DOWNEY | BRAND
ATTORNEYS LLP

Nicole E. Granquist
ngranquist@downeybrand.com
916/520-5369 Direct
916/520-5769 Fax

621 Capitol Mall, 18th Floor
Sacramento, CA 95814
916/444-1000 Main
916/444-2100 Fax
downeybrand.com

September 30, 2013

VIA U.S. MAIL AND ELECTRONIC MAIL

Ms. Dyan Whyte
Assistant Executive Officer
California Regional Water Quality Control Board
San Francisco Bay Region
1515 Clay Street, Suite 1400
Oakland, California 94612

Re: *TRE Workplan Progress Update (3rd Quarter) – June 27, 2013 Amended Water Code section 13267 Order, Order No. R2-2013-1005-A1, Directive 8*

Dear Ms. Whyte:

Enclosed, pursuant to the Regional Water Quality Control Board, San Francisco Bay Region's, ("Regional Water Board") June 27, 2013 amended Water Code section 13267 Order, Order No. R2-2013-1005-A1, ("Order"), Lehigh Southwest Cement Company ("Lehigh") timely encloses the Third Quarter TRE Workplan Progress Update in accordance with the May 28, 2013 Site-Specific TRE Workplan and Directive 8 of the Order. Please note that the lab reports accompanying the enclosure will be sent only via U.S. Mail, on a CD-ROM, given the large size.

If you or your staff have any questions regarding the enclosed TRE Workplan Progress Update, or would like to discuss further, please do not hesitate to contact me or Greg Knapp at Lehigh, or Mike Bryan and Ben Giudice of RBI.

Very truly yours,

Nicole E. Granquist

Cc: Brian Thompson, Regional Water Quality Control Board, San Francisco Bay Region
Julie Macedo, Counsel, State Water Resources Control Board
Greg Knapp, Director Environmental Region West, Lehigh
Scott Rickman, Regional Counsel, Lehigh Hanson

ROBERTSON - BRYAN, INC.
Solutions for Progress

TECHNICAL MEMORANDUM

Date:	September 30, 2013
Prepared for:	San Francisco Regional Water Quality Control Board
Prepared by:	Brant Jorgenson, Ph.D.
Reviewed by:	Ben Giudice, Ph.D., P.E.
Project:	Lehigh Southwest Cement Company *Ceriodaphni dubia* Toxicity Reduction Evaluation
Subject:	TRE Progress Update and Future TRE Activities

Introduction

The Lehigh Southwest Cement Company (Lehigh) operates the Permanente Quarry and Cement Plant (Permanente Facility) within the drainage of Permanente Creek. Monitoring of Pond 4A, Pond 9, Pond 13, and Pond 14 of the Permanente Facility for chronic 3-speicies whole effluent toxicity (WET) was initiated in the first quarter of 2013. Significant toxicity to *Ceriodaphnia dubia* was observed in Pond 4A samples collected the week of March 25, 2013 (laboratory report previously submitted). The Pond 4A survival EC25 was 16.6% site water, resulting in 6.0 chronic toxicity units (TUc, where TUc = 100/ EC25). The Pond 4A reproduction IC25 was 6.1% site water, resulting in 16.5 TUc (where TUc = 100/IC25). Samples collected from Ponds 13 and 14 also exhibited significant toxicity to *Ceriodapnia dubia,* though the magnitude of effect was less than at Pond 4A.

Accelerated monitoring was initiated, and similar significant toxicity to *Ceriodapnia dubia* was observed in samples collected from Pond 4A, Pond 13, and Pond 14 the week of May 6, 2013 (laboratory report previously submitted). The Pond 4A survival EC25 was 3.3% site water, resulting in 30.2 TUc and the reproduction IC25 was 2.9% site water, resulting in 34.7 TUc. Samples collected from Ponds 13 and 14 also exhibited significant toxicity to *Ceriodapnia dubia,* though the magnitude of effect was less than at Pond 4A. A formal Toxicity Reduction Evaluation (TRE) for *Ceriodaphnia dubia* was initiated at that time.

With the triggering of a formal TRE, a TRE Work Plan for *Ceriodaphnia dubia* was prepared and submitted to the San Francisco Regional Water Quality Control Board (San Francisco Water Board), detailing near-term activities to be taken by Lehigh in order to identify the cause and source of observed toxicity, and a schedule for reporting Lehigh's progress. In the TRE Work Plan, it was determined that due to the connectivity between Pond 4A, Pond 13, and Pond 14, limited intervening dilution sources, and the downstream profile of diminished toxic effect, the observed pond toxicity was associated with discharges from Pond 4A. Therefore, consistent with the TRE Work Plan, TIEs were performed only on Pond 4A samples in an attempt to characterize, isolate, and confirm the nature of the toxicant(s) at the source.

