

November 5, 2008

Chad Schlichtemeier
Wyoming Department of Environmental Quality
Air Quality Division / NSR Program Manager
Herschler Building
122 West 25th Street
Cheyenne, WY 82002



**Subject: Medicine Bow Fuel & Power LLC
Proposed Integrated Gasification and Liquefaction Plant
(PSD Air Quality Permit Application AP-5873)
Response to Public Comment/WDEQ Information Request**

Dear Mr. Schlichtemeier:

This letter is provided in response to a letter from Mr. James Nall, dated October 3, 2008, requesting additional information regarding the health risks associated with hazardous air pollutant (HAP) emissions from the proposed Medicine Bow Fuel & Power LLC (MBFP) industrial gasification and liquefaction (IGL) plant. Specifically, the WDEQ requested that MBFP submit the following additional information.

1. Revise the Tier 1 inhalation risk assessment to include all HAPs listed in the permit application (or provide justification for excluded HAPs) and to provide composite Hazard Quotient (HQ) values (referred to as Hazard Index, or HI values) for the chronic non-cancer and acute non-cancer risks, with a more refined assessment or an isopleth plot if either of the HI values exceed 1.0.
2. Search other sources of information to "more precisely" locate nearby residences.
3. Address the additive effects of non-inhalation risks relative to the proposed project.

These WDEQ requests referenced two public comment letters received in response to the WDEQ Permit Application Analysis for AP-5873: one letter from Earthjustice, dated August 1, 2008, and a letter from J. Johnson, dated August 4, 2008.

**MEDICINE BOW
EXHIBIT VV
PRE-HRG MEMO**

Copy Sent To: CH
Date: 1-5-09

Making Material Change

DEQ 001486

Revised Tier 1 Inhalation Risk Assessment

Attachment 1 to this letter is a report summarizing the revised Tier 1 Inhalation Risk Assessment for all HAP emissions from the proposed facility. Copies of modeling input and output files and output plots are on a CD enclosed with this letter.

The revised analysis addresses the following HAPs, as included in the permit application:

Acetaldehyde	Ethylbenzene	Propylene Oxide
Acrolein	Formaldehyde	Polycyclic Aromatic Hydrocarbons (PAH)
Benzene	Hexane	Toluene
1,3 Butadiene	Mercury	2,2,4-Trimethylpentane
Carbonyl Sulfide	Methanol	Xylene
Dichlorobenzene	Naphthalene	

It should be noted that the risks and hazards presented in this analysis are conservatively estimated (i.e., over-estimated) due to two key factors. First, note that emission rates for all HAPs in the MBFP permit application and in this risk analysis were calculated on the basis of uncontrolled HAP emissions. This is a very conservative estimate, due to the fact that oxidation catalyst will be employed for carbon monoxide (CO) and volatile organic compound (VOC) control on the three turbines, thus providing for 85-90% HAP control from the turbines. Second, the analysis considers maximum emission rates occurring during a facility startup year. Third, the short-term emission inventory used in the acute noncancer hazard analysis very conservatively assumes that all equipment at the facility is operating concurrently, although realistically, this will not occur. For example, the Black Start Generators will only run a short time during a startup year in order to startup the turbines, yet the analysis assumes all turbines and all Black Start Generators are operating at the same time.

As noted in your letter and recommended in the EPA's *Facility-Specific Assessment* guidance document (Volume 2 of the Air Toxics Risk Assessment Reference Library, EPA Document No. EPA-453-K-04-001B), cancer risks and noncancer hazards predicted for each individual HAP were summed in order to represent potential cumulative risks. This methodology is recommended based on a screening-level assumption that risks/hazards associated with individual chemicals in a mixture are additive. However, this approach is not recommended for acute noncancer inhalation hazards due to several complications cited by EPA which pertain only to acute noncancer analysis. More discussion on this point will be provided later in this section.

Detailed tables are provided in Attachment 1 listing all cancer risk estimates (expressed as "risk"), chronic noncancer hazards (expressed as HQs), and acute noncancer hazards (expressed as HQs). Table 1 provides a summary of these results. Note that summed HQ values are known as a Hazard Index (HI).

Table 1. Summed Maximum Cancer Risk Estimate and Noncancer Hazards for Proposed Facility. ¹

	Cancer Risk Estimate	Chronic Noncancer HI ²	Acute Noncancer HI ^{2,3}	
			(REL Basis)	(IDLH/10 Basis)
Sum, all HAPs at proposed facility	9x10 ⁻⁵	6x10 ⁻¹	4x10 ¹	5x10 ⁻²

Notes

1. As recommended in EPA's *Facility-Specific Assessment* guidance, one significant figure is used to express risk and hazard estimates.
2. The hazard Index (HI) is the summed value of the individual HQ for each HAP analyzed.
3. MBFP disagrees with the assertion that acute noncancer HQs should be expressed as a summed value, based on EPA guidance. Acute noncancer HQs are shown here, as calculated using two different types of acute dose-response values. The large disparity between these two values illustrates the points made in EPA guidance as to why summations are not advisable and shows why a summed HQ value (as calculated here) for acute noncancer hazards is meaningless.

Cancer Risk Estimate

As shown in Table 1, the maximum cancer risk estimate on a cumulative basis for all HAP at the proposed facility is 9 in 100,000. Note that this value represents the maximum cancer risk predicted at a single receptor point. A cumulative cancer risk contour plot is provided in Attachment 1, showing the extent of the varying levels of cumulative cancer risk up to this maximum value. The maximum cancer risks are located very near to the eastern fence line of the facility. Within 700 meters of the eastern fence line, the cancer risk drops to less than 1x10⁻⁶.

Chronic Noncancer Hazard

The maximum chronic noncancer HI shown in Table 1 is 0.6. According to the EPA guidance, an HQ value less than or equal to one indicates that noncancer effects are not likely to occur; therefore, the analysis shows that chronic noncancer effects are not likely to occur near the proposed facility. Per the request in WDEQ's October 3 letter and per EPA guidance, no further analysis has been conducted and a contour plot is not necessary.

Acute Noncancer Hazard

With regard to the acute noncancer HQ, interpretation of the two HI values shown in Table 1 is much less certain. MBFP is providing the summed result in response to WDEQ's specific request, but asserts (based on EPA's facility-specific guidance document) that both acute noncancer HI values are meaningless and do not represent a correct way to assess acute noncancer hazards in this screening analysis.

As shown in Table 1, the maximum acute noncancer HIs based on the REL and IDLH/10 acute AV data sets are 4×10^1 and 5×10^{-2} , respectively. This is a large range and is attributable to the different purposes and means of creating the REL and IDLH/10 AV data sets. A brief description of each AV data set is provided in Attachment 1, as well as in EPA's *Facility-Specific Assessment* guidance document. Clearly, the REL-based result is much greater than the "threshold value" of one, while the IDLH/10-based HI is much less than the value of one. Specifically, for the REL-based HI, the largest individual HQ is associated with acrolein, which has been assigned a dose-response value that is orders of magnitude less than REL values for many HAPs. Conversely, all IDLH/10 AVs fall within close range of each other, including that for acrolein. This illustrates the issues presented in the discussion above as to the complexity and possible inaccuracies in a summed acute noncancer HI value. An acrolein toxicological assessment is included in Attachment 2. This assessment compares acrolein concentrations from the Plant to the lowest concentrations at which effects of acrolein are actually perceived. Attachment 2 also discusses several potential inadequacies with regard to the derivation of the acrolein REL.

Detailed tables presented in Attachment 1 list the individual acute noncancer HQs and summed acute noncancer HI values for both the REL and IDLH/10 calculations.

Contour plots for both acute noncancer HI calculations (REL basis and IDLH/10 basis) have been provided in Attachment 1, showing the extent of the varying maximum HI levels for each receptor. As noted earlier, both REL and IDLH/10 data sets are incomplete with respect to the HAPs in this analysis. The acute noncancer HI contour plot based on the REL calculation considers the sum of individual HQs for which RELs exist. As a result, only eight HAPs are considered, because REL data does not exist for the remaining HAPs in this analysis. However, the acute noncancer HI contour plot based on the IDLH/10 AV data represents all HAPs: IDLH/10 AVs are available for all but three HAPs, and in order to create a contour plot based on a complete set of HAPs, values from the "TEEL-0" AV data set were substituted for the three missing HAPs to calculate individual HQs.

As mentioned earlier, MBFP asserts that summing acute noncancer HQs to determine the HI is technically indefensible. EPA states on pages 43-44 of its *Facility-Specific Assessment* guidance document that "Although this [summation of individual acute hazard quotients, or HQs, to calculate one acute hazard index, or HI] appears similar to the process for combining chronic HQs, the summing of acute HQs is complicated by several issues that do not pertain to chronic HQs," and "Risk assessors commonly

evaluate acute noncancer hazard using a variety of different acute values from different sources, and discuss the resulting hazard estimates considering only the purpose for which each value was developed...The significance of these HQs and [resulting] HIs would need to be considered in the context of the purpose of the risk assessment and the characteristics of the dose-response values, such as their purpose, averaging time, and health endpoints. EPA is working to provide a more comprehensive guidance on what benchmarks to rely upon and plan to develop an acute benchmark methodology.”

