pennsylvania
DEPARTMENT OF ENVIRONMENTAL PROTECTION
BUREAU OF AIR QUALITY

MEMO

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DATE: February 22, 2013

RE: Risk Assessment Analysis Review
EN-TIRE Logistics, Union County
Plan Approval Application, 60-00022A

The Pennsylvania Department of Environmental Protection (DEP) received a plan approval application in October 2011 for the construction of a waste tire to energy facility submitted by EN-TIRE Logistics of Milton, PA, LLC (ETL). This tire-derived fuel (TDF)-fired steam and power production facility, also referred to as the White Deer Energy (WDE) project, would be constructed adjacent to the existing National Gypsum Company facility in White Deer Township, Union County, Pennsylvania. The ETL plan approval application and inhalation pathway risk assessment were prepared by ALL4 Inc.

The facility will include a rotary kiln-style combustor followed by a furnace section/steam-generating unit. Shredded scrap tires will be incrementally charged to the rotary kiln. The power system will consist of a steam turbine and attached generator. Following the turbine, low pressure steam will exclusively be delivered to (and condensate returned from) the NGC facility via pipelines. Electricity generated will be used for WDE plant site consumption with excess power sold to the grid. The total electricity generation capacity will be 7 megawatts.

As part of the plan approval process the Department required the applicant to perform an inhalation risk assessment to allow the Department to evaluate potential cancer and noncancer inhalation risks from ETL's air emissions. Based on the authority of §127.12(a)(2), the Department can require that a facility must provide, as part of its permit application, "information that is requested by the Department and is necessary to perform a thorough evaluation of the air contamination of the source."[1] The information requested may include the requirement that an inhalation risk assessment and/or a multipathway risk assessment (MPRA) be performed on proposed construction, reconstruction or modifications.

The inhalation risk assessment for the proposed facility was performed using guidance included in the Department's 'Risk Assessment Guidelines for Facilities Burning Hazardous Waste' (RAG), portions of which are generally applicable to the performance of inhalation risk assessments at various types of facility. These guidelines were developed for facilities burning hazardous waste and describe the procedures for performing both an initial risk assessment screen and, if necessary, a MPRA. The United States Environmental Protection Agency (EPA) has determined that scrap tires removed from vehicles and managed under established tire collection programs are classified as non-hazardous secondary materials when burned in combustion units.[2] ETL has informed the Department that the tires to be used as fuel at the WDE facility will have been managed in such a manner as to qualify for EPA's non-hazardous secondary material categorization.

In December 2011 ETL submitted an inhalation risk assessment; additional revisions were received by DEP in May 2012 and again in July 2012. The inhalation risk assessment is based on the estimated chronic risks posed by the proposed facility operating at maximum operating load as well as two acute exposure scenarios posed by short term emissions. The risk assessment covers a selection of 35 compounds of potential concern (COPCs) which are considered by the Department to encompass the air toxics emissions expected to pose inhalation risk from the combustion of tire-derived fuel at this facility. The list of these COPCs (included in this document as Table 1) was prepared using data gathered from performance tests conducted on other facilities combusting tires. DEP and ETL developed emission rate estimates for these COPCs.

Results

Based on the information provided in the WDE risk assessment, the levels of risk posed by both chronic and acute exposure to the modeled COPCs do not exceed the Department's inhalation risk assessment benchmarks. It is recommended that permit conditions found in Attachment 1 be

[1] Pennsylvania Code, Title 25, Environmental Protection, January 8, 2011.
[2] 40 CFR Part 241, Identification of Non-Hazardous Secondary Materials That Are Solid Waste; Final Rule, published in the Federal Register 3/21/11. See pgs. 15550-15551 for specific details.

included in the plan approval to verify that the facility, after commencing operation, meets the Department's human health risk assessment benchmarks.

Inhalation Risk Assessment

The risk assessment process includes four primary steps: hazard identification, exposure assessment, toxicity assessment, and risk characterization. Hazard identification involves identifying the COPCs expected to be emitted from the proposed project considering factors such as fuel type, emissions control equipment, feedstock characteristics, *etc.* COPC emissions data from similar facilities and occasionally small scale tests were used in conjunction with manufacturers' specifications and engineering calculations to estimate emission rates from the proposed facility.

These emission rates, in conjunction with the results from the dispersion modeling conducted for this facility, were used to estimate the maximum hourly and annual ground level ambient concentrations of the COPCs over a defined grid outside the plant perimeter. The 'most exposed individual' (MEI) is assumed to live a 70 year lifetime at the point on the receptor grid where exposure concentrations are the highest. The risk assessment is based upon inhalation exposure to conditions at that maximum point.

ETL used the AERMOD air dispersion model to perform their analysis according to DEP guidance, which was also provided for various aspects of the modeling such as land use and terrain analysis, receptor grid, meteorological, building downwash, *etc.* The maximum ground level concentration (at the MEI location) was determined using a unitized COPC emission rate of 1 gram/sec. The unitized concentration is multiplied by the emission rate of each of the COPCs to provide the predicted inhalation exposure concentrations of each compound at the MEI location. This procedure was performed for both acute and chronic exposure scenarios. More detail pertaining to the air dispersion modeling is available in the memorandum written by the DEP Air Modeling Section.[3]

Toxicity assessment involves the identification of the adverse health effects posed by an individual compound and relates the development of these effects to the level of exposure. The toxic potential of a chemical may depend on exposure route and duration as well as the mode of action. In an inhalation risk assessment the health risk value potentially posed by each COPC is mathematically applied to the exposure concentration to provide the estimated risk level.