Significant progress has been made at identifying the cause and source of toxicity. Based on this information Lehigh has also proactively taken action to investigate methods of treatment and control of

this toxicity. Consistent with Lehigh's May 2013 TRE Work Plan for *Ceriodaphnia dubia*, the purpose of this memo is to update the San Francisco Water Board on Lehigh's TRE progress, and to detail Lehigh's plan for future related TRE activities.

Overview of TRE Activities Performed

A robust and comprehensive approach was taken towards toxicant identification, including:

- Phase I TIE testing;
- A synthetic simulated site water test;
- Toxicity persistence testing;
- Phase II TIE – nickel selective treatments;
- Phase II TIE – nickel and vanadium spiking experiments; and
- Toxicity control investigation.

An in-depth summary of each of these studies in general order of execution is provided in the Appendix. Toxicant identification testing and experimentation was performed by Pacific EcoRisk (Fairfield, California) on the accelerated monitoring Pond 4A sample collected on May 6, 2013. Individual laboratory reports detailing the above listed studies are provided as attachments to the Appendix.

Overall Conclusions from TRE Efforts

Based on the intensive study conducted to date, as summarized in further detail in the appendix and attached laboratory reports, the following evidence-based conclusions can be made as to the cause of toxicity to *Ceriodaphnia dubia* observed in Pond 4A on March 25 and May 6, 2013.

1. Nickel is the principal toxicant in Pond 4A site water. This conclusion is supported by demonstrated removal of nearly all toxicity when targeted treatments are employed to remove nickel. Moreover, this conclusion is supported by nickel spiking studies in laboratory culture water and in synthetic simulated site water, where empirically derived survival and reproductive effect thresholds (i.e., EC50 and IC25) are 2-10 times lower than baseline nickel concentrations in Pond 4A.
2. The mineral balance of the Pond 4A contributes marginally to observed Pond 4A toxicity. This conclusion is supported by demonstrated low-level reproductive toxicity in synthetic simulated site water, synthesized to represent the mineral balance of Pond 4A. Moreover, nickel effect thresholds in synthetic simulated site water suggest a possible indirect effect, where the mineral balance may cause added stress on the organism and thus result in slightly lower effect thresholds.
3. Vanadium may be a secondary contaminant and marginal contributor to observed Pond 4A toxicity. Removal of vanadium through use of anion exchange resin resulted in marginally lower toxicity, but spiking studies with vanadium do not definitively resolve whether vanadium itself is a contributing toxicant. Use of anion exchange resin also reduces bulk anions, and the reduced toxicity related to the anion exchange resin may also be related to the mineral balance of Pond 4A.

4. Lehigh evaluated two candidate pilot treatment plant control systems. The ABMet treatment system reduced nickel concentrations by 82% and eliminated observed toxicity in the influent. Evidence suggests that design of a full-scale treatment system that can remove nickel with similar efficiency would control toxicity observed in quarry seepage water and in Pond 4A.

Intention to Initiate Quarterly Monitoring

Based on the identification of the cause and source of toxicity to *Ceriodaphnia dubia* in Pond 4A and the identification of a sufficient treatment method, Lehigh intends to initiate a quarterly chronic WET monitoring schedule with *Ceriodaphnia dubia* in Pond 4A, Pond 13, and Pond 14 while design and construction of a full-scale treatment system is implemented. Quarterly chronic WET monitoring with *Ceriodaphnia dubia* will begin in the 4th Quarter of 2013. Upon installation and start-up of the full-scale treatment system, Lehigh will confirm the control of toxicity under the full-scale operational conditions of its treatment system. Thus, the return to a quarterly monitoring schedule would not mark a conclusion of the TRE, given Lehigh's need to confirm toxicity control upon construction of appropriate full-scale treatment facilities. Efforts at confirmation of toxicity control will occur at a time when Lehigh begins operation of its interim treatment process, anticipated in October 2014.

Continued toxicity in *Ceriodaphnia dubia* bioassays is anticipated until the treatment process is in operation. During this interim period of quarterly monitoring, Lehigh will not engage in further toxicant identification activities when the weight of evidence suggests the causes of toxicity are the same as already identified and described in this TRE Update Memo. In order to assist in distinguishing between previously characterized toxicity and toxicity to *Ceriodaphnia dubia* due to a new toxicant, Lehigh will do the following: (1) measure metals (including nickel and vanadium) and minerals in each of the samples (Pond 4A, 13, and 14), and (2) employ the criteria described below in determining whether a new cause of toxicity has been observed.