These statements acknowledge the complexity and variability inherent in acute noncancer hazard analysis. The acute noncancer HQ value for an individual HAP is calculated in a similar fashion to the chronic noncancer hazard in that a modeled concentration value is divided by a published reference factor associated with that HAP. In the case of chronic noncancer hazards, EPA has developed one set of recommended reference concentrations (RfC) to use in the hazard calculation. However, in the case of acute noncancer hazards, multiple sets of acute dose-response values (AV) have been created, representing varying time scales of exposure and varying short-term physical human responses. Two examples of AV sets are the “Reference Exposure Level” data set and the “IDLH/10” data set, both of which have been used to arrive at the acute noncancer HIs in Table 1. Each AV data set has been created for different purposes, meaning that some represent very mild effect levels, while some may represent more serious effects from exposure, and others may consider economics or technical feasibility when setting the dose-response data value. Additionally, these AV data sets are incomplete, such that MBFP cannot calculate an acute noncancer hazard for all HAPs using only one acute dose-response data set.

Although it is possible to use AVs from different data sets in order to calculate individual acute HQs for all HAPs in the analysis and then calculate a summed acute HI, it is obvious that there can be large discrepancies and errors resulting from such apples-and-oranges “mixing” of AVs. This is the basis of EPA’s cautionary statements on pages 43-44 of its guidance document against summing acute noncancer HQ values.

Despite these cautionary statements in EPA guidance of inherent errors, public commenters and the WDEQ requested that MBFP provide an HI for acute noncancer hazards. MBFP selected the reference exposure level (REL) and IDLH/10 AV data sets for the analysis, for three reasons. First, both data sets (with the exception of benzene in the REL data set) represent short-term effects and thus, correspond with the short-term concentrations provided by the air dispersion model. Second, the REL AV data set resulted in the most conservative analysis and the highest HI value when compared to the other data sets. Third, the IDLH/10 AV data set provided the most complete set of data for this analysis (data is missing for only three HAPs). Note that the REL AV data set is the most incomplete data set out of all AV data sets available from EPA for this analysis.

Nearby Residences

MBFP asserts that it has correctly identified the nearest residence (the Johnson residence) and noted it in the permit application. The Johnson residence is located on the west side of the Medicine Bow River where the Elk Mountain-Medicine Bow Road crosses the Medicine Bow River. The residence is located approximately 4.0 km (2.5 miles) south-southwest from the center of the proposed MBFP facility and about 3.7 km (2.3 miles) from the nearest point on the site's south boundary. As noted in the WDEQ letter, the permit application states on page 6-34 that the nearest residence is located approximately 3.3 km from the proposed facility. This estimate of 3.3 km (2.1 miles) appears to be slightly conservative because it places the residence approximately 0.3 km (0.20 miles) closer to the proposed facility than the residence actually is. Modeling plots showing predicted concentrations accurately depict the location of the Johnson residence.

Non-Inhalation Risks

Non-inhalation risk assessment, also referred to as multipathway risk assessment, is not specifically addressed in WDEQ major source or Prevention of Significant Deterioration (PSD) air modeling guidance, but is detailed in the EPA's "*Facility-Specific Assessment*" document that is referenced in the WDEQ modeling guidance. Relevant discussion points in this document are listed below.

- EPA states at the beginning of Chapter IV that "...multipathway risk assessment tools are less well developed..." and that EPA is continuing to revise guidance regarding facility-specific multipathway assessments.
- A multipathway risk assessment for HAPs is an assessment for "PB-HAP" compounds, meaning HAP compounds of concern for Persistence and Bioaccumulation. The terminology and focus on PB-HAPs are explained in Chapter II, Section 4 of the EPA document by stating that 'persistence' and 'bioaccumulation' are important chemical properties related to "persistence... in the environment (i.e., as determined by the HAP's half-life in air, water, soil, and sediment), and its potential to bioaccumulate...in plant or animal tissues (i.e., as determined by the steady-state ratio between environmental and tissue concentrations) and/or biomagnify in food chains." A list of PB-HAP compounds is provided as Exhibit 23, on page 78, of the EPA document.
- Acute exposure evaluations are generally not recommended for multipathway analyses because it is very unlikely that acute ingestion threats could exist under typical release conditions (Chapter IV, Sections 1.0 and 2.0, pages 77 and 79). Therefore, only chronic analyses focusing on longer-term emission rates are recommended.
- Only Tier 2 and Tier 3 methodologies are presented for multipathway analyses. EPA states that a Tier 1 methodology is under investigation and is

conceptualized as a simple lookup table or graph to identify threshold emission rates below which multipathway risks are not of concern. As stated by EPA, "the objective would be to allow a facility/source that emits small amounts of PB-HAP compounds to demonstrate that risk targets are met without the need for facility-specific modeling (as, for example, in a Tier 2 analysis)." (Chapter IV, Section 2, page 79)

The only PB-HAP compounds that will be emitted from the proposed MBFP facility are mercury and polycyclic aromatic hydrocarbons (PAH). (PAH is a subset of polycyclic organic matter and is comprised of compounds with multiple [fused] aromatic rings such as anthracene, benzo[a]pyrene, chrysene, naphthalene, and pyrene.) Estimated maximum annual emissions of mercury and PAH are 0.3 lbs and 41.9 lbs, respectively, which together account for less than 0.09 percent of the facility's total annual HAP emissions during the initial startup year and subsequent normal operation years. As stated earlier in this letter, facility HAP emissions were conservatively estimated on the basis of uncontrolled emissions. In reality, the oxidation catalyst on the turbines will provide for approximately 85-90% organic HAP control of turbine emissions; thus, expected maximum PAH emissions will be significantly reduced from these values. Note that high mercury removal is achieved by the carbon beds. It is reasonable to expect such low PB-HAP emission rates would fall under any thresholds established in a future Tier 1 multipathway analysis.

In summary, EPA guidance states that multipathway analyses are not recommended for acute exposure evaluations and that no Tier 1 approach currently exists. At this time, EPA believes that a future Tier 1 multipathway analysis approach would likely be based upon threshold emission rates. Due to the low PB-HAP emission rates and the lack of adequate guidance, MBFP does not believe that a multipathway risk analysis is appropriate for the proposed facility.

Conclusion

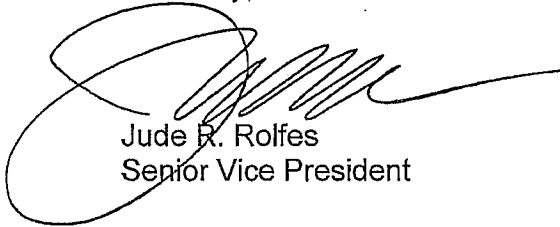
Based on additional HAP modeling and risk assessment, MBFP believes that it has adequately demonstrated that cancer, chronic noncancer, and acute noncancer risks are low. Risks near the facility fence line are low and risks in the vicinity of the nearest residence are even lower. This is true for individual HAPs and for conservative estimates of cumulative risks potentially associated with exposure to multiple HAPs. Although the acute noncancer risk and cumulative acute noncancer risk associated with acrolein appeared somewhat high near the facility based on one set of HQ and HI calculations, predicted acrolein concentrations are far below concentrations at which any human health effects are perceptible.

MBFP appreciates this opportunity to provide additional analysis to the WDEQ on issues raised during the public comment period. We hope this information is useful for

Chad Schlichtemeier
November 5, 2008
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you, and encourage you to contact us if you have any more questions or if you need clarification on any of the points raised in this letter.

Sincerely,



Jude R. Rolfes
Senior Vice President

cc: Andrew Keyfauver (WDEQ)
Robert Moss (DKRW)
Susan Bassett (URS)

Attachments: Attachment 1, Revised Risk Assessment for the Medicine Bow IGL
Plant
Attachment 2, Acrolein Toxicological Assessment

Enclosures: CD with model files

Attachment 1
Revised Risk Assessment by URS
for the
Medicine Bow IGL Plant

1.0 Introduction

This revised hazardous air pollutant (HAP) risk analysis for the Medicine Bow Industrial Gasification and Liquefaction (IGL) Plant (the Plant) has been developed in response to public comments submitted with regard to a WDEQ Permit Application Analysis for Permit Application AP-5873, dated June 19, 2008 (WDEQ 2008b). The previously submitted analysis considered risks for benzene, ethylbenzene, formaldehyde, hexane, methanol, toluene, and xylene. This revised report includes the following additions to the HAP risk assessment.

- Predicted concentrations for the following additional HAPs:
 - Acetaldehyde
 - Acrolein
 - 1,3 Butadiene
 - Carbonyl Sulfide
 - Dichlorobenzene
 - Mercury
 - Naphthalene
 - Propylene Oxide
 - Polycyclic Aromatic Hydrocarbons (PAH)
 - 2,2,4-Trimethylpentane

- Multi-pollutant summation of the following:
 - Risk_T – total individual cancer risk
 - HI_A – acute hazard index.

- Isopleths (Figures 3, 4, and 5) showing cumulative risk contours near the Plant and the residence located nearest to the Plant.

2.2 Plant Layout and Source Location

Figure 2 provides a close-up view of the Plant layout. The emission source labels included in Figure 2 correspond with the source IDs in Tables 1 and 2. As mentioned earlier, the complete receptor grid for this analysis is shown in Figure 1.