Risk Factors and Their Application

Risk characterization integrates the toxicity information with the exposure concentrations derived from the modeling process to project estimated cancer and non-cancer health risks. The estimated extra cancer risk is quantified in terms of additional predicted cases of cancer above the national average lifetime incidence rate (41.24% or four in ten for men and women based on the National Cancer Institute's 2007-2009 U.S. rates for incidence of all types of cancers).[4]

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[3] Summary of Air Quality Analysis for Inhalation Pathway Risk Assessment EN-TIRE Logistics of Milton PA, LLC Application for Plan Approval 60-00022A Proposed White Deer Energy Facility, White Deer Township, Union County, October 23, 2012.

[4] National Cancer Institute, http://seer.cancer.gov/statfacts/html/all.html (accessed 10/9/12).

The excess lifetime cancer risk (ELCR) is determined using a unit risk factor (URF) expressed with the units $1/(\mu g/m^3)$. The URF is multiplied by the individual COPC concentration (expressed in $\mu g/m^3$) to provide the ELCR value. That value is in a form estimating the increased lifetime cancer risk probability (*e.g.*, an ELCR of 3 per population of ten million or 3.0E-07, also written as 3.0×10^{-07}).

For noncancer risk the risk value is determined using a reference concentration (RfC) expressed in mass per cubic meter of air (typically mg/m^3). An RfC is "an estimate of a continuous inhalation exposure for a given duration to the human population (including susceptible subgroups) that is likely to be without an appreciable risk of adverse health effects over a lifetime."[5] The RfC is divided into the exposure concentration of the COPC (expressed in the same units of concentration) to derive the hazard quotient (HQ). An HQ of 1.0 or less indicates that a compound poses no threat of an adverse effect on a continuous exposure basis. The hazard quotients of all non-carcinogenic compounds are added to provide the 'whole body' hazard index (HI).

Inhalation risk assessment results are compared with the Department's risk management target levels for cancer and non-cancer health risks. These inhalation target values are 1 in 10 million or less aggregated ELCR and, on an individual COPC basis, a noncancer hazard quotient of 0.01 or less. For a proposed hazardous waste combustor subject to the Department's RAG these risk values represent the screening thresholds which, if exceeded, require the facility to perform a MPRA. For other types of facilities required by the Department to perform inhalation risk assessments these thresholds focus attention on any compounds that may exceed them. The reason for the elevated emission level(s) may be examined and particular attention paid to the results of future stack tests. On a facility-wide basis EPA Region 6 has proposed an ELCR limit of 1 in 100,000 and a total HI no greater than 0.25.[6] DEP follows these two standards. A failure to remain at or below the facility-wide risk levels may trigger imposition of operational changes and/or restrictions, emission controls, *etc.*, in order to bring emissions into an acceptable range. A MPRA could also be required to provide a clearer risk picture.

The acute health risk benchmark is the short term (one hour) exposure inhalation reference concentration. As with chronic RfCs, an HQ of 1.0 or less indicates that a compound poses no threat of an adverse effect, but on a one hour exposure basis rather than a chronic basis. The acute RfC for each COPC should not be exceeded by its respective maximum modeled hourly concentration - the predicted maximum one hour hazard quotient (a ratio of the acute exposure concentration divided by the acute RfC) should be no greater than 1.0 to meet the Department limit.

Risk Factor References

As a reference for chronic human health inhalation risk values from the COPCs, DEP follows the hierarchy provided by EPA's Office of Solid Waste in the "Human Health Risk Assessment Protocol" (HHRAP), Appendix 2-A. This list is recommended by EPA "for acquiring human

[5] EPA IRIS Glossary, http://www.epa.gov/iris/gloss8_arch.htm (accessed 8/21/12).

[6] Region 6 Risk Management Addendum – Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, EPA-R6-98-002, July 1998, pg. ADD-3, http://www.epa.gov/earth1r6/6pd/rcra_c/protocol/r6add.pdf (accessed 11/2/12).

health toxicity data to be used in performing risk assessments of hazardous waste combustion facilities" and is ranked as follows: [7]

1. EPA's Integrated Risk Information System (IRIS)
2. EPA's Provisional Peer Reviewed Toxicity Values (PPRTVs)
3. Other Toxicity Values (including California Environmental Protection Agency (Cal EPA) Reference Exposure Levels (RELs), Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels (MRLs), and Health Effects Assessment Summary Tables (HEAST) toxicity values.

The HHRAP also lists the following as sources for elevated acute (short-term) exposure risk levels. Note that WDE used a number of alternate acute risk values (*e.g*, STEL/40; 3 x TLV/20; 3 x PEL/20) in the Phase 2 calculations.