1. If for *Ceriodaphnia dubia* the sample (i.e., Pond 4A, Pond 13, or Pond 14) survival and reproduction TUc (as 100/EC25 or IC25, as appropriate) is ≤ 2, no further action is necessary.

2. If for *Ceriodaphnia dubia* the sample survival and reproduction TUc is > 2, and the sample total recoverable nickel concentration is ≥ 5.7 µg/L, the cause of toxicity is presumed to be related to nickel and no further action is necessary. The nickel concentration-based trigger of 5.7 µg/L is the empirical reproduction IC25 derived for *Ceriodaphnia dubia* buffered synthetic simulated site water (see Table 2 in the appendix).

3. If for *Ceriodaphnia dubia* the sample survival and reproduction TUc is > 2, and the sample total recoverable nickel concentration is < 5.7 µg/L, results for vanadium and standard minerals will be examined in combination with the nickel concentration and the magnitude of toxicity. If the weight of evidence suggests that the same combination of toxicants is responsible for the toxicity, no further identification activities will be performed. If the weight of evidence suggests that the cause of toxicity is not related to nickel, vanadium, mineral balance, or a combination thereof, but to a new toxicant or new source, Lehigh will proceed with the accelerated monitoring and TRE requirements of the 13267 Investigative Order.

APPENDIX
Summaries of Toxicity Testing and Experimentation

Phase I TIE Testing

A comprehensive Phase I TIE was performed (Attachment 1) utilizing the treatments listed **Table 1**. Results from the survival endpoint (shown in Table 1) were most definitive. Cation (Supelco LC-WCX), zeolite, and EDTA treatments strongly implicated a cationic metal(s). Anion and cation plus anion treatments also indicated that an anionic class of toxicant was contributing to toxicity, but to a much lesser degree. While the results for the carboxylesterase and BSA treatments would indicate a nonpolar organic toxicant, these treatments are also known to adsorb metals. Moreover, there was no corresponding decrease in toxicity with the C18 SPE treatment – a removal of toxicity would be expected if nonpolar organic toxicants were contributing to toxicity.

Aliquots of the baseline, cation, anion, cation plus anion, and zeolite treatments were submitted to Alpha Analytical for chemical analysis. Chemical analyses were limited to trace metals (EPA 200.8) and bulk minerals. Baseline nickel and vanadium concentrations (56 and 110 µg/L, respectively) were substantially elevated in Pond 4A site water relative to representative aquifer waters collected across dry climate regions of the United States (Ayotte et al. 2011).

Although nickel concentrations did not exceed EPA hardness-based aquatic life criteria, nickel toxicity is strongly pH dependent. Published *Ceriodaphnia dubia* 48-hour median lethal effect concentrations (i.e., LC50) for nickel at a test pH of 8-8.5 is 13 µg/L (Schubaur-Berigan et al. 1993), significantly lower than the baseline concentration of 56 µg/L. The cation treatment removed 98% of nickel (concentration of 1.2 µg/L) and the zeolite treatment removed 84% of nickel (concentration of 9.1 µg/L). Moreover, the pattern of increasing nickel toxicity with increasing pH was replicated in the pH treatments, where toxicity was mostly removed in the pH 6.5 treatment while there was no change in toxicity in the pH 8.5 treatment (ambient Pond 4A pH is approximately 8.0 to 8.2).

Vanadium is not a priority pollutant, and aquatic life criteria have not been adopted by EPA. Published *Ceriodaphnia dubia* 7-day median reproduction inhibition concentrations (i.e., IC50) for vanadium are as low as 362 µg/L (Puttaswamy and Liber 2012). While significantly higher than the baseline concentration of 110 ug/L, research indicates that nickel and vanadium can act additively at their highest sublethal concentrations (Puttaswamy and Liber 2012). Vanadium is an oxoanion, and baseline concentrations were completely removed by the anion treatment (i.e., non-detect).

Based on the magnitude difference in toxicity removed by the anion and the cation treatments, and the respective removals of nickel and vanadium by these treatments, it was concluded that nickel was the likely primary toxicant, and vanadium, possibly acting additively, was a secondary toxicant. There was also suspicion that ion imbalance could have a contributory effect, either directly, or indirectly through added organism stress. When evaluated in total, the following conclusions were drawn from the Phase I TIE and chemical analysis:

1. Nickel was the most likely principal toxicant;
2. Vanadium was a possible secondary contributor to toxicity; and
3. Ion imbalance could not be ruled out as a possible contributor to toxicity.

