scenario A	4.8E-07	2.1E-07	9.8E-07	3.5E-07
Maximum Off-site-Scenario B	6.9E-08	2.9E-08	5.1E-07	1.7E-07
Hypothetical Resident and Farmer Receptors at Boundary/Off-site Locations				
Receptor Name	**Resident Adult Cancer Risk**	**Resident Child Cancer Risk**	**Farmer Adult Cancer Risk**	**Farmer Child Cancer Risk**
Blue Creek- Scenario A	3.3E-07	1.4E-07	6.7E-07	2.4E-07
Blue Creek- Scenario B	5.0E-08	2.0E-08	3.5E-07	1.2E-07
Boundary 1- Scenario A	3.1E-07	1.3E-07	6.5E-07	2.3E-07
Boundary 1- Scenario B	4.3E-08	1.9E-08	3.4E-07	1.1E-07

TABLE 10-13
SUMMED EXCESS LIFETIME CANCER RISKS FOR ALL COPCS: ALL SCENARIOS INCLUDED

Boundary 2- Scenario A	1.2E-07	5.2E-08	2.5E-07	8.9E-08
Boundary 2- Scenario B	1.6E-08	7.5E-09	1.3E-07	4.4E-08
Boundary 3- Scenario A	3.1E-08	1.3E-08	6.4E-08	2.3E-08
Boundary 3- Scenario B	5.1E-09	1.9E-09	3.4E-08	1.1E-08
Boundary 4- Scenario A	3.3E-08	1.4E-08	6.9E-08	2.4E-08
Boundary 4- Scenario B	5.6E-09	2.0E-09	3.7E-08	1.2E-08
ATK Ranch Pond- Scenario A	9.7E-09	4.1E-09	2.1E-08	7.5E-09
ATK Ranch Pond- Scenario B	1.6E-09	6.5E-10	1.2E-08	4.0E-09

Scenario A - These chronic cancer risks were calculated using all sources (M-136 A1, A2, A3, B, C13, C14, M-225 A and M-225 B. This scenario is not realistic because of safety concerns, and would not occur. It represents an overestimation of risk.

Scenario B - These chronic cancer risks were calculated assuming all sources (M-136 A1, A2, A3, B, C13, C14, and M-225 A and B). In addition, chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017 because limited amounts of chromium and nickel are available for release compared with Scenario A emissions factors. This scenario represents ATK's best estimate of conservative but more likely risk and hazard levels associated with facility operations.

TABLE 10-14
EXCESS LIFETIME CANCER RISKS FOR A HYPOTHETICAL ADULT FARMER AT THE POINT OF MAXIMUM DEPOSITION ASSUMING DIFFERENT PERCENTAGES OF HEXAVALENT CHROMIUM

Variable Condition	ELCR – 45% Cr (VI)	ELCR – 100% Cr (VI)	ELCR – 14.3% Cr (VI)
Maximum Off-site (a)	9.8E-07	1.6E-06	6.5E-07
Maximum Off-site Adjusted (b)	5.2E-07	5.5E-07	5.0E-07

(a) These represent unadjusted values.
(b) Chromium was adjusted by a factor of 0.05, and nickel was adjusted by a factor of 0.017.

TABLE 10-15
SOME OF THE UNCERTAINTIES IN THE PROMONTORY RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect Risk Assessment Process	Assumption	Effect on the Risk Assessment
Emissions Tests	The worst case emissions from nine different tests is used to provide the emissions factors for the modeling of COPCs	Likely overestimates risk
Emissions Tests	The contribution from background is not subtracted from the emissions factors used to calculate COPC emissions rates	Likely overestimates risk
Emissions Tests	The chromium and nickel contribution from the stainless steel test trays likely creates artifacts that are not subtracted from the emissions factors used to calculate COPC emissions rates	Likely overestimates acute Hazard Indices
Emissions Tests	The contribution from non-detected PAH in the 1.3-Class tests was replaced with 1.1-Class emissions factors	Likely overestimates risk
Emissions Tests	The contribution from non-detected chemicals was shown to contribute an additional 7 to 25 percent of the risks	Shown to overestimate risk
Emissions Tests	Two PAH were eliminated from the COPC list	Likely underestimates risk
Air Quality Modeling	Acute air concentrations are calculated assuming all sources operate at the same time	Shown to overestimate risk
Air Quality Modeling	Assumes reasonable worst case meteorological conditions at the time of processing wastes	Likely overestimates risk

TABLE 10-15
SOME OF THE UNCERTAINTIES IN THE PROMONTORY RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect Risk Assessment Process	Assumption	Effect on the Risk Assessment
Air Quality Modeling	The model has a number of complex assumptions built in to represent plume rise, air dispersion, and particulate deposition. All have uncertainty.	Could overestimate or underestimate risk
Air Quality Modeling	The model has two components: OBODM and AERMOD, the operation of these two models together has not been validated.	Could overestimate or underestimate risk
Media Concentration Models	Soil concentrations are modeled based on deposition, release of COPCs to soil. COPCs may remain on released particles and not be released to soil.	Likely overestimates risk
Media Concentration Models	COPC uptake into plants from air is based on the assumption that higher molecular weight COPCs are in the vapor phase, when they are likely to be adsorbed to particulates.	Likely significantly over estimates risk
Media Concentration Models	Plant uptake of COPCs is based on chemical specific modeling, often using physical parameters and often un-validated assumptions	Likely significantly over estimates risk
Media Concentration Models	Bio-transfer factors for COPCs from plants-to-animals, plants-to-humans, animals-to-humans, and human-to-human is based on chemical specific modeling, often using physical parameters and often un-validated assumptions	Likely overestimates risk
Exposure Assumptions	Human exposure parameter assumptions are US EPA default and are based on Reasonable Maximum Exposure, these are conservative for the majority of the population, but may be exceeded in some instances.	Could overestimate or underestimate risk

TABLE 10-15
SOME OF THE UNCERTAINTIES IN THE PROMONTORY RISK ASSESSMENT,
AND A QUALITATIVE ASSESSMENT OF THEIR POTENTIAL IMPACT ON THE RISK ASSESSMENT

Aspect Risk Assessment Process	Assumption	Effect on the Risk Assessment
Exposure Assumptions	Human diet and intake exposure assumptions are US EPA default and are based on Reasonable Maximum Exposure, these are conservative for the majority of the population, but may be exceeded in some instances.	Will overestimate risk in this risk assessment
Exposure Assumptions	Human diet and intake exposure assumptions are unlikely at this location in Utah because the soil and water are of a quality that could not produce the assumed levels of plant and animal food for the farmer diet.	Will overestimate risk in this risk assessment
Toxicological dose-response	Risk assessment uses US EPA and other regulatory dose-response factors that are designed to be health protective for the majority of the population. By definition, these are conservative for the majority of the population, but may be exceeded in some instances.	Likely overestimates risk
Risk and Hazard Calculations	These calculations will multiply the conservative uncertainty in the parameters presented above, and will increase the uncertainty.	Likely overestimates risk

END OF REPORT