1. Cal EPA RELs
2. Acute Exposure Guideline Level 1 (AEGL-1)
3. Emergency Response Planning Guideline 1 (ERPG-1)
4. Temporary Emergency Exposure Level 1 (TEEL-1)

Increased impact of mutagens during childhood

In performing the excess lifetime cancer risk potion of the risk assessment, ETL followed EPA's "Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens" (March 2005) to account for the additional cancer risks due to childhood exposure to mutagenic air toxics. The following mutagenic COPCs were identified using EPA Region 3's "Regional Screening Level Summary Table June 2011"[8]:

- Chromium VI
- Chrysene
- Benz[a]anthracene
- Benzo[b]fluoranthene
- Benzo[a]pyrene
- Indeno[1,2,3-cd]pyrene

The Supplemental Guidance provides factors that take into consideration the potential for "early life exposure to make a greater contribution to cancers appearing in later life.":

"The following adjustments represent a practical approach that reflects the results of the preceding analysis, which concluded that cancer risks generally are higher from early-life exposure than from similar exposure durations later in life:

- For exposures before 2 years of age (*i.e.*, spanning a 2-year time interval from the first day of birth up until a child's second birthday), a 10-fold adjustment.
- For exposures between 2 and <16 years of age (*i.e.*, spanning a 14-year time interval from a child's second birthday up until their sixteenth birthday), a 3-fold adjustment.
- For exposures after turning 16 years of age, no adjustment." [9]

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[7] Human Health Risk Assessment Protocol, Appendix A-2, EPA530-R-05-006, September 2005. p. A-2-33. www.epa.gov/osw/hazard/tsd/td/combust/finalmact/ssra/05hhrapapa.pdf (accessed 8/3/12).

[8] http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/

[9] Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens, EPA/630/R-03/003F (March 2005), p.33. Expressed as a formula: ((2/70)x10)+((14/70)x3)+((54/70)x1)=1.66, which serves as a multiplier for the ELCR calculated for each of the mutagenic compounds.

Discussion

Carcinogenic Risk from Cobalt and Cobalt Compounds

The lack of applicable data from inhalation studies on the carcinogenicity of cobalt and cobalt compounds presents a particular problem when cancer risk values are required for an inhalation risk assessment. As summarized on the Air Toxics Website of the EPA Technology Transfer Network:

> "Human studies are inconclusive regarding inhalation exposure to cobalt, and the one available oral study did not report a correlation between cobalt in the drinking water and cancer deaths. EPA has not classified cobalt for carcinogenicity.... In one study on workers that refined and processed cobalt and sodium, an increase in deaths due to lung cancer was found for workers exposed only to cobalt. However, when this study was controlled for date of birth, age at death, and smoking habits, the difference in deaths due to lung cancer was found to not be statistically significant. In another study assessing the correlation between cancer deaths and trace metals in water supplies in the United States, no correlation was found between cancer mortality and the level of cobalt in the water."[10]

In a study by the National Toxicology Program (NTP) "cobalt sulfate heptahydrate exposure via inhalation resulted in increased incidences of alveolar/bronchiolar tumors in rats and mice." Yet, another animal study found that "inhalation of cobalt [oxide] over a lifetime did not increase the incidence of tumors in hamsters."[11]

The USEPA Office of Research and Development (ORD) has summarized the weight-of-evidence relevant to the potential of cobalt and cobalt compounds to present an inhalation cancer risk to humans. Under the EPA's 2005 Guidelines for Carcinogen Risk Assessment "cobalt sulfate (soluble) is described as 'likely to be carcinogenic to humans by the inhalation route,' based on both the limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals as shown by a statistically significant increased incidence of alveolar/bronchiolar tumors in both sexes of rats and mice, pheochromocytomas in female rats, and hemangiosarcomas in male mice.... While available studies in humans have suggested a possible association between exposure to cobalt and respiratory tumors in cobalt workers ... limitations within these studies, including small numbers of subjects, inadequate exposure assessment and potential exposure to other chemicals make them inadequate for assessing the carcinogenic potential of cobalt. Studies for evaluation of the oral carcinogenic potential for cobalt were not located."[12]

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[10] EPA Technology Transfer Network, Air Toxics Website, revised January 2000, http://www.epa.gov/ttn/atw/hlthef/cobalt.html (accessed 6/27/12)

[11] Ibid. The cited studies: NTP (National Toxicology Program). 1998. Toxicology and Carcinogenicity Studies of Cobalt Sulfate Heptahydrate (CAS No. 10026-24-1) in F344/N Rats and B6C3F1 Mice (Inhalation studies). U.S. Dept. of Health and Human Services, Public Health Service, National Institutes of Health. NTP Technical Report Series, No. 471; Wehner, A.P., B.O. Stuart and C.L. Sanders. 1979. Inhalation Studies with Syrian Golden Hamsters. Prog. Exp. Tumor Res. 24:177-198

[12] Provisional Peer Reviewed Toxicity Values for Cobalt, (8/25/08), p. 29. http://hhpprtv.ornl.gov/issue_papers/Cobalt.pdf (accessed 7/2/12); the cited guidelines: U.S. EPA. 2005. Guidelines for carcinogen risk assessment. Risk Assessment Forum; EPA/630/P-03/001F.