Table 1. Results of Phase I TIE experiments

Treatment	Change Relative to Baseline	Effect of Treatment on Survival
Baseline	0%	Sample Toxic (100% Mortality)
Aeration	0%	No change in toxicity
1 um Filtration	20%	No change in toxicity
C18 SPE	0%	No change in toxicity
C18 SPE Elution	100%	No toxicity present
25 ug/L PBO	40%	Slight reduction in toxicity
100 ug/L PBO	20%	No change in toxicity
Carboxylesterase	100%	Toxicity removed
BSA	80%	Most toxicity removed
Anion Exchange	40%	Slight reduction in toxicity
Cation Exchange	80%	Most toxicity removed
Anion and Cation Exchange	100%	Toxicity removed
Zeolite	100%	Toxicity removed
pH 6.5	80%	Most toxicity removed
pH 7.5	20%	No change in toxicity
pH 8.5	0%	No change in toxicity
STS at 10 mg/L	0%	No change in toxicity
STS at 25 mg/L	20%	No change in toxicity
EDTA at 3 mg/L	100%	Toxicity removed
EDTA at 8 mg/L	80%	Most toxicity removed

Note: Reductions (or increases) relative to baseline that are less/equal to 20% are characterized as "no change in toxicity".

C18 SPE	octyldecyl solid phase extraction
PBO	piperonyl butoxide
BSA	bovine serum albumin
STS	sodium thiosulfate
EDTA	ethylenediaminetetraacetic acid

Synthetic Simulated Site Water Test

The Phase I TIE indicated that toxicity was partially or completely removed by treatments affecting cations and anions. Standard TIE protocols utilizing cation exchange resins and zeolite call for replacement of calcium and magnesium in order to maintain test solution hardness. Therefore, the concentration and balance of these cations and the possible contribution to toxicity could not be evaluated directly through the Phase I TIE protocol. Moreover, the anion treatment substantially reduced baseline alkalinity and sulfate concentrations. Therefore, a test of water with the same major ionic makeup as Pond 4A water, but free of nickel and vanadium, was desired. This test was accomplished by preparing a synthetic simulated site water using reagent grade chemicals for the major ions - no nickel or vanadium were added.

There was no statistically significant mortality of *Ceriodaphnia dubia* in the synthetic simulated site water, although testing indicated a small reproductive effect (Attachment 2). The third brood IC25 was calculated to be 56.9% in simulated site water, corresponding to a reproduction TUc of 1.8. In

comparison, the Pond 4A sample collected May 6, 2013 had a reproduction TUc of 34.7 TUc. The following conclusions were drawn from the synthetic simulated site water testing:

1. Ion balance alone may contribute marginally to the observed *Ceriodaphnia dubia* reproduction effects in Pond 4A site water, but other toxicants are the cause of the majority of toxicity; and
2. Ion balance may amplify or mitigate nickel and vanadium toxicity (Puttaswamy and Liber, 2012), therefore Phase II TIE testing should include nickel and vanadium spiking studies in synthetic simulated site water.

Toxicity Persistence Testing

In the course of TIE testing, an originally collected sample may age considerably over the course of experimentation. Aging may affect the stability of a toxicant, and thereby affect the persistence of toxicity. In the case of a nonpolar organic toxicant, phase partitioning and natural degradation processes, albeit slowed by the storage of sample at <6°C and in the dark, can reduce the presence and/or bioavailability of the toxicant. Therefore, a toxicity persistence test serves the dual purpose of not only verifying the continued presence of toxicity, thereby allowing continued experimentation, but also informs on the stability, and thus, the class of toxicant – rapidly lost and impersistent toxicity may indicate the toxicant is organic in nature (such as a pesticide), while persistent toxicity may indicate the toxicant is inorganic in nature (such as a metal).

A stability test on the May 6, 2013 sample was executed on August 6, 2013, 93 days after initial sample collection. Results showed that toxicity was diminished, but persistent, where the survival EC25 was 27.4% site water, resulting in 3.7 TUc and the reproduction IC25 was 20.2% site water, resulting in 5.0 TUc. The following conclusions were drawn from the toxicity stability testing:

1. Persistent toxicity in the sample 93 days after collection indicates that the sample is suitable for continued TIE testing; and
2. Persistence of toxicity indicates that the toxicant is stable in matrix, suggesting that the toxicant may be inorganic in nature (i.e., metal) and not readily subject to degradation or chemical transformation.