Table 1. - Short-Term HAP Emissions (grams per second)

Source ID *	1,3-Butadiene	Acetaldehyde	Acrolein	2,2,4-Trimethylpentane	Benzene	Carbonyl Sulfide	Dichlorobenzene	Ethyl Benzene	Formaldehyde	Hexane	Mercury	Methanol	Naphthalene	PAH	Propylene Oxide	Toluene	Xylene
CTG1	4.25E-05	3.96E-03	6.33E-04	0.00E+00	1.19E-03	0.00E+00	0.00E+00	3.17E-03	7.02E-03	0.00E+00	1.25E-06	0.00E+00	1.29E-04	2.18E-04	2.87E-03	1.29E-02	6.33E-03
CTG2	4.25E-05	3.96E-03	6.33E-04	0.00E+00	1.19E-03	0.00E+00	0.00E+00	3.17E-03	7.02E-03	0.00E+00	1.25E-06	0.00E+00	1.29E-04	2.18E-04	2.87E-03	1.29E-02	6.33E-03
CTG3	4.25E-05	3.96E-03	6.33E-04	0.00E+00	1.19E-03	0.00E+00	0.00E+00	3.17E-03	7.02E-03	0.00E+00	1.25E-06	0.00E+00	1.29E-04	2.18E-04	2.87E-03	1.29E-02	6.33E-03
GHEAT1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	0.00E+00	3.11E-06	0.00E+00	1.95E-04	4.67E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.82E-06	0.00E+00
GHEAT2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	0.00E+00	3.11E-06	0.00E+00	1.95E-04	4.67E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.82E-06	0.00E+00
GHEAT3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	0.00E+00	3.11E-06	0.00E+00	1.95E-04	4.67E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.82E-06	0.00E+00
GHEAT4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	0.00E+00	3.11E-06	0.00E+00	1.95E-04	4.67E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.82E-06	0.00E+00
GHEAT5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	0.00E+00	3.11E-06	0.00E+00	1.95E-04	4.67E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.82E-06	0.00E+00
BSG1	6.56E-04	2.03E-02	1.26E-02	6.14E-04	5.21E-04	0.00E+00	0.00E+00	0.00E+00	1.30E-01	2.73E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.00E-03	4.52E-04
BSG2	6.56E-04	2.03E-02	1.26E-02	6.14E-04	5.21E-04	0.00E+00	0.00E+00	0.00E+00	1.30E-01	2.73E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.00E-03	4.52E-04
BSG3	6.56E-04	2.03E-02	1.26E-02	6.14E-04	5.21E-04	0.00E+00	0.00E+00	0.00E+00	1.30E-01	2.73E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.00E-03	4.52E-04
FIREPUMP	1.90E-05	3.73E-04	4.99E-05	0.00E+00	4.53E-04	0.00E+00	0.00E+00	0.00E+00	5.73E-04	0.00E+00	0.00E+00	0.00E+00	4.12E-05	0.00E+00	1.25E-03	1.99E-04	0.00E+00
AB	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-05	0.00E+00	9.78E-06	0.00E+00	6.11E-04	1.47E-02	0.00E+00	0.00E+00	4.97E-06	0.00E+00	0.00E+00	2.77E-05	0.00E+00
REGH	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.59E-06	0.00E+00	3.19E-06	0.00E+00	1.99E-04	4.79E-03	0.00E+00	0.00E+00	1.62E-06	0.00E+00	0.00E+00	9.04E-06	0.00E+00
REAH	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-06	0.00E+00	1.85E-06	0.00E+00	1.15E-04	2.77E-03	0.00E+00	0.00E+00	9.38E-07	0.00E+00	0.00E+00	5.23E-06	0.00E+00
HGT	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.76E-07	0.00E+00	3.29E-07	0.00E+00	2.06E-05	4.94E-04	0.00E+00	0.00E+00	1.67E-07	0.00E+00	0.00E+00	9.32E-07	0.00E+00
T _A through T _K	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.52E-02	0.00E+00	0.00E+00	1.09E-03	0.00E+00	1.42E-02	0.00E+00	6.87E-02	0.00E+00	0.00E+00	0.00E+00	1.63E-02	4.60E-03
V1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-01	6.72E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.29E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Total	2.11E-03	7.38E-02	3.97E-02	1.84E-03	2.48E-03	6.72E-03	3.07E-05	1.06E-02	4.14E-01	6.11E-02	3.75E-06	2.95E-01	4.36E-04	6.54E-04	9.86E-03	5.83E-02	2.49E-02

* These sources are described in the PSD permit application (MABFP 2007).

Table 2 - Annual HAP Emissions (grams per second)

Source ID *	1,3-Butadiene	Acetaldehyde	Acrolein	2,2,4-Trimethylpentane	Benzene	Carbonyl Sulfide	Dichlorobenzene	Ethyl Benzene	Formaldehyde	Hexane	Mercury	Methanol	Naphthalene	PAH	Propylene Oxide	Toluene	Xylene
CTG1	3.93E-05	3.65E-03	5.85E-04	0.00E+00	1.10E-03	0.00E+00	0.00E+00	2.92E-03	6.48E-03	0.00E+00	1.25E-06	0.00E+00	1.19E-04	2.01E-04	2.65E-03	1.19E-02	5.85E-03
CTG2	3.93E-05	3.65E-03	5.85E-04	0.00E+00	1.10E-03	0.00E+00	0.00E+00	2.92E-03	6.48E-03	0.00E+00	1.25E-06	0.00E+00	1.19E-04	2.01E-04	2.65E-03	1.19E-02	5.85E-03
CTG3	3.93E-05	3.65E-03	5.85E-04	0.00E+00	1.10E-03	0.00E+00	0.00E+00	2.92E-03	6.48E-03	0.00E+00	1.25E-06	0.00E+00	1.19E-04	2.01E-04	2.65E-03	1.19E-02	5.85E-03
GHEAT1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-07	0.00E+00	1.78E-07	0.00E+00	1.11E-05	2.67E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-07	0.00E+00
GHEAT2	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-07	0.00E+00	1.78E-07	0.00E+00	1.11E-05	2.67E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-07	0.00E+00
GHEAT3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-07	0.00E+00	1.78E-07	0.00E+00	1.11E-05	2.67E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-07	0.00E+00
GHEAT4	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-07	0.00E+00	1.78E-07	0.00E+00	1.11E-05	2.67E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-07	0.00E+00
GHEAT5	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-07	0.00E+00	1.78E-07	0.00E+00	1.11E-05	2.67E-04	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.03E-07	0.00E+00
BSC1	2.70E-05	8.44E-04	5.19E-04	2.52E-05	2.14E-05	0.00E+00	0.00E+00	0.00E+00	5.33E-03	1.12E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.12E-05	1.86E-05
BSC2	2.70E-05	8.44E-04	5.19E-04	2.52E-05	2.14E-05	0.00E+00	0.00E+00	0.00E+00	5.33E-03	1.12E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.12E-05	1.86E-05
BSC3	2.70E-05	8.44E-04	5.19E-04	2.52E-05	2.14E-05	0.00E+00	0.00E+00	0.00E+00	5.33E-03	1.12E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.12E-05	1.86E-05
FIREPUMP	1.08E-06	2.13E-05	2.56E-06	0.00E+00	2.59E-05	0.00E+00	0.00E+00	0.00E+00	3.27E-05	0.00E+00	0.00E+00	0.00E+00	2.35E-06	0.00E+00	0.00E+00	4.12E-05	1.86E-05
AB	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.71E-05	0.00E+00	9.78E-06	0.00E+00	6.11E-04	1.47E-02	0.00E+00	0.00E+00	4.97E-06	0.00E+00	0.00E+00	2.77E-05	0.00E+00
REGH	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.59E-06	0.00E+00	3.19E-06	0.00E+00	1.99E-04	4.79E-03	0.00E+00	0.00E+00	1.63E-06	0.00E+00	0.00E+00	9.04E-06	0.00E+00
REAH	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.23E-06	0.00E+00	1.85E-06	0.00E+00	1.15E-04	2.77E-03	0.00E+00	0.00E+00	9.38E-07	0.00E+00	0.00E+00	5.23E-06	0.00E+00
HGT	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.76E-07	0.00E+00	3.29E-07	0.00E+00	2.06E-05	4.94E-04	0.00E+00	0.00E+00	1.67E-07	0.00E+00	0.00E+00	9.12E-07	0.00E+00
T_A through T_K	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.52E-02	0.00E+00	0.00E+00	1.09E-03	0.00E+00	1.42E-02	0.00E+00	6.87E-02	0.00E+00	0.00E+00	0.00E+00	1.63E-02	4.60E-03
V1	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.27E-01	6.72E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.26E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Total	2.00E-04	1.35E-02	3.31E-03	7.56E-05	2.46E-01	6.72E-03	1.60E-05	9.85E-03	3.65E-02	3.83E-02	3.75E-06	2.95E-01	3.67E-04	6.03E-04	8.07E-03	5.22E-02	2.22E-02

* These sources are described in the PSD permit application (MBFP 2007).

3.0 Modeling Approach

HAP modeling was performed using the American Meteorological Society (AMS) / EPA Regulatory Modeling Program (AERMOD) version 07026. In order to be consistent with the previous risk assessment analysis performed by the WDEQ, the receptor grid, building downwash parameter values, meteorology, and stack parameter data sets provided by the WDEQ were used in this analysis. The model predicted maximum 1-hour and annual averaged concentrations at each receptor.

For additional details on HAP modeling input data and techniques, please refer to the PSD permit application (MBFP 2007) and the WDEQ Permit Application Analysis (WDEQ 2008b).

4.0 Tier 1 Inhalation Risk Assessment

This Tier 1 Inhalation Risk Assessment follows guidance established by EPA in its *Facility-Specific Assessment*, Volume 2 of the Air Toxics Risk Assessment Reference Library (EPA Document No. EPA-453-K-04-001B, April 2004). The risk assessment procedure integrates exposure and toxicity assessments to estimate risks from HAP emissions. Dispersion modeling is used to estimate exposure concentrations, which are divided by applicable dose-response values to generate a risk estimate.

Cancer risk is expressed in numerical terms (e.g., 1×10^{-6} , or 1 in a million) as the incremental chance an individual will develop cancer in their lifetime as a result of the long-term exposure.