Based on the limited data available, the International Agency for Research on Cancer (IARC) has concluded that:

> "Cobalt metal with tungsten carbide is *probably carcinogenic to humans (Group 2A)*. A number of working group members supported an evaluation in Group 1 [carcinogenic to humans] because:
> (1) they judged the epidemiological evidence to be sufficient, leading to an overall evaluation in Group 1; and/or (2) they judged the mechanistic evidence to be strong enough to justify upgrading the default evaluation from 2A to 1. The majority of working group members, who supported the group 2A evaluation, cited the need for either sufficient evidence in humans or strong mechanistic evidence in exposed humans.
> Cobalt metal without tungsten carbide is *possibly carcinogenic to humans (Group 2B)*.
> Cobalt sulfate and other soluble cobalt(II) salts are *possibly carcinogenic to humans (Group 2B)*."[13]

To date no chronic inhalation risk values for cobalt (either cancer or noncancer) have been developed for EPA's IRIS. When such values are not available in IRIS and are required by the Superfund program, ORD may develop cancer and/or noncancer Provisional Peer Reviewed Toxicity Values (PPRTVs), referred to previously as the second tier source for chronic human health risk values. IRIS values are intended to be used in all EPA programs; once an IRIS value becomes available the corresponding PPRTV is generally removed from the database.

PPRTVs differ in part from IRIS values in that PPRTVs do not receive the multiprogram consensus review provided for IRIS values. They are derived after a review of the relevant scientific literature using the methods, sources of data, and guidance for value derivation used by the U.S. EPA IRIS Program. "All provisional toxicity values receive internal review by two EPA scientists and external peer review by at least two scientific experts. A third scientific review is performed if there is a conflict between the two original external reviewers."[14] Yet caution is recommended when using PPRTVs: "U.S. EPA ORD is concerned that PPRTV's may be seen (and used) as equivalent to IRIS values" and, until recently, restricted direct access to these values except by EPA staff.[15] Although PPRTVs are used for risk assessment purposes for the Superfund program and for hazardous waste combustor multipathway risk assessments, EPA's Office of Air Quality Planning Standards does not include them in their "Prioritized Chronic Dose-Response Values" table (5/21/2012).[16] When considering a PPRTV for use in a risk assessment it is important to evaluate the basis for the risk value and decide whether its use in an assessment is appropriate and supportable since it may serve as a basis for regulatory action ultimately impacting public health.

Although a number of studies suggest the carcinogenic potential of cobalt and cobalt compounds, available human studies lack sufficient detail, particularly with regards to analysis of exposure concentration and cancer incidence. To date only one carcinogenicity study (Bucher et al.,

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[13] IARC Monographs Volume 86, Metallic Cobalt Particles(With or Without Tungsten Carbide) (2006), pg. 133. http://monographs.iarc.fr/ENG/Monographs/vol86/mono86-6.pdf (6/28/12)

[14] Human Health Risk Assessment Protocol, pgs. A-2-34 (accessed 7/5/12) http://www.epa.gov/osw/hazard/tsd/td/combust/finalmact/ssra/05hhrapapa.pdf

[15] Ibid.

[16] EPA's Technology Transfer Network, Sources of Chronic Dose-Response Information, http://www.epa.gov/ttn/atw/toxsource/chronicsources.html (accessed 8/23/12).

1999), exposing F344 strain rats and B6C3F$_1$ strain mice to a cobalt sulfate hexahydrate aerosol, provides a dose-response relationship. This study "was chosen as the principal study for the derivation of an inhalation unit risk, based on the dose-response relationship for statistically significant increased incidences of alveolar/bronchiolar (A/B) neoplasms (adenomas and carcinomas)."[17] The study was conducted over a period of two years during which the exposure to cobalt sulfate hexahydrate aerosol was conducted for 6 hours/day, 5 days/week.

Bucher et al. found that in both the rats and mice lung tumor incidence was greater in the exposed groups than in the controls and that "exposure to cobalt sulfate by inhalation resulted in increased incidence of alveolar/bronchiolar neoplasms in both sexes of rats and mice."[18] Additionally, the results revealed both gender and species response differences to the cobalt exposures. "Significant increases in alveolar/bronchiolar adenomas or carcinomas were seen in high-dose male rats, while significant increases in alveolar/bronchiolar [A/B] adenomas/carcinomas [referred to jointly as neoplasms[19]] were seen in the mid- and high-dose female rats."[20] Subtracting the control group rate of tumor incidence, male and female rats had equal incidence rates of tumors at the lowest exposure concentration (3/50 for both sexes). At the mid-level concentration 3/50 males developed A/B neoplasms; 15/50 females developed neoplasms. At the highest cobalt sulfate exposure concentration 6/50 males developed neoplasms; 15/50 females did so.[21]

Regarding the interspecies response difference, male mice showed a particularly elevated incidence of A/B carcinomas (malignant tumors) at all exposure levels when compared to male rats. After the control group rate of A/B carcinoma development is subtracted, 28.0% of male mice in the higher exposure group developed carcinomas; 2.0% of male rats did so. In the intermediate exposure groups 18.0% of male mice developed carcinomas; 6.0% of the male rats did so; the low exposure concentrations yielded a 16.0% rate for mice, 0.0% for rats. The same pattern is s true for neoplasm incidence at the two upper exposure concentrations: 34.0% of the high exposure group of mice developed neoplasms, 12.0% of the rats did so; 16.0% of the mice in the intermediate exposure group developed neoplasms, 6.0% of the rats did so.