Phase II TIE Testing – Nickel Selective Treatments

Phase II TIE testing was targeted to further evaluate and confirm the activity of nickel by utilizing a series of nickel selective treatments, including selective cation exchange resins, nickel selective chelator, and pH adjustments (see Attachment 4). Results from the survival endpoint were most definitive. Mortality was mostly removed when the pH of the sample was adjusted to 6.5, consistent with the pH dependence of nickel toxicity (Schubaur-Berigan et al. 1993). Similarly, the treatments utilizing the cation resins AG50W-X8 and IRC-748 , and the nickel-specific chelator dimethylglycoxime (DMG) reduced mortality. These cation resins and chelator were selected for testing because of their literature reported selectivity towards nickel.

Blank interference in the Supelco LC-WCX elution resulted in an uninterpretable treatment response. The Supelco LC-WCX cation resin was used in the previous Phase I TIE. The likely cause of the interference was the high conductivity of the eluent, which was >3 mS/cm.

When evaluated in total, the following conclusions were drawn from the Phase II TIE nickel selective treatments and chemical analysis:

1. The use of nickel selective resins and chelator resulted in diminished toxicity, indicating that nickel was a principal toxicant; and
2. No single treatment eliminated toxicity completely, indicating that one or more constituent(s) other than nickel are possibly contributing towards toxicity.

Phase II TIE Testing – Nickel and Vanadium Spiking Experiments

Further Phase II TIE testing was targeted at nickel and vanadium through the use of spiking experiments. In these experiments pure stock solutions of nickel (NiCl) and vanadium ($NaVO_3$) were spiked at increasing concentration into laboratory culture water (i.e., modified EPA moderately hard water) and into synthetic simulated Pond 4A site water (see Attachment 5). Because the feeding regimen of the bioassay test tends to increase pH over the course of the test, spiking experiments included sets buffered at a pH between 8 and 8.2 (equivalent to ambient Pond 4A pH) and sets that were unbuffered (pH allowed to increase over the course of the test).

As shown in **Table 2**, 7-day nickel EC50 and IC25 values are significantly lower than the baseline Pond 4A concentration of 56 µg/L, and thus can explain the mortality and reproductive effects observed in Pond 4A site water. Moreover, the EC50 values are very similar to the 48-hr LC50 thresholds published by Schubaur-Berigan et al (1993), thus providing added confirmation. When controlling for pH effects, the effect thresholds in buffered synthetic simulated site water are somewhat lower than the effect thresholds for buffered culture water, which possibly could be accounted for by an added stress on the organisms from the concentration and balance of minerals in Pond 4A site water.

As shown in Table 2, 7-day vanadium EC50 and IC25 values are within the range of the baseline Pond 4A vanadium concentration of 110 µg/L, although the large range in the water-type depended thresholds does not make it possible to definitively conclude vanadium's role in observed Pond 4A toxicity.

Table 2. Results of nickel and vanadium spiking experiments (µg/L)

Water	Nickel		Vanadium	
	Survival EC50	Reproduction IC25	Survival EC50	Reproduction IC25
Culture at unadjusted pH	11.7	6.9	377	168
Culture at buffered pH	23.6	9.2	707	396
Synthetic at unadjusted pH	17.7	11.2	124	69.3
Synthetic at buffered pH	17.5	5.7	437	319

The following conclusions were drawn from the nickel and vanadium spiking experiments:

1. Nickel concentrations in Pond 4A (i.e., 56 µg/L) are sufficiently high to account for nearly all of the *Ceriodaphnia dubia* toxicity observed in Pond 4A;
2. Based on the synthetic versus culture water spiking tests, Pond 4A mineral balance may contribute towards toxicity through added stress on the organism; and
3. Spiking tests provide some support in concluding that vanadium is contributing towards toxicity, but it is likely not as large of a contributor as nickel.

Source of Pond 4A Toxicity to *Ceriodaphnia dubia*

The sources of inflow to Pond 4A include seepage water from the quarry and discharges from the primary crusher. Primary crusher discharges are intermittent in nature, and contribute a very small fraction of total Pond 4A inflow. Moreover, the source of water to the primary crusher is Santa Clara Valley Water District which is low in toxic metals.