Noncancer hazard is expressed as a Hazard Quotient (HQ), which is the ratio of the estimated exposure to the noncancer dose-response value. Noncancer health effects data are usually available only for individual HAPs within a mixture. According to EPA's *Facility-Specific Assessment* guidance: "In these cases, the individual HQs can be summed together to calculate a multi-pollutant hazard index (HI) . . ." The guidance cautions against calculating an HI value for acute noncancer hazards, based on complications with acute noncancer dose-response values that do not pertain to chronic HQs. Specifically, EPA notes that:

1. "Acute dose-response values have been developed for purposes that vary more widely than chronic values. Some sources of acute values define exposures at which adverse effects actually occur, while other sources develop only no-effect acute values."
2. "Some acute values are expressed as concentration-time matrices, which others are expressed as single concentrations for a set exposure duration."
3. "Some acute values may specifically consider multiple exposures, whereas others consider exposure as a one-time event."

4. "Some sources of acute values are intended to regulate workplace exposures, assuming a population of healthy workers (i.e., without children, seniors, or other sensitive individuals). Such occupational values may also consider cost and feasibility."

Notwithstanding this caution against calculating HI values for acute noncancer hazards, the HQ or HI value effectively normalizes risk relative to a specific reference level. EPA's *Facility-Specific Assessment* guidance states the following [bold text appears in EPA's guidance]:

"Based on the definition of the RfC, a HQ less than or equal to one indicates that adverse noncancer effects are **not likely to occur**. With exposures increasingly greater than the RfC, (i.e., HQs increasingly greater than 1), the **potential for adverse effects increases**. However, note the following: **The HQs should not be interpreted probabilistically because the overall chance of adverse effects may not increase linearly as exposures exceed the RfC.**" [EPA, 2004, p.41]

4.1 Chronic Risk Assessment

The annual average emission scenario was modeled for the chronic (long-term) exposure risk assessment. There were two sub-analyses completed for this assessment, long-term cancer risk exposure and a chronic noncancer exposure.

4.1.1 (Long-term) Cancer Risk

The long-term cancer risk assessment estimates the potential cumulative cancer risk for all pollutants classified as carcinogens, with a cancer risk factor listed in the Prioritized Chronic Dose-Response Values Table (EPA Table 1, 2007). IURs are included in EPA's Table 1. In this document, Table 3 lists the modeled concentrations (EC_L), the Inhalation Unit Risk Estimate (IUR), and the calculated maximum cancer risk for each HAP, as well as the maximum cumulative (all HAPs) cancer risk at a discrete receptor. HAPs with no cancer risk factor are noted as "NA" in the IUR column.

The cancer risk is calculated as the product of the modeled concentration and the IUR:

$$\text{Risk} = EC_L \times IUR$$

This individual cancer risk is expressed as an upper-bound risk of contracting cancer over a lifetime.

Table 3 - Cancer Risk Analysis			
HAP	Factors/Risk		
	EC _L (µg/m ³)	IUR [1/(µg/m ³)] ¹	Maximum Risk
Acetaldehyde	0.0069	0.0000022	1.52E-08
Acrolein	0.0041	NA	--
Benzene	11.3109	0.0000078	8.82E-05
1,3-Butadiene	0.0002	0.00003	6.60E-09
Carbonyl Sulfide	0.3343	NA	--
Dichlorobenzene	0.0001	0.000011	7.70E-10
Ethyl benzene	0.0071	NA	--
Formaldehyde	0.0465	5.50E-09	2.56E-10
Hexane	0.1242	NA	--
Methanol	11.3214	NA	--
Mercury	0.0000	NA	--
Naphthalene	0.0002	0.000034	5.78E-09
PAH ²	0.0002	0.0011	2.53E-07
Propylene Oxide	0.0035	0.0000037	1.31E-08
Toluene	0.1035	NA	--
2,2,4-Trimethylpentane	0.0002	NA	--
Xylene	0.0294	NA	--
Maximum Cumulative Risk³	NA	NA	8.82E-05

1. IURs are published in EPA's Table 1, 2007.

2. The PAH IUR value in this table equals the Benzo-Pyrene IUR value in EPA Table 1, per EPA guidance in the *Facility-Specific Assessment* (EPA 2004).

3. The highest pollutants risk summation (cumulative total) at a specific receptor

Figure 3 presents the cumulative cancer risk contour plot for all receptors in the analysis.

4.1.2 Chronic Noncancer Risk

HQs were calculated for the chronic noncancer analysis using Reference Concentration Levels (RfCs) obtained from the Prioritized Chronic Dose-Response Values Table (EPA Table 1, 2007). Individual HQs for each HAP are calculated by dividing the estimate of continuous (long-term) inhalation exposure, or modeled concentration (EC_C), by the appropriate RfC value:

$$HQ = EC_C \div RfC$$

Individual HQs, when summed together, result in the multi-pollutant Hazard Index (HI) which indicates the hazard quotient for the mixture. As stated earlier, HQ and HI values less than or equal to one indicate that adverse noncancer effects are not likely to occur.

Note, HAPs with no listed RfC value are noted as "NA" in the RfC column of Table 4.

Table 4 – Chronic Noncancer Risk Analysis			
HAP	EC_c (µg/m³)	RfC (µg/m³)	HQ
Acetaldehyde	0.0069	9	0.00077
Acrolein	0.0041	0.02	0.20650
Benzene	11.3109	30	0.38
1,3-Butadiene	0.0002	2	0.00011
Carbonyl Sulfide	0.3343	NA	--
Dichlorobenzene	0.0001	800	0.00000
Ethyl benzene	0.0071	1,000	0.00001
Formaldehyde	0.0465	9.8	0.0047
Hexane	0.1242	700	0.00018
Mercury	0.0000	0.30	--
Methanol	11.3214	4,000	0.003
Naphthalene	0.0002	3	0.00006
PAH	0.0002	NA	--
Propylene Oxide	0.0035	30	0.00012
Toluene	0.1035	5,000	0.00002
2,2,4-Trimethylpentane	0.0002	NA	--
Xylene	0.0294	100	0.00029
HI (Cumulative HQ)	NA	NA	0.59266

As shown in Table 4, the HI for all HAPs with a valid RfC was below 1; therefore, chronic noncancer effects are not likely to occur. A chronic noncancer HI plot is not presented with this analysis, due to this result.

4.2 Acute Noncancer Risk Assessment Dose-Response Values

Results from the maximum short-term emission scenario were used to assess acute noncancer risks. Similar to chronic noncancer hazard analysis, acute noncancer HQ values for each HAP were calculated by dividing the estimate of inhalation exposure (in this case, short-term exposure, or EC_{ST}), by the appropriate reference value, which in this case is the acute dose-response value, or AV:

$$HQ_A = EC_{ST} \div AV$$

The acute dose-response values (AVs) for each HAP are provided in the Acute Dose-Response Values for Screening Risk Assessments Table (EPA Table 2, 2007). A notable complexity in the acute noncancer hazard analysis is the fact that multiple sets of AV data exist and can be used to generate a wide distribution of acute noncancer HQs for any one HAP. EPA Table 2 presents the following sets of AV data:

AEGL-1 (1-hr)	MRL
AEGL-1 (8-hr)	REL
AEGL-2 (1-hr)	IDLH/10
AEGL-2 (8-hr)	TEEL-0
ERPG-1	TEEL-1
ERPG-2	

The full titles of each data set, and brief descriptions, are provided in the EPA *Facility-Specific Assessment* guidance document. Each data set is developed to match specific timescales of exposure and desired effect levels (e.g., no-effect or mild reversible effects). Therefore, the basis for each AV set is quite different, and should be taken into consideration when performing the acute noncancer hazard assessment.

For this analysis, the Acute Reference Exposure Level (REL), 1/10 Levels Imminently Dangerous to Life and Health (IDLH/10), and Temporary Emergency Exposure Limits (TEEL-0) data sets were used as AV values to calculate two sets of acute noncancer HQ values. These reference levels are explained below. Text marked with an asterisk (*) was taken directly from EPA's *Facility-Specific Assessment* document (EPA, 2004).

The REL AV data set was chosen as a conservative measure, due to the higher ECST values generated with this data set. However, the REL data set is the most incomplete data set and is missing values for many of the emitted HAPs. The IDLH/10 AV data set was selected because it provided the most complete data set for the emitted HAPs.

Acute Reference Exposure Levels (REL values)*

The REL is a chemical-specific acute exposure level estimate for noncancer effects (with an uncertainty spanning an order of magnitude) that is not likely to cause adverse effects in a human population after acute exposure to inhaled chemicals other than criteria air pollutants. RELs are developed by the California Office of Environmental Health Hazard Assessment (OEHHA), and more information about RELs is available at their website (EPA 2004).

1/10 Levels Imminently Dangerous to Life and Health (IDLH/10 Values)*

IDLH values are exactly as described, and are intended to trigger immediate evacuation of work areas. However, levels one-tenth of the IDLH tend to be similar to mild effect levels such as AEGL-1s, and are included with EPA/OAQPS's [Office of Air Quality Planning and Standards] acute values table on this basis. The IDLH/10 has been used commonly as the level of concern (superceded by AEGL values) in the Agency's emergency planning programs pursuant to the Emergency Planning and Community Right-to-Know Act (EPCRA) and Section 112r of the

Clean Air Act. IDLHs are developed by the National Institute for Occupational Safety and Health (NIOSH) (EPA, 2004).