The most pronounced difference between rat and mouse neoplasm incidence rates was between the control groups of male rats and mice: 1/50 control group rats (2%) developed A/B neoplasms; 11/50 control group mice (22%) developed A/B neoplasms. In the two control groups of females, 0/50 (0%) of the rats developed A/B neoplasms; 4/50 (8%) of the female mice developed neoplasms. The high spontaneous tumor development rate (11/50 or 22%) among the male mice could obscure the true incidence of cobalt-induced neoplasms.[22] Clearly, the use of a mouse strain (B6C3F$_1$) with such a high rate of spontaneous tumor development in this study is

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[17] Provisional Peer Reviewed Toxicity Values for Cobalt, p. 33. The cited study: Bucher, J.R., J.R. Hailey, J.R. Roycroft et al. 1999. Inhalation toxicity and carcinogenicity studies of cobalt sulfate. Toxicol. Sci. 49:56-67; NTP (National Toxicology Program).

[18] Bucher, J.R., J.R. Hailey, J.R. Roycroft et al. 1999. Inhalation toxicity and carcinogenicity studies of cobalt sulfate. Toxicol. Sci. 49:56-67; NTP (National Toxicology Program), pg. 66.

[19] A neoplasm is a tumor; an adenoma is a benign neoplasm of epithelial tissue; a carcinoma is a malignant neoplasm of epithelial tissue (National Toxicology Program, U.S. Department of Health and Human Services, http://ntp.niehs.nih.gov/ntp/roc/twelfth/Glossary.pdf).

[20] Provisional Peer Reviewed Toxicity Values for Cobalt, pgs. 20-21.

[21] Ibid., pg. 21.

[22] Ibid., Tables 3 and 4 on pgs.21-22.

questionable. At least one study has been conducted on spontaneous neoplasms in both of these rodent stains in order to provide researchers with some perspective on this phenomenon.[23]

Considerable assumption and extrapolation were required to derive a human inhalation cancer risk value based on the limited data derived from this study. As mentioned above, the study's exposures were conducted for 6 hours/day, 5 days/week over a period of two years, and had to be expanded to a 70-year human lifetime of continuous exposure. The intermittent exposure concentrations of the study were converted to continuous exposure concentrations by the rather simple expediency of multiplying the exposure concentration level:

$$\text{Concentration}_{\text{adjusted}} = \text{Concentration} \times [(5 \text{ days/week})/(7 \text{ days/week})] \times [(6 \text{ hrs./day})/(24 \text{ hrs./day})].$$

The adjusted concentrations derived from the preceding calculations were then multiplied by the factor 0.2243 to account for the cobalt molecular weight percentage in cobalt sulfate hexahydrate ($CoSO_4(H_2O)_6$) resulting in "duration-adjusted concentrations of 0, 0.012, 0.040 and 0.120 mg cobalt/m^3, respectively, for exposure to cobalt sulfate hexahydrate at 0.0, 0.3, 1.0 and 3.0 mg/m3 exposure levels."[24]

"Using the RDDR [regional deposited dose ratio] computer program… human equivalent concentrations… were calculated at each exposure level for each species and sex using body weight default values."[25] The EPA IRIS website defines the RDDR as the "dose calculated for a given exposure in the animal species of interest to the regional deposited dose of the same exposure in a human. This ratio is used to adjust the exposure effect level for interspecies dosimetric differences to derive a human equivalent concentration...."

> "In accordance with the U.S. EPA (2000) BMD methodology, the default benchmark response (BMR) of 10% increase in extra risk was used as the basis for the BMD… Lung tumors in female rats were chosen as the endpoint for use as a point of departure for derivation of the inhalation unit risk….The BMDL [benchmark dose lower confidence limit] for this endpoint was the lowest for all study groups (*i.e.*, male and female rats and mice) and was based on a model that showed a good fit to the data …after dropping the high exposure group … [which was dropped following EPA procedure] when no models achieve adequate fit using all exposure levels. Although this left only two exposure levels (in addition to the control group), these exposure levels are in the low-dose portion of the curve within the region of the dose-response relationship in which response is increasing with exposure level (*i.e.*, the region of interest for deriving the point of departure) and bracket the derived BMD."[26] When applied to the data from the female rat cohort, these calculations resulted in a PPRTV inhalation cancer URF for cobalt of 9.0E-03$(\mu g/m^3)^{-1}$.