The majority of inflow to Pond 4A is seepage water from the quarry. Quarry dewatering operations shunt water through a series of intermediary sedimentation tanks and open-air ponds prior to being pumped out of the quarry and into Pond 4A. Seepage water metals concentrations are equivalent to those measured in Pond 4A. As such, the source of Pond 4A toxicity is believed to be seepage water from the quarry.

Toxicity Control Investigation

In its efforts to investigate selenium control strategies in quarry seepage water, Lehigh evaluated two candidate pilot treatment plant control systems. While in operation, samples of influent and treatment plant effluent were collected and subject to chronic WET testing with *Ceriodaphnia dubia* (Attachment 6 and Attachment 7). Influent samples were collection from Pond 950. Pond 950 is used to collect and integrate seepage water from various points around the quarry prior to pumping to Pond 4A. As such, Pond 950 is the last point at which quarry seepage water is added prior to pumping to Pond 4A.

Effluent samples were collected from the final process effluent stream of each pilot treatment system. Systems tested were ABMet and ICB, both anaerobic bioreactor-based treatment systems specifically operated to target selenium removal. No effort was made to modify operations to target nickel removal prior to sample collection. Results of testing are summarized in **Table 3**.

Chronic WET testing of ABMet occurred in June, 2013 and testing of ICB occurred in July, 2013. Mortality was not observed in influent samples from either sampling event. There was sufficient nickel in the samples to cause expected mortality, except that influent sample pH was approximately 7.7 which may have contributed to lower mortalities due to the pH dependence of nickel. Reproduction effects were similar between influent samples. The ABMet treatment system reduced nickel concentrations by 82% and eliminated observed toxicity in the influent. The ICB treatment system reduced nickel concentration by only 23% and caused in increase in mortality and reproductive effects. The cause of this increased toxicity in the ICB system is not known.

Although the magnitude of initial toxicity in this testing was smaller than that observed in March and May of 2013 at Pond 4A, the WET testing of ABMet effluent further supports concluding that nickel is the principal toxicant. Evidence suggests that design of a full-scale treatment system that can remove nickel with similar efficiency would control toxicity observed in quarry seepage water and in Pond 4A. Based on a variety of design, operation, maintenance, and reliability factors, Lehigh will select and design a treatment process that will be capable of removing metals with similar efficiency as the ABMet process. An interim treatment process is scheduled for operation in October 2014. A final process will be designed based on results from the interim system.

The following conclusions were drawn from testing of the pilot treatment systems:

1. The increased nickel removal efficiency of the ABMet process relative to the ICB process, and the associated elimination of toxicity with the ABMet process further indicates that nickel is the principal toxicant in quarry seepage water and in Pond 4A;
2. A system with similar metals removal efficiencies as the ABMet process is a suitable method for toxicity control.

Table 3. Results of pilot treatment plant WET testing

	ABMet Treatment System		ICB Treatment System	
	Influent	Effluent	Influent	Effluent
Survival (TUc) [1]	<1	<1	<1	3
Reproduction (TUc) [2]	5.2	1.0 [3]	6.7	13.3
Nickel (µg/L)	35	6.3	35	27
Vanadium (µg/L)	70	46	70	55

[1] TUc based on 100/EC25
[2] TUc based on 100/IC25
[3] TUc based on 100/NOEC – there was no statistically significant difference between the lab control or site water dilutions.

Citations

Ayotte, JD, JM Gronberg, and LE Apodaca. 2011. *Trace Elements and Radon in Groundwater Across the United States, 1992–2003*. U.S. Geological Survey Scientific Investigations Report 2011–5059.

Puttaswamy, N, K Liber. 2012. Influence of inorganic anions on metals release from oil sands coke and on toxicity of nickel and vanadium to *Ceriodaphnia dubia*. *Chemosphere*. 86:521-529.

Schubauer-Berigan, MK, JR Dierkes, PD Monson, GT Ankley. 1993. pH-dependent toxicity of Cd, Cu, Ni, Pb, and Zn to *Ceriodaphnia dubia, Pimephales promelas, Hyalella aztea, and Lumbriculus variegatus*. Environ. Toxicol. Chem. 12:1261-1266.

ATTACHMENT 1
Phase I TIE Experiments

ATTACHMENT 2

Synthetic Simulated Site Water Test

ATTACHMENT 3

Toxicity Persistency Test

ATTACHMENT 4
Phase II TIE Tests – Nickel Selective Experiments

ATTACHMENT 5

Phase II TIE Tests – Nickel and Vanadium Spiking Experiments

ATTACHMENT 6
ABMet Pilot Treatment System Testing

ATTACHMENT 7
ICB Pilot Treatment System Testing