Temporary Emergency Exposure Limits (TEEL-0) Values (EPA Table 2, 2007)

TEEL-0 values are US Department of Energy temporary emergency exposure limits for no effects for 1-hour exposure. Values for mild, transient effects were also established and are referred to as TEEL-1. They are derived according to a tiered, formula-like methodology and do not undergo peer review. EPA does not recommend their use as the basis for regulatory decision-making, but in some cases they are the only acute dose-response values available for certain chemicals.

4.2.1 Acute Noncancer HQs and the HI value

As noted in the beginning of Section 4.0 of this revised HAP Risk Assessment Report, EPA cautions against calculating an HI value for acute noncancer hazards (HI_A), based on possible differences between AV data within a reference set and certain differences between AV data sets. However, in response to public comment and WDEQ request, HI_A values have been calculated. Two HI_A values are presented; one from the REL-based HQ_A s, and the other primarily from the IDLH/10-based values, with substitution as necessary for missing data (in order to provide a calculation from all HAPs) from the TEEL-0 AV data set.

For purposes of this analysis, it is assumed that acute noncancer HI_A values follow the same threshold rule as chronic noncancer HI values: values less than or equal to one indicate that adverse noncancer effects are unlikely, while values greater than one indicate increased potential for adverse noncancer effects.

Table 5 presents the maximum modeled 1-hour averaged concentration (EC_{ST}), the REL and IDLH/10 acute noncancer dose-response levels (AV), and the calculated acute noncancer risk (HQ_A) for each HAP. For those HAPs with no corresponding REL AV, "NA" has been entered in the REL AV column. For the three HAPs with no corresponding IDLH/10 AV, the corresponding TEEL-0 AV has been substituted so that an HQ_A value can be calculated.

As shown in the bottom row of Table 5, this approach results in one acute noncancer HI_A value greater than one (based on REL values), and one acute HI_A value less than one (based on IDLH/10 values). The disparity between these two results highlight the need for caution when assessing acute noncancer risks and emphasizes the cautionary statements in the EPA facility-specific guidance. Note that the IDLH/10 HI is based on the most complete set of AV data.

Figures 4 and 5 present HI_A contour plots illustrating the maximum 1-hour HI_A s at all receptor points. Figure 4 presents the REL-based HI_A values, and Figure 5 presents the IDLH/10-based HI_A values (using the TEEL-0 substitutions as described above).

Table 5 - Acute Noncancer Risk Analysis					
HAP	EC_{ST} (µg/m³)	REL AV (µg/m³)	REL HQ	IDLH/10 AV (µg/m³)	IDLH/10 HQ
Benzene	254.9806	1300	0.1961	160000	0.0016
Ethyl benzene	0.2431	NA	--	350000	6.94E-07
Formaldehyde	75.0750	94	0.7987	2500	0.0300
Methanol	254.1677	28000	0.0091	790000	0.0003
Hexane	3.4552	NA	--	390000	8.86E-06
Toluene	3.6410	37000	0.0001	190000	1.92E-05
1,3-Butadiene	0.3795	NA	--	440000	8.62E-07
Acetaldehyde	11.8796	NA	--	360000	3.30E-05
Acrolein	7.3022	1.90E-01	38.4327	460	0.0159
2,2,4-Trimethylpentane	0.3551	NA	--	350000 ³	1.01E-6 ³
Carbonyl Sulfide	7.5432	NA	--	3100 ³	0.0024 ³
Dichlorobenzene	0.0023	NA	--	90000	2.53E-08
Naphthalene	0.0188	NA	--	130000	1.44E-07
PAH ¹	0.0065	NA	--	15000 ³	0.0005
Propylene Oxide	0.5713	3100	0.0002	95000	6.01E-06
Mercury	0.00004	2	2.22E-05	NA	NA
Xylene	1.0279	22000	4.67E-05	390000	2.64E-06
HI_A (Cumulative HQ_A)²			39.4370		0.0508³

1. Polycyclic aromatic hydrocarbon (PAH) reference levels are based on the TEEL-0 value for pyrene.
2. EPA guidance cautions against summing acute noncancer HQs to assess an acute noncancer HI.
3. TEEL-0 AV values are substituted for 2,2,4 TMP, COS, and PAH in order to assess risk for all emitted HAPs.

5.0 References

EPA, 2004. Air Toxics Risk Assessment Reference Library, Volume 2, Facility-Specific Assessment. http://www.epa.gov/ttn/fera/data/risk/vol_2/volume_2-april_2004.pdf.

EPA Table 1, 2007. Prioritized Chronic Dose-Response Values. <http://www.epa.gov/ttn/atw/toxsource/summary.html>.

EPA Table 2, 2007. Acute Dose-Response Values for Screening Risk Assessments Table. <http://www.epa.gov/ttn/atw/toxsource/summary.html>.

MBFP, 2007. Medicine Bow Fuel & Power LLC Prevention of Significant Deterioration Permit Application dated December 31, 2007.

WDEQ, 2008a. Wyoming Department of Environmental Quality/Air Quality Division Guidance for Submitting Major Source/PSD Modeling Analyses. http://deq.state.wy.us/AQD/downloads/cbm/MajorSrc_ModelGuide_31Jan08.doc.

WDEQ, 2008b. Wyoming Department of Environmental Quality Permit Application Analysis dated June 19, 2008.

WDEQ, 2008c. Wyoming Department of Environmental Quality letter to Medicine Bow Fuel & Power LLC dated October 3, 2008.