As discussed previously, there is a body of evidence that at least some chronic inhalation exposures to cobalt and certain cobalt compounds may be linked to respiratory tumor

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[23] Haseman, Joseph K., Hailey, James R., and Morris, Richard W. 1998. Spontaneous Neoplasm Incidences in Fisher 344 Rats and B6C3F$_1$ Mice in Two-Year Carcinogenicity Studies. Toxicol Pathol 1998 26:428. http://tpx.sagepub.com/content/26/3/428.full.pdf

[24] Provisional Peer Reviewed Toxicity Values for Cobalt, pg. 33.

[25] Ibid.

[26] Ibid., pg. 34. The reference cited: U.S. EPA. 2000. Benchmark Dose Technical Guidance Document [external review draft]. EPA/630/R-00/001. Online: http://www.epa.gov/iris/backgr-d.htm.

development. The Bucher rat and mouse study offers support for the link between inhalation exposure to cobalt and an increased probability of the development of lung tumors in rats and mice. Nonetheless, some of the results of this study have raised concerns on the part of DEP about the use of the PPRTV URF derived from the study as a basis for regulatory action, particularly considering the lack of additional animal and human studies providing supporting dose/response data.

In the results referred to above there was a pronounced disparity between the responses of the male and female rats used to establish the risk value. Female rats had a much higher incidence of A/B neoplasms (30.0%) at both the intermediate and highest exposure concentrations than male rats (male A/B tumor incidence was 6.0% and 12.0% respectively). The data from the female rats was used as the basis for the PPRTV URF but, with the two highest data points being equal (30.0% incidence rate), the three female rat data points failed to adequately fit any of the models. Consequently the data point from the highest exposure level was eliminated in order to provide a fit to one of the models.

The Bucher study also revealed an interspecies response difference to cobalt exposure: male mice showed a particularly elevated incidence of A/B carcinomas at all exposure levels when compared to male rats (specific details provided above). The same is true for neoplasm incidence at the two upper exposure concentrations. Although the difference is not so pronounced,

The different response rates, in one instance between genders of the species on which the risk value is based, and in the other between two very closely related species of rodent, plus the use of only two data points for risk model selection, serve to weaken confidence in the results of risk extrapolation between female rats and the very distantly-related human species to the extent that the Department would have difficulty in supporting and enforcing a regulatory requirement based on this risk value. Therefore the provisional cobalt cancer risk value is not used in the DEP's calculation of excess lifetime cancer risk for the WDE facility. Since there are no alternate cobalt inhalation cancer risk values available for evaluation in the list of acceptable inhalation risk value sources (detailed previously), no cancer risk for cobalt is included in the Department's risk calculations.

Noncarcinogenic Risk from Cobalt and Cobalt Compounds

Regarding noncancer human health effects, numerous studies have linked inhalation of cobalt and cobalt compounds with a range of deleterious symptoms and conditions. "Chronic exposure to cobalt by inhalation in humans results in effects on the respiratory system, such as respiratory irritation, wheezing, asthma, decreased lung function, pneumonia, and fibrosis." Non-respiratory effects including cardiac effects (functional effects on the ventricles, enlargement of the heart) plus congestion of the liver, kidneys, and conjunctiva, and immunological effects have been noted in humans from inhalation exposure to cobalt. [27]

Based on human and animal data, ORD concluded that respiratory effects are the most sensitive and easily observable noncancer endpoints of cobalt inhalation exposure. "Symptoms of respiratory tract irritation and altered pulmonary function have been widely reported in workers

[27] EPA Technology Transfer Network, Air Toxics Website, revised January 2000, http://www.epa.gov/ttn/atw/hlthef/cobalt.html (accessed 7/13/12).

exposed to cobalt-containing airborne media. Of the four human epidemiology studies … [reviewed by ORD in the course of deriving the chronic inhalation provisional-RfC (p-RfC)], the study by Nemery et al. (1992) provides the strongest basis for derivation of the p-RFC."[28]

The Nemery study details cobalt exposure and respiratory effects in a group of 194 workers engaged in cutting and polishing diamonds using equipment that exposed them to airborne cobalt dust. Both work area and personal air samples were collected to measure worker exposure. The study was of particular value in establishing a RfC because the range of exposure concentrations of metallic cobalt dust permitted the calculation of a "no observed adverse effects level" (NOAEL). A NOAEL "represents the highest exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effect between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse, nor precursors to adverse effects…." Qualitatively similar but on an expanded time scale, an RfC is "an estimate of a continuous inhalation exposure for a given duration to the human population (including susceptible subgroups) that is likely to be without an appreciable risk of adverse health effects over a lifetime."[29]