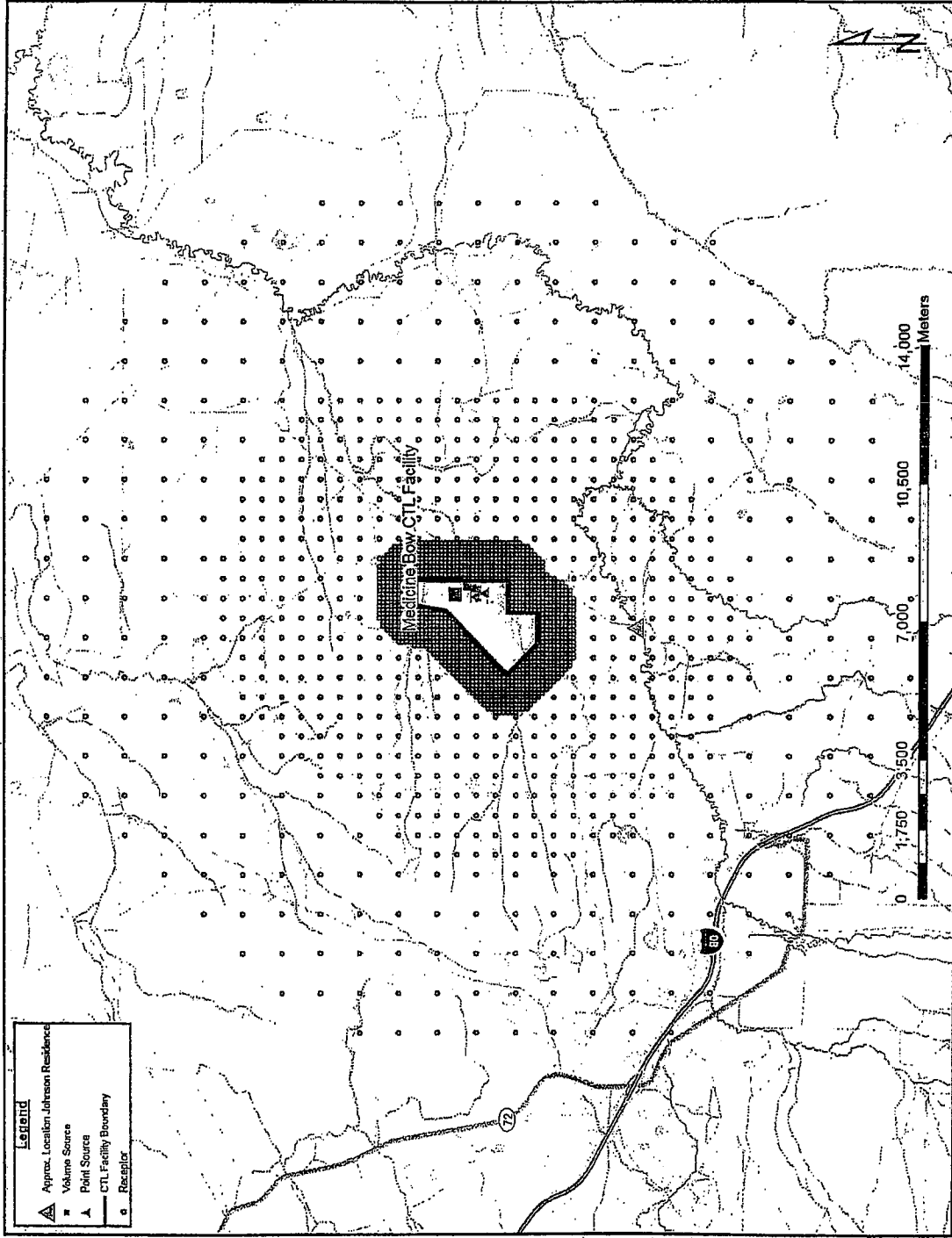


Figure 1. Plant Location and Receptors

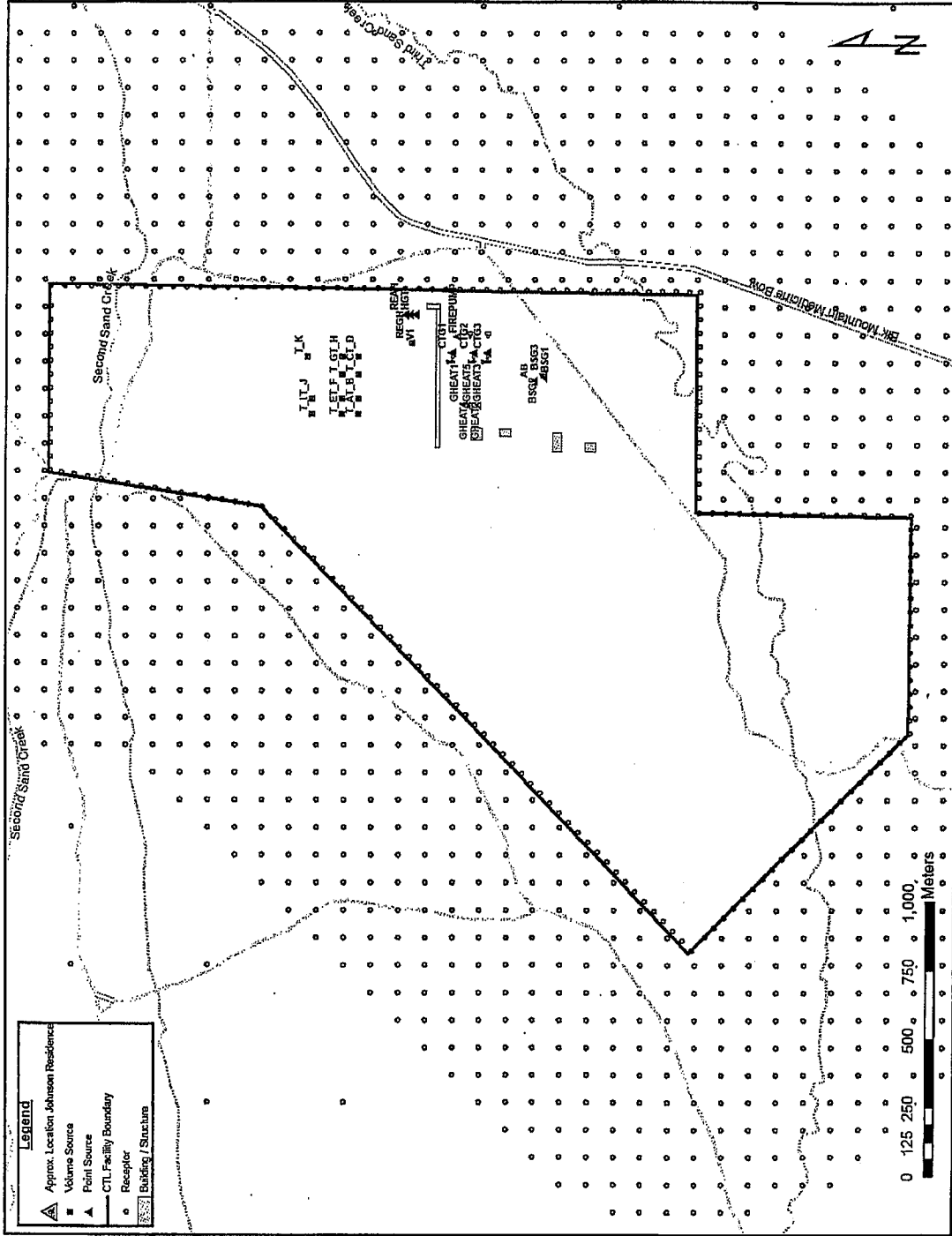


Figure 2. Plant Layout

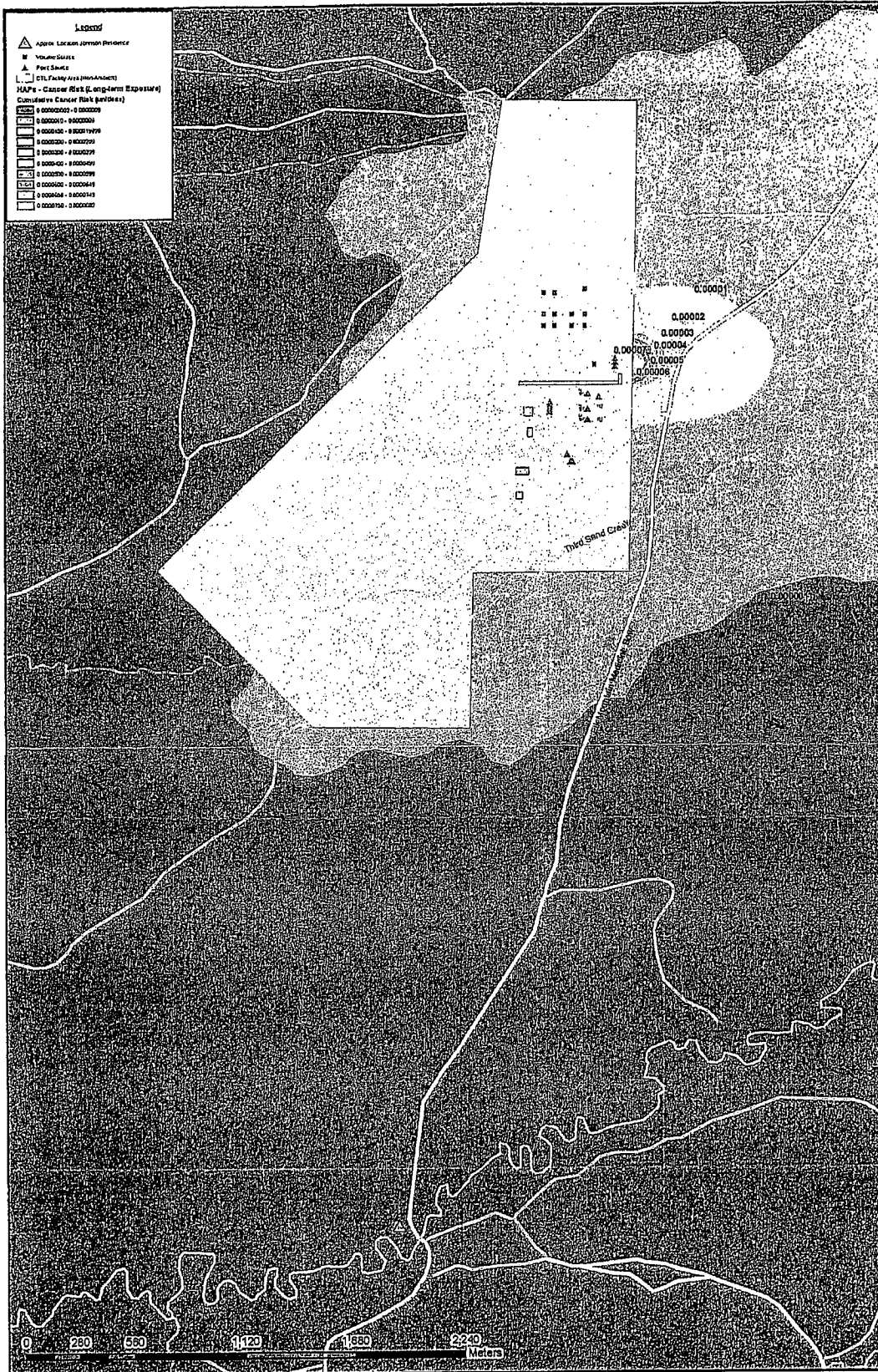


Figure 3. Cumulative Cancer Risk

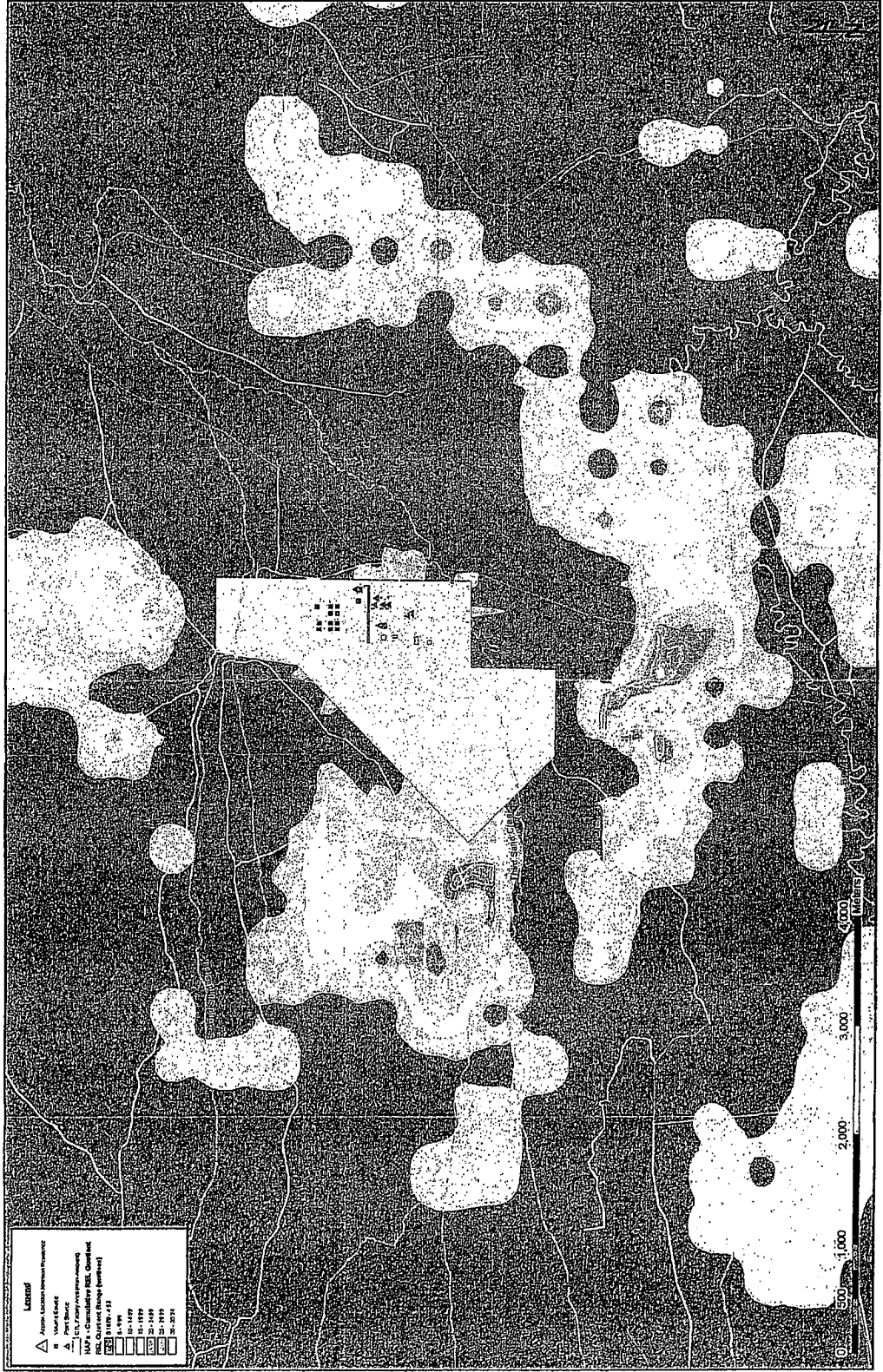


Figure 4. REL-Based Acute Noncancer Hazard Index (HL)

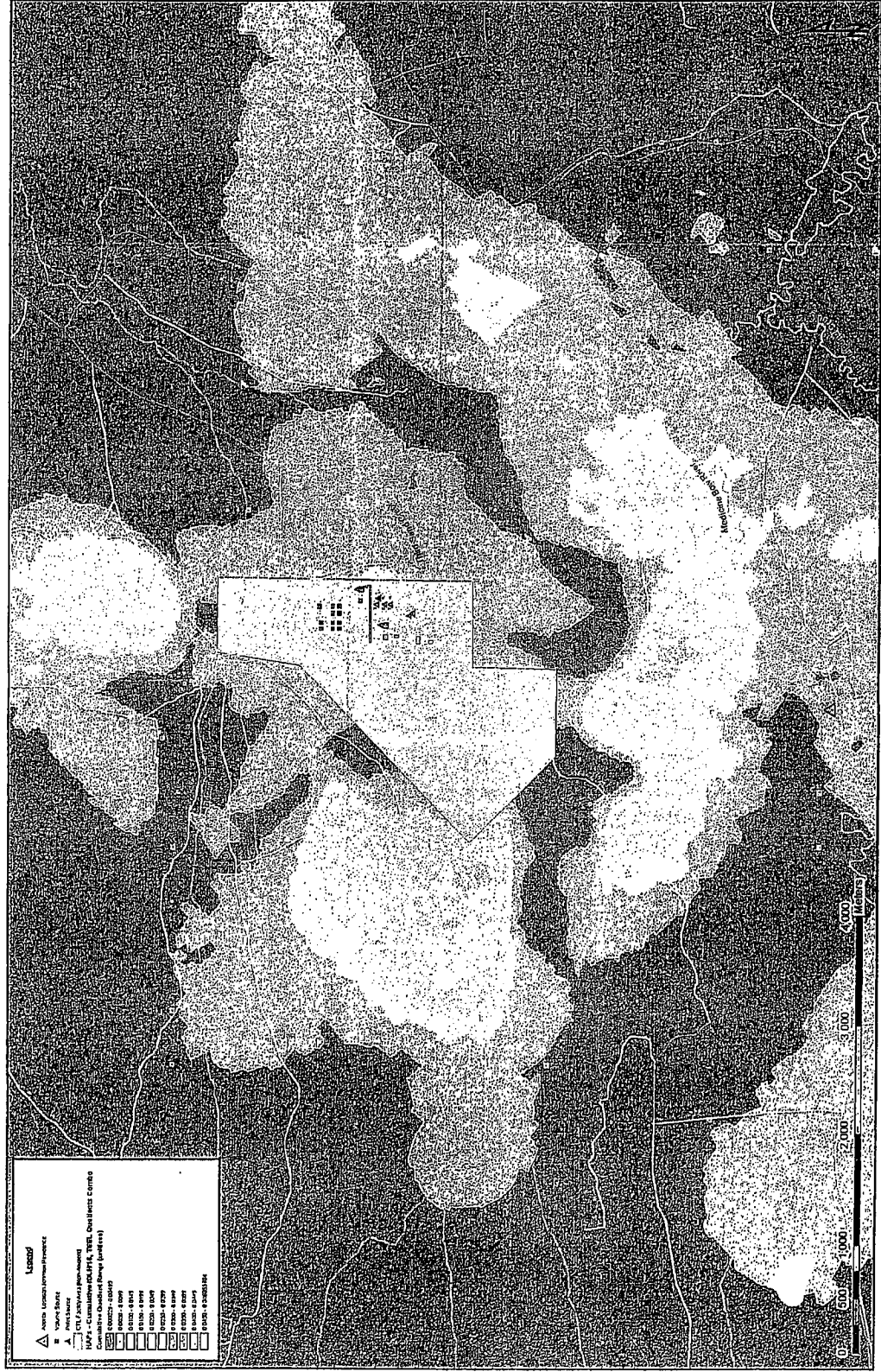


Figure 5. IDLH/10-Based Acute Noncancer Hazard Index (HI)

Attachment 2

Acrolein Toxicological Assessment by URS Toxicologist Jenifer Heath PhD

1.0 Introduction

This overview summarizes information related to short-term effects of acrolein. The purpose of this overview is to provide additional context for the acute hazard quotient (HQ) of 38, obtained based on a maximum short-term concentration of $7.3 \mu\text{g}/\text{m}^3$ ($0.0073 \text{ mg}/\text{m}^3$ or approximately 0.003 ppm), as predicted by the hazardous air pollutant (HAP) modeling described in Attachment 1.

2.0 Acrolein Effects

Acrolein is highly reactive, binding irreversibly to biologically important molecules in tissues. This can cause both direct adverse effects as well as secondary or indirect effects. The main means by which acrolein causes adverse effects is point-of-contact irritation (Agency for Toxic Substances and Disease Registry [ATSDR] 2007) on mucous membranes, including the nasal epithelium and eyes. Typical effects include nasal and throat irritation, eye irritation, and lacrimation (tearing of the eyes). Acrolein gas affects nasal passages through inhalation and subsequent contact with nasal and throat tissues. Acrolein gas affects eyes through dermal or direct contact.

Children are not more susceptible to the adverse effects of acrolein than adults. Furthermore, acrolein effect levels on mucous membranes occur at similar exposure levels in humans and laboratory animals. This is one reason that most "regulatory" levels are based on data from animal models. This summary focuses on human effect levels.

3.0 Acrolein Exposure

As described in the accompanying letter and in Attachment 1, acrolein exposure is conservatively estimated by modeling maximum acrolein emissions that would not actually occur. Maximum hourly emissions were calculated based on the assumption that all equipment at the Plant operates concurrently. The largest sources of acrolein at the Medicine Bow Fuel & Power LLC (MBFP) industrial gasification and liquefaction plant (the Plant), are the three Black Start Generators (BSG). These generators operate only during plant startup and, thereafter, for short (generally less than one hour) periods of time for testing and