> "Decreased pulmonary function and respiratory tract irritation were identified as the co-critical effects for derivation of the subchronic and chronic p-RfCs. Assuming the personal air samples to be more representative of worker exposure than the area air samples, the study by Nemery et al. (1992) identified a NOAEL of 5.3 μg/m3 …for metallic cobalt for effects on pulmonary function (e.g. forced expiratory volume (FEV), forced vital capacity (FVC) and forced expiratory flow [referred to as MMEF]) and an increased prevalence of symptoms of respiratory tract irritation (e.g. nose/throat irritation, cough, phlegm, dyspnea). … The NOAEL for occupational exposure was adjusted to continuous exposure as follows:
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> $$5.3\ \mu g/m^3\ (10\ m^3/day\ /\ 20\ m^3/day)\ (5\ days\ /\ 7\ days) = 1.9\ \mu g/m^3$$ [30]
>
> Dividing the $NOAEL_{ADJ}$ of 1.9 $\mu g/m^3$ by a composite UF [uncertainty factor] of 300 yields a chronic p-RfC of 6E-6 mg/m^3 for metallic cobalt as follows:
>
> $$\text{Chronic p-RfC} = NOAEL_{ADJ} \div UF$$
> $$= 1.9\ \mu g/m^3 \div 300$$
> $$= 0.000006 \text{ or } 6E\text{-}6\ mg/m^3$$
>
> "The composite UF of 300 is composed of three uncertainty factors: 3 to account for extrapolating from an assumed subchronic exposure duration to a chronic exposure duration, 10 for database insufficiencies and 10 for human inter-individual variability…. Since Nemery et al. (1992) did not report duration for any worker in this study, it is possible that exposure duration may have been subchronic or longer for some workers. A factor of 10 is applied to account for database insufficiencies due to the lack of inhalation developmental toxicity studies and a multi-generation reproduction study. A factor of 10 is applied to account for human variability, including sensitive subgroups. Individuals with underlying respiratory diseases (asthma, chronic obstructive pulmonary

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[28] Provisional Peer Reviewed Toxicity Values for Cobalt. The cited study: Nemery, B., P. Casier, D. Roosels et al. 1992. "Survey of cobalt exposure and respiratory health in diamond polishers." Am. Rev. Resp. Disease 145:610-616.

[29] EPA IRIS Glossary, http://www.epa.gov/iris/gloss8_arch.htm (accessed 8/21/12).

[30] Provisional Peer Reviewed Toxicity Values for Cobalt. p 27-28.

disease) may be more sensitive to the respiratory effects of inhaled cobalt. This chronic p-RfC may not be protective for people with hypersensitivity to cobalt."[31]

The fact that the study basis for this chronic inhalation noncancer PPRTV involved a fairly large number of human subjects, provided dose/response data over an exposure concentration range encompassing the level at which the selected health endpoints appeared, and is also supported by other human and animal studies recommends its use in estimating noncancer inhalation risk for cobalt for the purposes of this risk assessment.

Modeling with Alternate Meteorological Data

The Department performed modeling with two additional meteorological data sets, one from Williamsport Regional Airport, the other from Penn Valley Regional Airport, in order to provide a comparison with the Montour set used by ETL. The supplementary data spans a greater length of time (five years compared with 18 months of Montour data) but was collected at a greater distance from the proposed WDE project.

In both of these alternate sets the acute values increase greatly and the chronic values decrease. The acute values increase by 48.0% (Williamsport) and 20.4% (Penn Valley), the chronic values decreasing by about 2.5% and 27.7% respectively. The highest acute risk HQ resulting from the 48.0% increase using the Williamsport data is the Phase 2 HQ for hydrogen chloride which was modeled to increase from 0.011 to 0.016, still well below the Department benchmark acute HQ of 1.0.

Conclusions

Based on the information provided in the WDE risk assessment, the levels of risk posed by both chronic and acute exposure to the modeled COPCs do not exceed the Department's inhalation risk assessment benchmarks.

With the inclusion of the Provisional Peer Reviewed Toxicity Value for inhalation cancer risk from cobalt and cobalt compounds, ETL's evaluation of the chronic inhalation risks estimated the facility-wide excess lifetime cancer risk to the MEI to be approximately 6.4 in 10,000,000. According to the ETL evaluation cobalt contributed the majority of the excess cancer risk: 5.7 in 10,000,000. Hexavalent chromium, formaldehyde, arsenic, nickel and cadmium were found to be the other primary contributors to cancer risk. However, for the reasons detailed above the Department does not include the PPRTV for excess lifetime cancer risk from cobalt in assessing the potential ELCR from the proposed facility. Minus the provisional value for cobalt the highest projected ELCR for an individual compound (hexavalent chromium) is 0.34 in 10,000,000. The facility-wide projected excess cancer risk is 0.75 in 10,000,000, which is below the 1 in 100,000 limit which DEP applies to cancer risk: risk exceeding this level would trigger actions discussed in the preceding section 'Risk Factors and Their Application'.

[31] Ibid, p 28.

Cobalt, followed by chlorine, hydrogen chloride and hydrogen bromide, made the greatest projected contributions to non-cancer risk. Cobalt had the highest chronic non-cancer HQ: 0.0105 (1.0E-2). This modeled cobalt noncancer hazard quotient approximates the Department's level of concern. The whole body hazard index of 1.8E-02 (which includes the cobalt HQ) is below the Department's facility-wide threshold of 2.5E-01 and is considered acceptable.