maintenance purposes. Total BSG operating time for each generator during the worst-case, initial startup year is 360 hours. However, it is extremely unlikely that the BSGs would operate continuously for 360 hours (15 days). Maximum hourly acrolein emissions are conservatively estimated to be 0.0397 grams/second (g/s). Average annual acrolein emissions are conservatively estimated to be only 0.00331 g/s (or less than 9 percent of maximum hourly emissions). Typical exposure to acrolein will

be at concentrations far less than those described in this document as the maximum short-term exposure concentration.

4.0 Dose-Response Values

As described in the attached letter, dose-response values for acrolein vary greatly. While the immediately dangerous to life and health divided by 10 (IDLH/10) HQ for acrolein is less than 1, the recommended exposure limit (REL)-based HQ is approximately 38. The high REL-based HQ is due to the extremely low acute California Environmental Protection Agency (CalEPA) REL for acrolein of $0.19 \mu\text{g}/\text{m}^3$.

4.1 California's REL

CalEPA's REL of $0.19 \mu\text{g}/\text{m}^3$ acrolein is based on a study by Darley et al that was published in 1960. Review of the original literature is outside the scope of this overview. However, none of the other authoritative reviews considered herein cites to the Darley et al study. CalEPA 1999 does not give sufficient information about the study to provide comfort that the results form a sound basis for decision making. For example, although we know that the study included 36 healthy human volunteers who wore carbon filter respirators, we do not know their gender(s) and we do not know whether controls were used (and in particular whether there was a control group who also wore the respirators, which may be uncomfortable and unfamiliar to most people). Furthermore, we do not know whether lower concentrations were used that caused no adverse effects; a no observed adverse effect level (NOAEL)-based REL would have been much higher than a Lowest observed adverse effect level (LOAEL)-based REL because of the uncertainty factors applied. In addition, to extrapolate from the 5-minute study duration to a 1-hour REL the effects level concentration was reduced by a factor of about 5. This appears to be overly conservative because there is evidence in other studies that eye effects plateau with increasing duration of exposure to the same concentration. CalEPA RELs are developed following specific guidance. Interpretation of an HQ based on the acrolein REL should be tempered by an understanding of the basis of this specific REL.