Two acute risk sets were modeled under normal maximum conditions (*i.e.*, the highest annual one hour average), one using CALEPA acute Reference Exposure Levels (RELs), EPA Acute Exposure Guideline Levels (AEGLs), Emergency Response Planning Guideline-one hour (ERPG-1), and Temporary Emergency Exposure Limits-one hour (TEEL-1) values as benchmarks, the other set using DEP-calculated levels from short-term exposure limits (STELs), threshold limit values (TLVs), permissible exposure levels (PELs), and ceiling/10 values. Modeling results using both sets of acute inhalation RfC values predict that all hazard quotients will be less than the Department's acceptable threshold of 1.0, the level at which no adverse effect would be expected from the exposure. Hydrogen chloride had the highest acute HQ with a Phase 2 value of 0.011, more than an order of magnitude below the DEP threshold. Cobalt, hydrogen bromide, formaldehyde, cadmium, chlorine, arsenic, and nickel had the relatively higher HQs in both of the calculated acute risk sets but were below 1.0 by at least two orders of magnitude and are therefore considered acceptable.

It is recommended that permit conditions found in Attachment 1 be included in the plan approval to verify that the facility, after commencing operation, meets the Department's human health risk assessment benchmarks.

Attachment 1

Recommended Risk Assessment Based Plan Approval Conditions

As a result of the risk assessment review specific conditions are recommended for inclusion in the plan approval. These requirements are recommended to ensure that the risks at the facility, once operating, are reflective of those projected in the risk assessment. It is recommend that the human health risks be re-assessed after the facility is operational based on actual emissions data gathered from performance tests.

The following risk assessment related conditions are recommended for inclusion in the plan approval and facility operating permit:

1. Within 180 days after commencement of operation, a stack test shall be conducted to verify that the emission rates for each of the compounds included in the risk assessment do not exceed the estimated emission rates used in the risk assessment portion of the plan approval application. These compounds are listed in Table 1.

2. Within ninety (90) days of receiving approval from the Department on the acceptability of the stack test results, the permittee shall perform the risk assessment using the stack test emission rates, the exhaust parameters from each test, and the dispersion modeling techniques as approved by the Department for all compounds submitted to the Department for approval.

   The Department may waive this risk assessment requirement if the measured pollutant levels are below those used in the application, the volumetric flow rate has not significantly changed from the value used in the application, and the stack gas temperature has not significantly changed from the value used in the application.

3. If the aggregated risks based upon stack testing exceed a 1 in one hundred thousand cancer risk, or if the non-cancer risk is more than 0.25, a multi-pathway risk assessment will be required by the Department. A protocol for this analysis shall be submitted to the Department within sixty (60) days of receipt of a notice from the Department that the multi-pathway risk assessment is required. Upon approval of the protocol for the analysis, a multi-pathway risk assessment shall be submitted to the Department within one hundred eighty (180) days.

4. The operation of the kiln and boiler shall at no time result in the emission of the following contaminants at rates exceeding the limits identified in pounds per hour and verified by annual stack testing:

Arsenic	4.46E-05
Cadmium	7.40E-05
Chromium VI	4.81E-05
Cobalt	1.75E-03
Copper	2.81E-03
Lead	1.33E-03
Manganese	4.57E-04
Mercury	8.90E-04
Nickel	3.22E-04
Zinc	5.62E-02
Benzene	6.93E-03
Chloromethane	2.07E-02
Formaldehyde	3.14E-02
Naphthalene	1.07E-03
Chlorine	1.85E-02
Hydrogen bromide	3.02E-01
Hydrogen chloride	7.40E-01

Note: the Department may modify this list, and the frequency of stack testing, based upon the stack test results.

5. The exhaust gas temperature, measured at the inlet to the fabric filter, shall not exceed 200 °F. The compliance determination will be determined on a 4-hr block arithmetic average. This requirement may be waived if a satisfactory demonstration is made that an equivalent control of condensable heavy metals and toxic organics can be achieved at higher exhaust temperatures through the use of alternate technologies.

Table 1. Risk Assessment Compounds of Potential Concern

Analyte	CAS#
Aluminum	7429-90-5
Antimony	7440-36-0
Arsenic	7440-38-2
Barium	7440-39-3
Beryllium	7440-41-7
Cadmium	7440-43-9
Chromium (total)	7440-47-3
Chromium VI	18540-29-9
Cobalt	7440-48-4

(continued on next page)

Copper	7440-50-8
Lead	7439-92-1
Manganese	7439-96-5
Mercury	7439-97-6
Nickel	0000-00-7
Selenium	7782-49-2
Zinc	7440-66-6
Chlorine	7782-50-5
Hydrogen Chloride	7647-01-0
Hydrogen Bromide	10035-10-6
Hydrogen Fluoride	7664-39-3
Benzene	71-43-2
Chloromethane	74-87-3
Toluene	108-88-3
Formaldehyde	50-00-0
Acetaldehyde	75-07-0
Chrysene	218-01-9
Fluorene	86-73-7
Naphthalene	91-20-3
Benz[a]anthracene	56-55-3
Benzo[b]fluoranthene	205-99-2
Benzo[a]pyrene	50-32-8
Indeno[1,2,3-cd]pyrene	193-39-5
Total PCBs	various
Dioxins & furans (total) and (TEQ)	various

cc: Andrew Fleck, Air Quality Modeling Section (e-copy)
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