4.2 Human Health Studies

Table 1 presents information related to effects of short-term exposure of humans to acrolein in air. Much of the literature related to short-term exposure of humans to acrolein is anecdotal and/or does not provide useful or quantitative information about exposure levels/concentrations. Therefore, Table 1 focuses on human studies in which concentration information is provided.

As shown in Table 1, 0.06 ppm is the lowest effect level (subjective reports of eye irritation) identified in humans. This study is the basis of the CalEPA REL, but is not cited by authoritative federal sources reviewed in this document. In addition, inadequate information is provided about the study design, which included personal protective equipment that is not normally worn by most people. However, the next lowest effect level, 0.09 ppm ("a little" eye irritation after 5 minutes), is only slightly higher. The modeled short-term concentration of 0.003 ppm is 20 times lower than the lowest known concentration that has caused eye irritation in humans. In other words, at the modeled

concentration, people would be exposed to acrolein concentrations that are approximately 5 percent of the level that caused irritation of the eyes in laboratory humans.

Table 1. Acute and Subchronic Human Data for Acrolein in Air

Citation	Study Population	Year of Publication	Concentration in Air (ppm)	Exposure Duration	NOAEL	LOAEL	Adverse Effects
Weber-Tschopp et al aci ATSDR 2007; also CalEPA 1999 (although some details seem to be reported differently here) ¹	Male and female student volunteers	1977	0.3	40 minutes		X	Mild nasal irritation shortly after irritation; throat irritation after 10 minutes; 10% decrease in respiratory rate after 10 minutes in 47% of subjects; 20% decrease in respiratory rate (time unknown).
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.26	Gradually increasing levels for 35 minutes	X	X	LOAEL for Nose irritation and increased eye blink. NOAEL for throat irritation.
Weber-Tschopp et al aci ATSDR 2007	Male and female student volunteers	1977	0.43	Gradually increasing levels for 35 minutes	X	X	LOAEL for Throat irritation. NOAEL for decreased respiratory rate.
Weber-Tschopp et al aci ATSDR 2007	Male and female student volunteers	1977	0.6	Gradually increasing levels for 35 minutes		X	Decreased respiratory rate
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.3	90 seconds		X	"a little" eye irritation
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.6	90 seconds		X	Nasal irritation
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.15	90 seconds	X		Nasal irritation

Table 1. Acute and Subchronic Human Data for Acrolein in Air

Citation	Study Population	Year of Publication	Concentration in Air (ppm)	Exposure Duration	NOAEL	LOAEL	Adverse Effects
Weber-Tschopp et al aci ATSDR 2007	Male and female student volunteers	1977	0.09	At the 5 – minute mark of gradually increasing concentration		X	“a little” eye irritation
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.3	10 minutes		X	Throat irritation; “a little” eye irritation. Eye blink had reached a steady state by this time.
Weber-Tschopp et al aci ATSDR 2007; USEPA 2003	Male and female student volunteers	1977	0.3	40 minutes		X	“medium” irritation; decreased respiratory rate
Sim and Pattle aci ATSDR 2007; USEPA 2003	Humans	1957	0.81	20 seconds		X	Lacrimation
Kane and Alarie aci ATSDR 2007 ²	Humans	1977	0.5	10 minutes or less		X	Lacrimation and possibly other evidence of eye irritation
Darley et al., aci CalEPA 1999. ³	Healthy volunteers	1960	0.06	5 minutes; eye-only exposure (breathed through carbon-filter respirators).		X	Not specified, except “subjective reports of eye irritation”. Also not clear whether there were unexposed people who also wore the respirators.

NOTES:

¹ This study is the basis of ATSDR’s acute minimal risk level (MRL) of 0.003 ppm and the National Academy of Sciences’ (NAS’) emergency exposure guidance level (EEGL) of 0.05 ppm.

² Details unclear in secondary source.

³ This study is the basis of CalEPA’s acute REL. Details unclear in secondary source. Not mentioned in any of the other secondary sources reviewed.

5.0 Comparison to Acrolein in Indoor Air

Table 2 provides summary information about levels of acrolein present in indoor air. Application of this table is limited by its overview nature. Information about dates, potential sources of acrolein, etc., is not included in the table. However, a couple of generalizations can be made. First, based on ATSDR 2007, acrolein concentrations are typically greater in indoor air than in ambient air. Second, indoor air in some homes can have acrolein present at concentrations approaching or exceeding the maximum modeled short-term concentration. Acrolein in indoor (residential) air is associated with low air exchange rates in homes, temperature, and cooking (being formed as animal and vegetable fats are heated). It is also known to be released from lumber used in new homes and from wood burning (Seaman et al 2007; Gilbert et al 2005; USEPA Region 1, 2008; ATSDR 2007).

Table 2. Typical Acrolein Concentrations

Location	Concentration ($\mu\text{g}/\text{m}^3$)
<i>Annual average predicted concentrations</i>	
Near facility	0.0041
Near closest residence ¹	0.00017
<i>Maximum predicted concentrations</i>	
Near facility	7.3
Near closest residence ¹	0.78
<i>Residential Indoor Air²</i>	
Raleigh, NC	0.85 – 4.62
Woodland, CA (Range)	Not quantifiable (below detection limit of 2.0) to 29
Woodland, CA (Average)	7.1
Windsor, Ontario	0.4 – 8.1
Hamilton, Ontario	<0.05 – 5.4
Toronto, Ontario	16 - 23

¹ Maximum concentrations modeled within 500 meters of the Johnson residence.

² Source: ATSDR 2007, primarily from Table 6-5, page 121.

6.0 Acrolein Regulatory Thresholds

Table 3 summarizes select air criteria for acrolein. Note that additional criteria are listed in the response to comments document and related report. Application of the information in this table is limited because, with the exception of the minimal risk levels (MRLs), the criteria in Table 3 were not developed for use related to exposure of the general public, but rather for workers. The worker criteria may carry the inherent assumption of a

healthy workplace population, an assumption that may not apply to the general public. In addition, some worker criteria may assume that minor, reversible adverse effects are acceptable or that workers' bodies adapt to exposure over time; these assumptions also do not apply to the general public. Nevertheless, two observations can be made on the basis of information in Table 3.

Table 3. Select Regulatory Criteria for Acrolein in Air

		Applicable Timeframe	Criterion or modeled concentration (ppm)	Intended Population
Maximum predicted concentrations				
Near facility		1 hour	0.003	
Near closest residence ²		1 hour	0.0003	
Agency	Threshold			
ATSDR	Acute MRL	1 to 14 days	0.003	General public
ACGIH	TLV (ceiling limit)	Instantaneous	0.1	Workplace
NIOSH	REL (10-hr TWA)	10-hour average	0.1	Workplace
NIOSH	STEL	15-minute average	0.3	Workplace
NIOSH	IDLH	Immediate	2	Workplace
OSHA	PEL (8-hr TWA)	8-hour average	0.1	Workplace

ATSDR 2007. Section 8 and especially Table 8-1.

- ACGIH American Conference of Governmental Industrial Hygienists.
- IDLH Immediately dangerous to life and health. Guideline to indicate when respirators should be used.
- MRL Minimal risk level.
- NIOSH National Institute of Occupational Safety and Health.
- PEL Permissible exposure limit.
- REL Recommended exposure limit.
- STEL Short-term exposure limit.
- TLV Threshold limit value. At no time should a ceiling limit be exceeded.
- TWA Time-weighted average. Average value over a specified time-frame.

7.0 Conclusions

Although the short-term HQ for acrolein based on 1/10th of the IDLH is less than 1, the short-term HQ for acrolein of 38 (based on the CalEPA REL) exceeds 1. The CalEPA REL is based on a study that is not cited in other authoritative reviews (ATSDR 2007 and USEPA 2003 and 2008), and insufficient detail is provided in CalEPA 1999 to evaluate the reliability or applicability of the selected study.

It is clear that even the modeled short-term estimate of exposure concentration is significantly lower than concentrations that have caused eye irritation (and no other adverse effects) in humans (factor of about 20). In addition, the modeled short-term estimate of exposure concentration is less than available occupational standards by a factor of about 100, and is similar to the protective ATSDR MRL.

This information provides a useful context for evaluating whether it is reasonable to be concerned about worst-case, short-term emissions of acrolein from the proposed plant.

8.0 References

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9.0 Acronyms

ACGIH	American Conference of Governmental Industrial Hygienists
ATSDR	Agency for Toxic Substances and Disease Registry
CalEPA	California Environmental Protection Agency
EEGL	Emergency Exposure Guidance Level
g/s	grams per second
HAP	Hazardous Air Pollutant
HQ	Hazard Quotient
IDLH	Immediately dangerous to life and health
LOAEL	Lowest observed adverse effect level
$\mu\text{g}/\text{m}^3$	Microgram per cubic meter
MBFP	Medicine Bow Fuel & Power LLC
MRL	Minimal Risk Level
MSDS	Material Safety Data Sheet
NAS	National Academy of Sciences
NIOSH	National Institute of Occupational Safety and Health
NOAEL	No observed adverse effect level
PEL	Permissible Exposure Limit
ppm	Parts per million
REL	Recommended Exposure Limit
STEL	Short-Term Exposure Limit
TLV	Threshold Limit Value
TWA	Time-weighted average
$\mu\text{g}/\text{m}^3$	micrograms per meter cubed
USEPA	U.S. Environmental Protection Agency